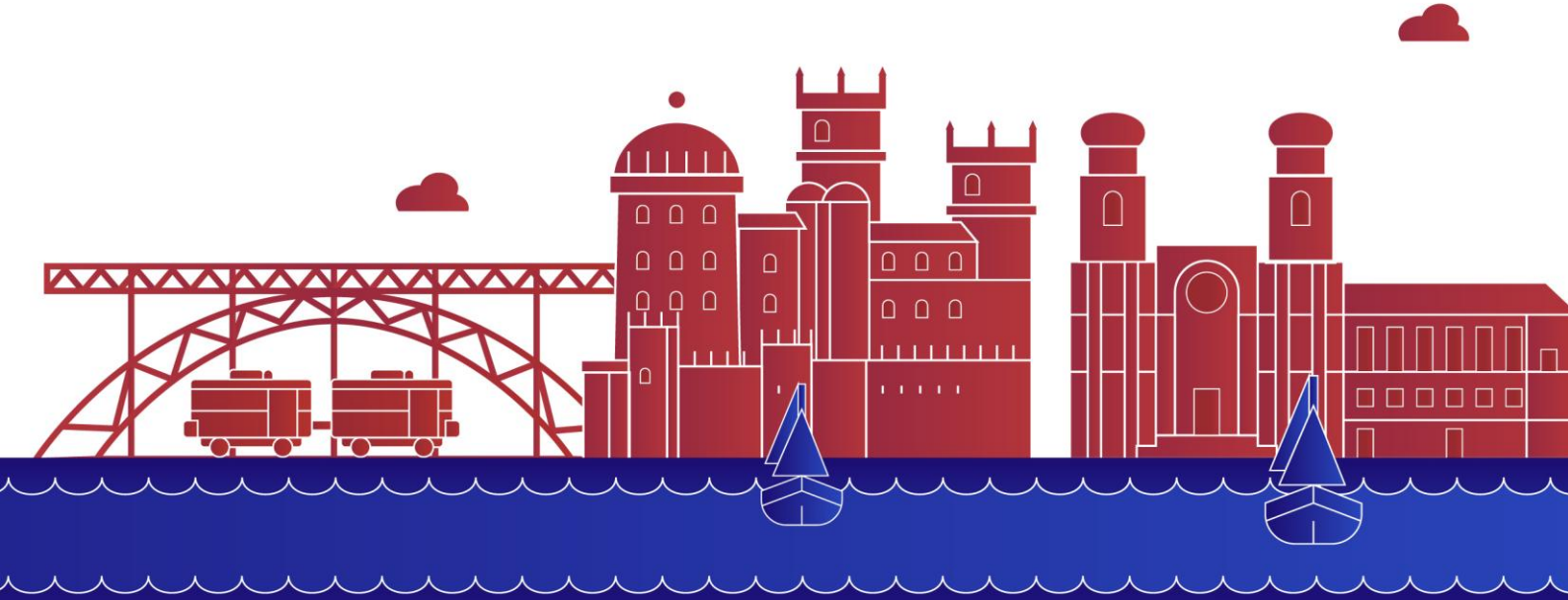
ICT 2025

29th International Congress on Thrombosis
Porto, Portugal

6-8 November 2025
Sheraton Hotel

BOOK OF ABSTRACTS



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Oral Communications

OC08

From PE to CTEPH: still lost in translation

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Background:
Chronic thromboembolic pulmonary hypertension (CTEPH) is a potentially curable cause of pulmonary hypertension. It has been hypothesized that a prior episode of acute pulmonary embolism (PE) may facilitate earlier recognition and diagnosis of CTEPH at a less severe stage.
Methods:
We conducted a single-center retrospective study that included **95 consecutive patients** diagnosed with CTEPH between 2005 and 2024. Data were collected on prior PE events, CTEPH risk factors (hypothyroidism, splenectomy, inflammatory bowel disease, myeloproliferative disorders, antiphospholipid syndrome, and cancer), WHO class, echocardiographic parameters (right atrial and ventricular size, sPAP, TAPSE), and hemodynamic variables (mPAP, PAWP, CI, PVR, arterial O2 saturation). Patients were stratified by the presence of prior PE, and among those with prior PE, according to time from PE to CTEPH diagnosis (<12 vs ≥12 months).
Results:
Of the 95 patients included, 66 (69%) had a documented history of PE. The median time from PE to CTEPH diagnosis was 21.5 months in patients with prior PE and 19.0 months from first symptoms to CTEPH diagnosis in those without prior PE (p=ns), indicating that prior PE did not lead to a significantly earlier diagnosis. Clinical, echocardiographic and hemodynamic characteristics were also comparable, suggesting that prior PE did not identify a lower-risk phenotype (table 1).The prevalence of established CTEPH risk factors (hypothyroidism, splenectomy, inflammatory bowel disease, myeloproliferative disorders, antiphospholipid syndrome, cancer) was also similar between groups. Among patients with prior PE, 21 (32%) were diagnosed within 12 months and 45 (68%) after ≥12 months. No significant differences were found between these groups regarding NYHA class, right atrial size, right ventricular dilatation, sPAP, cardiac index, mPAP, PAWP, PVR, or arterial oxygen saturation. The only significant difference was observed in TAPSE, which was lower in patients diagnosed <12 months (16.2 [14–20] vs 19.0 [16–22] mm, p=0.049) (table 2).
Conclusions:
The diagnosis of CTEPH in our cohort was still late and at a high-risk stage. Having a prior PE did not result in a shorter time to CTEPH diagnosis or a less severe phenotype at presentation. The presence of PE in the medical history does not appear to enable earlier recognition of CTEPH or diagnosis at a lower-risk stage. Among patients with prior PE, only TAPSE was significantly lower in those diagnosed within 12 months, suggesting that post-PE investigations are likely performed mainly in more symptomatic patients rather than as part of systematic screening. Systematic post-PE screening strategies might be needed to detect CTEPH patients at an earlier and potentially lower-risk stage.

Table 1 - Comparison of patients with and without prior PE

Variable	PE (n=66)	No PE (n=29)	p-value
Time do diagnosis (months)	21.5 [9.3-69.3]	19.0 [6.0-60.0]	ns
NT pro-BNP (pg/mL)	967 [108-3555]	952 [120-3200]	ns
PASP (mmHg)	80.9 ± 30.5	81.7 ± 31.1	ns
TAPSE (mm)	18.4 ± 4.7	18.5 ± 5.3	ns
mPAP (mmHg)	44.3 ± 13.0	43.3 ± 13.8	ns
PVR (WU)	9.0 ± 5.1	9.5 ± 4.9	ns
NYHA III-IV (%)	72.7%	72.4%	ns

Data are presented as mean ± SD for normally distributed variables, and as median [IQR] otherwise.

A p-value < 0.05 was considered statistically significant.

Table 2 - Patients with prior PE: <12 vs ≥12 months to diagnosis

Variable	<12m (n=21)	≥12m (n=45)	p-value
NT pro-BNP (pg/mL)	959 [108-3555)	975 [113-2065]	ns
sPAP (mmHg)	79.1 ± 30.6	81.8 ± 30.8	ns
TAPSE (mm)	16.8 ± 3.9	19.2 ± 4.9	0.015
mPAP (mmHg)	47.2 ± 12.8	42.9 ± 13.0	ns
CI (L/min/m^2)	2.4 [1.9-2.7]	2.1 [1.9-2.5]	ns
PVR (WU)	10.5 ± 5.6	9.5 ± 5.4	ns
NYHA III-IV (%)	78.3%	69.8%	ns

Data are presented as mean ± SD for normally distributed variables, and as median [IQR] otherwise.

A p-value < 0.05 was considered statistically significant.

OC17

Gene expression profiles linking ADAM22 and CTNNB1 downregulation to increased atherothrombotic complications in familial hypercholesterolemia

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Background:

Individuals with familial hypercholesterolemia (FH) exhibit a substantially higher risk of premature atherosclerotic cardiovascular disease (ASCVD) than those with comparable LDL-C levels but without FH, primarily due to lifelong exposure to elevated LDL-C. However, FH is characterized by marked heterogeneity in the severity and progression of ASCVD, even among individuals carrying the same causal mutation. This study aimed to identify differential gene expression profiles to plaque vulnerability and atherothrombotic processes in statin-treated FH patients with and without clinical evidence of ASCVD, to elucidate the potential contribution of these genes to premature cardiovascular events.

Methods:

Patients with a genetic diagnosis of FH (*N*=82) from the SAFEHEART Cohort were included in the study (nASCVD= without clinical event, *N*=39; ASCVD=with clinical event, *N*=43).

Differential gene profiles were identified by Next Generation sequencing (NGS) and validated by real-time PCR from whole blood samples. Bioinformatic *in silico analysis* using Cytoscape and STRING tools was performed to identify the molecular interaction between immune response and thrombosis.

Results:

A differential gene pattern of 107 genes (>2 fold-change) was identified by NGS between FH-patients with premature clinical events and those at lower risk, who remained free of clinical cardiovascular disease during an 8-year follow-up. Of them, by *in silico* analysis, 5 genes related to immune response and thrombosis (ADAM22, AIFM1, CTNNB1, CXCL8 and SCRIB), which were down regulated in FH-patients with premature ASCVD, were identified and selected for further analysis. Real-time PCR confirmed that the disintegrin and metalloproteinase (ADAM22) and the catenin Beta 1 / β-catenin (CTNNB1) were significantly reduced (*P*=0.006 and *P*=0.012, respectively) in FH-patients with active disease progression, presenting major clinical events (MACE: acute MI, sudden death, unstable angina) after inclusion. Receiver operating characteristic (ROC) analysis demonstrated that expression levels of both ADAM22 and CTNNB1 significantly distinguished FH patients who experienced major adverse cardiovascular events (MACE) during follow-up from those in the nASCVD group, who remained free of clinical ASCVD (AUC: 0.762; *P*=0.004). Additionally, the decreased ADAM22 and CTNNB1 levels were linked to a significantly higher rate of MACE over 8 years (Kaplan–Meier HR=4.41; *P*<0.001).

Conclusion:

Downregulated gene expression of ADAM22 and CTNNB1 is observed in the total blood of FH patients with an active ASCVD phenotype characterized by disease progression and incident acute clinical cardiovascular events. These results suggest that reduced expression of ADAM22 and CTNNB1 may promote plaque vulnerability, thereby increasing the risk of atherothrombotic complications.

Gene Expression Profiles Linking ADAM22 and CTNNB1 Downregulation to Increased Atherothrombotic Complications in Familial Hypercholesterolemia

Rafael Escate[1,2], Maria Borrell-Pages[1,2], Montse Gomez-Pardo[1], Manuel J Romero[3], Pedro Mata[4], Lina Badimon[1,2,5], Teresa Padro[1,2]

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Background: Individuals with familial hypercholesterolemia (FH) exhibit a substantially higher risk of premature atherosclerotic cardiovascular disease (ASCVD) than those with comparable LDL-C levels but without FH, primarily due to lifelong exposure to elevated LDL-C. However, FH is characterized by marked heterogeneity in the severity and progression of ASCVD, even among individuals carrying the same causal mutation. This study aimed to identify differential gene expression profiles to plaque vulnerability and atherothrombotic processes in statin-treated FH patients with and without clinical evidence of ASCVD, to elucidate the potential contribution of these genes to premature cardiovascular events.
Methods: Patients with a genetic diagnosis of FH (*N*=82) from the SAFEHEART Cohort were included in the study (nASCVD= without clinical event, *N*=39; ASCVD=with clinical event, *N*=43). Differential gene profiles were identified by Next Generation sequencing (NGS) and validated by real-time PCR from whole blood samples. Bioinformatic *in silico analysis* using Cytoscape and STRING tools was performed to identify the molecular interaction between immune response and thrombosis.
Results: A differential gene pattern of 107 genes (>2 fold-change) was identified by NGS between FH-patients with premature clinical events and those at lower risk, who remained free of clinical cardiovascular disease during an 8-year follow-up. Of them, by *in silico* analysis, 5 genes related to immune response and thrombosis (ADAM22, AIFM1, CTNNB1, CXCL8 and SCRIB), which were down regulated in FH-patients with premature ASCVD, were identified and selected for further analysis. Real-time PCR confirmed that the disintegrin and metalloproteinase (ADAM22) and the catenin Beta 1 / β-catenin (CTNNB1) were significantly reduced (*P*=0.006 and *P*=0.012, respectively) in FH-patients with active disease progression, presenting major clinical events (MACE: acute MI, sudden death, unstable angina) after inclusion. Receiver operating characteristic (ROC) analysis demonstrated that expression levels of both ADAM22 and CTNNB1 significantly distinguished FH patients who experienced major adverse cardiovascular events (MACE) during follow-up from those in the nASCVD group, who remained free of clinical ASCVD (AUC: 0.762; *P*=0.004). Additionally, the decreased ADAM22 and CTNNB1 levels were linked to a significantly higher rate of MACE over 8 years (Kaplan–Meier HR=4.41; *P*<0.001).
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Funding: Institute of Health Carlos III (ISCIII): PMP22/00108 from the ISCIII with Next Generation EU funds from the Recovery and Resilience Mechanism (RRM) Program

OC18

Thromboprophylaxis in ambulatory cancer patients: concordance between Khorana score and clinical practice

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Unidade Local de Saúde de Gaia/Espinho

Background:
Venous thromboembolism (VTE) is a frequent complication in cancer patients and the second leading cause of death in this population, with approximately 80% of events occurring in ambulatory settings. Although prophylactic anticoagulation can prevent VTE, it remains a challenge to select patients who may benefit from thromboprophylaxis. The Khorana Score (KS) is the most validated risk assessment model for predicting VTE in ambulatory cancer patients and is guideline-recommended to guide thromboprophylaxis. Despite this, its adoption in routine clinical practice remains low. Objective: To assess the concordance between KS-recommended and actual prescription of prophylactic anticoagulation in a real-world cohort of ambulatory cancer patients.

Material and methods:
Single-center retrospective analysis of patients with a cancer diagnosis prior to a VTE event, followed between 2017 and 2024 at the Internal Medicine–VTE outpatient clinic. Demographic data, cancer-related variables and VTE characteristics were retrieved from clinical records. KS was calculated in patients who had received chemotherapy. Data analysis was performed using IBM SPSS Statistics, version 29.

Results:
A total of 70 patients were included. The median age at VTE diagnosis was 69.5 years (IQR: 59.5–77.0) and 61.4% were male. Most patients had a good performance status (ECOG PS 0-1, 87.1%). The most common cancer type was colorectal (18.6%), and the disease was mainly localized (47.1%) vs. locally advanced or metastatic (44.3%). Treatment modalities mainly included surgery alone (n=16, 22.9%) or a combination of surgery and systemic therapy (n=41, 58.6%). Pulmonary embolism was the most frequent VTE event (92.9%), and 55.7% of events were symptomatic. Of the 45 patients who received chemotherapy, 13 (28.9%) had a KS ≥ 2, and were thus eligible for primary thromboprophylaxis. Despite this, none of them received prophylactic anticoagulation.

Conclusions:
Despite guideline recommendations for primary thromboprophylaxis in ambulatory cancer patients, a significant gap exists in the real-world implementation, potentially leading to an increased risk of preventable VTE. This study highlights the importance of improving prophylactic strategies in this population. Although limited by its retrospective, single-center design and focus on a population with established VTE, this study underscores the critical need for interventions to improve adherence to guidelines. Future studies should investigate the barriers to prophylaxis implementation and develop strategies to bridge this care gap.

OC19

Is there a cure for CTEPH? Long-term functional and hemodynamic outcomes after PEA and BPA

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Background:

Pulmonary endarterectomy (PEA) is currently the first-line treatment for chronic thromboembolic pulmonary hypertension (CTEPH), offering the potential for cure. Balloon pulmonary angioplasty (BPA) has recently emerged as a valid alternative for patients deemed inoperable, with consistent long-term improvements in resting hemodynamics. This study compared long-term health-related quality of life (HRQoL) and exercise-induced hemodynamic responses after BPA or PEA, to determine whether either intervention achieves true physiological resolution of disease.

Methods:

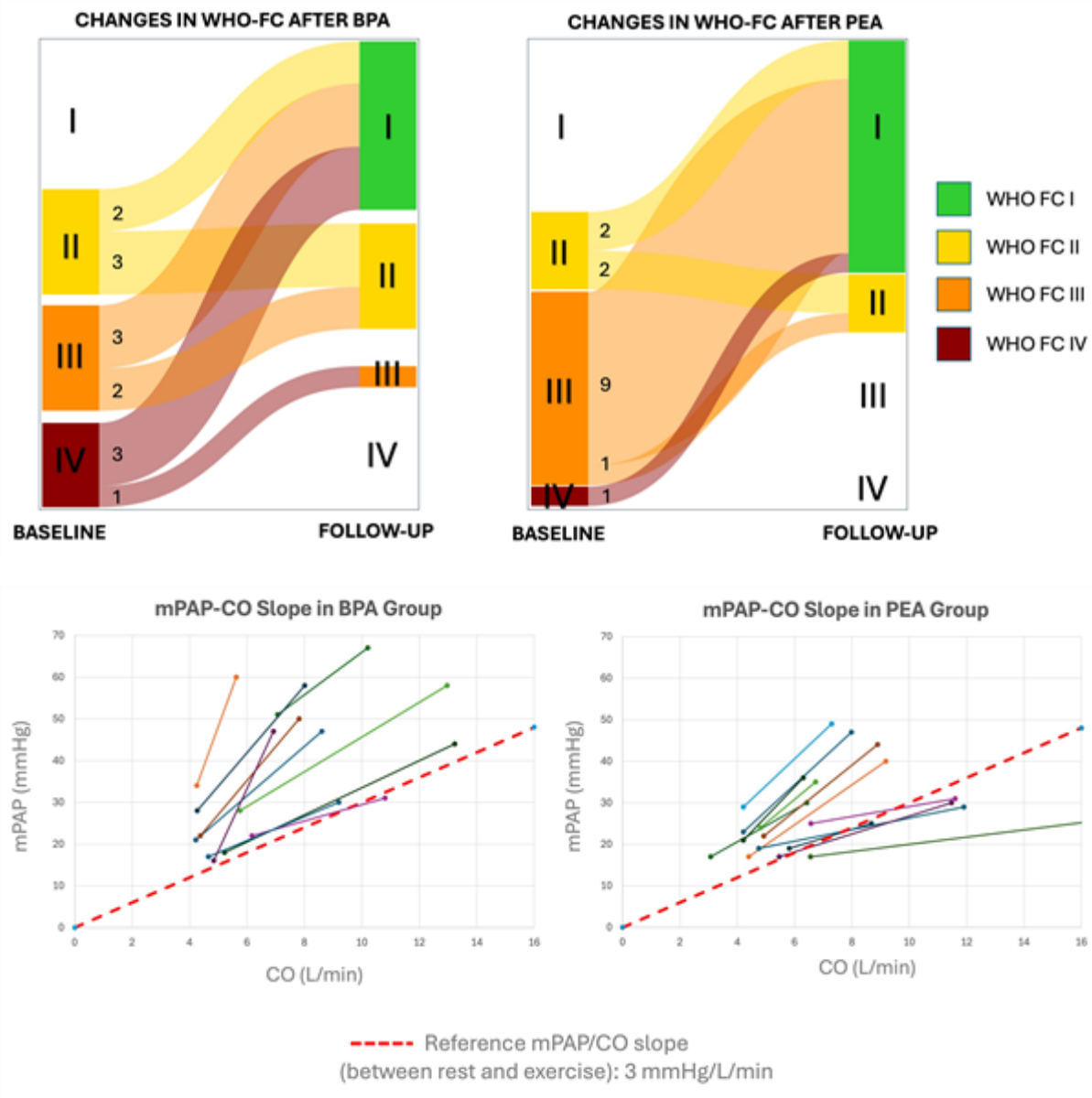
We analyzed a prospective single-center registry (2017–2023) including 14 patients who completed BPA and 15 who underwent PEA, with a median follow-up of 50 months (IQR 36–61). Treatment allocation was decided by a multidisciplinary CTEPH team. Clinical assessment included WHO Functional Class (WHO FC), 6-minute walk distance (6MWD), transthoracic echocardiography, and right heart catheterization (RHC) at rest. At long-term follow-up, RHC was repeated under exercise conditions to evaluate the mean pulmonary arterial pressure/cardiac output (mPAP/CO) slope as an index of pulmonary vascular reserve. Health-related quality of life (HRQoL) was evaluated in both treatment groups at long-term follow-up using the RAND 36- Item Short Form Health Survey, version 1 (SF-36). Physical (PCS) and mental (MCS) component summary scores were calculated from the eight subscales, and each domain score was normalized against a norm-based U.S. population reference.

Results:

Baseline characteristics were similar between groups. Both BPA and PEA led to sustained improvements in functional class over long-term follow-up (Figure 1). Right ventricular (RV) reverse remodeling was observed in both groups, with significant improvement in RV fractional area change (FAC) after BPA (29.1 ± 11.9 → 47.1 ± 4.1; $p < 0.001$) and PEA (31.7 ± 9.9 → 38.8 ± 7.6; $p = 0.024$). Both interventions also significantly reduced mPAP (BPA: 44.8 ± 12.4 → 26.1 ± 9.3 mmHg; PEA: 42.1 ± 12.9 → 22.6 ± 5.4 mmHg; both $p < 0.001$) and pulmonary vascular resistance (BPA: 9.8 ± 4.6 → 3.0 ± 1.3 WU; PEA: 9.0 ± 5.4 → 2.9 ± 1.9 WU; both $p < 0.001$). Despite these improvements, the mPAP/CO slope was not significantly different between groups (7.0 ± 5.6 vs. 4.0 ± 2.3 mmHg/L/min; $p = 0.108$), and abnormal slopes (> 3.0 mmHg/L/min) were frequent in both (80.0% vs. 58.3%; $p = 0.268$). Individual mPAP/CO slopes for each treatment group are shown in Figure 2. At long-term follow-up, PCS scores remained below the population norm of 50 and were comparable between BPA and PEA groups (44.4 ± 12.7 vs. 44.5 ± 7.3; $p = 0.899$). MCS scores tended to be lower after BPA (42.7 ± 14.3 vs. 49.9 ± 14.1; mean difference −7.2, 95% CI −17.1 to 2.8; $p = 0.212$), although this difference was not statistically significant.

Conclusion:

Both BPA and PEA yield sustained improvements in resting hemodynamics, yet abnormal exercise responses and reduced physical quality of life persist in most patients. These findings underscore that, despite major therapeutic advances, CTEPH remains a **chronic—rather than curable—condition**.


CHANGES IN WHO-FC AFTER BPA
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WHO FC II
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WHO FC IV
mPAP-CO Slope in BPA Group
mPAP (mmHg)
CO (L/min)
mPAP-CO Slope in PEA Group
mPAP (mmHg)
CO (L/min)
Reference mPAP/CO slope
(between rest and exercise): 3 mmHg/L/min

OC20

Altered HDL proteomic profile reflects enhanced atherothrombotic risk in familial hypercholesterolemia

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Background:

Familial hypercholesterolemia (FH) is an autosomal dominant disorder characterized by lifelong elevated LDL cholesterol, leading to accelerated atherosclerosis and premature cardiovascular events (CVE). However, substantial variability in CVE incidence suggests additional risk modifiers beyond LDL levels. Increasing evidence highlights the relevance of HDL composition rather than HDL-cholesterol (HDL-c) levels as a key determinant of its atheroprotective and antithrombotic properties. Experimental studies indicate that HDL's protective functions—such as prevention of oxidation, reduction of thrombosis, and promotion of endothelial repair—may be impaired under hypercholesterolemic conditions. Nevertheless, the relationship between HDL proteomic composition and the occurrence of premature clinical CVE in FH remains unclear. This study aimed to characterize the HDL proteomic profile in subjects with FH and to identify the differential proteomic signature associated with premature CVE (before 55 years of age) in this high-risk population.

Methods:

HDL proteomic characterization was conducted in participants from the Spanish SAFEHEART cohort, including FH and non-FH subjects. Two statin-treated FH groups were analysed: individuals with early CVE (myocardial infarction or unstable angina before 55 years, N=76, 50% women) and resilient subjects who reached 70 years without clinical CVE. A non-FH healthy group (N=30, 50% women) served as reference. Mean age at inclusion was 57 years. In the CVE group, clinical events occurred almost one year before biological sample collection. HDL fractions were isolated from serum by density gradient ultracentrifugation, and proteomic profiles were analysed using an untargeted mass-spectrometry (LC-MS/MS). Differential protein signatures were identified through bioinformatic analysis.

Results:

Seventy-four proteins were consistently detected in HDL fractions, of which 16 showed significant differences between FH and non-FH subjects. Four proteins involved in thrombosis—SERPINA10, C3, CETP, and PLTP—were reduced in FH, whereas immunoglobulin A1 (IGHA1), linked to increased thrombosis risk, was >1.5-fold elevated. Within FH, individuals with previous CVE displayed a distinct 12-protein signature compared with resilient FH patients. Immunoglobulins IGHM and IGHA1, fibrinogen alpha chain (FGA), and protease inhibitor A2M were >1.5-fold increased, while antioxidant and anti-inflammatory proteins (PON1, PON3, APOA4, CLU, LCAT) and lipid-transfer proteins (CETP, LCAT) were significantly reduced in HDL from CVE subjects. The proteomic pattern was largely consistent across sexes, except CETP, which was lower in women with CVE but not in men. ApoA levels correlated positively with LCAT and CETP and negatively with FGA, IGHA1, and A2M. The correlation between ApoA and FGA appeared only in the CVE subgroup, suggesting a shift toward a prothrombotic, proinflammatory HDL phenotype. Notably, HDL-c levels did not correlate with the differential protein signature, underscoring that HDL functionality depends on composition rather than cholesterol content.

Conclusion:

The HDL proteomic profile identifies a distinct signature associated with premature cardiovascular events in FH, independent of HDL-c levels. Enrichment of coagulation and inflammatory related-proteins, along with depletion of antioxidant and lipid-transfer components, supports a dysfunctional HDL phenotype contributing to increased atherothrombotic risk. Although based on a limited cohort and untargeted proteomic analysis, these findings suggest that HDL composition may modulate atherothrombotic processes in FH and warrant confirmation in larger, targeted studies.

OC21

Sex-dependent variations in platelet tissue factor expression in familial hypercholesterolemia: implications for thrombotic risk

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Background:

Familial hypercholesterolemia (FH) is a genetic disorder characterised by high levels of LDL cholesterol (LDLc) from birth, which increases the risk of premature atherosclerotic cardiovascular events (CVE). FH is associated with a proinflammatory and prothrombotic state, in which platelets play a key role. Platelet activation promotes the expression of proteins such as P-selectin and tissue factor (TF), as well as chemokines such as regulated upon activation, normal T-cell expressed and secreted (RANTES), which contribute to leukocyte adhesion, coagulation and vascular inflammation. Here, we aimed to characterize the proinflammatory-prothrombotic platelet profile by measuring P-selectin, RANTES, and tissue factor in FH patients with a history of CVE treated for over two years post-event.

Material and methods:

Platelet extracts were obtained from blood samples of subjects enrolled in the SAFEHEART cohort (n = 83), including genetically confirmed FH subjects (n = 57) and their unaffected relatives (Non-FH subjects, n = 26). Forty-two percent of FH-subjects had suffered a clinical CVE (FH/CVE; n = 24) at least 2 years prior entering the cohort study and were under statin treatment for more than 2 years. A group of FH subjects without evidence of CVE (FH/Non-CVE; n = 33) was also investigated. Platelet P-selectin, tissue factor (TF) and RANTES levels were quantified by enzyme-linked immunosorbent assay (ELISA) in platelet extracts from citrated blood.

Results:

Patients with FH under optimal statin therapy, according to clinical guidelines, showed no significant differences in platelet P-selectin, TF and RANTES compared to non-FH controls. These markers were independent on age. In sex-stratified analysis, no differences were observed for these platelet activation markers within the non-FH group. However, among FH-subjects, women tended to have higher platelet TF levels than men (1.2-fold increase; *p=0.066*). This sex difference was significant in FH subjects without previous CVE (TF in FH/Non-CVE: Women=40.5 [32.1-46.5] pg/mg protein; Men=27.6 [20.5-33.7] pg/mg protein, *p=0.021*). Notably, this difference was not observed in FH subjects with prior events due to a significant increase in TF levels in men who experienced cardiovascular events compared to FH men without events (FH/CVE vs. FH/Non-CVE =39.4 [33.4-43.1] vs 27.6 [20.5-33.7] pg/mg of protein, *p=0.014*).

Conclusions:

Our findings demonstrate that long-term guideline-based statin therapy in familial hypercholesterolemia patients stabilizes platelet activation markers to levels comparable to unaffected non-FH relatives. The observed sex-specific differences in tissue factor expression may indicate subtle variations in thrombotic risk, warranting further investigation. These results underscore the importance of sustained risk management in FH to mitigate prothrombotic and inflammatory states

OC28

Antiplatelet therapy and prognosis in patients with spontaneous coronary artery dissection: a single-center cohort study

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Background:
Spontaneous coronary artery dissection (SCAD) is an uncommon cause of acute coronary syndrome (ACS), characterized by non-traumatic and non-iatrogenic obstruction of the coronary artery lumen. Current knowledge claims that single antiplatelet therapy (SAPT) management is generally safe and preferred in stable patients, as thrombolytic and antiplatelet therapies may increase bleeding risk, promote extension of intramural hematoma and delay vessel healing. However, evidence on the prognostic impact of SAPT in SCAD remains limited. This study aimed to characterize a consecutive cohort of SCAD patients in terms of clinical, sociodemographic, and angiographic features, and to evaluate the association between single versus dual antiplatelet therapy and adverse cardiac and hemorrhagic events.

Materials and methods:
This is a single-center, retrospective, longitudinal observational study including 84 patients diagnosed with SCAD admitted between January 2010 and February 2024, accounting for 104 SCAD events. Patients were stratified by antiplatelet therapy at discharge: 37 received single antiplatelet therapy (SAPT) and 44 received dual antiplatelet therapy (DAPT). Adverse outcomes assessed included the composite outcome which comprises SCAD/ACS recurrence, hospitalizations due to heart failure, stroke and all-cause mortality. Hemorrhagic events were also evaluated in this study.

Results:
The median age was 55 years, and 83.3% of patients were women. Twenty-seven patients (32.2%) had at least one predisposing factor, the most frequent being multiparity (n = 17, 21%). Sixteen patients (19%) had no identifiable cardiovascular risk factors. Non–ST-elevation myocardial infarction was the presenting manifestation in 64.3% of cases. The left anterior descending artery was most frequently affected (56%), and angiographic type 2 SCAD was the predominant pattern (77%). A conservative approach was the initial management strategy in most patients (n = 67, 79.8%), while 17 (20.2%) underwent percutaneous coronary intervention. SCAD recurrence occurred in 17 patients. There was no statistically significant difference in baseline characteristics between SAPT and DAPT group, The group under DAPT association presented a higher occurrence of heart failure hospitalizations (SAPT: 0 [0.0%] vs. DAPT: 6 [13.6%], p= 0.029), with no significant difference on the composite outcome or occurrence of hemorrhagic events.

Conclusions:
Single antiplatelet therapy was not associated with an increased risk of adverse cardiac events, when compared with dual antiplatelet therapy. These findings support a conservative antiplatelet approach in stable SCAD patients, suggesting that routine DAPT may not provide additional benefit for preventing recurrent cardiac events. Given the potential risks associated with dual therapy, including bleeding and delayed vessel healing, individualized antiplatelet strategies should be considered. Further prospective studies are needed to establish optimal antiplatelet regimens and to clarify the balance between thrombotic and hemorrhagic risks in SCAD.

OC39

Thrombotic events after prothrombin complex administration in chronic liver disease and cirrhosis' patients: a tertiary care hospital retrospective study

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Background:

Hemostasis is rebalanced in chronic liver disease, especially in cirrhosis. Recent literature recognizes that these patients do not need empirical correction of prolonged coagulation times before several invasive procedures.

When correction is required, fresh frozen plasma (FFP) is discouraged due to risks of circulatory overload and transmission of infections. Prothrombin complex concentrates (PCC), due to a lesser volume of administration and faster action, are preferred.

Material and methods:

Retrospective study between April 1st 2024 and April 30th 2025. Inclusion criteria: adults with chronic liver disease and cirrhosis who received PCC during this period at a tertiary care hospital. Primary endpoint: evaluate potential thrombotic complications within three months of drug administration.

Results:

From 560 patients who received PCC, 54 met the study inclusion criteria. Of these, 10 had chronic liver disease (19%) (etiologies: alcoholic, metabolic dysfunction-associated steatohepatitis [MASH]) and 44 patients had liver cirrhosis (81%) (etiologies: alcoholic, MASH, hepatitis C, hemochromatosis).

Regarding the studied population, average age was 65 years (minimum 49 years, maximum 85 years) (approximately 19% female and 81% male).

The average dose of PCC administered was 22IU/kg (7-41IU/kg).

Seventeen patients were on prior anticoagulant therapy (31%): 47% medicated with vitamin K antagonists (63% atrial fibrillation, 13% bileaflet mechanical aortic valve prosthesis, 13% biological mitral valve prosthesis, 13% superior mesenteric vein thrombosis) and 53% under DOACs (atrial fibrillation).

The most common clinical indications for administering PCC were: hemostasis laboratory abnormalities associated to hemorrhage (43% of patients), hemostasis laboratory abnormalities prior to an invasive procedure (approximately 43%) [Table 1].

Thrombotic events within 3 months after therapy with PCC occurred in 6 patients (11%) [Table 2].

Conclusions:

From this study, we observe that the studied population was predominantly male, as reported in the literature. The average dose of PCC administered per kilogram was within the recommended range, however additional data are necessary to determine the most effective and safe dosing regimen in this context.

The most prevalent clinical criteria for administering PCC were active bleeding and prophylactic correction of hemostasis laboratory abnormalities before invasive procedures, even though there was no active bleeding.

Within three months of PCC administration, thrombotic events occurred in 11% of these patients. Compared to previous reports involving oral anticoagulation reversal, patients with chronic liver disease and cirrhosis appear to exhibit a slightly increased incidence of thrombotic events following PCC administration. Patients who were previously on anticoagulation (higher thrombotic risk) exhibited a similar rate of thrombotic events compared to those who were not. Although data on efficacy and safety are still scarce in the literature, the thrombotic risk is recognized and warrants individual assessment.

The high mortality rate observed in this population (39%) likely reflects the burden of multiple comorbidities associated with chronic advanced liver disease.

Moreover, we need continuous investigation and education, namely regarding point-of-care tests for assessing hemorrhagic risk in these patients, as well as well-designed, prospective trials that are urgently required to provide the high-quality evidence necessary to define the best practices.

Clinical criteria	Patients		
	N		N Total (%)
	Patients without prior anticoagulation	Patients previously on oral anticoagulation	
Hemostasis laboratory abnormalities + Hemorrhage	17	6	23 (43%)
Hemostasis laboratory abnormalities prior to an invasive procedure	20	3	23 (43%)
Hemorrhage + anticoagulation reversal	0	5	5 (9%)
Anticoagulation reversal prior to an invasive procedure	0	3	3 (6%)

Table 1: Indications for prothrombin complex concentrate administration in the studied population between April 1st 2024 and April 30th 2025

Caption: Hemorrhage characterization: gastrointestinal, intracerebral, osteoarticular, hemothorax, hemoperitoneum, oropharynx

Occurrence of thrombotic events within 3 months following administration of Prothrombin Complex concentrate	Yes N (%)	6 (11%)	Thrombosis of the distal superior mesenteric vein*1	LMWH
			Cardioembolic ischemic stroke in the territory of the left middle cerebral artery*2	LMWH
			Non-ST-elevation myocardial infarction*2	DOAC + dual antiplatelet therapy
			Progression of pre-existing portal vein thrombosis	LMWH
			Thrombosis of the portal vein	LMWH
			Left fronto-temporo-parieto- occipital stroke	Deceased
	No N (%)	23 (43%)	-------	-------
Deceased		21 (39%)	-------	-------
Lost to follow-up		4 (7%)	-------	-------

Table 2: Monitoring of adverse events of prothrombin complex concentrate focusing on thrombotic events within 3 months after therapy and other hemostasis modulators' therapeutics
*Patients previously on oral anticoagulation (*1 due to superior mesenteric vein thrombosis; *2 due to atrial fibrillation)

Caption: Direct oral anticoagulant (DOAC); low-molecular-weight heparin (LMWH)

OC44

Spontaneous haematomas associated with Enoxaparin: a retrospective study

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Background:

Haemorrhage is the most frequent and clinically significant complication of anticoagulant therapy. Spontaneous intramuscular and retroperitoneal haematomas are uncommon but often life-threatening. Their incidence has increased with the widespread use of low-molecular-weight heparin LMWH for venous thromboembolism prevention and treatment.

Although LMWH is pharmacologically predictable, certain high-risk groups—especially those with renal impairment, advanced age or extremes of body weight—show marked pharmacokinetic variability, leading to accumulation and bleeding even at therapeutic doses. Anti-factor Xa monitoring may improve safety, yet remains inconsistently applied.

Spontaneous intramuscular haematomas can present insidiously, with diagnostic delay contributing to high mortality. Computed tomography is the diagnostic standard, and transarterial embolisation shows high success in stable patients. However, data on LMWH-related spontaneous haematomas remain limited. This study aimed to describe their clinical features, management, and predictors of in-hospital mortality in the Unidade Local de Saúde Almada-Seixal.

Material and methods:

A retrospective study included all adults admitted between 1 January and 31 December 2023 with radiologically confirmed spontaneous haematomas under enoxaparin. Neuroaxial location, trauma, surgery-related or vascular lesions were excluded. Demographics, comorbidities, anticoagulation regimen, laboratory data, treatment and outcomes were collected from medical records.

Bleeding severity followed ISTH criteria. Statistical analysis used SciPython libraries: Fisher's exact test for binary, Mann–Whitney U for continuous, and chi-square for categorical variables, with significance at $p < 0.05$. Ethical approval and consent waiver were obtained.

Results:

Thirty-seven patients were identified (mean age 78.9 ± 9.2 years; 59.5 % female). Most received therapeutic enoxaparin (97 %), with adequate renal adjustment in all cases except one. Hypertension (91.9 %), atrial fibrillation (70.3 %), heart failure (54.1 %), renal impairment (37.8 %) and diabetes (29.7 %) were common. Baseline bleeding risk was high (HAS-BLED 1.97 ± 1.09; VTE-BLEED 2.96 ± 1.24).

Haematomas were located in the abdominal wall (37.8 %), retroperitoneum (27.0 %) or lower limbs (27.0 %). Reversal therapy was used in 32 %, embolisation in 2.7 % and surgery in 8.1 %. Vasopressors were required in 27 %. Anti-Xa activity was assessed in only three patients.

The overall in-hospital mortality was 48.6 % (18/37), of which twelve patients were directly attributed to the acute haemorrhagic event and with over half of the patients dying within 48 hours. Mortality correlated with thrombocytopenia ($p = 0.063$), major bleeding ($p = 0.042$), retroperitoneal or multisite haematomas and vasopressor use ($p = 0.029$). Survivors more often received reversal therapy ($p = 0.038$) and resumed anticoagulation ($p = 0.003$); none developed ischaemic complications despite high thrombotic risk.

Conclusions:

Spontaneous enoxaparin-related haematomas occurred mainly in elderly, comorbid, high-risk patients, with almost half dying—mostly within two days. Mortality was associated with deep or retroperitoneal bleeding, vasopressor requirement, thrombocytopenia and major bleed classification. Early reversal and anticoagulation reintroduction were protective.

These results show that outcomes depend not only on baseline fragility but also on bleeding severity, location and haemodynamic collapse. Anti-Xa monitoring was underused and could aid early detection of excessive anticoagulation. Prompt recognition, reversal and stabilisation remain crucial to improve survival.

Variable	Deceased (n=18)	Alive (n=19)	p-value
Haemorrhage classification			
Major bleed	17 (94.4%)	12 (63.2%)	0.042
Clinically relevant minor bleed	1 (5.6%)	7 (36.8%)	
Haemorrhage location			
Thoracic wall	3 (16.7%)	1 (5.3%)	
Abdominal wall	5 (27.8%)	7 (36.8%)	
Intraperitoneal	1 (5.6%)	0 (0.0%)	0.085
Retroperitoneal	8 (44.4%)	2 (10.5%)	
Upper limb	1 (5.6%)	1 (5.3%)	
Lower limb	2 (11.1%)	8 (42.1%)	
Multisite	3 (16.7%)	0 (0.0%)	
Lowest haemoglobin level, g/L	49.7 ± 37.2	41.1 ± 34.8	0.669
Transfusion of packed red blood cells	14 (77.8%)	11 (57.9%)	0.295
Use of reversal agents	9 (50.0%)	16 (84.2%)	0.038
Protamine sulphate	9 (50.0%)	5 (26.3%)	0.184
Prothrombin complex	1 (5.6%)	0 (0.0%)	0.487
Fresh frozen plasma	5 (27.8%)	2 (10.5%)	0.232
Procedures			
Embolisation	1 (5.6%)	0 (0.0%)	0.4865
Surgery	1 (5.6%)	2 (11.2%)	1.000
Use of vasopressors	8 (44.4%)	2 (10.5%)	0.029
Admission to high dependency or intensive care units	6 (33.3%)	3 (15.8%)	0.269
Ischaemic complication	1 (5.6%)	1 (5.3%)	1.000
Infectious complication	0 (0.0%)	1 (5.3%)	1.000
Anticoagulation reintroduction	5 (27.8%)	15 (78.9%)	0.003
Time without anticoagulation	11.2 ± 14.4	13.5 ± 9.3	0.391
Recurrence of haemorrhage	2 (11.1%)	2 (10.5%)	1.000

Table 3. Characteristics of haemorrhage and clinical evolution.

Variable	Deceased (n=18)	Alive (n=19)	p-value
Indications for LMWH Prophylactic Therapeutic	 0 (0.0%) 18 (100.0%)	 1 (5.3%) 18 (94.7%)	1.000
Concomitant antiaggregation	3 (16.7%)	1 (5.3%)	0.340
Latest haemoglobin level before haemorrhage, g/L	77.4 ± 46.8	67.2 ± 52.1	0.616
Anaemia before haemorrhage	13 (72.2%)	12 (63.2%)	0.728
Latest platelet count before haemorrhage, x10^9/L	255 ± 145	270 ± 102	0.323
Thrombocytopaenia before haemorrhage	3 (16.7%)	1 (5.3%)	0.340
Latest TP before haemorrhage, sec	15.3 ± 4.0	13.4 ± 1.7	0.125
Latest INR before haemorrhage	1.4 ± 0.3	1.2 ± 0.2	0.132
Latest APTT before haemorrhage, sec	36.6 ± 6.8	34.8 ± 5.3	0.374
Latest creatinine clearance before haemorrhage, ml/min/1.73m²	65.4 ± 26.1	61.7 ± 24.2	0.658
LMWH dose adjusted to renal function	17 (94.4%)	19 (100.0%)	0.487
Anti-Xa monitoring performed	3 (22.2%)	0 (0.0%)	0.105
Anti-Xa levels Infratherapeutic Therapeutic Supratherapeutic	 0 (16.7%) 2 (66.6%) 1 (33.3%)	 0 (0.0%) 0 (0.0%) 0 (0.0%)	1.000

Table 2. Latest patient characteristics before haemorrhage.

Variable	Deceased (n=18)	Alive (n=19)	p-value
Mean age, years	77.5 ± 10.9	80.3 ± 7.3	0.681
Gender Male Female	 9 (50.0%) 9 (50.0%)	 6 (31.6%) 13 (68.4%)	0.325
Weight, kg	81.2 ± 13.9	72.7 ± 16.2	0.204
Baseline haemoglobin level, g/L	93.0 ± 54.3	72.6 ± 53.9	0.316
Baseline anaemia	9 (50.0%)	7 (36.8%)	0.515
Baseline platelet count, x10^9/L	208 ± 95	237 ± 90	0.255
Thrombocytopaenia	7 (38.9%)	2 (10.5%)	0.063
Baseline creatinine, mg/dl	1.1 ± 0.5	1.1 ± 0.6	0.636
Baseline creatinine clearance, ml/min/1.73m²	67.6 ± 25.0	60.4 ± 21.9	0.359
Baseline creatinine clearance below 60	7 (38.9%)	7 (36.8%)	1.000
Systemic arterial hypertension	16 (88.9%)	18 (94.7%)	0.604
Type II diabetes mellitus	6 (33.3%)	5 (26.3%)	0.728
Chronic heart failure	10 (55.6%)	10 (52.6%)	1.000
Atrial fibrillation	11 (61.1%)	15 (78.9%)	0.295
Coronary artery disease	1 (5.6%)	4 (21.1%)	0.340
Cerebrovascular disease	2 (11.1%)	6 (31.6%)	0.232
Peripheral artery disease	5 (27.8%)	2 (10.5%)	0.232
Venous thrombosis	3 (16.7%)	2 (10.5%)	0.660
Chronic liver failure	3 (16.7%)	3 (15.8%)	1.000
Active malignancy	2 (11.1%)	0 (0.0%)	0.230
Previous anticoagulation	14 (77.8%)	16 (84.2%)	0.693
Previous antiagreggation	4 (22.2%)	2 (10.5%)	0.405
CHA_2DS_2-VASc score	4.3 ± 2.1	5.1 ± 1.4	0.356
HAS-BLED score	1.9 ± 0.9	2.0 ± 1.3	0.700
VTE-BLEED score	3.0 ± 1.4	2.9 ± 1.1	0.743

Table 1. Patient characteristics at baseline.

OC47

Massive blood transfusion rates in the care of trauma injuries

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Background:

Management of trauma patients with massive intraoperative bleeding and the usual accompanying massive blood transfusion (MBT) is challlenging. For optimal management of MBT patient, communication between clinicians, transfusion and laboratory services is essential. We aimed to identify massive transfusion rates, basic characteristics and outcome of target patient population as a first step in order to establish the best practices and appropriate protocols for improvement of MBT patient outcomes in our hospital.

Materials and methods:

The total number of patients admitted to Traumatology Clinic, University Hospital Centre Sestre milosrdnice, Zagreb, Croatia between years 2015 and 2023. with number of MBT cases was retrospectively recorded. The amount of red blood cell units (RBCu), age, gender, type of injury, lenght of hospitalization and outcome of MBT cases was analyzed. MBT was defined as ≥10 blood units administrated in 24h. The Mann-Whitney test was used for group comparisons with $P < 0.05$ (MedCalc Software, Ostend, Belgium).

Results:

Overall rate of MBT in all hospitalized patients was 0.4%. Among 102 MBT patients (aged 19 to 91; 73% males), burn injuries were most represented (33%). Median of hospitalisation lenght was 25 days. Overall mortality among MBT patients was 27.5% (N=27). In total, 32550 RBCu were administrated, with 1488 related to MBT with median of 11 doses per MBT patient. 10.8% of patients required MBT twice and 2.9% three times. No significant difference was found between survivors and non-survivors considering gender (p=0.314), median age (67 vs. 66; p=0.887) and hospitalisation lenght (25.5 vs. 22.5 days; p=0.168), respectively. The number of RBCu administreated per non-survivals was 2.5 higher than in survivos (25 vs.10, respectively).

Conclusion:

Number of transfunded RBCu in massive blood loss within 24h could be early indicator of survival. Extended investigation and follow up of other indicators should be done in order to set up appropriate protocols.

OC49

Venous thromboembolism and cancer: hospital characterization and VTE as a potential sentinel event

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Background:
Up to 30% of venous thromboembolism (VTE) episodes occur in patients with active cancer, particularly within the first 12 months, when the risk is up to ninefold higher than in the general population. Conversely, VTE may precede an occult malignancy, with up to a fourfold increased cancer risk during the first year after thrombosis. This study aimed to characterize the clinical, laboratory, and oncologic profiles of patients hospitalized for VTE, distinguishing cancer-related from non-oncologic events, to identify predictive patterns and implications for clinical practice.

Materials and methods:
Retrospective, observational, multicenter study including all patients admitted for VTE to the Internal Medicine department of a Portuguese hospital between 2021–2023. Data collection included clinical, laboratory, imaging, and oncologic variables. Cancer status was categorized as: (1) No cancer; (2) Active <1 year; (3) Active ≥1 year; and (4) Post-VTE (cancer diagnosed ≤12 months after the index thrombotic event).

Results:
A total of 275 patients were included: No cancer 116 (42.2%), Active <1 year 53 (19.3%), Active ≥1 year 7 (2.5%), and Post-VTE 99 (36.0%), yielding 57.8% cancer-associated VTE. Thrombotic events comprised 106 pulmonary embolisms (PE), 115 deep vein thromboses (DVT), and 54 thromboses at unusual sites (41 splanchnic, 6 upper limb/cervical). Non-cancer patients showed a higher proportion of central PE (59.7%) and more frequent hemodynamic instability, though without statistical significance (p=0.08). Among cancer-associated VTE, the most prevalent primaries were gastrointestinal (18.8%), hematologic (16.8%), lung (16.8%), and prostate (8.9%). Post-VTE cancers occurred predominantly in colon (68%) and hematologic malignancies (71%), without statistical significance compared with other sites (p=0.15 and p=0.13, respectively).

Conclusions:
More than half of VTE cases were cancer-related, with a substantial proportion diagnosed after the thrombotic event—supporting VTE as a potential sentinel marker for occult cancer, particularly colorectal and hematologic malignancies. Although statistical significance was not reached, the observed trends and their proximity to significance suggest that a larger sample might confirm these associations. These findings underscore the need for structured oncologic surveillance following unprovoked VTE, integrating early multidisciplinary referral and context-driven cancer screening. Larger multicenter studies are warranted to validate these results and strengthen evidence for standardized post-VTE cancer screening protocols.

OC52

Heparin-induced thrombocytopenia managed with a direct oral anticoagulant: a case report

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Background:

Heparin-induced thrombocytopenia (HIT) is a rare but potentialy life-threatening, immune-mediated complication of heparin exposure. It results from antibodies directed against platelet factor 4 (PF4)–heparin complexes, leading paradoxicaly to platelet activation and thrombosis despite thrombocytopenia. We report a case of severe HIT successfuly treated with apixaban, highlighting the feasibility of direct oral anticoagulants (DOACs) as an alternative in se tings where parenteral non-heparin anticoagulants are unavailable.

Material, methods and Result:

A 50-year-old man was admited for cerebral venous thrombosis involving the superior sagital and left lateral sinuses, associated with bilateral parieto-occipital hemorrhagic transformation. She received unfractionated heparin for 72 hours, then was switched to low-molecular-weight heparin. On day 14, his platelet count decreased from 217 ×10?/L to 133 ×10?/L (a 45% fal). No bleeding occurred, but clinical evolution was marked by a deterioration in consciousness and the onset of respiratory distress, requiring transfer to the intensive care unit for intubation and mechanical ventilation. Folow-up brain CT combined with thoracic CT angiography revealed bilateral proximal pulmonary embolism and worsening cerebral edema with a 30 mm midline shift. The 4Ts score was calculated and estimated at 6, indicating a high probability of HIT. Screening for anti-PF4/heparin antibodies was performed using an immunoturbidimetric assay (HemosIL® HIT-Ab, ACL TOP analyzer, Werfen®). This fuly automated method measures the turbidity change caused by the formation of antigen–antibody complexes, alowing rapid, quantitative, and reliable detection of HIT antibodies. The test result was strongly positive 11.4 UI/ml. confirming the diagnosis of immune-mediated HIT. Heparin was immediately discontinued. Because fondaparinux and other parenteral non-heparin anticoagulants (such as argatroban or danaparoid) were unavailable in our institution, apixaban 5 mg twice daily was initiated as a therapeutic alternative. Platelet count rose progressively to 274×10?/L. No new thrombotic or bleeding events occurred. The subsequent clinical course was favorable, with stable platelet counts and resolution of thrombosis at the 3-month folow-up. The patient presented only with mild residual right hemiparesis. A comprehensive etiological work-up was initiated, including immunological and thrombophilia testing, antiphospholipid antibody screening, thoracoabdomino-pelvic CT scan, colonoscopy, upper endoscopy, breast ultrasound, cervical smear, infectious workup, and serum protein electrophoresis, al of which were unremarkable.

Conclusions:

This case ilustrates that DOACs, especialy apixaban, may represent a safe and e fective therapeutic option in the absence of specific treatments for HIT, alowing prompt anticoagulation while ensuring close clinical and laboratory monitoring.

OC53

Activated charcoal neutralizes xaban interference in vitro: a practical and efficient approach

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Contexte:

Direct oral anticoagulants (DOACs), particularly xabans, are increasingly used for thromboprophylaxis but can interfere with routine coagulation assays, leading to potential misinterpretation. Activated charcoal (AC) has been proposed as an in vitro method to neutralize these anticoagulants. This study aimed to evaluate the impact of apixaban and rivaroxaban on standard hemostasis tests (PT, aPTT) and lupus anticoagulant screening (dRVVT), and to assess the e ficacy of AC in restoring assay reliability.

Matériel et méthodes:

This cross-sectional, analytical, and experimental study was conducted over two years (2023–2025) at the Biological Hematology Laboratory of Sahloul Hospital, Sousse. Blood samples were colected from patients receiving generic formulations of apixaban or rivaroxaban at both peak and trough plasma concentrations. Standard coagulation assays (PT, aPTT), dilute Russel's viper venom time (dRVVT), and anti-Xa activity were measured before and after in vitro neutralization with powdered activated charcoal (Norit Carbomix®, Kela Pharma). The eficacy of AC was assessed by the percentage of normalization of coagulation parameters compared to baseline values. Sixteen healthy plasma samples, not receiving any DOACs, were included as controls to evaluate potential nonspecific e fects of AC.

Résultats:

A total of 67 patients receiving xaban-type DOACs were included, comprising 51 on apixaban and 16 on rivaroxaban. In the apixaban group, at peak plasma concentration, PT and aPTT were prolonged in 75% of samples, and dRVVT was prolonged in 77.2%, whereas at trough, these rates decreased to 53.2% for PT/aPTT and 53.8% for dRVVT. In the rivaroxaban group, PT and aPTT were prolonged in 100% of samples at peak and 93.3% at trough, with dRVVT prolonged in 100% and 88.8%, respectively. Anti-Xa activity showed strong correlations with PT ($r = –0.64$ for apixaban; $r = –0.85$ for rivaroxaban) and dRVVT ($r = 0.72$ and 0.82, respectively). In vitro neutralization with activated charcoal was performed on 63 samples (48 apixaban, 15 rivaroxaban), resulting in significant normalization of coagulation parameters, with mean neutralization rates of 91.6% for apixaban and 84.3% for rivaroxaban. Correction rates for PT, aPTT, and dRVVT reached 100%, 80.6%, and 87.5% under apixaban, and 92.3%, 85.7%, and 90% under rivaroxaban. No e fect of AC was observed in control plasma samples.

Conclusions:

Xabans markedly interfere with routine coagulation and lupus anticoagulant assays, potentialy compromising clinical interpretation. In vitro neutralization with powdered activated charcoal (Norit Carbomix®) reliably restores test accuracy, providing a simple and practical solution for laboratories managing patients on DOAC therapy.

OC55

ROTEM® Meets Xabans: Tracking Anticoagulation in Real Time

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Background:

Direct oral anticoagulants (DOACs), particularly factor Xa inhibitors (Xabans), have transformed anticoagulation management. However, biological monitoring may stil be necessary in certain clinical situations. Rotational thromboelastometry (ROTEM®) could serve as a rapid and reliable tool to assess the anticoagulant e fect of these drugs. The aim of this study was to investigate the impact of apixaban and rivaroxaban on ROTEM® parameters (EXTEM and INTEM) and to evaluate their correlation with specific anti-Xa activity.

Material and methods:

A cross-sectional analytical study was conducted at Sahloul Hospital, Sousse, from March 2023 to April 2024. Demographic and clinical data, including age, sex, and indication for anticoagulation, were recorded. Thirty-three patients receiving factor Xa inhibitors (apixaban or rivaroxaban) were included. For each patient, two venous blood samples were colected: one at trough and one at peak plasma concentration, yielding a total of 66 ROTEM® assays (33 at nadir, 33 at peak). ROTEM® analyses included EXTEM and INTEM assays to evaluate extrinsic and intrinsic pathway coagulation dynamics. Simultaneously, specific anti-Xa activity was measured using a chromogenic assay validated for two DOACs. Statistical analyses compared ROTEM® parameters between nadir and peak levels and assessed correlations with anti-Xa activity.

Results:

Among the 33 patients included, 75.8% (n = 25) were on apixaban and 24.2% (n = 8) on rivaroxaban. The population was predominantly elderly (87.8%), with embolic non-valvular atrial fibrilation as the main indication for anticoagulation (78.1%). Anti-Xa activity increased from nadir to peak plasma concentrations, with a mean 1.7-fold rise under apixaban and a 3.2-fold rise under rivaroxaban. In line with these changes, ROTEM® analysis of 66 assays showed that apixaban induced only modest changes in coagulation parameters, whereas rivaroxaban caused a more pronounced prolongation of coagulation time (CT), particularly in the EXTEM assay, while INTEM parameters were less a fected. A positive correlation was observed between anti-Xa activity (apixaban and rivaroxaban) and EXTEM CT, indicating that this parameter most accurately reflects the degree of anticoagulation by Xabans.

Conclusions:

ROTEM® profiles in patients treated with Xabans confirm the sensitivity of EXTEM CT to anticoagulation intensity. The test could provide a rapid and practical alternative for assessing anticoagulation in emergency situations or prior to invasive procedures. Further multicenter studies including patients with bleeding or thromboembolic complications are warranted to validate these findings and clarify the clinical utility of ROTEM® in monitoring DOAC therapy.

OC56

Evaluation of pretest clinical score (4Ts) for diagnosis of heparin-induced thrombocytopenia - a retrospective study of a tertiary hospital

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Background:
Heparin induced thrombocytopenia (HIT) is a pro-thrombotic and potencially life-threatening complication of heparin therapy. HIT is characterized by a drop in platelet count, usually beginning 5-14 days after heparin exposure. HIT diagnosis relies on clinical suspicion determined by 4Ts score (degree of thrombocytopenia; timing of platelet decline after heparin administration; presence of thrombosis or other HIT sequelae; and the probability of other causes of thrombocytopenia) and immunoassays through testing for anti-PF4/heparin antibodies (HITAb). Current guidelines recommend use of the 4Ts score before ordering laboratory tests as a measure of pretest probability. The aim of our study was to evaluate the utilization of the 4Ts score before ordering HITAb testing for HIT diagnosis at São João University Hospital.

Material and methods:
A retrospective observational study was performed on patients admitted to our hospital between May 1, 2024 and April 30, 2025 that had HITAb test ordered. Laboratory tests included commercial lateral flow or chemiluminescence immunoassay kits. Given the retrospective nature of our study, confirmatory testing was not performed on all patients, and therefore all patients with a positive HITAb test were assumed to be truly positive. After calculating the 4Ts score retrospectively, we calculated the proportion of HITAb tests which were not indicated due to a low risk 4Ts score. Scores of 0-3, 4-5 and 6-8 were classified as low, intermediate and high risk, respectively.

Results:
A total of 90 patients that underwent HITAb testing were analysed (52 male and 38 female). Mean age was 63,8 ± 19,4 years. 50% of the patients were at the intensive care unit (ICU), 26,7% and 23,3% were at medical and surgical departments respectively. Pre-test probability using the 4T score revealed 39 patients (43,3%) in the low risk category, 36 (40,0%) in the intermediate risk category and 15 (16,7%) in the high risk category. Analysis of testing trends showed that only 2 of the 39 patients (14,3%) with low 4Ts score tested positive for HITAb, while 11 of the 15 patients (78,6%) with high risk score tested positive for HITAb. 64,3% of the patients who had a positive HITAb test were receiving treatment with unfractionated heparin (UFH).

Conclusions:
HIT is one of the most common immune-mediated adverse drug reactions. However, several clinical scenarios may cause thrombocytopenia in a patient receiving heparin, namely sepsis or liver disease, commonly seen in critical patients at ICU. Our study showed that in our hospital, the 4Ts score is not always accounted for when managing a patient with suspected HIT. This seems to result in unnecessary laboratory testing causing great financial burden. The use of 4Ts score for assessing the pretest probability of HIT has the potential to simplify and improve the process of identifying patients at different risk of HIT. In particular, a low risk 4Ts score is suitable for ruling out HIT in most clinical settings. We hope to improve practice trends in our institution through a multidisciplinary approach.

OC61

Outpatients over 75 with a first venous thromboembolic event: a prospective observational study of therapeutic management after the acute phase in the first 12 months (SOCRATE study)

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Introduction:

The 2019 ESC guidelines strongly recommend anticoagulant therapy for the first 3 months following an acute venous thromboembolism (VTE). However, the net benefit of extended anticoagulation remains uncertain, particularly in elderly and frail patients, who are at increased risk of bleeding. These patients are underrepresented in major clinical trials on direct oral anticoagulants (DOACs) and in extended-treatment studies such as AMPLIFY-EXT and EINSTEIN CHOICE, despite being increasingly encountered in routine outpatient practice. Balancing the risk of thrombotic recurrence against bleeding complications remains a major clinical challenge, especially in older patients. Recently developed predictive models (CHAP and VTE-PREDICT) may assist in risk stratification, although their real-world applicability has not yet been fully established. This study aims to provide real-world evidence from outpatient practice to address this increasingly relevant clinical challenge in an aging population.

Materials and methods:

218 patients aged over 75 were enrolled at the outpatient clinics of the Angiology Unit of Padua Azienda-Ospedale between Jan 2022 and Dec 2024, following a first VTE event. Baseline data were collected for each patient and they underwent serial outpatient scheduled visits over the follow-up period. The primary outcome of the study was to evaluate the incidence of thromboembolic recurrences, major bleeding complications, all-cause mortality, and the composite of adverse events occurring within 18 months after the index event. This study was approved by the local Ethics Committee (CE).

Results:

The cohort consisted of 62.8% female patients, with a mean age of 83 years. Chronic arterial disease was present in 46.5% of cases (including 30.1% with peripheral artery disease, 7% with transient ischemic attack, and 9.4% with ischemic heart disease). Reduced mobility was observed in 50.6% of patients, and 34.7% fell within the extreme BMI categories. The VTE event was predominantly provoked and presented as lower-limb deep vein thrombosis (DVT) in 68.6% of cases, with distal vein involvement in 63.1%. Active cancer was identified in 23% of patients. A direct oral anticoagulant (DOAC), most commonly edoxaban, was prescribed in 86.5% of cases. The cumulative incidence of overall adverse events was 21.1%, decreasing to 14.1% when excluding the acute phase (46 events in total, including 20 all-cause deaths, 14 clinically relevant bleedings, 0 major bleedings, and 3 VTE recurrences). At 12 months, half of the cohort had discontinued DOAC therapy; among those who continued treatment, 77% were receiving a reduced dose.

Conclusion:

This study provides real-world evidence supporting the use of reduced-dose DOACs (as an alternative to full-dose regimens) for extended VTE prevention in elderly, frail patients at risk of recurrence, demonstrating a significant reduction in bleeding risk while maintaining comparable antithrombotic protection.

Table 2. Venous Thromboembolism (VTE) characteristics

	Patients (N=218)
VTE Characteristics N (%)	
Provoked	150 (68.6)
Major transient risk factors	
Surgery	31 (20.8)
Trauma with fracture	46 (31.2)
Acute infection	27 (18.1)
Minor transient risk factors	
Catheter-related	10 (6.9)
Persistent oncologic risk factor	
Active cancer	50 (22.9)
Brain	2 (4.9)
Gastrointestinal	12 (24.4)
Lung	12 (24.4)
Genitourinary	2 (4.9)
Breast	7 (14.6)
Hematological	10 (19.5)
Otorhinolaryngologic	1 (2.4)
Pancreas	1 (2.4)
Hepatobiliary	1 (2.4)
Active chemotherapy	26 (52.1)
Metastatic cancer	10 (19.5)
History of cancer	30 (13.5)
Smoke	23 (10.9)
Thrombophilia	1.7 (15.3)
Type of thrombosis N (%)	
Isolate pulmonary embolism	8 (3.7)
pulmonary embolism and deep venous thrombosis	21 (9.9)
deep venous thrombosis	188 (86.4)
Site of thrombosis N (%)	
Lower limbs	194 (92.3)
Proximal	71 (36.9)
Distal	29 (63.1)
Prescribed therapy N (%)	
DOAC	188 (86.5)
Apixaban	74 (34.0)
Edoxaban	99 (45.7)
Rivaroxaban	44 (20.3)
Dabigatran	0.0
Low-molecular-weight heparin/ fondaparinux	29 (13.5)
DOAC dose in the acute phase	
Full dose/Reduced dose	157 (72.3) / 61 (27.7)

Table 3. Comorbidities by Anticoagulant Therapy Status

	Patients (N=218)	**Therapy discontinuation (N= 107)**	**Therapy continuation (N=111)**	**p value**
Comorbidies				
Hypertension (%)	69.5	72.4	66.7	0.26
Dyslipidemia (%)	53.1	58.6	47.8	0.18
PAD (%)	31.1	35.6	26.7	0.41
Diabetes (%)	14.1	13.8	14.4	0.46
Chronic ischemic heart disease (%)	10.2	16.1	4.4	0.12
Previous stroke/TIA (%)	7.9	11.5	4.4	0.46
CKD (%)	25.4	28.7	22.2	0.22
Anemia (%)	10.7	29.7	5.6	**0.02**
Thrombocytopenia(%)	1.1	2.3	0	0.09
Liver disease (%)	1.7	3.4	0	**0.03**

Table 4. Composite outcome at 18 months after acute VTE

Outcome N (%)	**Patients (N=218)**
composite Outcome	46 (21.1)
VTE Recurrences	3 (1.4)
active cancer	2 (66.7)
non-oncologic patients	1 (33.3)
Bleeding	14 (6.4)
18-Month Mortality	20 (9.1)

Table 1. Cohort characteristics

	Patients (N=218)
Baseline characteristics	
Mean Age ± Standard Deviation	83 ± 4.9
Male (%)	38.2
Comorbidities	
Hypertension (%)	67.8
Dyslipidemia (%)	52.3
Peripheral arterial disease (%)	30.1
Diabetes (%)	14.0
Chronic ischemic heart disease(%)	9.4
Previous stroke/TIA (%)	7.0
Chronic Kidney Disease (%)	24.8
Anemia (%)	10.9
Thrombocytopenia(%)	1.3
Liver disease (%)	1.9
Frailty	
Dementia (%)	15.8
History of falls (%)	15.0
Reduced mobility(%)	50.6
Body Mass Index (BMI) BMI < 20 (%) BMI 20-29 (%) BMI ≥ 30 (%)	 21.7 65.3 13.0

OC69

Pretreatment with P2Y12 inhibitors in STEMI patients referred to primary PCI – insights from a national registry

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Background:

There is no conclusive evidence regarding the optimal timing for initiation of dual antiplatelet therapy (DAPT) in patients with ST elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (pPCI).

Therefore, the aim of this study is to compare the impact of pretreatment with $P2Y_{12}$ inhibitors on angiographic and clinical outcomes in STEMI patients undergoing pPCI, with a particular emphasis on patients transferred from hospitals without pPCI capability.

Material and methods:

This study included patients from a nationwide registry with the diagnosis of STEMI managed with pPCI and $P2Y_{12}$ inhibitors during hospitalization.

Patients who were on $P2Y_{12}$ inhibitors or anticoagulants before the index event were excluded. Were also excluded patients on cardiac arrest at first medical contact (FMC).

Patients were divided into pretreatment group (PTG), if the $P2Y_{12}$ inhibitor loading dose was administered before admission to the Catheterization Laboratory and no pretreatment group (no-PTG), if loading dose was administered during the pPCI.

Primary endpoint is a composite of angiographic and adverse clinical events occurring during the index hospitalization, namely: TIMI flow<3 at the end of pPCI, death, cardiac arrest, re-infarction, cardiogenic shock, or stroke. Primary safety endpoint is severe bleeding, defined according to GUSTO investigators.

A subgroup analysis was planned for patients requiring transport from non-PCI hospitals to PCI centres, where longer FMC-to-pPCI delays were expected.

Results:

Among 7953 participants selected, 4109 patients were included in the PTG and 3844 in the no-PTG. Patients on PTG were younger than those in the no-PTG and had more frequently a past myocardial infarction and PCI. There were no significant differences between groups with respect to gender, risk factors, or history of CABG, stroke or chronic kidney disease. Systolic blood pressure at admission was higher in PTG patients, while no significant difference was observed in heart rate.

Patients in PTG more frequently presented to non-PCI hospitals (26.4% compared to 22.9% in no-PTG), while initial contact with medicalized emergency medical services (EMS) was less common in this group (28.5% versus 36%, p<0.001).

The incidence of the primary endpoint was similar in both groups- It was met in 400 (11.1%) patients in the PTG and in 348 (10.6%) patients in no-PTG (OR=1.06, 95%CI:0.90-1.23, p= 0.46). However, a significant interaction between pretreatment effects and location of FMC was found, with PTG patients presenting to non-PCI hospitals showing a reduction in the risk of primary endpoint (OR 0.73; 95%CI: 0.54-0.98, p=0.04) as opposed to an increase in primary endpoint incidence in those transported by EMS (OR=1.37, 95%CI:1.05-1.79). These differences persisted after adjustment for age, past MI and systolic blood pressure.

No significant differences were observed in safety endpoint, either in overall population or subgroup analysis.

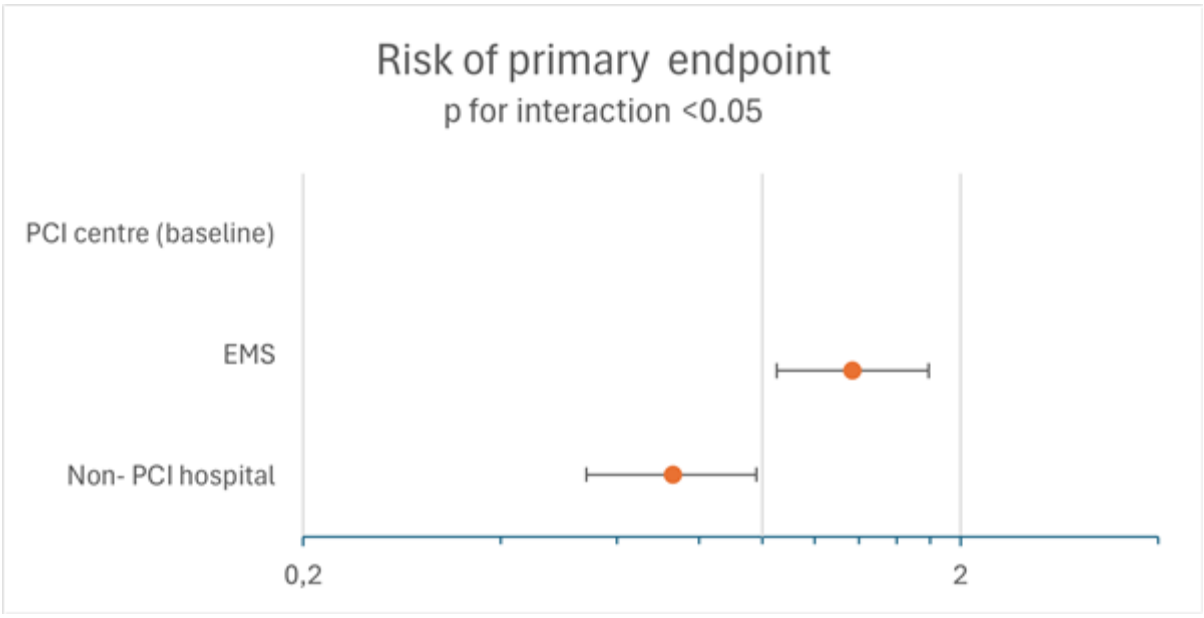
Conclusions:

In STEMI patients undergoing pPCI routine pretreatment with $P2Y_{12}$ inhibitors is not associated with a better outcome. However, in patients who need transfer from non-PCI hospitals to a PCI center, with longer FMC to pPCI delay, pretreatment may play a role. Our data support the need for further studies in this later population.

Baseline characteristics of PTG and no-PTG patients

Variable	PTG n =4109	Missing	No-PTG n =3844	Missing	p
Age, years - mean(sd)	62.1 (13.2)	4	62.7	5	0.04
Female Sex	970 (23.6)	2	889 (23.1)	14	0.67
Risk factors					
Hypertension - n (%)	2287 (55.7)	-	2198 (57.2)	-	0.17
Diabetes - n (%)	902 (22.3)	-	854 (22.6)	3	0.77
Dyslipidemia - n (%)	1987 (48.4)	-	1930 (50.2)	-	0.10
Current Smoker - n (%)	1650 (42)	-	1537 (40.0)	1	0.89
Past medical history					
MI – n (%)	298 (7.3)	-	355 (9.2)	-	0.001
PCI – n (%)	258 (6.3)	-	307 (8.0)	-	0.003
CABG – n (%)	29 (0.7)	-	25 (0.7)	-	0.76
CVD– n (%)	160 (3.9)	-	181 (4.8)	-	0.06
CKD – n (%)	110 (2.7)	3	130 (3.4)	8	0.07
FMC		54		231	
PCI centre – n (%)	1830 (45.1)		1484 (41.1)		
EMS – n (%)	1155 (28.5)		1302 (36.0)		
Non PCI centre – n (%)	1070 (26.4)		827 (22.9)		0.001
STEMI location		33		4	
Anterior/LBBB – n (%)	1984 (48.7)		1912 (49.8)		
Non anterior – n (%)	1912 (51.3)		1928 (50.2)		0.32
Killip class (admission)		82		3	
I	3478 (86.9)		3386 (88.5)		
II	319 (8.0)		265 (6.9)		
III	74 (1.8)		62 (1.6)		
IV	133 (3.3)		111 (2.9)		
Vital signs (at FMC)					
HR, bpm - mean (SD)	77 (19)	12	78 (18)	4	0.17
SBP, mmHg - mean (SD)	136 (29)	12	133 (29)	4	0.001
Lab values (admission)		322		375	
Creatinin,mg/dl -median(IQR)	0.9 (0.8-1.1)		0.9 (0.8-1.1)		0.23
$P2Y_{12}$ inhibitors*					
Clopidogrel	2873 (69.9)		2594 (67.5)		
Ticagrelor	1461 (35.6)		1333 (34.7)		
Prasugrel	5 (0.1)		167 (4.3)		

* Switch between $P2Y_{12}$ inhibitors occurred during hospitalization


Risk of primary endpoint
p for interaction <0.05
PCI centre (baseline)
EMS
Non- PCI hospital
0,2
2

OC76

Implementation of a thrombotic thrombocytopenic purpura management protocol in a tertiary hospital

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Background:
Thrombotic thrombocytopenic purpura (TTP) is a rare but life-threatening thrombotic microangiopathy requiring prompt diagnosis and treatment. Despite advances in therapeutic options including plasma exchange (PEX), caplacizumab, and rituximab, standardized management protocols remain underutilized in many healthcare settings. We propose the implementation of structured protocol for patients with the confirmed diagnosis of immune TTP in a tertiary hospital.

Material and methods:
The proposed protocol encompasses three main phases: (1) Early diagnosis featuring standardized clinical evaluation, comprehensive analytical assessment including immediate ADAMTS13 testing with proper sample handling; (2) Acute phase therapy with prompt referral to designated inpatient units, PEX initiation within 8 hours of suspicion, high-dose corticosteroids (methylprednisolone 500mg-1g daily), caplacizumab administration (10mg IV then 10mg/day SC for at least 30 days post-PEX), and rituximab therapy (375mg/m² weekly for 4 weeks) within 3 days of immune TTP confirmation; (3) Structured follow-up with weekly evaluations for the first month, monthly assessments for 3 months post-caplacizumab, and long-term monitoring every 3-months with serial ADAMTS13 measurements, with preemptive rituximab therapy for patients with documented ADAMTS13 relapses. The protocol will be implemented by a multidisciplinary working group comprising specialists from Internal Medicine, Intensive Medicine, Nephrology and Emergency Services.

Results:
The implementation of the protocol will lead to an uniformized management of TTP patients. Our main goals are, firstly, the early diagnosis of patients with TTP and timely institution of acute phase therapy. Regarding the follow-up we intend to actively search for ADAMTS13 responses (partial or complete), thus identifying patients with higher relapse risk. Adequate follow-up will also allow the early identification of ADAMTS13 relapses and, hopefully, prevent any clinical relapses.

Conclusions:
Implementation of this comprehensive TTP management protocol in a tertiary hospital provides a standardized, evidence-based approach that integrates current therapeutic advances with systematic patient monitoring. The protocol's emphasis on early ADAMTS13 testing, rapid multimodal therapy initiation, and structured long-term follow-up addresses critical gaps in TTP management. We except to achieve improved treatment response rates and reduced relapse risk through proactive monitoring and intervention.

OC80

D-Dimer testing in the emergency department: low specificity and high imaging burden in older and comorbid patients

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Background:
D-dimer testing is widely used to exclude venous thromboembolism (VTE) due to its high negative predictive value in patients with low to intermediate pretest probability scores. However, its specificity is reduced in older and multimorbid patients, as D-dimer levels may be elevated in various clinical scenarios, such as malignancy, systemic infection or cardiovascular diseases. This study aimed to evaluate the correlation between serum D-dimer levels and patient comorbidities, as well as to assess impact of applying an age-adjusted cutoff for patients over 50 years of age, particularly regarding its implications for imaging use in clinical practice related to VTE diagnosis in clinical practice.

Material and methods:
A retrospective observational study was conducted involving 1128 patients who underwent D-dimer testing at the Emergency Department (medical area) at the Emergency Department of São João University Hospital between October 1 and December 31, 2024. The prevalence of elevated D-dimer levels, associated comorbidities, imaging utilization, and confirmed VTE cases were analysed. D-dimer concentrations >500 µg/L were considered positive, according to the manufacturer's specifications.

Results:
Baseline characteristics are summarized in Table 1. Positive D-dimer levels were observed in 49.1% of patients, of whom 55.6% underwent imaging studies. However, only 64 (11.6%) patients were diagnosed with VTE (table 2). Cardiovascular risk factors were more prevalent among patients with positive D-dimer results (Table 3). Elevated D-dimer levels also appear to be associated with active infections and malignancies (Table 3). Among patients over 50 years old, 18 underwent imaging that could have been avoided if an age-adjusted cutoff had been applied. Data collection further revealed that D-dimer testing is often ordered as part of routine blood work by some physicians, regardless of pre-test clinical probability, which may contribute to unnecessary investigations.

Conclusions:
While D-dimer testing remains a valuable diagnostic tool for excluding VTE in selected low- to intermediate-risk patients, this study raises concerns about its frequent use outside recommended clinical pathways. In this study, nearly half of the patients with positive D-dimer results did not undergo subsequent imaging, suggesting either limited clinical relevance of the test in these cases or that results did not influence management decisions. This is especially evident in patients with comorbidities - such as active infection, cancer, heart failure, and cardiovascular risk factors - that are associated with elevated D-dimer levels and are highly prevalent in the studied population, as reflected in our results. Moreover, applying age-adjusted cutoffs and combining D-dimer testing with validated clinical probability scores may also improve diagnostic accuracy, reduce unnecessary imaging, and minimize potential harms.
A notable limitation of this study is the lack of detailed clinical documentation in many emergency department records, which limited our ability to retrospectively assess pre-test probability and fully understand the rationale behind test ordering. These findings emphasize the importance of ordering D-dimer testes based on the specific clinical scenario and highlight the need to enhance clinician education to ensure proper and evidence-based use of this diagnostic tool in emergency settings.

Comorbidities, n (%)	Positive DD Population*	Negative DD Population
Cardiovascular risk factors		
Hypertension	237 (48.4)	154 (26.8)
Dislipidemia	206 (42.0)	114 (19.9)
Diabetes	108 (22,0)	64 (11.1)
Smoking	92 (18,8)	82 (14.3)
Obesity	87 (15.7)	65 (11.3)
Active infection	204 (41.6)	44 (7.7)
Heart failure	131 (26,7)	51 (8.9)
Chronic inflammatory disease**	103 (21,0)	67 (11.7)
Active Cancer, n (%)	75 (15.3)	17 (2.9)
CKD	42 (8,6)	8 (1.4)
AF	37 (7,6)	33 (5.7)

Table 3 – Comparison of comorbidity prevalence between D-dimer positive and negative groups. *Patients with VTE diagnosis were excluded. ** Includes COPD, asthma, IBD, RA, SLE. AF – atrial fibrillation. CKD – chronic kidney disease. COPD – chronic obstructive pulmonary disease. IBD – inflammatory bowel disease. RA – rheumatoid arthritis. SLE – systemic lupus erythematosus.

Characteristics of the Positive DD Population	
Imaging studies, n (%)	
Yes	308 (55.6)
No	246 (44.4)
Total	554 (100)
VTE diagnostic, n (%)	
Yes	64 (11.5)
No	490 (88.4)
Total	554 (100)
Type of VTE, n (%)	
PE	41 (64.1)
DVT/SVT	23 (35.9)
Total	64 (100)

Table 2 – Distribution of imaging studies, venous thromboembolism (VTE) diagnosis, and VTE type among individuals with positive D-dimer results. DD – D-dimer. DVT – Deep Venous Thrombosis. PE – Pulmonary Embolism. SVT – Superficial Venous Thrombosis. VTE – Venous Thromboembolism.

Characteristics	
Sex, n (%)	
Female	624 (55.3)
Male	504 (44.7)
Total	1128 (100)
Age, median (*sd*), years	
Female	61 (20.9)
Male	64 (18.6)
D-dimer, n (%)	
Positive	554 (49.1)
Negative	574 (50.9)
Total	1128 (100)

Table 1 – Baseline characteristics

Posters

PO02

Concentration- and time-dependent effects of pseudomonas aeruginosa lipopolysaccharides on blood clotting

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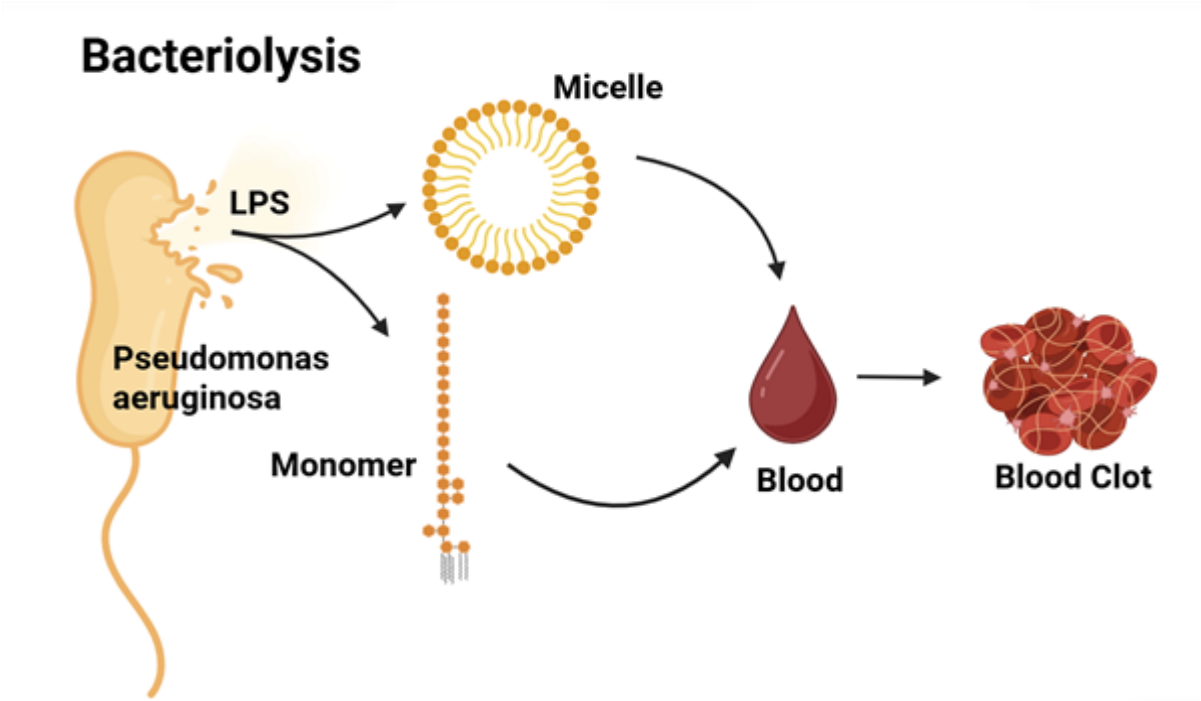
Introduction:
Pseudomonas aeruginosa bloodstream infections, particularly in sepsis, are associated with high morbidity due to disrupted hemostasis. Lipopolysaccharide (LPS) from *P. aeruginosa* contributes by inducing inflammation and directly interacts with coagulation pathways. Emerging evidence shows that LPS modulates coagulation in a concentration- and time-dependent manner through supramolecular aggregation, affecting thrombin activity and clotting. However, the mechanisms behind these biphasic effects remain poorly understood.

Materials and methods:
The physicochemical properties of LPS were analyzed using a Zetasizer to determine particle size distribution and surface charge. Prothrombin time (PT) and activated partial thromboplastin time (aPTT) were conducted using 5 µg/mL and 25 µg/mL *P. aeruginosa* LPS incubated in human citrate platelet-poor plasma (PPP) for 15 minutes or 30 minutes. aPTT utilized the Pacific Hemostasis aPTT-XL reagent incubated in plasma for 3 minutes and treated with ~6.7 mM calcium chloride. Clotting time (CT) was conducted using ~6.7 mM calcium chloride and 5 µg/mL or 25 µg/mL LPS incubated with PPP. PT, aPTT and CT were all measured using the KC4 Delta by Trinity Biotech. A 96-well microplate was incubated with 5 nM thrombin and 0 to 100 µg/mL of LPS from *P. aeruginosa* in 20 mM HEPES buffer supplemented with 150 mM NaCl at 37°C for 15 minutes. 50 µL of S2238 was added to each well and the absorption at 405 nm was measured over 1 hour.

Results:
LPS formed stable, negatively charged aggregates in solution (~220 nm diameter; zeta potential −31 mV). Coagulation assays revealed a biphasic, time- and dose-dependent effect on hemostasis. At low concentrations (5 µg/mL), LPS significantly reduced PT at both 15 and 30 minutes, suggesting a procoagulant effect via the extrinsic pathway. At 25 µg/mL, LPS had no significant effect at 15 minutes and tended to prolong PT at 30 minutes, possibly due to interference with coagulation factors or altered aggregate behavior. APTT was unchanged at 15 minutes but significantly reduced at both concentrations after 30 minutes, indicating delayed activation of the intrinsic or contact pathway. Clotting time assays supported this biphasic pattern: low LPS delayed clot formation, while higher concentrations accelerated it after longer incubation. Thrombin activity assays showed no increase across 0–100 µg/mL; instead, a mild, dose-dependent reduction was observed at higher doses. Overall, these results suggest LPS modulates coagulation via concentration- and time-dependent supramolecular rearrangements that differentially affect coagulation pathways.

Conclusions:
These findings suggest that LPS may modulate coagulation in a concentration- and time-dependent manner. Low concentrations appear to promote procoagulant activity, while higher doses or prolonged exposure might impair coagulation, highlighting a potentially complex role of LPS in infection-associated coagulopathies.


Bacteriolysis
Micelle
LPS
Pseudomonas
aeruginosa
Monomer
Blood
Blood Clot

PO03

Primary thromboprophylaxis and onkoTEV score in pancreatic cancer: real-world evidence from a portuguese cohort

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Hospital Espírito Santo Évora

Background:
Pancreatic cancer is associated with one of the highest risks of cancer-associated thrombosis (CAT). OnkoTEV is a validated predictive score for thromboembolic events (TEE). Real-world data are essential to better characterize the incidence, type of thrombotic events, and the effectiveness of prophylactic strategies.

Material and methods:
We conducted a retrospective study including patients diagnosed with pancreatic cancer between January 2021 and December 2024 in a Portuguese hospital. Clinical records were reviewed to collect patient and tumor-related characteristics, TEE occurrence, and use of primary prophylaxis. The OnkoTEV score was calculated using history of prior TEE, lymphovascular compression by the tumor, presence of metastatic disease, and the Khorana score (hemoglobin, leukocyte and platelet counts, and body mass index). Descriptive statistical analysis was performed using Excel and IBM SPSS. Comparisons between groups were performed using Fisher's test.

Results:
A total of 61 patients were included (59% female; median age 69 years, range 39–92). At diagnosis, 67% presented metastatic disease. Twenty-one patients (34.4%) received primary thromboprophylaxis (9 rivaroxaban 10 mg/day, 7 apixaban 2.5 mg twice daily, and 5 enoxaparin 40 mg/day), from which three developed TEE (14%). In the group without primary prophylaxis (n=40), 10 patients developed TEE (25%). The incidence of TEE was numerically lower with prophylaxis but this difference was not statistically significant (p=0.51).
Overall, 13 patients (21%) developed TEE: 4 pulmonary embolisms, 4 deep vein thromboses, 3 ischemic strokes, 1 splenic vein thrombosis and 1 portal vein thrombosis. The median time from diagnosis to thrombosis was 93 days (0–543). Three thrombotic events occurred under prophylaxis, all in patients receiving direct oral anticoagulants (DOACs) at prophylactic doses (n=16); all were subsequently switched to low-molecular-weight heparin (LMWH). No thrombotic events were observed under therapeutic LMWH (n=5). Given the limited sample size, no conclusions can be drawn regarding differential efficacy between prophylactic agents. There was a higher incidence of TEE in patients with higher OnkoTEV scores (0: 0%, 1: 7.7%, 2: 30.8%, 3: 61.5%) with the majority of events occurring in patients with a score ≥2. This trend supports the discriminative ability of the OnkoTEV score in identifying high-risk patients within our cohort.

Conclusions:
In this real-world pancreatic cancer cohort, thrombosis occurred in approximately one-fifth of patients, frequently within the first 3 months after diagnosis. Primary prophylaxis was associated with a numerically lower incidence of TEE, but this difference did not reach statistical significance, most likely due to the small sample size. The OnkoTEV score showed a clear correlation with TEE risk, supporting its potential role as a predictive tool in this population. These findings reinforce the indication for thromboprophylaxis in pancreatic cancer according to current guidelines and highlight the importance of larger real-world studies to better evaluate prophylactic strategies and agents.

PO04

Early cardiorenal risk factors in patients with preserved eGFR: integration of vascular and renal biomarkers

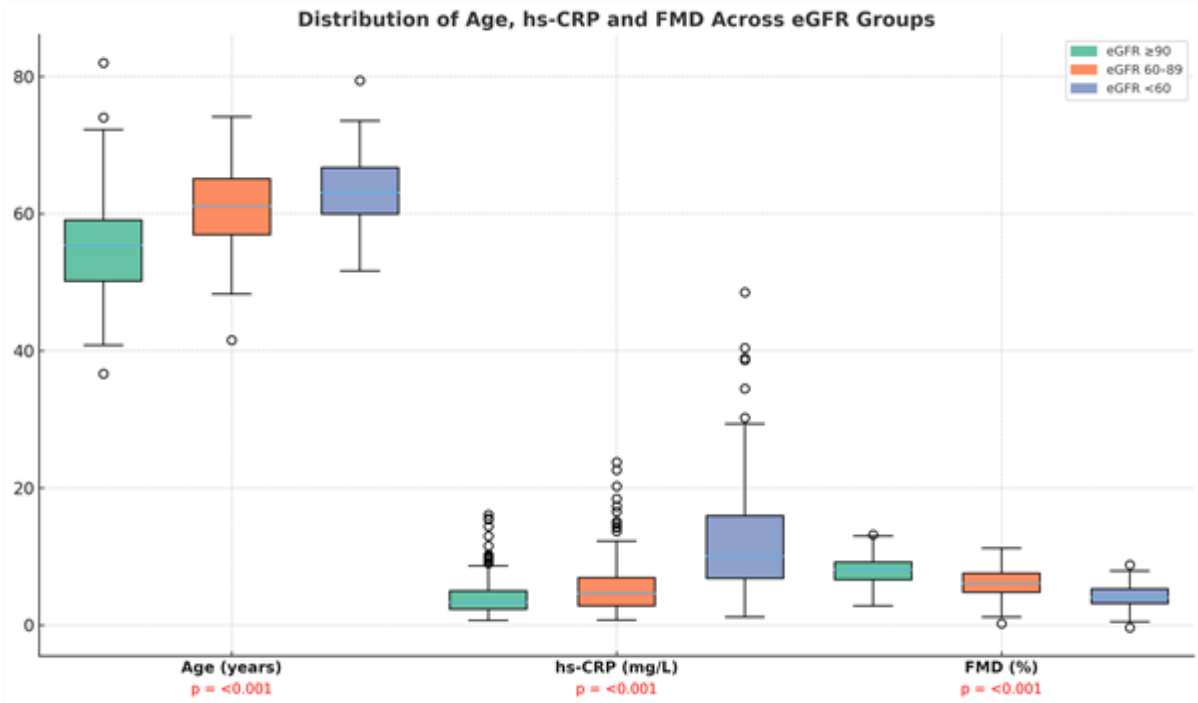
Ruslan Najaf Najafov[1]; Elman Alekberov[2]; Elbrus Huseynov[1]
[1] Scientific Research Institute of Cardiology
[2] Diamed Medical Group

Background:
Chronic kidney disease (CKD) and cardiovascular disease (CVD) share common pathophysiological pathways, forming the cardiorenal continuum. Patients with preserved estimated glomerular filtration rate (eGFR) may still present early vascular and renal risk markers that increase cardiovascular morbidity and mortality.

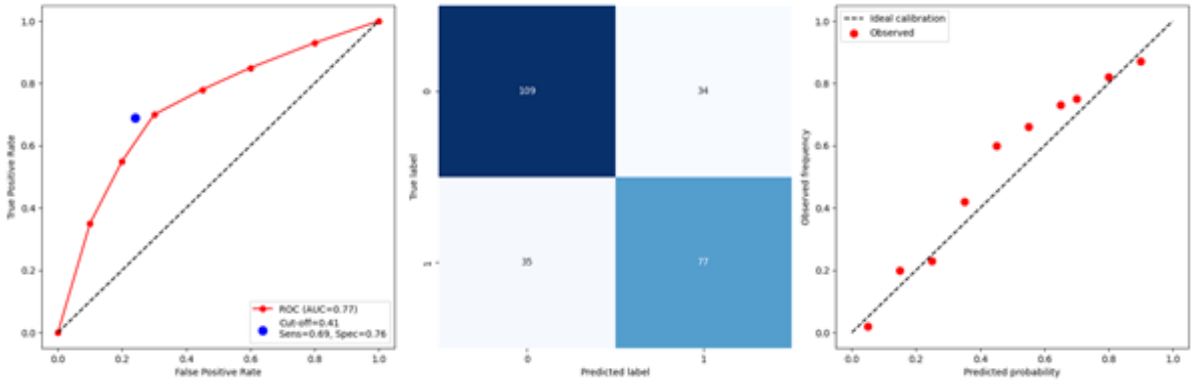
Methods:
We prospectively analyzed 847 patients (mean age 58 years; 52% male) without overt clinical CVD. Patients were stratified by eGFR (≥90, 60–89, <60 mL/min/1.73m²). Early risk factors assessed included preclinical atherosclerosis (cIMT ≥0.8 mm and/or plaque), arterial stiffness (pulse pressure >60 mmHg), endothelial dysfunction (FMD <7%), ankle-brachial index (ABI <0.9), hs-CRP >3 mg/L, microalbuminuria, proteinuria, and NT-proBNP. Logistic regression was applied to identify predictors of major adverse cardiovascular events (MACE).

Results:
Despite preserved eGFR, early risk markers were frequent: preclinical atherosclerosis (15.0–24.2%, p<0.01), hs-CRP >3 mg/L (37–61%, p<0.001), FMD <7% (56%, p<0.001), and ABI <0.9 (13.8%, p=0.018).
MACE incidence increased stepwise with renal impairment (36.4% in eGFR ≥90 vs. 69.5% in eGFR <60, p<0.001). Independent predictors of MACE included preclinical atherosclerosis (OR 3.18, 95% CI 1.9–5.3, p<0.001), diabetes mellitus (OR 1.73, 95% CI 1.1–2.6, p=0.014), age (OR 1.19, 95% CI 1.1–1.3, p=0.002), and microalbuminuria (OR 1.65, 95% CI 1.0–2.7, p=0.048).

Conclusions:
Even with normal eGFR, patients demonstrate significant early cardiorenal risk. Integrating vascular and renal biomarkers into risk stratification may enable earlier intervention and reduce long-term cardiovascular burden. This biomarker-based approach could improve early risk detection and guide preventive strategies in clinical practice.



Risk Marker	eGFR ≥90 (n=300)	eGFR 60–89 (n=400)	eGFR <60 (n=147)	*p*-value
Plaque or increased IMT (%)	15.0	19.8	24.2	<0.01
hs-CRP >3 mg/L (%)	37.0	49.5	61.0	<0.001
FMD <7% (%)	56.0	56.0	56.0	0.002
ABI <0.9 (%)	13.8	13.8	13.8	0.018
MACE incidence (%)	36.4	52.7	69.5	<0.001



PO06

Clinical aspects of protein C, protein S and antithrombin deficiencies, and activated protein C resistance: a retrospective analysis

Maatamri; Rahma Belhadj; Yassine Gaiech; Ahmed Amine Sabbagh; Wided Maatamri; Nejia Braham
hopital Farhat Hached Sousse

Background:
Thrombophilia can result from congenital deficiencies in natural anticoagulants or from acquired conditions. Interpretation of thrombophilia screening is challenging, as results are influenced by clinical context and the timing of testing.
Methods:
We conducted a retrospective study of patients with pathological thrombophilia screening results, collected between January 2019 and July 2025, defined by deficiencies in protein C, protein S, antithrombin III, or by the presence of activated protein C resistance (APCR).
Results:
A total of 127 patients were included (age range: 3–78 years; median age: 36 years). We noted a female predominance (sex ratio 0,41).
Clinical indications included prior thromboembolic events (55.6%), recurrent miscarriage or intrauterine fetal demise (23.8%), suspicion of thrombophilia (10.3%), nephropathy-related complications (6.3%), and preoperative assessment (3.2%). Family history of thrombosis was rare (0.8%).
Laboratory findings showed protein C deficiency in 42.9% of patients, protein S deficiency in 42.1%, antithrombin deficiency in 44.4%, and RPCA positivity in 14.3%. Combined abnormalities were frequent (34.6%).
Overall, 52.4% of abnormalities were considered constitutional, 40.5% acquired, and 7.1% non-interpretable.
Acquired causes included hepatic disease (24%), renal disease (16%), pregnancy (14%), autoimmune disease (12%), active malignancy (8%), HIV (4%), cardiac disease with thrombotic risk (4%), chronic inflammation (4%), and undetermined cause (14%).
Non-interpretable results were mainly related to acute thrombosis (n=5) or testing under anticoagulants (n=4).
Conclusion:
These findings highlight the need for careful interpretation and suggest that further studies are needed to clarify the etiological factors and to optimize the timing of testing.

PO07

Congenital coagulation factor deficiency: a report of 160 cases

Maatamri; Amani Dali; Wided Maatamri; Ahmad Amine Sabbagh; Sondos Hizem; Nejia Braham; Yosra Ben Youssef
hopital Farhat Hached Sousse

Background:
Congenital coagulation factor deficiencies represent a group of rare disorders. This study was conducted to describe their epidemiological, clinical, and biological characteristics and to improve their diagnostic and therapeutic management.
Material and methods:
This is a cross-sectional descriptive study carried out over a period of 9 years, from January 2016 to December 2024. We included in our study patients with VWF, fg, FII, FV, FVII, FIX, FX, FXII, FXIII, PK, KHPM deficiency.
Results:
A total of 160 cases were included: Hemophilia accounted for 49% of cases, followed by factor VII deficiency (21%) and von Willebrand disease (14%). The diagnosis was made early in hemophilia cases, with a median age of 9 months, whereas it was later for factor V deficiency, with a median age of 50 years. Hemophilia A accounted for 80% of hemophilia cases. Type 2 von Willebrand disease was the most frequent subtype (45%). repeated miscarriages were observed in one patient.
The predominant bleeding manifestations were mucocutaneous, while they were mainly deep (hematomas and hemarthroses) in 55% of hemophilia patients. Hemophilia patients required prophylactic treatment in 22% of cases and were treated with recombinant factor VIII or plasma-derived factor VIII in 67% and 33% of cases, respectively. The development of inhibitors was observed in 12% of patients, all with hemophilia A, and 25% of them received immune tolerance induction. FVII deficiency was discovered incidentally in 88% of cases. Mucocutaneous hemorrhagic syndrome was not correlated with the severity of the deficiency. Severe deficiency (FVII<10%) was found in 8 patients (23%), of whom only three had minor hemorrhagic manifestations (menorrhagia, ecchymosis). Factor XI deficiency was observed in 9 patients with 5 severe deficiencies (FXI <20%). Bleeding history was absent in 66% of cases. Afibrinogenemia was discovered following severe bleeding at the fall of the umbilical cord, FII deficiency following diffuse bruising, and both FV and FX deficiency following severe post-traumatic hemorrhages (postpartum or dental extraction). Factor XII deficiency, prekallikrein deficiency, and high molecular weight kininogen deficiency mainly affected women and were asymptomatic.
Conclusions:
Congenital coagulation factor deficiencies remain an underdiagnosed health problem. Greater awareness, the establishment of national registries, and national guidelines are essential to improve early diagnosis and management of these conditions.

PO09

Impact of dextran sulfate and exogenous antithrombin on anti-Xa levels in patients receiving low-molecular-weight heparin

Tamara Rojnik; Mojca Božic Mijovski
University Medical Centre Ljubljana

Background:

Anti-Xa is the method of choice for monitoring low-molecular-weight heparin (LMWH) in clinical practice. This chromogenic test determines heparin concentration by measuring the residual activity of exogenous FXa after its inhibition by the heparin-antithrombin (AT) complex in plasma. Despite efforts to standardize different anti-Xa assays, considerable variability between them exists, potentially impacting anticoagulation management. This variability is partly due to differences in reagent composition, particularly the presence or absence of dextran sulphate (DS) and exogenous AT. DS releases protein-bound heparin for the measurement of total heparin concentration, while exogenous AT compensates for differences in endogenous AT levels. Their individual effects on anti-Xa levels have been studied in vitro and showed a significant effect of exogenous AT, especially in samples with low endogenous AT (<68%), but no effect of the addition of DS. The aim of our study was to evaluate these effects under similar conditions, but in ex-vivo samples from patients receiving LMWH.

Material and methods:

A total of 59 citrate plasma samples from patients receiving LMWH therapy with a broad range of AT activity (42–108%) and anti-Xa levels (0.17–1.04 IU/mL) were included. Anti-Xa was measured on an ACL TOP 500 CTS coagulation analyzer (Werfen) with four anti-Xa assays (Hyphen BioMed) that differed only in the presence or absence of DS (DS+/DS−) and exogenous AT (AT+/AT−). The Friedman test and Wilcoxon signed-rank test were used as post-hoc analyses to assess statistical differences, while Bland-Altman and Passing-Bablok regression assessed agreement and bias between assays.

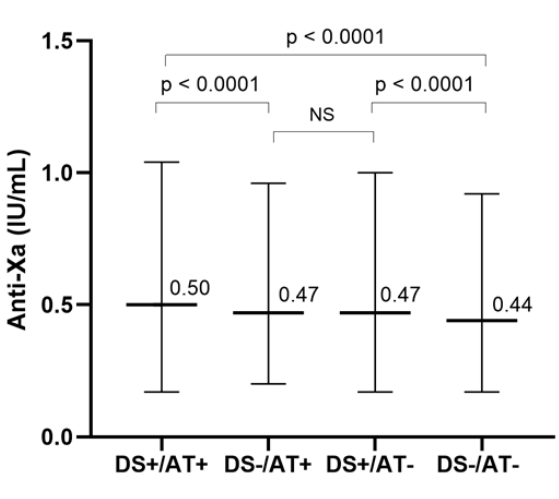
Results:

Anti-Xa levels were the highest if measured with the DS+/AT+, while the lowest anti-Xa levels were obtained with the DS-/AT- assay (both $p<0.001$). Anti-Xa levels obtained with DS-/AT+ and DS+/AT- gave comparable results, which were significantly lower compared to DS+/AT+ and significantly higher compared to DS-/AT- (Figure). Both AT+ assays (DS+/AT+ and DS-/AT+) yielded consistently higher anti-Xa levels regardless of the endogenous AT activity. The absolute differences in the measured anti-Xa levels between the assays were up to 0.32 IU/mL, which could influence clinical decisions in approximately 14% of samples.

Conclusions:

Both DS and exogenous AT significantly increased the measured anti-Xa levels in patients treated with LMWH. This is in contrast to the in vitro results, which showed a significant effect only for exogenous AT. This discrepancy could be due to a higher concentration of heparin-binding proteins in the patient samples, making the effect of DS more pronounced. In addition, only a small proportion of our patients had reduced endogenous AT activity (24%), which may have attenuated the effect of exogenous AT. Further research is needed to fully elucidate the effects of DS, AT and other assay parameters and to determine their influence on treatment decisions, especially in patients with AT deficiency.

Figure: Anti-Xa levels measured with four different assay combinations regarding the presence of DS and exogenous AT. Medians and ranges (min to max) are shown. NS, non-significant.


p < 0.0001
p < 0.0001
p < 0.0001
NS
1.5
1.0
0.5
0.0
Anti-Xa (IU/mL)
0.50
0.47
0.47
0.44
DS+/AT+
DS-/AT+
DS+/AT-
DS-/AT-

PO10

Agreement between classical and two automated platelet aggregometers in the assessment of response to antiplatelet therapy

Tamara Rojnik; Mojca Božic Mijovski
University Medical Centre Ljubljana

Background:

Classical optical aggregometry is still considered the gold standard for assessment of platelet function, despite being poorly standardized and cumbersome. Recently, automated systems became available that allow for broader routine application. The aim of this study was to compare platelet aggregation measurements obtained by two automated analyzers and a classical optical aggregometry.

Material and methods:

49 participants were included in the study: 32 on antiplatelet therapy (24 on aspirin monotherapy, 1 on clopidogrel monotherapy and 7 on both aspirin and clopidogrel) and 17 control subjects without any antiplatelet therapy. None of the participants had any hematological disorders. From each participant blood was drawn in vacuum tubes containing sodium citrate. Platelet-rich plasma (PRP) was prepared by centrifugation for 10 minutes at 167 × g and platelet-poor plasma (PPP) with additional centrifugation for 15 minutes at 2,640 × g. Platelet count in PRP was not adjusted. All measurements were performed within 2–4 hours after blood collection. Platelet aggregation was determined by classical optical aggregometry using the Chrono Log Model 700 (Chrono Log, USA; CH) with the arachidonic acid (AA) and adenosine diphosphate (ADP) (Bio/Data Corporation, USA) and two automated analyzers: Thrombomate XRA (Behnk Elektronik, Germany; TR) and CS-2500 (Sysmex, Japan; CS) with AA and ADP (Behnk Elektronik, Germany, for both analyzers). For all three analyzers 1 µM AA and 10 µM ADP were used and maximal aggregation (MA) in % was recorded. The lower reference value was 62 % for AA on all three analyzers, while for ADP it was 30 % on the CH, and 47 % on both TR and CS as validated in the local laboratory.

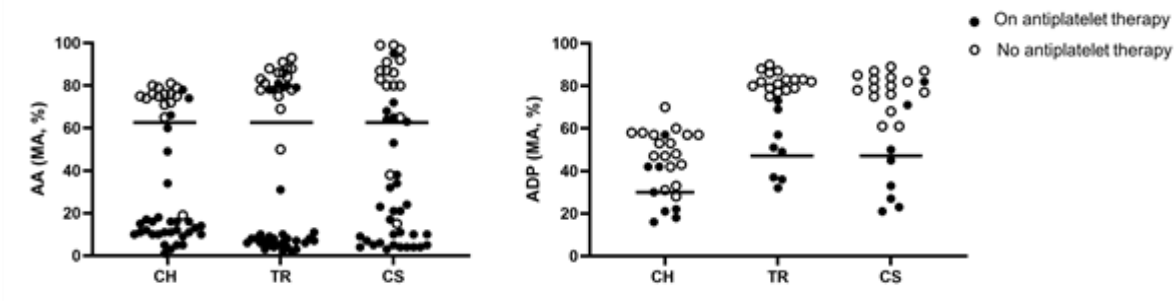
Results:

The results of all measurements are shown in Figure. The following kappa coefficients were obtained of AA-induced platelet aggregation: 0.78 (0.60 – 0.96, 95%CI) between CH and TR, 0.73 (0.53 – 0.93) between CH and CS and 0.66 (0.45 – 0.88) between TR and CS. For ADP the obtained kappa coefficients were 0.34 (0.00 – 0.83) between CH and TR, 0.69 (0.36 – 1.00) between CH and CS and 0.49 (0.07 – 0.91) between TR and CS.

Conclusion:

Our study showed good agreement between all three analysers for AA-induced platelet aggregation and moderate agreement between all three analysers for ADP-induced aggregation. Better results could probably be obtained on a larger group of patients and with strictly established reference values for agonist/analyzer combination. Nevertheless, the automated platelet aggregometry seems to be a promising tool for a more widely use in routine practice.

Figure: Platelet aggregation induced by arachidonic acid (AA) left, and adenosine diphosphate (ADP) right. Maximal platelet aggregation (MA) in % is shown. CH – Chrono-Log Model 700, TR – Thrombomate XRA, CS – CS-2500.



PO12

Challenges on the detection of lupus anticoagulant:experience of our laboratory

Maatamri Wided; Yassine Gaiech; Rahma Belhadj; Wided Maatamri; Ahmed Amine Sabbagh; Nejia Braham
Farhat Hached Hospital

Background:
The confirmation of lupus anticoagulant (LAC) remains a diagnostic challenge due to the variability of clinical indications and multiple interferences with testing, particularly anticoagulant therapy.

Material and methods:
We conducted a retrospective study in the Department of Biological Hematology, reviewing all requests for LAC testing between January 1, 2024, and September 10, 2025. The testing protocol included two sensitive assays:
? Screening and confirmatory test using the Dilute Russell's Viper Venom Time (DRVVT) were performed simultaneously. Tests were considered positive if Normalized ratio was over 1.2.
? Screening tests using Silica Clotting Time (SCT-s) and DRVVTs were performed. In cases of positivity (SCT-s ratio>1,16 and or DRVVT-s ratio >1,2), a confirmatory test and mixing test with low phospholipids concentration were performed simultaneously. Tests were considered positive if the Normalized ratio (NR) was over 1.16 for SCT and/or over 1,2 for the DRVVT and the mixing test was positive.

Results:
A total of 237 requests were collected, of which 56 (27.4%) patients tested positive. Among these, 32 patients (57%) were hospitalized, suggesting testing during acute illness, while 24 (42.8%) were outpatients. Six patients (including four outpatients) presented with an INR over 1.5, raising the possibility of ongoing antivitamin K therapy. Fifteen patients (26.8%) were treated with low-molecular-weight heparin and one with unfractionated heparin.
Clinical indications were poorly documented in 14 (25%) cases; other reasons included deep vein thrombosis (23.2%), systemic lupus erythematosus (10.7%), and recurrent miscarriages (5.4%).
Positivity rates varied by testing protocol without a significant difference (p>0,05): DRVVT alone identified 39 of 153 patients as positive (25.5%), while combined DRVVT and SCT identified 18 of 83 (21.7%).
Comparison of SCT-s/SCT-c results with mixing assays (patient plasma mixed 50/50 with control plasma) revealed inconsistencies. While the mix test confirmed results in most cases (n=5, 71.4%), it produced one probable false positive linked to anticoagulant treatment and one probable false negative, likely due to antibody dilution by control plasma. Notably, none of the positive patients underwent a second confirmatory test.
Notably, none of the positive patients underwent a second confirmatory test.

Conclusions:
These findings underscore the challenges in detecting and confirming lupus anticoagulant antibodies. Systematic clinical documentation, careful interpretation in the context of therapy, and repeat confirmatory testing are essential to ensure accurate diagnosis and management of antiphospholipid syndrome.

PO13

Early bioprosthetic valve thrombosis prompted by endocrine disease

Catarina Gonçalves Coelho; Nuno Cotrim; Beatriz Andrade; Sofia Lázaro Mendes; Kevin Domingues; Vítor Paulo Martins
HOSPITAL DISTRITAL DE SANTARÉM, E.P.E.

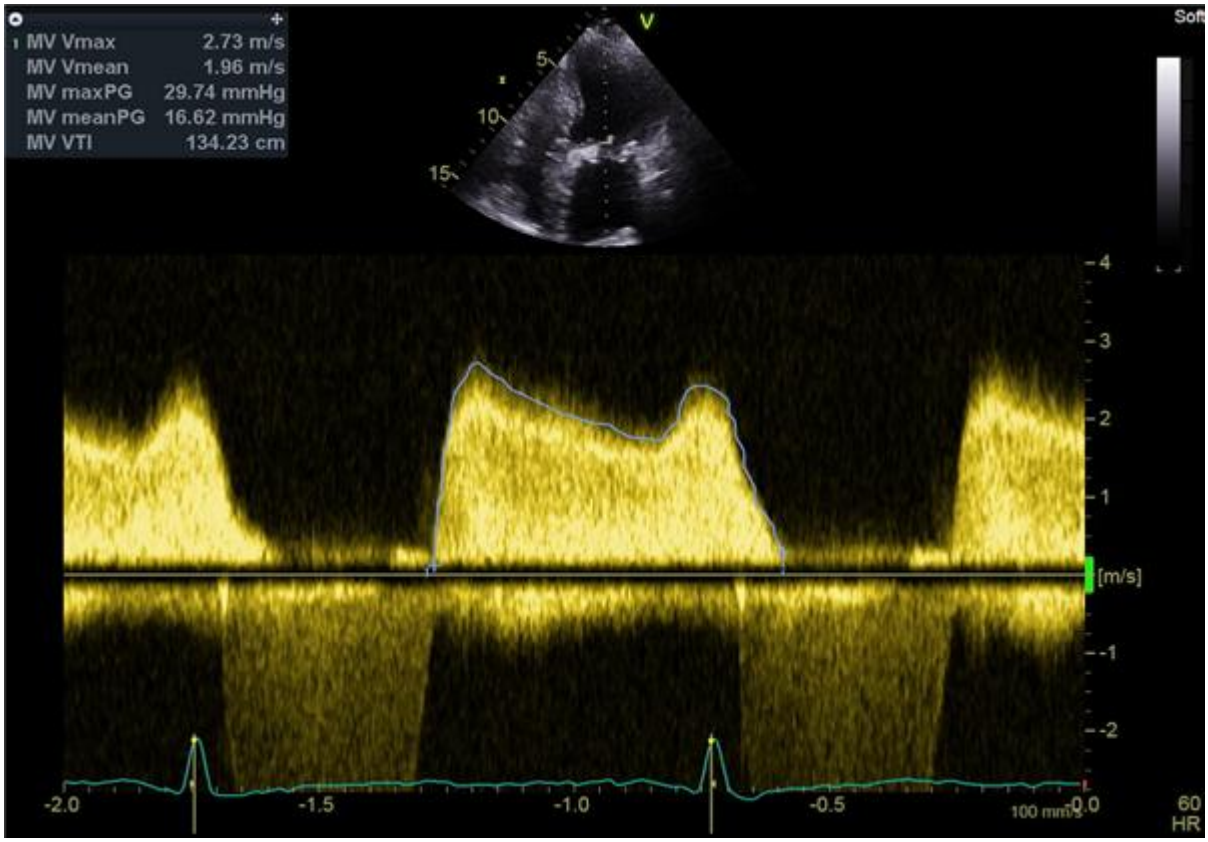
Clinical Case

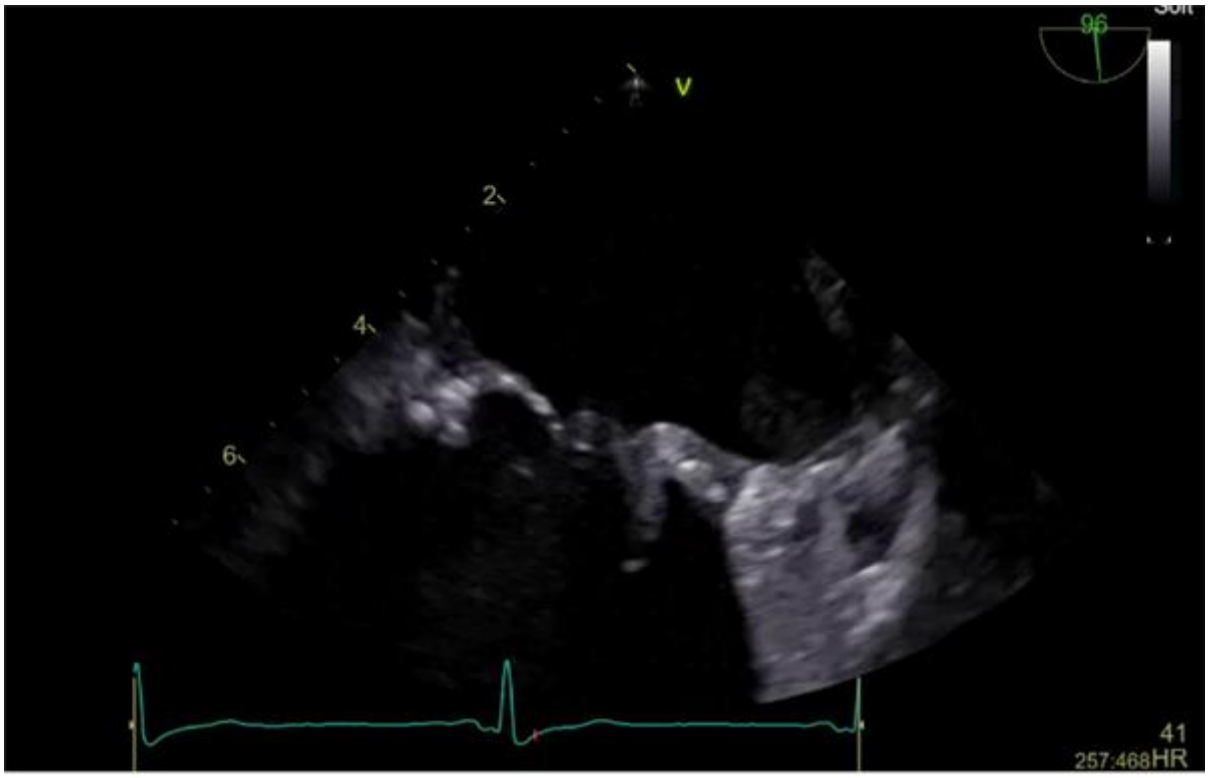
Introduction:
Biological valvular prosthesis are a great improvement for the treatment of heart valve disease, compared to the mechanical options that were first made available. The main disadvantage to the patients comes from faster structural deterioration, resulting in the need for further valvular treatment, either percutaneous or surgical, and their respective possible complications.

Case:
This is the case of an 81 years-old female patient, who received a bioprosthesis for symptomatic severe mitral regurgitation due to rheumatic valve disease. Past medical history included arterial hypertension, nephrectomy due to Grawitz tumour and chronic gastritis. She was medicated with vitamin K antagonist for three months after surgery. A follow-up echocardiogram six months later showed increased trans-prosthetic gradients. The patient presented mild symptoms, mostly exertional fatigue. A cardiac computerized tomography confirmed the hypothesis of valve thrombosis. She was treated with anticoagulation, with initial mediocre results given the difficulty in therapeutic compliance. There was never an evident need for prompt thrombolytic treatment.
After several trials of anticoagulation, the thrombosis cleared, but the valve still presented with pannus. Before proper valvular treatment, we needed to identify the specific risk factors for such a precocious finding. In the accompanying investigation, the patient was diagnosed with **primary hyperparathyroidism**.

Discussion:
Primary hyperparathyroidism can indirectly contribute to accelerated degeneration, through a process known as **dystrophic calcification**. This can then lead to clinically relevant thrombosis, due to turbulent flow and a vicious cycle of limitation of movement further contributing to mechanical leaflet stress and degeneration. Also, the increased calcium in circulation facilitates valvular calcification and thrombus formation, highlighting the need for maintaining anticoagulation until normal calcium levels are re-established.

Conclusion:
Early bioprosthetic valve thrombosis can develop in some patients, despite being an infrequent finding. It is more common in prosthesis in mitral position.
As early stage, subclinical thrombosis contributes to accelerated valve degeneration, anticoagulation in the first months after valve implantation is essential.
Multi-modality imaging is very helpful to understand the mechanisms of valve dysfunction.
When there is thrombosis, the duration of anticoagulation is not well established, and must be decided on an individual basis.


MV Vmax 2.73 m/s
MV Vmean 1.96 m/s
MV maxPG 29.74 mmHg
MV meanPG 16.62 mmHg
MV VTI 134.23 cm
V
5
10
15
Soft
4
3
2
1
[m/s]
-1
-2
-2.0
-1.5
-1.0
-0.5
0.0
100 mm/s
60
HR


96
V
2
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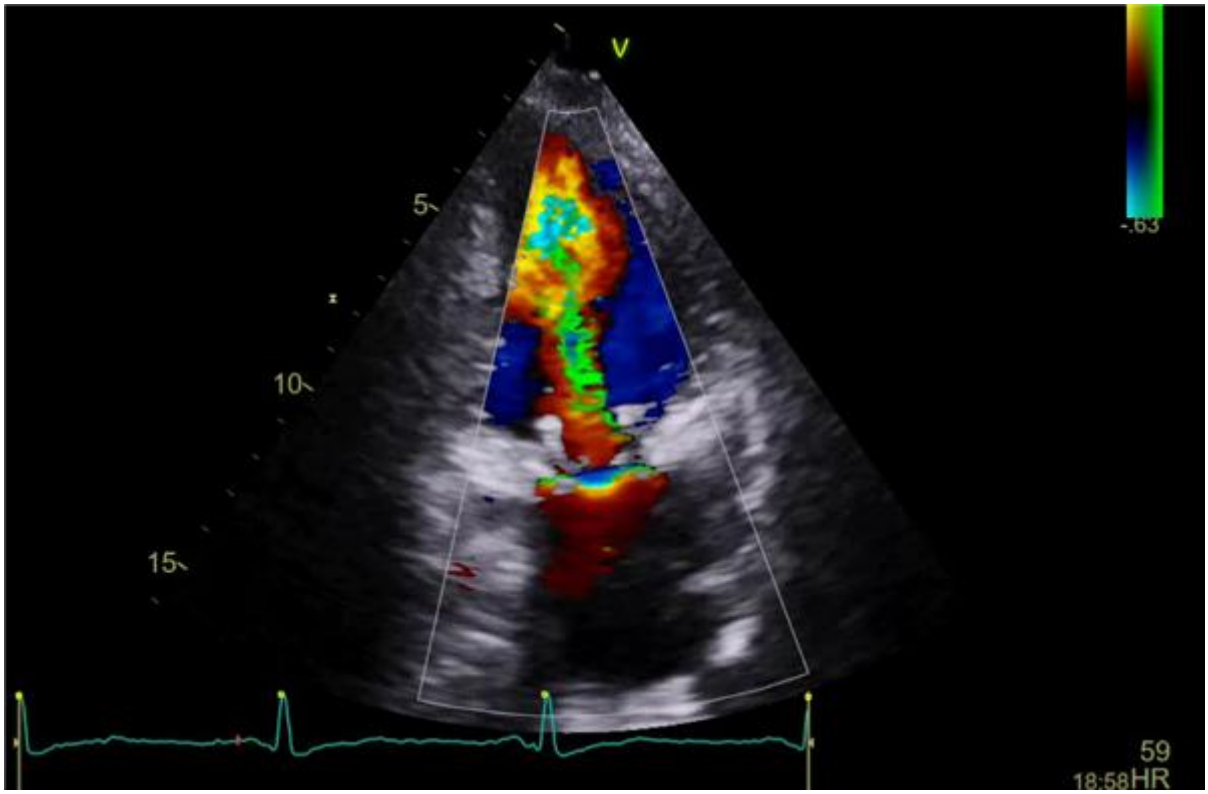
257:468HR


V
5
10
15
-.63
59
18:58HR

PO14

Aortic dissection - related spinal cord infarction with concomitant pulmonary embolism: a diagnostic and therapeutic dilema

Tiago Serrano Constantino; Miguel João Pinheiro; Inês Veiga Dias; António Epifânio Mesquita; João Fonseca Oliveira; Augusto Ribeirinho; Vitor Brotas
Unidade Local de Saúde de São José

Background:

Spinal cord infarction secondary to aortic dissection is a rare but clinically significant entity associated with diagnostic complexity[1,2]. The concomitant occurrence of pulmonary embolism and aortic dissection, particularly when the descending aorta is affected, is exceedingly rare, and its pathophysiology remains poorly understood[3]. Patients with acute aortic dissection complicated by spinal cord infarction may develop a hypercoagulable state which, combined with immobilization due to neurological deficits, increases venous thromboembolism risk[1,3]. Prompt recognition of aortic dissection with spinal cord infarction and vascular complications is crucial, as delayed diagnosis can cause irreversible neurological injury and multisystem failure[2].

Material and methods:

A 92-year-old woman with a history of epilepsy, without recent seizures or cognitive decline, presented with sudden-onset paraplegia, complete sensory loss below T7, and urinary retention. Three days earlier, she had been hospitalized for progressive weakness, during which atrial fibrillation with rapid ventricular response was diagnosed and anticoagulation initiated. On admission, cardiovascular, respiratory, and systemic examinations revealed no abnormalities. Spinal magnetic resonance imaging (MRI) and computed tomographic angiography (CTA) of the thoracic, abdominal, and pelvic regions were performed to evaluate vascular causes, along with routine laboratory tests and electrocardiography. Supportive management was provided throughout hospitalization.

Results:

Initial spinal MRI revealed findings consistent with spinal cord infarction extending from T3 to T9, characterized by high T2 and STIR signal intensity and restricted diffusion on DWI sequences. Subsequent CTA confirmed a Stanford type B aortic dissection originating at the distal aortic arch and extending through the descending aorta. CTA also demonstrated a saddle thrombus within the main pulmonary artery trunk and both pulmonary branches, along with hepatic vein opacification indicative of right ventricular strain. A subtle perfusion defect in the distal aortic arch and descending aorta suggested left renal infarction. Despite parenteral anticoagulation and close hemodynamic monitoring, the dissection progressed, leading to hypotension, ischemia of the upper left limb, and ultimately cardiac arrest.

Conclusions:

This case underscores the diagnostic and therapeutic challenges posed by the coexistence of aortic dissection and pulmonary embolism in elderly patients with comorbidities. Although uncommon, spinal cord infarction secondary to aortic dissection should be considered in acute paraplegia. The proximity of the descending aorta to the pulmonary vasculature may contribute to thrombus formation and embolic events. Clinicians must balance anticoagulation against hemorrhagic risk. Early CTA and MRI are essential for timely diagnosis and management. Greater awareness of these rare but life-threatening presentations is crucial to improving outcomes.

References:

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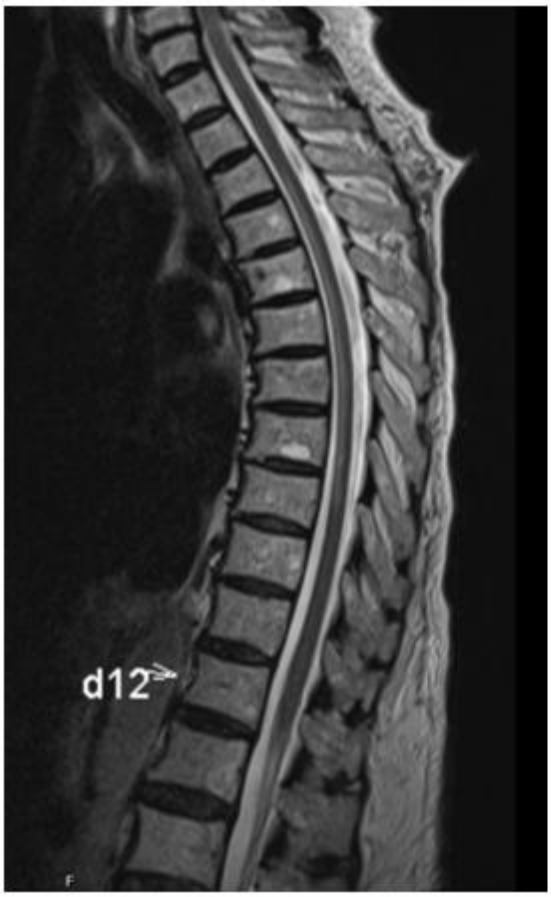

Figure 2. Sagittal T2-weighted spinal MRI showing hyperintense signal from T3 to T9, consistent with spinal cord infarction.

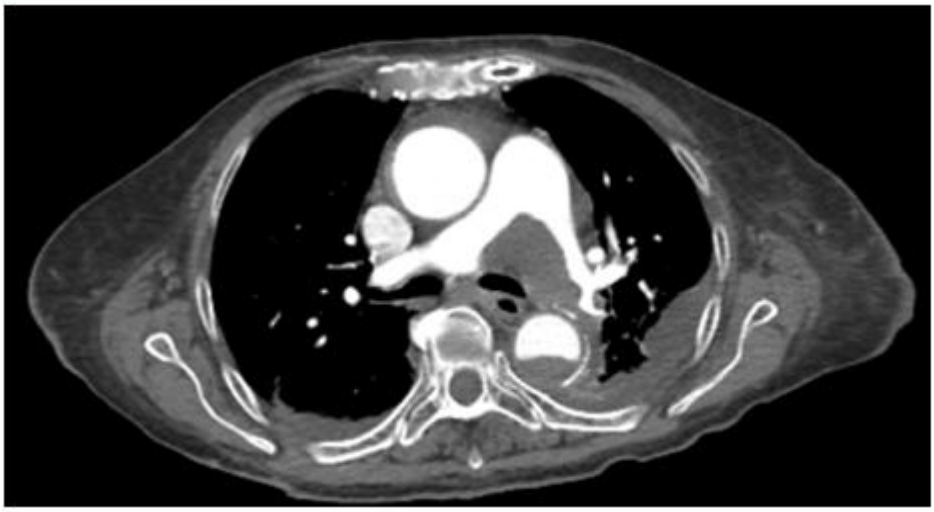

Figure 3. CTA image showing a saddle thrombus within the main pulmonary artery trunk extending into both right and left pulmonary arteries.

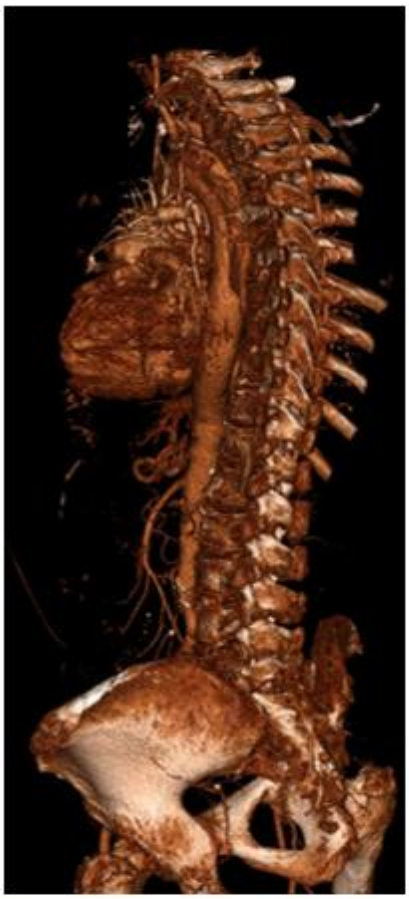

Figure 1. Volume-rendered CTA image demonstrating a Stanford type B aortic dissection originating at the distal aortic arch and extending through the descending aorta.

PO15

Bilateral thalamic and midbrain infarction due to artery of percheron occlusion: a case report

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Unidade Local de Saúde de São José

Background:

The artery of Percheron (AOP) is a rare anatomical variant arising from a single perforating branch of the proximal posterior cerebral artery that supplies both paramedian thalami and the rostral midbrain[1,2]. Occlusion of this vessel results in bilateral thalamic infarction, an uncommon subtype of ischemic stroke accounting for approximately 0.1% of all ischemic strokes and about 4% of thalamic infarctions[1]. Because the thalamus plays a crucial role in regulating consciousness, cognition, and ocular motor control, AOP infarction typically presents with altered mental status, varying degrees of cognitive dysfunction, and supranuclear vertical gaze palsy[3]. Early diagnosis is often challenging due to nonspecific clinical presentation and the low sensitivity of early non-contrast computed tomography (CT) imaging[3]. Recognition of this rare vascular variant is essential for accurate diagnosis and appropriate management[2,3].

Material and methods:

A 93-year-old Portuguese woman, partially dependent due to advanced osteoarthritis, was found unresponsive about eight hours after last being seen well. On admission, she was comatose with minimally reactive pupils, preserved corneal reflexes, reduced tone, and limited withdrawal to pain. Initial non-contrast CT showed chronic microvascular changes and an old right lacunar infarct without acute findings. Forty-eight hours later, computed tomography angiography (CTA) and brain magnetic resonance imaging (MRI) were performed.. During hospitalization, echocardiography, Holter monitoring, and carotid ultrasound were conducted to assess ischemic evolution and potential embolic sources.

Results:

During hospitalization, the patient remained comatose, exhibiting only minimal motor responses to painful stimuli. Repeat CT at 48 hours revealed bilateral thalamic hypodensities, more pronounced on the left, extending toward the mesencephalon. CTA confirmed occlusion of the artery of Percheron (Figure 1). DWI and FLAIR sequences demonstrated acute ischemic lesions in the bilateral paramedian thalami with extension to the ventral midbrain (Figure 2), as well as additional small infarcts in the right cerebellum and right mesial temporal region.

Echocardiography, Holter monitoring, and carotid ultrasonography did not reveal structural, embolic, or hemodynamic abnormalities to explain the etiology.

Conclusions:

Artery of Percheron occlusion is a rare cause of bilateral thalamic infarction, often presenting with nonspecific neurological findings that delay diagnosis. This case highlights the importance of suspecting AOP infarction in cases of unexplained coma or altered consciousness. Early MRI with DWI/FLAIR and CTA are essential for accurate diagnosis and management. Greater clinician awareness may prevent diagnostic delays and improve outcomes.

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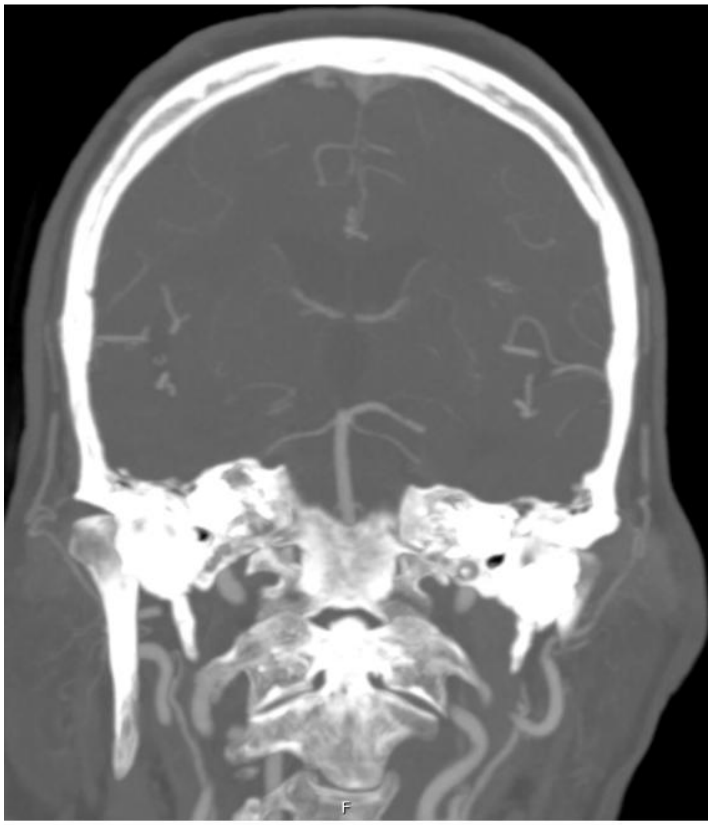

Figure 2. Coronal maximum intensity projection (MIP) from computed tomography angiography (CTA) showing absence of opacification of the right posterior cerebral artery at its proximal segment, consistent with occlusion involving the origin of the artery of Percheron.

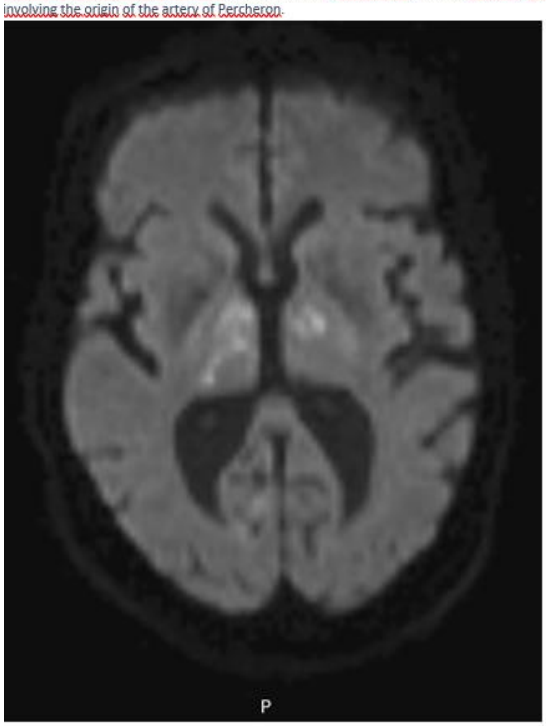

Figure 1. Axial diffusion-weighted MRI (DWI) showing areas of restricted diffusion consistent with acute ischemic lesions in the bilateral paramedian thalami.

PO22

Listening to the heart: Frank's sign as a non-invasive indicator of coronary artery disease

Beatriz Vargas Andrade[1]; Nuno Cotrim[1]; Catarina Coelho[1]; Rita Veiga[2]; Vítor Martins[1]
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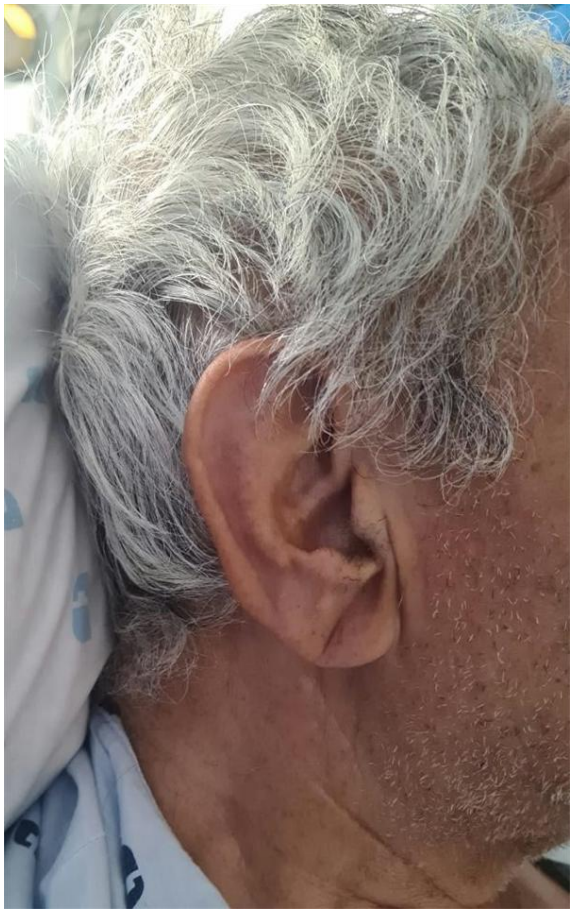
[2] Hospital Particular do Algarve

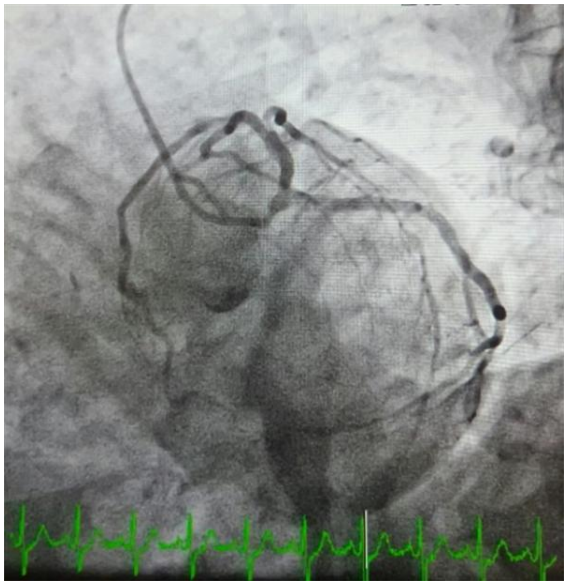
Background:
Cardiovascular diseases continue to be the leading cause of death worldwide. Effective prevention relies on accurate cardiovascular risk assessment through validated scoring systems. Nonetheless, the identification of physical signs indicative of atherosclerosis remains a valuable clinical approach for early detection and risk stratification. Frank's sign, also known as the diagonal earlobe crease (DELC), was first described in 1973 by Sanders T. Frank as a potential dermatological predictor of coronary artery disease (CAD). Subsequent studies have consistently demonstrated that this crease is independently associated with both the presence and severity of atherosclerosis, showing diagnostic sensitivities exceeding 75% and specificities greater than 85%.

Case presentation:
A 71-year-old man presented to the Emergency Department with exertional precordial pain. His past medical history included hypertension and type 2 diabetes mellitus. On examination, bilateral Frank's sign was evident (Figure 1). The ECG demonstrated ST-segment depression and T-wave inversion from leads V4 to V6. High-sensitivity cardiac troponin T peaked at 38 ng/L. Coronary angiography revealed severe coronary artery disease with 90% ostial stenosis of the left main coronary artery and 90% ostial stenosis of the circumflex artery (Figure 2). The patient was referred for coronary artery bypass grafting.

Discussion:
This case highlights the potential of DELC as a simple, non-invasive clinical marker indicative of underlying atherosclerotic disease. Although its pathophysiological basis is not fully understood, oxidative stress and microvascular dysfunction have been proposed as shared pathways linking dermal changes to vascular aging and atherogenesis. Numerous observational studies have validated the association between Frank's sign and angiographically confirmed CAD, independent of conventional cardiovascular risk factors. Additionally, DELC has been correlated with increased carotid intima-media thickness, reinforcing its role as a marker of systemic atherosclerosis. Given that these processes also overlap with age-related vascular remodeling, some evidence suggests that the diagnostic relevance of DELC may be greater in men under 60 years, particularly when the crease is complete, bilateral, deep and accompanied by accessory lines.

Conclusion:
The presence of DELC is an easily observable, non-invasive bedside marker associated with increased risk of CAD, making it a valuable tool to prompt further cardiovascular assessment in at-risk individuals.





PO23

Unmasking radiation-induced cardiac injury: acute coronary syndrome during cancer treatment

Beatriz Vargas Andrade
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Backround:

Radiotherapy (RT) is known to increase the risk of ischemic heart disease (IHD) due to incidental exposure of cardiac structures to ionizing radiation. Thoracic RT can damage multiple cardiac components such as the myocardium, pericardium, valves, coronary arteries, and conduction system. Despite technological advances that have reduced doses to normal tissues, significant heart radiation exposure remains unavoidable, particularly in women treated for left-sided breast cancer who receive substantially higher heart doses than those with right-sided tumors. While multiple studies have demonstrated an elevated risk of IHD after RT in left-sided breast cancer, the occurrence of acute coronary syndrome (ACS) during the course of radiotherapy is uncommon.

Case description:

We present a 51-year-old female patient with type 2 diabetes, dyslipidemia, and asthma, who was diagnosed with HER2-positive left breast cancer. She received neoadjuvant chemotherapy (doxorubicin, cyclophosphamide, paclitaxel) followed by mastectomy and axillary lymph node dissection. Subsequent adjuvant therapy included trastuzumab and radiotherapy to the left chest wall and axilla (50 Gy), with an additional boost to the surgical scar (total 60 Gy). During radiotherapy, she experienced recurrent episodes of oppressive precordial chest pain at rest without additional symptoms. Initial diagnosis considered radiation-induced pericarditis or costochondritis, and treatment with non-steroidal anti-inflammatory drugs provided only partial relief. One month later, echocardiogram showed preserved systolic function but new hypokinesia of the basal inferior wall segment. Electrocardiogram revealed new T-wave inversion in aVL lead. Cardiac troponin levels were negative. Further cardiologic evaluation with cardiac magnetic resonance confirmed localized hypokinesia and minimal late subendocardial enhancement with perfusion defect, while coronary computed tomography identified a 70-99% stenosis in the distal right coronary artery. Medical therapy with bisoprolol, aspirin, atorvastatin, and amlodipine was initiated with symptomatic control.

Discussion:

This case exemplifies an uncommon presentation of IHD induced by thoracic radiotherapy, manifesting as ACS during treatment. Radiation promotes inflammatory and fibrotic changes characterized by increased myofibroblast and macrophage activity leading to intimal proliferation and a pro-thrombotic vascular milieu. Although RT-related cardiac damage is typically clinically silent for years, this case shows ACS occurrence can happen during RT. Early recognition, baseline cardio-oncology risk assessment, and careful monitoring during and after radiotherapy are essential to optimize cardiovascular outcomes without compromising oncological treatment goals.

Conclusion:

This case reinforces the importance of multidisciplinary collaboration to manage and prevent radiation-associated cardiac complications effectively.

PO24

Neoplasia, obesity and SARS-CoV infection: risk factors for pulmonary thromboembolism

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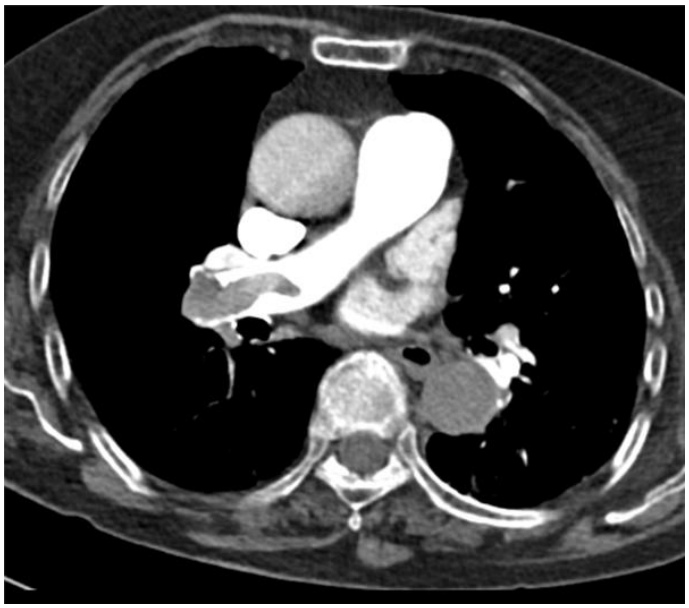
Background:

Neoplasia, obesity, and SARS-CoV infection are risk factors for pulmonary thromboembolism. Mortality is increased in underdiagnosed cases of pulmonary thromboembolism. Material and methods: Our work includes patients admitted for Pulmonary Thromboembolism from the Emergency Department through research of the discharge report. Results: The first patient was a 93-year-old obese woman presents to the Emergency Department with dyspnea on mild exertion and asthenia. Laboratory tests: creatinine 1.3 mg/dl, D-Dimers >20000 ng/ml, NTproBNP 11220 pg/ml, Troponin T 0.138 ng/ml, electrocardiogram sinus rhythm, chest angiotomography showing extensive filling defect in the right and left main pulmonary arteries, characterizing a saddle thrombus (cavalryman-like), and other thrombi in the proximal lobar and segmental arterial branches of the middle lobe, lingula, and lower lobes, compatible with bilateral pulmonary thromboembolism. The Pulmonary Embolism Severity Index (PESI) was 173 points, class V, intermediate-high risk. Echocardiogram: mild left ventricular systolic dysfunction, left ventricular diastolic dysfunction. She was medicated with enoxaparin 1 mg/kg 12/12 H for 6 days. She was discharged with edoxaban 30 mg/day. The second patient was a 85-year-old woman, obese, with hypocoagulated atrial fibrillation and apixaban 2.5 mg 1 tablet/day, presented to the Emergency Department due to vomiting and headache. Laboratory tests: creatinine 1.45 mg/dl, D-Dimers >800 ng/ml, NTproBNP 13479 pg/ml, Troponin T 0.067 ng/ml, subtherapeutic apixaban levels, electrocardiogram showing atrial fibrillation and rapid ventricular response. Chest angiotomography showed altered perfusion of the subsegmental branches of the right inferior lobe artery, compatible with subsegmental pulmonary thromboembolism. Brain tomography showed an expansile lytic lesion in the frontoparietal cranial vault, with extracranial and intracranial extension, suggesting a malignant neoplastic lesion, causing slight molding of the adjacent brain parenchyma. Echocardiogram: normal left ventricular systolic function. She was medicated with enoxaparin 1 mg/kg q12h for 6 days. She was discharged with apixaban 5 mg twice daily. The third patient was a 68-year-old man underwent left nephrectomy due to renal cell carcinoma. Postoperatively, he developed low-risk pulmonary embolism and was treated with anticoagulation therapy with 4 mg of acenocoumarol. Three months later, he was hospitalized with COVID-19 pneumonia and switched to enoxaparin 1 mg/kg twice daily for 6 days. He was discharged with apixaban 5 mg twice daily. Conclusions: In these cases, the prognosis was favorable due to early diagnosis of risk factors for pulmonary thromboembolism: neoplasia, obesity and SARS-CoV infection. The choice of anticoagulation therapy should be individualized according to the patient's characteristics.



PO26

Sickling of red blood cells contributes to the enhanced venous thrombosis in sickle cell trait mice

Rafal Pawlinski; Karnsasin Seanoon; Izabela Pawlinski; Patrick Ellsworth; Fatima Trebak; Chatphatai Moonla; Nigel S. Key
UNC Chapel Hill

Background:
Individuals with sickle cell trait (SCT) are heterozygous carriers of the sickle beta-globin gene. Analyses of large cohort studies showed that SCT is a risk factor for venous thrombosis (VT). We previously reported that enhanced experimental VT observed in humanized SCT mice was attenuated by the treatment with Gardos channel inhibitor, Senicapoc (SEN).

Aims:
Determine if sickling of SCT red blood cells (RBCs), triggered by hypoxia and/or dehydration, contributes to VT and if SEN reverse these changes.

Methods:
Ex vivo clot contraction assay was performed on whole blood (WB) from control mice expressing two copies of normal human hemoglobin beta globin (AA) or heterozygous for mutated sickle hemoglobin beta globin (AS) under hypoxia (pO2 = 20 mmHg) or after exposure to 7.5 μM ionophore (ION) in the presence or absence of SEN (200 ng/mL). RBC deformability, as a surrogate of sickling, was measured by the Laser Optical Rotational Red Cell Analyzer (LORRCA).

Results:
During clot contraction, excess RBCs are extruded from the thrombus. To evaluate whether SEN attenuates enhanced VT in AS mice via reducing retention of sickled RBC in the clots, we measured the number of RBCs extruded from the WB clots. To mimic venous oxygen concentration, WB from AA (n=11) and AS (n=12) mice was preincubated in hypoxia for 4 hours, clotting was then initiated, and clot weight and serum RBC content were assessed 1 hour later. Under hypoxia conditions, the number of AS RBC (1.0±0.4x10^6/μL) extruded from clot was reduced compared with the number of AA RBC (1.6±0.35x10^6/μL; $p < 0.001$). Consequently, the weight of AS clots (100±9.3 mg) was significantly increased compared to AA clots (85±8.2 mg; $p < 0.001$). Importantly, compared to vehicle treated WB, SEN pretreatment during hypoxia significantly increased the extrusion of AS RBC during clot contraction (0.92±0.29 vs 0.68±0.23x10^6/μL; $p < 0.001$; n=15).
Stimulation with ION also attenuated RBC extrusion (1.0±0.1 vs 2.3±0.5x10^6/μL) and increased weight (107±7 vs 78±9 mg) of AS clots compared to AA controls (n=3; $p<0.05$ for both parameters). Next, AS WB was incubated with vehicle (n=8) or SEN (n=8) prior to incubation with ION. Compared to vehicle treated blood, SEN promoted the extrusion of AS RBC from these clots (1.1±0.4 vs 1.7±0.5x10^6/μL; $p < 0.001$).
Lastly, we used LORCA to evaluate whether the effect of ION on RBC extrusion was mediated by changes in RBC deformability (sickling). Blood from AS mice (n=5) was incubated with ION in the presence or absence of SEN. Compared to unstimulated blood, ION led to dehydration (shown by reduction of maximum elongation index Elmax; 0.495±0.01 vs 0.212±0.05; $p<0.05$) and sickling of AS RBCs, identified by a clear point of sickling (PoS; 76±4.8 mmHg). Pre-treatment with SEN decreased the PoS (26±5.4 mmHg; $p < 0.01$) and increased the Elmax (0.445±0.025; $p < 0.0001$) demonstrating that SEN partially prevented ionophore-induced AS RBC dehydration and rendered them less prone to sickling.

Conclusions:
These findings demonstrate that Gardos channel inhibition partially reverses the hypoxia- and dehydration-induced retention of RBCs within AS clots.

PO27

Relationship of JAK2 mutation burden with thrombosis and survival in polycythemia vera

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[3] FUNDACIÓN BURGOS POR LA INVESTIGACIÓN DE LA SALUD

Abstract

Background:

Patients with polycythaemia vera (PV) have an increased risk of arterial and venous thrombosis. The European VETS (VEnous Thrombosis Score) prognostic system includes previous thrombosis and high JAK2V617F mutation burden (VAF (Variant Allele Fraction) ≥ 50%) as risk factors for venous thrombosis. Ninety-five per cent of patients diagnosed with PV have a somatic mutation of the JAK2 gene in exon 14, which causes a change in aminoacid 617 and produces a change from valine (V) to phenylalanine (F) (V617F). This mutation activates the JAK-STAT pathway, causing the proliferation of haematopoietic cells, increasing the risk of thrombotic complications and promoting progression to myelofibrosis or leukaemia. Patients with higher JAK2 V617F VAF (VAF ≥50%) have a more serious risk of developing thrombosis and lower survival rates. The aim of the study was to analyse the relationship between the VAF load of the JAK2V617F mutation and thrombotic complications, as well as to analyse the relationship between the VAF load of the JAK2V617F mutation and survival in patients with polycythaemia vera diagnosed at our centre between January 2000 and June 2025.

Material and methods:

Retrospective study of patients with PV at the University Hospital of Burgos, Spain. Epidemiological data, the percentage of VAF at diagnosis, pre- and post-diagnosis thrombotic complications, and survival were collected. Statistical analysis was performed using SPSS statistical analysis software.

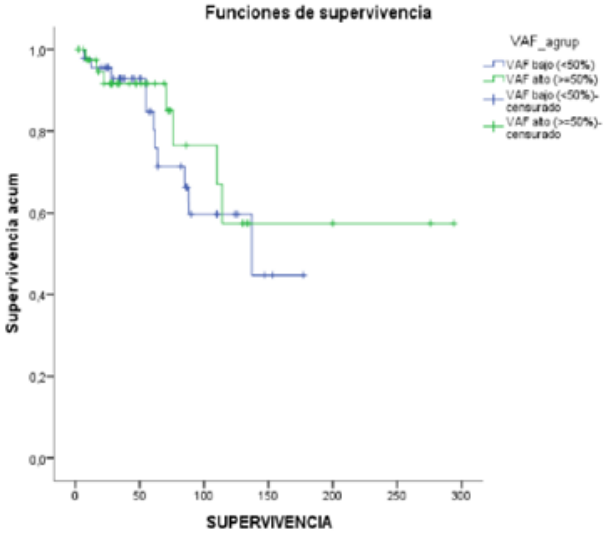
Results:

Eighty-seven patients were analysed, 37 women (42.53%) and 50 men (57.47%), with a mean age of 69.15 years (SD ±15.09). Of these patients, 45 (51.72%) had a VAF < 50%, while 42 (48.28%) had a VAF ≥ 50%. Twenty-one patients (24.14%) had thrombosis prior to diagnosis: 10 (22.22%) in the VAF < 50% group and 11 (26.19%) in the VAF ≥ 50% group. Fifteen patients (17.24%) had thrombosis after diagnosis: 11 (24.44%) in the VAF < 50% group and 4 (9.52%) in the VAF ≥ 50% group. The median survival was 50 months. By June 2025, 18 patients (20.69%) had died. In the VAF <50% group, 11 (24.44%) and in the VAF ≥50% group, 7 (16.67%) (p 0.371).

Table 1 shows the characteristics of the patients. The Kaplan-Meier method was used to obtain the survival curves. Graph 1

Conclusions:

The group of patients with JAK2 V617F ≥50% did not present greater thrombotic complications than the JAK2 V617F <50% group. Survival in the JAK2 V617F ≥50% group was lower, although the difference was not statistically significant.



			VAF				p-valor
			<50%		≥50%		
AGE	*media±deviation*		67,62	16,3	70,79	13,67	0,331
SEX	*n/%*	WOMAN	16	35,56%	21	50,00%	0,173
		MEN	29	64,44%	21	50,00%	
VAF	*median (IQR) [min;max]*		31	(15;42)[4;49]	66	(55;79)[50;95]	
THROMBOSIS PRIOR	*n/%*	No	35	77,78%	31	73,81%	0,666
		Sí	10	22,22%	11	26,19%	
THROMBOSIS AFTER	*n/%*	No	34	75,56%	38	90,48%	0,066
		Sí	11	24,44%	4	9,52%	
OVERALL SURVIVAL	*median (IQR) [min;max]*		55	(32;87) [7; 177]	45	(21;75) [2; 294]	0,246
EXITUS	*n/%*	No	34	75,56%	35	83,33%	0,371
		Sí	11	24,44%	7	16,67%	

PO29

Impact of age and NYHA class on clinical outcomes of fibrinolysis versus surgery in prosthetic valve thrombosis: a systematic review and meta-regression *Ana Marta C. Pinto[1]; Bernardo Resende[2]; Emídio Mata[1]; Margarida Castro[1]; Sílvia Ribeiro[1]; João Gameiro[2]; António Lourenço[1]; Gonçalo Ferraz-Costa[2]; Lino Gonçalves[2]*

[1] ULS Alto Ave

[2] ULS Coimbra

Background:

Obstructive prosthetic valve thrombosis (PVT) is a rare, life-threateningcondition with high morbidity and mortality. Urgent surgery has been standard, but it s risks and evidence supporting low-dose, slow infusion fibrinolysis protocols have prompted reconsideration. Age and NYHA functional class may influence outcomes, as older or more symptomatic patients often carry higher surgical risk.

Objective: To perform a meta-analysis and meta-regression comparing surgery versusfibrinolysis in PVT, evaluating age and NYHA class III-IV prevalence as moderators oftreatment effect.

Methods:

CENTRAL, SCOPUS, Web of Science, EMBASE, and PubMed were searched (July20–August 15, 2025) for studies comparing fibrinolysis with surgery for PVT. Random-effects meta analyses estimated pooled risk ratios (RRs) with 95% confidence intervals. Mixed-effects meta-regression evaluated the influence of age and NYHA III/IV prevalence on in-hospitalmortality, complete valve restoration, and recurrence.

Results:

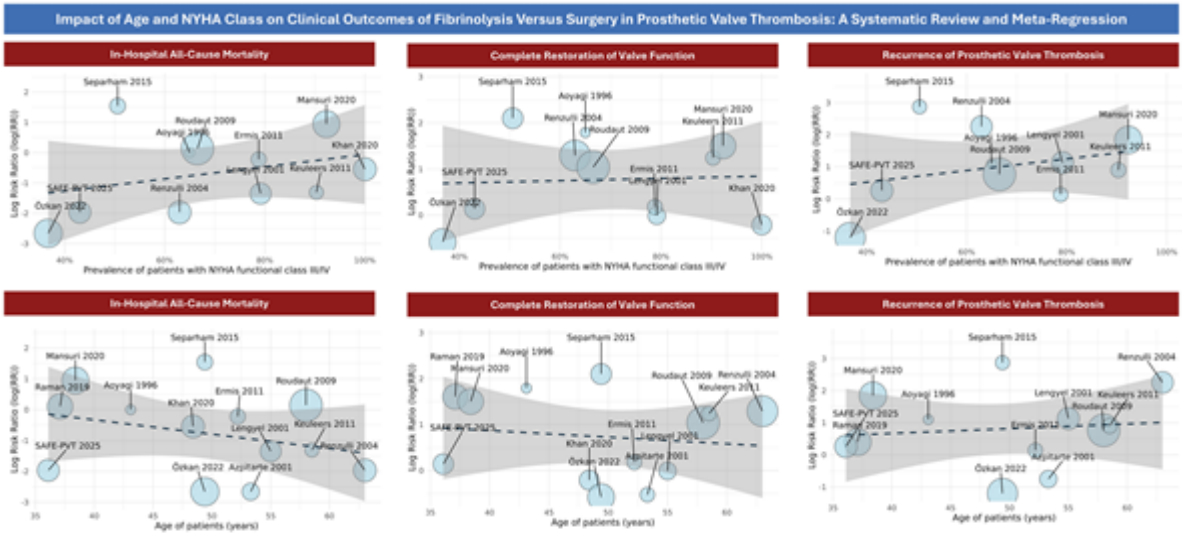
Thirteen studies (12 observational, 1 randomized control trial; 586 fibrinolysis, 714surgery) were included. Mean age ranged from the mid-30s to early 60s, with over half ofpatients in NYHA class III-IV.

Meta-regression showed NYHA class did not significantly moderate outcomes. In a hypothetical NYHA I-II cohort, baseline RRs were 0.17 [0.01–3.24] for in-hospital mortality,1.24 [0.21–7.43] for valve restoration, and 0.66 [0.04–11.80] for recurrence. Each 10% increasein NYHA III-IV prevalence modestly favored surgery by a non-significant factor of 1.08 to1.25(mortality QM(1) = 0.79, p = 0.37; valve restoration QM(1) = 0.33, p = 0.56; recurrence QM(1)= 1.08, p = 0.30).

Age similarly did not showed significant moderation. In a cohort with mean age 50, baselineRRs were 0.64 (95% CI, 0.32–1.31) for mortality, 2.02 (95% CI, 1.22–3.36) for valverestoration, and 2.46 (95% CI, 1.27–4.80) for recurrence. Each 10-year increase had negligible effect (mortality QM(1) = 0.52, p = 0.47; valve restoration QM(1) = 0.06, p = 0.80; recurrenceQM (1) = 0.58, p = 0.45).

Conclusions:

Interpretation is limited by substantial heterogeneity, high risk of bias, and potential confounding from non-random treatment allocation, reflecting possible selection bias not fully accounted for by meta-regression. Nevertheless, the lack of significant moderation by age or NYHA class may suggests that treatment outcomes may be independent of patient age or symptomatic status at presentation.



Meta-regression analysis of age and NYHA functional class on outcomes of fibrinolysis versus surgery in prosthetic valve thrombosis (PVT).
Bubble plots illustrate the association between NYHA class III–IV prevalence (top row) and mean age (bottom row) with log risk ratios (log[RR]) for in-hospital all-cause mortality, complete restoration of valve function, and recurrence of prosthetic valve thrombosis. Each bubble represents an individual study, with bubble size proportional to study weight in the random-effects model. Dashed lines indicate regression slopes, and shaded areas show 95% confidence intervals. No significant moderating effect of NYHA class or age was observed for any clinical outcome.

PO30

Influence of valve position on outcomes of surgery versus fibrinolysis in prosthetic valve thrombosis: a meta-analysis and meta-regression

Ana Marta C. Pinto[1]; Bernardo Resende[2]; Emídio Mata[1]; Margarida Castro[1]; Sílvia Ribeiro[1]; João Gameiro[2]; António Lourenço[1]; Gonçalo Ferraz-Costa[2]; Lino Gonçalves[2]

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Background:

Obstructive prosthetic valve thrombosis (PVT) is a rare but life-threateningcomplication. While urgent surgery has traditionally been the standard of care, fibrinolytictherapy has emerged as a less invasive alternative in selected patients. The influence of the affected valve position on treatment outcomes remains unclear.

Objective:

To compare surgery and fibrinolysis for prosthetic valve thrombosis using meta-analysis and meta-regression, examining valve position as a potential modifier of treatmenteffect.

Methods: A systematic search of five databases identified studies comparing fibrinolysis withsurgery for prosthetic valve thrombosis. Random-effects meta-analyses were conducted toestimate pooled risk ratios (RRs) with 95% confidence intervals, and mixed-effects meta-regression was used to evaluate the influence of valve position on in-hospital mortality,complete valve restoration (without associated death), and recurrence.

Results:

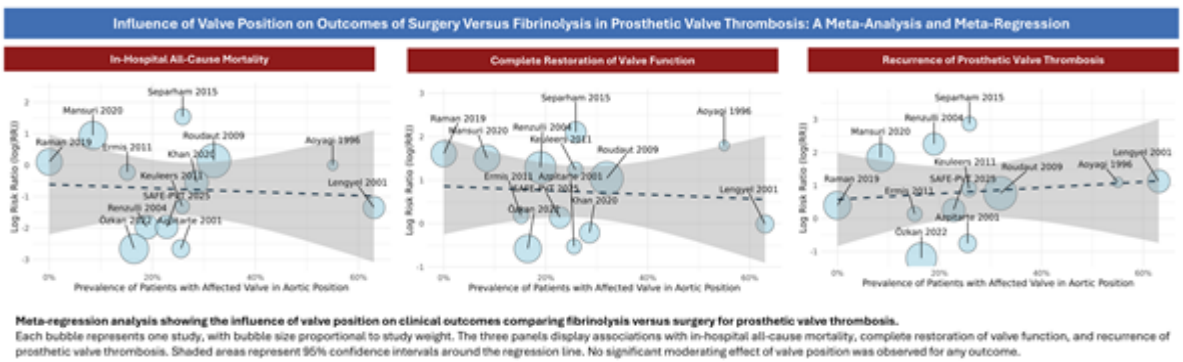
Thirteen studies (12 observational, 1 randomized control trial; 586 fibrinolysis, 714surgery) were included. The distribution of affected valves varied across studies: aortic (A) 8.5–55.0%, mitral (M) 54.8–93.7%, tricuspid (T) 1.5–20.0%, and combined valve involvement (A–M or M–T) 0.7–10.6%.

Meta-regression showed no significant moderating effect of valve position on any outcome. Forin-hospital mortality, the baseline risk ratio (RR) for a fully non-aortic cohort was 1.01 [0.32–3.17], and each 10% increase in aortic valve involvement was associated with a non-significant14% relative decrease in RR (0.86 [0.60–1.24] QM(1)=0.64, p=0.42).

For complete valve restoration, the baseline RR for a fully non-aortic cohort was 2.67 [1.10–6.48], indicating a significant advantage for surgery over fibrinolysis, while each 10% increasein aortic valve prevalence corresponded to a non-significant 10% decrease in RR (0.90 [0.66–1.21] QM(1)=0.53, p=0.47). For recurrence, the baseline RR for a fully non-aortic cohort was2.10 [0.57–7.68], with each 10% increase in aortic valve prevalence associated with a non-significant 7% relative increase in RR (1.07 [0.66–1.74] QM(1)=0.08, p=0.78).

Discussion:

Surgical treatment remained associated with higher rates of complete valverestoration, while mortality and recurrence were similar between strategies, irrespective of valvelocation. These findings suggest that treatment choice should primarily consider patient clinicalstatus and procedural risk rather than valve position alone.



Meta-regression analysis showing the influence of valve position on clinical outcomes comparing fibrinolysis versus surgery for prosthetic valve thrombosis.
Each bubble represents one study, with bubble size proportional to study weight. The three panels display associations with in-hospital all-cause mortality, complete restoration of valve function, and recurrence of prosthetic valve thrombosis. Shaded areas represent 95% confidence intervals around the regression line. No significant moderating effect of valve position was observed for any outcome.

PO31

Haemorragic stroke, a pulmonary headache

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Introduction:

Hemorrhagic stroke is a medical emergency consisting of an intraparenchymal or subarachnoid hemorrhage. Most commonly associated with high intensity headaches with sudden onset. No matter the cause, blood pressure, besides other cardiovascular risk factors control is essential.

The lack of mobility associated with the hemorrhagic stroke either through hemiparesis or the lack of patient mobilization in the infirmary increases the risk of thrombotic events. On top of that hemorrhagic stroke is an absolute contraindication for anticoagulation. There are preventive measures, namely elastic socks or pneumatic compression socks. Nevertheless even these measures can fail and we might see ourselves forced to anticoagulate an hemorrhagic stroke.

Clinical case:

previously independent fatient for daily tasks is brought to the emergency room with right brachial hemiparesis, nausea, exhaustion and progressive dysarthria. The emt refers high blood pressure with systolic's 190 mmhg.

In the emergency room the physical examination reveals conjugated eye gaze to the left, miotic pupils, right side hemiplegia and dysphagia. A ct-scan was performed showing an extensive hemorrhagic stroke. Patient was discussed with Neurosurgery. Patient was to be under surveillance and risk factor control. Since admission, patient kept a difficult to control high blood pressure, with the need for a labetalol perfusion during 5 days, after which oral medication sufficed.

On the fifth day of admission the patient started having fever, with an elevation of inflammatory parameters. Empirical antibiotics were started with amoxicillin- clavulanate acid. Urine sample microbiology isolated Morganella morganii, susceptible to amoxicillin- clavulanate acid. A 7 day regimen was administered.

On the 19th day of admission a sudden, intense dyspnea, with signs of poor peripheral perfusion and hypotension prompted the performance of a thoracic ct-scan that revealed a central pulmonary embolism with cardiac right side cavities enlargement.

Hemorrhagic Stroke is one of the absolute contraindication for anticoagulation.Because of that the patient had, since day one, elastic compression socks.

Patient was discussed with a specialized Pulmonary Embolism team and neurology, and a non-fractioned heparin perfusion for 72 hours was decided as best approach. The fibrinolytic therapy went without any mishaps. After the 72 hours, enoxaparin was initiated and no further issues.

The neurological deficits remained stable.

The patient was discharged to a rehabilitation center.

Conclusion:

Even with proper surveillance and proper care, life-threatening situations may arise that jeopardize our patients' lives and forces to make risk full, although pondered decisions in order to reestablish health. And when the time comes, quick but decisive measures must be taken in order to prevent further damage.

PO32

Characterization of patients followed in a venous thromboembolism outpatient clinic at a district hospital: a six-month experience

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Introduction:
Venous thromboembolism (VTE) is a common and potentially life-threatening condition, associated with significant short- and long-term morbidity and mortality. The establishment of dedicated VTE outpatient clinics enables a structured approach to patient management, promoting standardized follow-up, comprehensive etiological investigation, and therapeutic optimization. The aim of this study was to describe the clinical, etiological, and follow-up characteristics of patients managed in a dedicated VTE outpatient clinic.

Material and methods:
We conducted a retrospective observational study including all patients evaluated at the VTE outpatient clinic between February and July 2025. Demographic characteristics, type and circumstances of the thrombotic event, personal and family history, etiological work-up, treatment phase, number of consultations, complications, and anticoagulation-related adverse events were collected and analyzed descriptively. **Results:**
A total of 21 patients were included, with a mean age of 62 years, and 57% were male. The index thromboembolic event was pulmonary embolism (PE) in 9 cases (43%), deep vein thrombosis (DVT) in 5 (24%), superficial vein thrombosis (SVT) in 2 (10%), and other events in 2 (10%). Most events were unprovoked (n = 18; 86%), while 3 events (14%) were provoked. A positive personal history of VTE was identified in 5 patients (24%), and a positive family history in 3 patients (14%). The etiological study was negative in 20 patients (95%), with only one positive result. Patients attended an average of 2 follow-up visits, and 16 patients were in the primary treatment phase at the time of data collection. Complications related to the event were observed in 2 cases, mainly post-thrombotic syndrome. Anticoagulation-related adverse events occurred in 2 patients, and no VTE recurrences were recorded during the observation period.

Conclusion:
Most patients seen in this specialized VTE consultation experienced unprovoked events, and during follow-up, they had few complications and no recurrences. The high proportion of negative etiological studies underlines the importance of structured clinical follow-up, individualized risk assessment, and careful therapeutic monitoring. These findings underscore how specialized VTE consultations can enhance patient care and help avert long-term complications.

PO33

Post-thrombotic syndrome following pregnancy- associated deep vein thrombosis: a retrospective cohort study

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Background:
In the chronic phase after deep vein thrombosis (DVT), 20-50% of non-pregnant patients develop post-thrombotic syndrome (PTS). However, data on the incidence of PTS following pregnancy-associated DVT remain limited. Epidemiological studies suggest that approximately 42% of women with pregnancy-related DVT report some degree of PTS based on self-reported Villalta scores.

Materials and methods:
We conducted a retrospective cohort study of women treated in our outpatient clinic between 2015 and 2024, who experienced lower extremity DVT during pregnancy or within six weeks postpartum. All patients received therapeutic doses of low-molecular-weight heparin. Treatment lasted at least 3 months or 6 weeks postpartum, whichever was longer. At a follow-up visit, conducted on average 10 months postpartum, patients underwent objective assesment for PTS using Villalta score. Villalta score ≥5 was considered diagnostic for PTS. Recanalization of the affected vein was evaluated via duplex ultrasonography.

Results:
Among 120 women (mean age 31.8 ± 5.4 years), DVT occurred in 49.6% during the first trimester, 19.3% in the second, 20.2% in the third and 10.9% postpartum. Proximal DVT was present in 66.7% of cases. 105 women were assessed postpartum for the presence of PTS. Although 63.8% reported at least one symptom according to the Villalta score, only 7.6% met the criteria for PTS (mean Villalta score: 5.6 ± 0.9). No cases of severe PTS (Villalta score >15) were observed. No differences between groups or predictors of PTS were identified (including BMI, age, trimester of occurrence, or thrombosis location). However, incomplete venous recanalization, observed in 31.7% of patients, showed a non-significant trend toward association with PTS (OR 3.99; 95% CI 0.9–20.5; p = 0.07).

Conclusion:
Compared to previous studies available, our cohort demonstrated a lower incidence of post-thrombotic syndrome following pregnancy-associated DVT. This could be due to more objective assessement of PTS in our study and younger subjects compared to studies with non-pregnant populations. The limited number of PTS cases may have reduced the statistical power to detect significant associations. Further prospective studies with larger sample sizes are warranted to validate these findings and explore potential predictors of PTS in pregnancy.

PO34

Direct oral anticoagulants in patients diagnosed with venous thromboembolism secondary to inherited thrombophilias

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Introduction:
Inherited thrombophilias are characterized by genetic predisposition for developing venous thromboembolism (VTE). The most common genetic defects in clinical practice are deficiencies in natural anticoagulants, namely antithrombin (AT), protein C (PC), and protein S (PS), as well as the presence of polymorphisms such as Factor V Leiden and prothrombin gene mutation (PT20210a). Anticoagulant therapy is essential to prevent associated secondary complications. Direct oral anticoagulants (DOACs) have been increasingly identified as an alternative to vitamin K antagonists (VKAs) due to their efficacy and safety profile. Given this diagnosis, the choice of anticoagulant should be individualized, considering the patient's clinical characteristics, thrombotic and bleeding risk, and the location and extent of VTE.

Objectives:
Evaluation of the efficacy and safety profile of DOACs prescription in patients diagnosed with VTE associated with inherited thrombophilias.

Material and methods:
Retrospective and descriptive study of patients with VTE associated with inherited thrombophilia followed up in the Immuno-hemotherapy consultation at the Gaia/Espinho Local Health Unit, between January 2021 and December 2024. Data were obtained by consulting the SClinico® program and processed using SPSS® and Excel® software.

Results:
A total of 18 patients were evaluated, 59% male, with a median age of 43 years (min. 18 years; max. 55 years). Regarding venous thromboembolic events, 11 presented deep vein thrombosis (DVT) of the lower limbs (61.1%); 5 patients with low/intermediate risk pulmonary thromboembolism (PTE) (27.8%) and 2 patients (11.1%) with thromboses in unusual sites (portal vein thrombosis and renal vein thrombosis). Twelve patients (66.7%) were hypocoagulated with apixaban 5 mg 12/12 h, 5 patients with rivaroxaban 20 mg (27.8%) and 1 patient (5.6%) with edoxaban 60 mg. The patient's initial comorbidities were serially assessed during follow-up consultations, and a complete blood count, renal and liver function, and hemostasis study (with DOAC measurement - antifactor Xa activity if clinically justified) were requested. During the etiological investigation, a thrombophilia study was performed 3 to 6 months after the acute phase, given the previous diagnosis of unprovoked or idiopathic VTE events, with appropriate temporary suspension of anticoagulant therapy. Four patients (22.2%) with PC deficiency, three patients (16.7%) with PS deficiency, and two patients (11.1%) with AT deficiency were identified, as wells as five patients (27.8%) with homozygosity for factor V Leiden and four patients (22.2%) for the prothrombin gene mutation. During the 48 months of the study, there was no recurrence of thrombotic events or clinically significant bleeding episodes, with maintenance of DOAC therapy without the need for dosage adjustments or pharmacological class optimization.

Conclusion:
Prescribing DOACs in patients with inherited thrombophilias complicated by VTE appears to be a pharmacological option with high therapeutic efficacy and without increased bleeding complications, as demonstrated in this retrospective study. These results require external validation, with prospective studies using more robust and representative samples.

PO35

Recombinant activated factor VII as salvage treatment in life-threatening postpartum haemorrhage

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Background:
Recombinant activated factor VII (rFVIIa) was approved in 2022 in Europe for severe postpartum haemorrhage (PPH), after uterotonics failure.

Material and methods:
We reviewed two clinical cases of pregnant women with increased bleeding risk during delivery who developed PPH, requiring massive transfusion, hysterectomy and rFVIIa to control haemorrhage.

Results:
Case 1: Pregnant 32 years-old, previous history of preeclampsia and preterm birth. Admitted at 27 weeks(W)+1day(D) of gestation (day 0/D0), due to placenta accreta plus metrorrhagia; newly diagnosed with superficial venous thrombosis (cephalic vein), treated with low molecular weight heparin (LMWH).
At 35W+5D (D60), she underwent caesarean section (CS) with hysterectomy, bilateral salpingectomy and partial bladder resection due to multiorgan placenta accreta's infiltration; live male newborn (Apgar 6/7/6). Previous coagulation tests were normal. During surgery, patient progressed to haemorrhagic shock, requiring vasopressor support (lactate 11mmol/L), monitored also through viscoelastic testing (VET); minimal Hb 4.5g/dL, fibrinogen 118mg/dL, platelets 54.000/uL, D-dimers 1.38µg/mL.
The patient underwent massive transfusion (10 units of red blood cell concentrate (pRBC), 12 units of plasma (FFP), 2 platelet pool (PP)), perfusion of tranexamic acid (TXA), 6g fibrinogen and rFVIIa (5mg≈72µg/kg). She was admitted to the intensive care unit (ICU) under mechanical ventilation (MV); postoperative coagulation testing without abnormalities.
On D62, prophylactic LMWH was resumed and maintained till 6 weeks postpartum. Puerpera was discharged on D67.
Case 2: Pregnant 31 years-old, 40W+4D, admitted to the Obstetrics ward due to oligohydramnios.
On D2 she underwent CS due to labour induction's failure (live female newborn, Apgar 3/6/8).
She developed PPH with haemorrhagic shock secondary to uterine atony, was transfused with 10pRBC+9FFP+3PP (VET) [minimal Hb 5.5g/dL, thrombocytopenia 49.000/uL). She was transferred to the ICU under MV. CT angiography showed moderate hemoperitoneum in the pelvic cavity with active high-flow/arterial haemorrhage from the peri-uterine arterial vessels.
rFVIIa (6mg≈82µg/Kg) was administered. Patient underwent exploratory laparotomy, where uterine rupture was diagnosed. Hysterectomy was performed.
Intermediate-low risk pulmonary embolism was diagnosed on D5. She started therapeutic LMWH, switching afterwards to oral anticoagulation.
Puerpera was discharged on D19.

Conclusions:
The administration of rFVIIa followed current scientific guidelines for PPH unresponsive to conventional treatments (uterotonics, volume resuscitation, hemostatic agents, blood products, hysterectomy).
According to the literature, the recommended dosage ranges from 16.7-120µg/kg. However, a formal consensus has yet to be established in the Obstetrics field. These recommendations align with the dosages here administered. rFVIIa was key in achieving haemostatic control.
Puerperium presents an increased risk of developing thromboembolic events up to 3–6 weeks postpartum. This risk is significantly higher in cases of severe postpartum haemorrhage. Therefore, establishing a causal relationship between rFVIIa administration and subsequent thromboembolic events is not straightforward. Nevertheless, available clinical data suggest an acceptable safety profile for rFVIIa, though prophylactic LMWH administration should be considered.
To establish the efficacy and appropriate timing of rFVIIa administration in severe PPH, further clinical trials are needed. In fact, rFVIIa may contribute to reducing blood loss, transfusion requirements, and the need for invasive procedures, thereby preserving fertility and decreasing the risk of postoperative complications.

PO36

Recurrent thrombosis under therapeutic anticoagulation as the first sign of occult gastric cancer

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Background:

Cancer-associated thrombosis (CAT) is one of the most frequent and complex complications in oncology. Cancer patients have a markedly increased risk of venous thromboembolism (VTE), which may exceed 20% over the course of their disease. In some cases, thrombosis precedes the diagnosis of malignancy, with recurrent or idiopathic events serving as early warning signs of occult cancer. Recurrent thrombosis despite appropriate anticoagulation should prompt renewed diagnostic assessment. This case illustrates recurrent thrombosis as a first manifestation of an occult gastric adenocarcinoma and highlights the diagnostic and therapeutic challenges involved.

Materials and methods:

A retrospective analysis of a single patient with cancer-associated thrombosis was performed, complemented by a review of relevant scientific literature.

Results:

A 65-year-old man, with no recent surgery, trauma, immobilization, or known thrombophilia, presented with pain and swelling of the left lower limb. Venous Doppler ultrasound confirmed femoropopliteal deep vein thrombosis (DVT). Therefore, anticoagulation with rivaroxaban was initiated (15 mg twice daily for 21 days, followed by 20 mg once daily). Initial laboratory evaluation, including renal, hepatic, and thyroid function tests, autoimmune panel, and hereditary thrombophilia screening, was unremarkable. As the event was idiopathic, further imaging studies were suggested to investigate potential underlying causes, but the patient declined additional examinations. Six weeks later, he presented with acute dyspnea and pleuritic chest pain. CT pulmonary angiography revealed bilateral segmental pulmonary embolism, occurring despite full-dose rivaroxaban and good treatment adherence. Anticoagulation was switched to low-molecular-weight heparin (enoxaparin 1 mg/kg twice daily).A new etiological investigation was performed, and a thoraco-abdominopelvic CT showed irregular thickening of the gastric wall, multiple abdominal lymph nodes, and several hepatic lesions suggestive of metastases. The patient then consented to upper endoscopy, which revealed an ulcerated antral lesion; biopsy confirmed poorly differentiated gastric adenocarcinoma. He started palliative chemotherapy and maintained therapeutic anticoagulation with enoxaparin, with no further thrombotic events despite radiological disease progression during follow-up.

Conclusions:

From a therapeutic standpoint, this case illustrates the challenge of managing anticoagulation in the context of cancer, where tumor-driven hypercoagulability may persist despite optimal treatment, requiring individualized management. Recurrent thrombosis under therapeutic anticoagulation should be regarded as a clinical warning sign and prompt thorough investigation for an underlying malignancy. Early recognition of occult cancer may not only guide oncologic treatment but also improve thrombotic control and overall outcomes. At present, the patient remains on low-molecular-weight heparin with stable anticoagulant control. A switch back to an oral anticoagulant is being carefully considered to improve quality of life, though this remains a therapeutic challenge given the persistent risk of recurrence and bleeding in the setting of active malignancy.

PO37

Factor V Leiden and pregnancy loss: a clinical case report

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Background:
Thrombophilia refers to an increased predisposition to form blood clots (thrombi), which can lead to clinically significant complications. There are several haemostatic markers indicative of thrombophilia, which may have either congenital or acquired origins. Pregnancy represents a physiological acquired hypercoagulable state, essential for ensuring efficient placental perfusion and minimizing the risk of bleeding during gestation and childbirth. During this period, there is a progressive increase in procoagulant factors (i.e., VII, VIII, X, fibrinogen, and von Willebrand factor), accompanied by a decrease in anticoagulant activity (i.e., reduced protein S levels and resistance to activated protein C), and an inhibition of the fibrinolytic system. Additionally, hemodynamic changes and potential vascular injuries during childbirth further contribute to the increased thrombotic risk. In this context, the Factor V Leiden mutation plays a central role, namely by conferring resistance to degradation by the activated protein C complex, it promotes excessive thrombin generation and, consequently, an increased risk of venous thrombosis. The association between this mutation and obstetric complications such as recurrent pregnancy loss, foetal growth restriction, severe preeclampsia, and placental abruption highlights the need for targeted screening in high-risk scenarios, with the aim of optimizing clinical monitoring and intervention.

Objective:
The aim of this publication is to review the pathophysiology and clinical implications of the Factor V Leiden mutation in obstetrics, emphasizing its relevance in the prevention and management of perinatal complications, through the presentation of a clinical case.

Methods:
A non-systematic literature review was conducted on the Factor V Leiden mutation, focusing on coagulation changes in pregnant women. In addition, a clinical case is presented of a pregnant woman heterozygous for Factor V Leiden, with no history of thrombosis, who experienced intrauterine foetal death at 22 weeks of gestation, followed by severe haemorrhage and uterine rupture after curettage, requiring emergency hysterectomy and massive transfusion of blood products and prothrombin complex concentrate. The collected data were processed in order to maintain the anonymity of the participant and preserve their confidentiality.

Discussion:
Although the isolated Factor V Leiden mutation does not necessarily cause adverse clinical complications, its interaction with hemodynamic and haemostatic changes during pregnancy can lead to severe events. In this case, the intense activation of coagulation and fibrinolysis (D-dimers > 20,000 ng/mL) and the need for massive transfusion underscore the importance of prior thrombotic risk assessment. The identification of Factor V Leiden allows for prophylaxis consideration, reducing obstetric complications. Current evidence does not support universal screening but emphasizes the need for individualized assessment, especially in women with a history of unexplained fetal loss or placental vascular complications.

Conclusions:
Factor V Leiden remains a clinically relevant mutation with significant implications for maternal and fetal health. Awareness of its impact and the use of personalized prophylaxis are essential to prevent thrombotic and obstetric complications. This case highlights the educational value of early recognition of hereditary thrombophilias.

PO38

Impact of plasminogen activator inhibitor-1 homozygosity in obstetric context: report of three clinical cases

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Background:

Plasminogen activator inhibitor type 1 (PAI-1) is a serine protease belonging to the serpin family and a key regulator of fibrinolysis. It inhibits the activators tPA and uPA, thereby preventing the conversion of plasminogen into plasmin.

The 4G/5G polymorphism in the promoter region of the *SERPINE1* (PAI-1) gene influences its expression, with the 4G/4G genotype being associated with higher plasma levels of PAI-1.

During pregnancy, a physiologically hypercoagulable state, active fibrinolysis protects placental and foetal vascularization. However, PAI-1 overactivity, particularly in individuals carrying the 4G/4G genotype, may impair this protective mechanism, leading to hypofibrinolysis. This mechanism has been implicated in obstetric complications, including preeclampsia, intrauterine growth restriction, placental abruption, and intrauterine foetal death, possibly due to thrombosis-induced placental insufficiency.

Objective:

The main objectives were the following: i) to review the pathophysiology of the PAI-1 4G/4G gene polymorphism; ii) to present three clinical cases of homozygous patients with obstetric complications; and iii) to emphasize the relevance of genetic and laboratory diagnosis for the early identification of this risk profile.

Method:

An observational and descriptive study of three cases with obstetric complications, in which hereditary thrombophilia screening, including PAI-1 genotyping, was performed. Relevant clinical and laboratory data were collected. It should be highlighted that the data were processed in order to maintain the anonymity of the participants and preserve their confidentiality.

Results:

The first case refers to a 47-year-old woman who experienced severe postpartum haemorrhage requiring hysterectomy, with a prior history of recurrent hematologic disorders. The second case involves a 26-year-old woman with three first-trimester spontaneous abortions, none requiring curettage. The third case concerns a 35-year-old patient with two first-trimester spontaneous miscarriages.

In all cases, homozygosity for the 4G allele of the PAI-1 gene was identified, with no other clinically significant genetic thrombophilia abnormalities. Thus, the identification of this polymorphism allowed subsequent and more targeted obstetric and hematologic follow-up.

Conclusions:

The homozygous PAI-1 4G/4G polymorphism is associated with an increased risk of repeated implantation failure and recurrent pregnancy loss, although its clinical significance remains debated due to variability among studies. Nonetheless, PAI-1 homozygosity may represent an important risk factor in recurrent obstetric complications. Genetic testing, although not universally recommended, contributes to risk stratification and personalized management. The findings from these three cases reinforce the potential role of PAI-1 homozygosity as a factor associated with gestational complications, highlighting the importance of integrating laboratory investigation into obstetric clinical practice.

PO40

Post-surgical hemorrhage in senile entropion due to factor XIII deficiency

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Abstract

Background:

When diagnosing post-surgical bleeding with normal coagulation tests, factor XIII (F XIII) deficiency must be ruled out. This circulates in plasma, mainly bound to fibrinogen, and has a half-life of 7 to 14 days. F XIII acts as a fibrin stabiliser and is involved in tissue repair and healing. In F XIII deficiency, coagulation tests are normal because it does not participate in thrombus formation but rather in its stabilisation, both in congenital and acquired deficiency. Patients with FXIII levels below 1 U/dl are at high risk of early onset bleeding (with umbilical cord haemorrhage). With factor levels between 1-4 U/dl, the haemorrhagic phenotype is severe or moderate. With values above 5 U/dl, haemorrhagic symptoms are occasional. However, cases of mild deficiency (factor XIII activity levels of 30-60%) have been reported in which abnormal post-traumatic bleeding may occur. Treatment ranges from specific F XIII concentrate or fresh frozen plasma to antifibrinolytic drugs in mild cases.

Material and methods:

78-year-old patient, no toxic habits, with a history of high blood pressure, dyslipidaemia, type II diabetes mellitus and ischaemic heart disease. Two episodes of hospitalisation for anaemia of digestive origin. He had undergone transurethral resection of the prostate, infrarenal aortic aneurysm repair with endoprosthesis, and cataract surgery in both eyes without haemorrhagic complications. He was being treated with rosuvastatin, valsartan, bisoprolol, furosemide, and linagliptin. He required surgery for senile entropion of the lower eyelid of the right eye under local anaesthesia. Preoperative tests, including a complete blood count, biochemistry, and coagulation, showed normal values. A lateral tarsal strip was performed to anchor the outer edge of the eyelid to the orbit to tighten it. Forty-eight hours after the operation, the patient presented with continuous bleeding from the surgical wound, hyposphagma, and a large haematoma on the lower eyelid. The surgical wound was examined on three occasions and, despite local compression and haemostatic measures, recurrent bleeding continued in the surgical bed for two weeks, requiring urgent attention on several occasions.

Results:

Subsequently, the study was expanded to include plasma factor determination, finding only a Factor XIII value of 42%. After ruling out other causes of haemorrhagic diathesis, Factor XIII deficiency was considered to be the cause of the condition. Treatment with tranexamic acid at a dose of 1 g every 8 hours orally helped to resolve the condition.

Conclusions:

Outpatient entropion surgery resulted in moderate haemorrhagic complications that complicated the postoperative period. The mild Factor XIII deficiency caused recurrent bleeding that required interventional and pharmacological treatment with antifibrinolytics.

PO41

Warfarin use during pregnancy in a woman with double mechanical valve prostheses: between thrombosis and teratogenicity

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Background:

Pregnancy in women with mechanical heart valves (MHVs) poses major clinical challenges, as maternal thromboembolic and bleeding risks coexist with significant fetal hazards. Vitamin K antagonists (VKAs) offer the most reliable protection against valve thrombosis but carry a dose-dependent teratogenic risk during the first trimester. In contrast, low-molecular-weight heparin (LMWH) avoids fetal toxicity but is associated with higher thrombosis rates when subtherapeutic or poorly monitored. Current international guidelines recommend using VKAs outside the organogenesis period and switching to heparin near term to minimize peripartum hemorrhage.

Material and methods:

Single-case report based on a structured review of the medical record from a tertiary hospital. Clinical data included maternal background, anticoagulation regimen by trimester, laboratory and INR monitoring, and obstetric assessments.

Clinical case:

A 30-year-old gravida 2, para 1 woman with rheumatic heart disease and double mechanical prostheses (mitral St. Jude 29 mm and aortic St. Jude 21 mm, implanted in October 2021) required lifelong anticoagulation. Before pregnancy, she was treated with warfarin, presenting INR variability but no valve thrombosis or major bleeding. During the current pregnancy, LMWH (enoxaparin) was used throughout the first trimester. At 15 weeks + 1 day, obstetric evaluation described a “probable embolic infarct in the first trimester,” though the affected territory was unspecified. Fetal ultrasound was normal, with low-risk combined screening. By 19 weeks + 3 days, a multidisciplinary team agreed to transition to warfarin for the second trimester (INR target 2.0–3.0) and to continue until 36 weeks, followed by a planned peripartum heparin strategy. No maternal hemorrhagic complications, valve thrombosis, or fetal malformations were reported.

Conclusions:

This case exemplifies a guideline-aligned, trimester-specific anticoagulation approach for a high-risk patient with double MHVs: LMWH during organogenesis, warfarin during mid-pregnancy for optimal maternal protection, and reintroduction of heparin at 36 weeks to reduce delivery bleeding. The plan balances the teratogenic potential of warfarin during weeks 6–12 against its superior efficacy in preventing valve thrombosis compared with LMWH, particularly in mitral prostheses. The normal mid-trimester ultrasound and low-risk screening reinforce the fetal safety of deferring VKA use until after organogenesis, while the second-trimester reintroduction of warfarin mitigates the well-documented maternal risks associated with exclusive heparin therapy.

Overall, this case supports a dose, trimester, and risk adapted anticoagulation pathway that safeguards maternal outcomes without compromising fetal safety.

PO42

When coagulation unmasks cancer: disseminated intravascular coagulation as the first clue to gastric adenocarcinoma

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Background:

Disseminated intravascular coagulation (DIC) is a critical, life-threatening systemic coagulopathy. It is characterized by uncontrolled activation of coagulation and secondary fibrinolysis, leading to consumption of platelets and clotting factors. Among noninfectious causes, malignancy-associated DIC is particularly relevant in solid tumors with bone marrow infiltration, notably gastric adenocarcinoma. Early recognition is essential, as supportive correction of coagulopathy must be balanced with prompt oncologic treatment of the underlying trigger.

Material and methods:

This single-patient case report was based on a structured review of medical, laboratory, and imaging records from a tertiary university hospital. Data were collected from the emergency admission, observation, intensive care unit (ICU), and inpatient follow-up notes. Diagnostic criteria followed the International Society on Thrombosis and Haemostasis (ISTH) scoring system for overt DIC, and therapeutic decisions were guided by serial coagulation testing and viscoelastic monitoring.

Clinical case:

A 41-year-old woman (past medical history: Graves' disease; no other relevant conditions) presented with hemoptysis and a rapidly enlarging hematoma of the left upper limb following bronchoscopy. At presentation, she was hemodynamically stable but appeared anxious and pale. Laboratory studies revealed normocytic anemia (Hb 9.7 g/dL), severe thrombocytopenia (14 × 10?/L), markedly elevated D-dimer (> 100 µg/mL), and hypofibrinogenemia (92 mg/dL) with mildly prolonged PT and aPTT—fulfilling ISTH criteria for overt DIC (score = 5). A peripheral smear showed left-shifted myeloid precursors without schistocytes. Chest CT demonstrated bilateral ground-glass opacities compatible with alveolar hemorrhage and mediastinal lymphadenopathy.

Over the next 24 hours, hemoptysis worsened and hemoglobin dropped to 6 g/dL. Despite aggressive transfusional support with fresh frozen plasma, fibrinogen concentrate, and platelet pools, bleeding persisted, prompting ICU admission due to respiratory compromise. Antifibrinolytic therapy (tranexamic acid) was administered, and coagulation correction was guided by rotational thromboelastometry (ROTEM®).

Bone marrow aspiration revealed clusters of poorly differentiated malignant cells. Immunohistochemistry confirmed CDX2 positivity, consistent with a gastric origin. CT staging demonstrated gastric wall heterogeneity, a single hepatic lesion, and a vertebral focus at D3, leading to the diagnosis of stage IV HER2-positive gastric adenocarcinoma with bone marrow infiltration. Paraneoplastic DIC was identified as the cause of the bleeding diathesis. Immuno-hematologic therapy with FOLFOX and trastuzumab was initiated, along with corticosteroids for symptom control. Over the following days, bleeding subsided, respiratory function improved, and transfusion requirements decreased progressively.

Conclusions:

This case illustrates disseminated intravascular coagulation as the initial manifestation of metastatic gastric carcinoma with bone marrow infiltration. Paraneoplastic DIC may present subtly, with bleeding disproportionate to thrombocytopenia, and often precedes the oncologic diagnosis. Early identification using ISTH DIC scoring and viscoelastic testing is critical for adequate supportive care. The patient's improvement after tumor-specific chemotherapy reinforces that control of the underlying malignancy remains the cornerstone of treatment. Close collaboration among transfusion medicine, intensive care, and oncology teams is essential to optimize outcomes in cancer-related coagulopathies.

PO43

Acquired hemophilia A: the essential role of the laboratory in diagnosis

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Background:
Acquired hemophilia A (AHA) is a rare, potentially life-threatening autoimmune bleeding disorder caused by autoantibodies against coagulation factor VIII. It typically affects elderly patients and presents with spontaneous hematomas and an isolated prolongation of activated partial thromboplastin time (aPTT), which may represent the first and only analytical clue. Prompt recognition and laboratory investigation are critical to guide therapy and improve prognosis.

Methods:
We report the case of an 84-year-old man with dementia and multiple comorbidities admitted for extensive spontaneous hematomas of the thoracic and abdominal wall, in the absence of trauma or anticoagulant therapy. Initial coagulation screening showed a markedly prolonged and isolated aPTT, leading to an extended laboratory investigation including factor assays and inhibitor testing.

Results:
Factor VIII activity was < 1%, with a high-titer inhibitor (50 Bethesda Units), confirming acquired hemophilia A. Other coagulation factors were within reference intervals, and screening for viral and autoimmune disorders was negative. High-dose prednisolone (80 mg/day) was initiated, resulting in progressive improvement of aPTT (82″ → 36.9″) and recovery of FVIII activity (124%) after several weeks. The patient remained clinically stable, with resolution of hematomas and no new bleeding episodes.

Conclusion:
This case illustrates the pivotal role of the clinical laboratory in diagnosing acquired hemophilia A. The analytical finding of an isolated aPTT prolongation prompted further testing that was decisive for confirming an FVIII inhibitor and guiding appropriate treatment. Early recognition by the laboratory team is fundamental to prevent morbidity and mortality in this rare and often underdiagnosed disorder.

Table 1 – Chronological evolution of laboratory parameters

Date	aPTT (s)	Hemoglobin (g/dL)	Factor VIII (%)	Inhibitor (Bethesda U)	Comments
17 Nov 2023	77.7	6.2	—	—	Emergency admission
18 Nov 2023	79.5	7.7	—	—	—
20 Nov 2023	82.2	6.2	—	—	—
21 Nov 2023	76.7	9.1	1	50	Mixing test corrected
23 Nov 2023	87.2	8.3	—	—	—
27 Nov 2023	74.1	10.5	1	—	—
04 Dec 2023	56.9	11.3	—	—	Start of improvement
12 Dec 2023	45.0	—	49	Negative	Inhibitor negative
19 Dec 2023	37.2	—	100	Negative	—
26 Dec 2023	38.4	—	125	—	—
03 Jan 2024	36.9	13.6	124	Negative	Clinical discharge
25 Jan 2024	35.7	—	177	—	—
12 Feb 2024	31.6	12.6	—	—	Re-admission

PO45

Occult malignant disease: the saga of multiple thromboembolic events

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Background:

Venous thromboembolism is a common complication and the leading cause of death in patients with malignant disease. In 1844, Trousseau described the principals of clinical evidence between VTE and cancer, which often appears as a harbinger of hidden malignant disease. These circumstances require a diagnostic and therapeutic approach carefully adapted to the tumor characteristics, where the antithrombotic treatment constitutes a medical challenge.

Material and methods:

This report describes the clinical case of a patient referred to an immunohematology appointment for evaluation and therapeutic guidance due to a history of thrombosis. Clinical information was extracted from the patient's record, containing data from the previous 3 years. A 48-year-old woman, ceramist, was referred due to multiple thrombotic events, mostly while undergoing anticoagulation therapy. There was no personal or family history. No risk factors were identified. She experienced thrombophlebitis of the left lower limb (LL), deep vein thrombosis of the right LL, left pulmonary thromboembolism and an extension of the thrombosis to the right, with occlusion of the right iliac vein and involvement of the inferior vena cava. Due to the severity, a filter was placed in the inferior vena cava, with partial thrombus formation, after 6 months of therapeutic anticoagulation. The clinical history with additional tests supports the hypothesis of an underlying malignant disease. A thrombophilia study was carried out and oncological disease was screened.

Results:

The functional thrombophilia study revealed 3 free PS assays that were slightly diminished, with no alterations to the PROS1 gene. Molecular studies revealed no major risk factors or mutations in the JAK2 gene. On a CT scan was detected a spiculated opacity of 14 mm in the lingula of the lung; with intense FDG avidity on PET/CT. Through transthoracic lung biopsy, it was possible to identify the presence of cells with features suggesting lung adenocarcinoma. Oral chemotherapy was started immediately, with a good clinical response. The patient was under therapeutic anticoagulation during all events. LMWH was the treatment of choice. Switching to oral anticoagulation occurred as soon as it was verified that there were no contraindications. Two years after the last thrombotic event, the patient is clinically stable, undergoing chemotherapy and oral anticoagulation with Rivaroxaban. There are no bleeding complications or new thrombotic events. The imaging findings have improved, with filter patency and re-permeabilization of the right iliac vein and inferior vena cava.

Conclusions:

In this case, venous thromboembolism was the first manifestation of an occult malignant disease. A multidisciplinary approach enabled the diagnosis, appropriate treatment and follow-up. Spontaneous thrombotic events, in individuals with no relevant personal medical history or triggering risk factors, pose a multidisciplinary clinical challenge to all medical specialties. This clinical case highlights the importance of detecting occult oncological disease and adopting an individualized therapeutic approach.

PO46

Clinical case report: peripartum anticoagulation management in pregnant women with a mechanical aortic valve

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Background:

Pregnancy in women with mechanical aortic valve prostheses represents a particularly high-risk clinical scenario, especially during labour. Mechanical heart valves in pregnancy are associated with considerable risks, including valve thrombosis, maternal mortality, and severe morbidity. The hypercoagulable state induced by pregnancy -marked by increased clotting factors and reduced anticoagulant proteins- further heightens the risk of thrombotic events in this patient group. Managing anticoagulation is especially challenging, as insufficient therapy may result in valve thrombosis, whereas excessive anticoagulation can lead to bleeding complications, including obstetric hemorrhage.

Striking the right balance in anticoagulation is essential to minimise both maternal and fetal complications, creating a complex clinical dilemma that necessitates multidisciplinary input and tailored anticoagulation protocols throughout pregnancy and the peripartum period.

Materials and methods:

This report details the case of a 33-year-old pregnant woman at 38 weeks and 1 day of gestation, who underwent an elective caesarean section due to her history of mechanical aortic valve replacement (2018) and ongoing anticoagulation with warfarin. Warfarin was discontinued at 36 weeks and replaced with low molecular weight heparin (LMWH), administered at a therapeutic dose of 100 mg subcutaneously twice daily. Thirty-six hours prior to the caesarean section, LMWH was switched to unfractionated heparin (UFH), delivered as a 5,000 IU intravenous bolus followed by an infusion at 18 IU/kg/h, with the target activated partial thromboplastin time (aPTT) set at ≥2 times the control value (≥56 seconds).

On the day of the caesarean, UFH was stopped four hours before surgery, and aPTT was measured four hours after cessation of UFH. Prophylactic antibiotics for bacterial endocarditis (cefazolin 2 g IV) were administered prior to incision. Enoxaparin was restarted six hours after the procedure during the postoperative period.

Results:

aPTT levels were kept within the desired range prior to caesarean delivery (aPTT 83.1 seconds), in line with current European Society of Cardiology guidelines. Immediately before the caesarean, an aPTT of 23 seconds (<1.3 times control, i.e. <36.4 seconds) permitted the procedure to proceed safely. The operation was completed without any hemorrhagic or thrombotic complications.

Conclusions:

This case underscores the critical importance of thorough multidisciplinary planning and strict adherence to evidence-based management protocols, both of which are essential for minimising maternal morbidity and optimising perioperative outcomes in this high-risk patient population. Furthermore, it demonstrates that, despite the significant risks of thrombosis and hemorrhage associated with pregnancy in women with mechanical heart valves, the use of individualised anticoagulation regimens -combined with close monitoring and robust interdisciplinary collaboration- can yield positive maternal outcomes without an increased risk of bleeding complications.

PO48

PEG-asparaginase–related venous thromboembolism in adult acute lymphoblastic leukemia: a case of coexisting thrombotic and bleeding risks

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Venous thromboembolism (VTE) is a frequent and potentially severe complication of PEG-asparaginase-based regimens for acute lymphoblastic leukemia/lymphoma (ALL), particularly in adults. Despite limited evidence regarding the optimal prevention strategy, current guidelines recommend pharmacological thromboprophylaxis during the induction phase of chemotherapy, together with antithrombin (AT) monitoring and repletion throughout treatment.

We report the case of a 26-year-old woman with obesity who was diagnosed with T-cell ALL. At presentation, she exhibited supradiaphragmatic lymphadenopathy and pericardial and bilateral pleural effusions requiring pericardiocentesis and thoracentesis, with minimal bone marrow infiltration. The patient started a pediatric-inspired chemotherapy protocol including PEG-asparaginase. Pharmacological thromboprophylaxis was initially withheld due to severe thrombocytopenia and severe menometrorrhagia, prompting initiation of a combined oral contraceptive pill.

On day 10 of induction therapy, the patient developed sinus tachycardia and exertional dyspnea. Electrocardiography, chest X-ray, and serum biomarkers (troponin, NT-proBNP and D-dimer) were unremarkable. Persistent tachycardia prompted an echocardiogram, which showed no significant findings. Further investigation with CT pulmonary angiography demonstrated an acute bilateral central pulmonary embolism. Lower limb venous doppler ultrasound ruled out deep vein thrombosis. Therapeutic anticoagulation with low-molecular-weight heparin (LMWH) was initiated with platelet transfusion support to maintain counts above 50×10?/L. AT activity was normal, therefore repletion was not required. Therapeutic anti-Xa activity was achieved and an intrauterine device was placed.

At discharge, the patient showed clinical improvement, with CT/PET-FDG demonstrating a good partial response and bone marrow assessment revealing negative minimal residual disease. PEG-asparaginase was continued at a 50% dose reduction. After two months, anticoagulation with LMWH was switched to apixaban, which is to be maintained throughout PEG-asparaginase treatment. At the three-month follow-up, a repeat CT scan demonstrated near-complete resolution of the thrombus. Currently, the patient is in her sixth month of treatment, with no recurrent VTE, bleeding events, or evidence of relapse.

This case highlights the clinical dilemma of managing concurrent thrombotic and bleeding risks in adults receiving PEG-asparaginase. In practice, thromboprophylaxis is often withheld due to thrombocytopenia, yet the high incidence of PEG-asparaginase–related VTE calls for reconsideration of prophylactic strategies. Continuing anticoagulation despite thrombocytopenia may benefit selected high-risk patients. Moreover, it is crucial to determine whether PEG-asparaginase should be continued, dose-reduced or omitted, as treatment modifications may compromise outcomes of this malignant disease, further underscoring the importance of effective thromboprophylaxis.

In the past decade, a few case reports have documented successful treatment of PEG-asparaginase-related VTE with direct oral anticoagulants (DOACs). Unlike LMWH, whose effect depends on AT activity and costly AT replacement, DOACs act independently of AT, which may be advantageous in this setting. However, prospective studies are needed to confirm their safety and efficacy in this population.

In summary, we describe a case of PEG-asparaginase-related VTE successfully managed with LMHW followed by apixaban in a young adult with ALL. This case highlights the need for prospective studies to establish optimal strategies for prevention and management of PEG-asparaginase–related thrombosis in adults.

PO50

Cost-effective strategy for the optimization of ADAMTS13 activity testing in Croatia

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Background:

Albeit being a rare disease, thrombotic thrombocytopenic purpura (TTP) is a life-threatening thrombotic microangiopathy that requires prompt diagnosis and treatment. The diagnosis depends on clinical expertise and is confirmed with a very low activity (<0.10 kIU/L, <10%) of a disintegrin-like and metalloproteinase with thrombospondin type 1 motif 13 (ADAMTS13). ADAMTS13 determination represents a crucial step for establishing or ruling-out the diagnosis of TTP. Optimization of ADAMTS13 testing is needed for prompt patient management, taking also in account laboratory resources and testing availability. The aim of this study was to determine the best cost-effective strategy for ADAMTS13 testing based on testing priority and disease activity.

Materials and methods:

Starting from 2021, ADAMTS13 activity testing in Croatia is performed solely in the coagulation laboratory of the Department of Laboratory Diagnostics, University Hospital Centre Zagreb, Zagreb, Croatia. In order to optimize ADAMTS13 testing, we have designed a specific query form for shipping samples for ADAMTS13 testing from other Croatian hospitals. This query form is also incorporated in the national recommendations for the diagnosis and treatment of immune thrombotic thrombocytopaenic purpura in adults of the Working group for benign hematological diseases of the Croatian cooperative group for hematological diseases, published in May 2023**.** One of the major queries addressed to the treating physician was to indicate whether an urgent testing is required, such as a great suspicion of first acute episode of TTP, clinical relapse or routine follow-up determination for patients in remission. Another important information to be provided in the query form was whether the sample was collected before or after plasma exchange. Total cost of reagents utilized per year for ADAMTS13 activity testing, was calculated following possible two scenarios: immediate performance of sample analysis upon admission (on-demand scenario) and batch sample analysis (serial testing scenario), according to information provided in the completed query forms. The calculation included the number of all analysed tests, comprised of quality control and patient samples in the period from May 2021 to July 2024. The on-demand scenario implies the determination of a quality control sample with each run, meaning that every single testing for ADAMTS13 is accompanied with at least one control sample determination. The serial testing scenario assumed weekly batch analysis of two or more non-urgent samples that were selected according to provided query form data.

Results:

The comparison of obtained reagent cost per year of both scenarios before the publication of national guidelines yielded a cost saving of 3.976,57€ when a batch-testing strategy was applied (total of 2-year requests for ADAMTS13, N=336). After the implementation of guidelines, the calculated reagent cost saving per year was 2.4-fold higher (9.515,31€, N=311) than before the publication of guidelines.

Conclusions:

The introduction of a strategy based on provided information from a treating physician in the received completed query form, resulted in the reduce of unnecessary reagent costs and unnecessary single sample testing. The implemented cost-effective ADAMTS13 testing strategy led to significantly higher reagent cost saving per year and confirmed the importance of publishing and adherence to national guidelines.

PO54

Dual presence of anti-factor V I antibody and lupus anticoagulant: a rare and confounding case

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Background:

Lupus anticoagulant (LA) and anti-Factor VIII (anti-FVIII) antibodies are distinct acquired autoimmune inhibitors of coagulation. Although they share similar laboratory features, they difer fundamentaly in their pathophysiology and clinical expression: LA is associated with a prothrombotic tendency, whereas anti-FVIII antibodies cause hemorrhagic diathesis. The coexistence of these inhibitors is exceedingly rare, with only a few cases documented in the literature.

Material, methods and Results:

A 61-year-old man presented with sweling and pain in the left calf that worsened upon mobilization, with no history of trauma or anticoagulant therapy. Radiological exploration revealed the left calf hematoma, hemoperitoneum, and retroperitoneal and intramuscular (psoas muscle) hematomas. The patient was subsequently transferred to the intensive care unit. Initial laboratory evaluation revealed a hemoglobin level of 9.1 g/dL, a hematocrit of 27.1%, a platelet-count of 409 x 10?/L, and a white blood cel count of 20.6 x 10?/L. Standard coagulation studies showed a normal prothrombin time (PT) of 99% and a prolonged activated partial thromboplastin time (APTT) with a ratio of 1.95. To investigate this prolongation, a mixing study was performed immediately and after a 2-hour incubation at 37°C, which demonstrated a failure to correct the APTT, suggesting the presence of a circulating inhibitor. Given the hemorrhagic presentation, a one-stage clo ting activity assay for intrinsic pathway factors (VIII, IX, and XI) was performed on an ACL Top Family analyzer. The results showed Factor VIIIc, IXc,XIc and XIIc levels of 4,6%, 100,5%, 56,1% and 56,2% . LA screening was concurrently carried out in accordance with the recommendations of the International Society on Thrombosis and Hemostasis (ISTH). The results were as folows: SCT: Low phospholipid concentration = 86.3 s; High phospholipid concentration = 68.2s; Normalized ratio = 1,26; dRVVT: Screen = 44.1 s; Confirm = 29.5s; Normalized ratio = 1,49. Given the confirmed presence of LA, a chromogenic Factor VIII assay was performed ON SYSMEX®2500 analyzer, which revealed a level of 5,2%, thereby confirming the diagnosis of acquired hemophilia A coexisting with LA. The patient was initiated on corticosteroid therapy (1 mg/kg/day) for the acquired hemophilia and received recombinant activated Factor VII (NovoSeven® at 90 µg/kg), fresh frozen plasma, and packed red blood cel transfusions. The etiological workup for acquired hemophilia, including viral serologies and tumor markers, returned negative or within normal limits.

Conclusions:

The coexistence of Lupus Anticoagulant and an anti-Factor VIII antibody represent a hematologic crisis. It necessitates close colaboration between the clinician and the specialized coagulation laboratory to avoid diagnostic errors. The use of chromogenic assays is essential for an accurate diagnosis. Management must be highly individualized, carefuly weighing the benefit/risk balance of immunosuppressive, hemostatic, and anticoagulant therapies.

PO57

Case Report: Acquired von Wilebrand Disease Secondary to Chronic Lymphocytic Leukemia

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Background

Acquired von Wi lebrand disease (AvWD) is a rare bleeding disorder that may develop in association with lymphoproliferative, autoimmune, or cardiovascular conditions. It results from increased clearance or inhibition of von Wi lebrand factor (vWF), leading to variable bleeding manifestations. Early recognition is crucial to prevent life-threatening complications.

Material and methods

We report the case of an 81-year-old male with a history of B-cel chronic lymphocytic leukemia (CLL) diagnosed in 2013, currently on third-line therapy with ibrutinib since November 2021, who was admi ted for fever of unknown origin. During hospitalization, pulmonary aspergi losis was diagnosed, leading to discontinuation of ibrutinib and initiation of voriconazole. The patient was also receiving prednisolone (1 mg/kg/day), acyclovir, and cotrimoxazole prophylaxis. Subsequently, he developed cholestatic liver enzyme abnormalities and underwent a liver biopsy, after which he presented with hemodynamic instability. Computed tomography revealed hemoperitoneum, requiring urgent surgical intervention and transfusional support.

Results

Coagulation studies showed a prolonged activated partial thromboplastin time of 48 seconds (reference values 20,9-34.9 secunds) with incomplete correction in mixing studies, suggesting the presence of an inhibitor. Lupus anticoagulant was positive. Given the severe bleeding complication, further laboratory testing demonstrated decreased vWF antigen and activity (FV I 61.9%; vWF:RCo/vWF:Ag 21/53.6), consistent with acquired von Wi lebrand disease, most likely secondary to CLL. The patient was managed supportively with transfusions, plasma products.

Conclusions

This case i lustrates the diagnostic chalenge of acquired von Wi lebrand disease in a patient with CLL presenting with severe bleeding folowing an invasive procedure. The coexistence of lupus anticoagulant further complicated the evaluation. Awareness of this rare entity and prompt laboratory investigation are essential for accurate diagnosis, timely management, and prevention of recurrent bleeding in patients with hematologic malignancies.

PO60

Platelet transfusions to support anticoagulation management in thrombocytopenic hemato-oncologic patients: a retrospective analysis

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[2] CH TRAS OS MONTES

Background:

The management of anticoagulation in patients with hemato-oncologic disease and thrombocytopenia is a complex clinical challenge that requires an effective balance between thrombotic and bleeding risks. For this reason, platelet transfusions are frequently needed to allow the initiation, maintenance, or reintroduction of anticoagulation. Thus, individualized patient management and the clinician's experience become key points.

Material and methods:

A retrospective observational analysis was conducted of all platelet transfusions performed in the Hemato-Oncology Unit (HOU) of ULS São João, between 01/01/2025 and 06/30/2025, in patients with a platelet count ≤50,000/µL. For each transfusion, age, gender, clinical diagnosis, platelet count, transfusion indication, as well as data regarding anticoagulation, thrombosis, bleeding, and outcome were collected.

Results:

A total of 1,542 transfusions were performed, of which those administered to patients with platelet counts below 10,000/µL (1,132; 73.4%) were excluded from the extended analysis. Among the remaining transfusions, 74 (18.1%) were administered to patients with an indication for anticoagulation, whose characteristics are detailed in Table 1.

Conclusions:

In this cohort, platelet transfusions were often required not only for bleeding but to initiate, continue, or resume anticoagulation. These findings underscore the role of transfusion support in managing thrombotic and bleeding risks in thrombocytopenic hemato-oncologic patients and highlight the need for clear, individualized transfusion strategies.

PO66

Protein S deficiency and antiphospholipid syndrome: are they related?

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Background:

Protein S (PS) is a vitamin K–dependent glycoprotein that acts as a cofactor of activated protein C, playing a key anticoagulant role. Its deficiency can be congenital or acquired, the latter often secondary to consumption (disseminated intravascular coagulation), liver disease, vitamin K deficiency, or the presence of autoantibodies.
Several cases of PS deficiency associated with Antiphospholipid Syndrome (APS) have been reported in the literature, potentially linked to an increased thrombotic risk.
Characterizing this phenomenon is relevant, as early recognition may influence both therapeutic strategy and patient follow-up.

Material and methods:

This is a case report of a 46-year-old man with a medical history of hypertension, multiple sclerosis, and degenerative cervical spine disease, under treatment with amlodipine, presented to the emergency department with an ischemic stroke. He was discharged on aspirin, clopidogrel, and atorvastatin.
Approximately one year later, he experienced a new ischemic stroke.
Given the recurrence of arterial thrombosis and a relevant family history (father with intestinal ischemia), a thrombophilia work-up was requested, following clinical stabilization, in the Immunohematology outpatient setting.

Results:

Laboratory results revealed: aPTT 59.9 s (ref. 27–38 s), positive lupus anticoagulant (ratio 1.76), anticardiolipin IgG 43 U/mL (positive >40 U/mL), antibodies to β2-glycoprotein I IgG 29 U/mL (positive >10 U/mL), and Protein S level of 31% (ref. 55–140%), with no other relevant abnormalities.
Three months later, repeat testing confirmed persistence of: positive lupus anticoagulant (ratio 1.68), anticardiolipin IgG 323 U/mL, antibodies to β2-glycoprotein I IgG 430 U/mL, and PS 20%.
Molecular analysis of the PROS1 gene, including large deletion/duplication screening by MLPA, showed no pathogenic variants explaining the deficiency.
These findings confirmed a triple-positive Antiphospholipid Syndrome diagnosis and suggested an acquired PS deficiency mediated by autoantibodies.

Conclusions:

APS is a systemic autoimmune disorder characterized by persistent antiphospholipid antibodies and thrombotic and/or obstetric events.
Although PS deficiency is typically congenital, anti-PS autoantibodies have been described in association with APS, though their pathogenic mechanism remains unclear.
In this case, the combination of persistently high antiphospholipid antibody titers, absence of PROS1 variants, and progressive reduction in PS levels supports the diagnosis of an acquired deficiency.
Clinically, the coexistence of these two thrombophilic conditions may explain the recurrence of arterial thrombotic events despite antiplatelet therapy.
This case adds to the limited number of reports describing this association and reinforces the need to consider autoimmune mechanisms in the evaluation of anticoagulant protein deficiencies, particularly in patients with recurrent thrombosis and no identifiable genetic mutations.

PO67

Temporary mechanic aortic valve dysfunction – a clinical dilemma to solve

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Background:

Mechanical prosthetic heart valves are a great option for valvular substitution in younger patients, when paired with sustained anticoagulation. Late complications are rare but possibly life-threatening.

Case summary:

A 65-years old male patient presented to the emergency department due to intermittent oppressive chest pain, pallor and fainting sensation, after a short period of physical exercise. The patient had received a mono-disc mechanical aortic prosthesis 14 years before, due to symptomatic severe aortic stenosis, and was medicated with warfarin. He also mentioned shortness of breath and wheezing for about 15 minutes, specifically referring to not hearing the usual metallic cardiac sounds for a while.

At physical examination, the patient was clammy, blood pressure 90/50 mmHg, heart rate 80 bpm, slight tachypnoea and adequate peripheral perfusion. Cardiac auscultation was rhythmic, with metallic timbre. He had hypoxia, with bilateral rales on auscultation. The first electrocardiogram, in pre-hospital care, showed diffuse ST-segment depression, with ST-elevation in aVR. At admission, the ECG had almost completely normalized.

Emergent coronary angiography showed no coronary lesions, thrombus or dissections. Fluoroscopy displayed normal valvular position and motion. Blood analysis revealed elevated troponin I and myoglobin, as well as mild renal impairment.

Discussion:

Throughout the years the patient presented stable values of INR. He had no other relevant past history. During hospitalization, the patient kept asymptomatic and hemodynamically stable. Computerized tomography showed normal position and motility of the prosthesis and no evident signs of thrombosis; there was some pannus in the sub-annular anterior right position. There were no coronary anomalies.

Cardiac magnetic resonance disclosed mildly depressed left ventricular function with global hypokinesis. The STIR sequences showed diffuse subendocardial hypersignal. Late-enhancement gadolinium was diffusely present, in subendocardial position, in every ventricular wall.

The patient was started on medical therapy and discharged after 20 days. Low-dose acetilsalicilic acid was added to the anti-thrombotic therapy.

Conclusion:

The overall findings and clinical evolution point to the possibility of transitory prosthetic dysfunction, with acute severe aortic regurgitation (*fixed-open position*). This could have resulted in a severe enough coronary steal and cardiac hypoperfusion, explaining the multi-vessel territory involvement with a clear angiography. The main issue here is which treatment would be the most appropriate for the patient. Specifically, if the prosthesis should be replaced or just keep tbe patient under vigilant follow-up. This was discussed in Heart Team, with both the patient and his family, and watchful waiting was decided for the time being.

Fig. 1 A, B and C - Short axis view of T2 images.

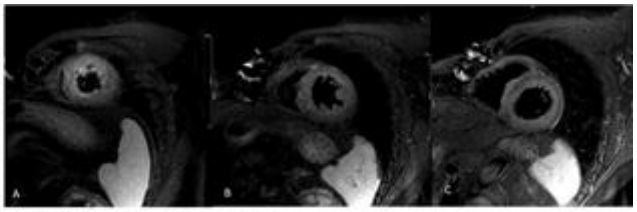

Fig. 2 A, B and C - Short axis view of late enhancement gadolinium.

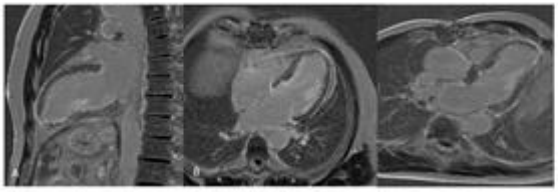

Fig. 3 A, B and C - Two, four and three chambers views in T2w images.

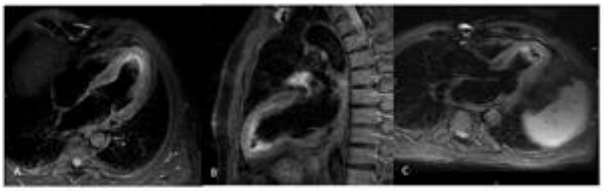

Fig. 4 A, B and C - Four, two and three chambers view of late enhancement gadolinium.

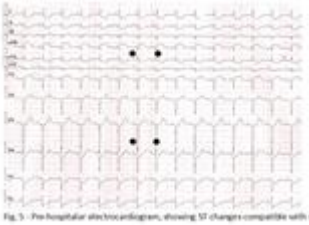

Fig. 5 - Pre-hospitalar electrocardiogram, showing ST changes compatible with multivessel coronary event.

Fig. 6 - Valve angle opening in CT scan.

Fig. 7 - Valve opening in fluoroscopy.

PO70

Prevalence and clinical relevance of IgA anticardiolipin and IgA anti-ß2 glycoprotein I antiphospholipid antibodies in splanchnic vein thrombosis

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Background:
Splanchnic venous thrombosis (SVT) is a rare but potentially serious and unusual localization of venous thromboembolic disease (VTE). The role of non-conventional antiphospholipid antibodies in SVT is still debated. The aim of our study was to determine the prevalence and assess the clinical relevance of IgA anticardiolipin and IgA anti-β2-glycoprotein I antibodies in patients with splanchnic vein thrombosis.

Material and methods:
This cross-sectional, comparative, case-control study (January 2019–August 2021) included 32 patients with SVT and 30 healthy blood donors. All patients and controls were screened for anticardiolipin (IgG, IgM, and IgA) and anti-β2-glycoprotein I (IgG, IgM, and IgA) antibodies using ELISA. Clinical and biological characteristics were recorded from medical records.

Results:
The mean age was 50, with a sex ratio of 0.77. Eight patients (25%) had a history of VTE. The portal vein was the most frequent location (69%). Cirrhosis was the most frequent etiology (n=17), followed by chronic inflammatory bowel disease (n=4). The IgA isotype was identified in 34% (n=11) of patients versus 0% in the control group (p=0.001). *Isolated IgA positivity was found in 8 patients (25%): 4 IgA anti-β2GPI, 1 IgA aCL, and 3 with both IgA anti-β2GPI and IgA aCL.* Anti-β2GPI antibodies were found in 11 patients versus 1 control (p=0.002). The IgA anti-β2GPI was the most common isotype, found in 10 patients (31%, p=0.002): 8 isolated IgA, 1 IgA+IgM, and 1 IgA+IgM+IgG. aCL antibodies were found in 8 patients versus none in controls. IgA aCL was the most common isotype, identified in 6 patients (19%, p<0.001): 4 isolated IgA, 1 IgA+IgG, and 1 IgA+IgM+IgG.

Conclusions:
IgA anticardiolipin and IgA anti-β2-glycoprotein I antiphospholipid antibodies may play a role in the pathogenesis of SVT. Larger prospective studies are needed to better assess their potential pathogenic significance.

PO71

Early identification of platelet and fibrinogen deficits with Rotem

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Background and objective:

Thrombocytopenia and hypofibrinogenemia increase bleeding risk in surgical and critically ill patients. Rapid, point-of-care assessment is essential for timely hemostatic intervention. This study evaluated the predictive performance of ROTEM parameters (EXTEM and FIBTEM) for detecting thrombocytopenia and hypofibrinogenemia.

Methods:

This retrospective study included all ROTEM tests performed between November 2022 and June 2025 for which platelet counts and plasma fibrinogen measurements were obtained simultaneously. Patient profiles were reviewed, and ROTEM parameters (EXTEM and FIBTEM), specifically A5, A10, and maximal clot firmness (MCF), were retrieved. Platelet counts were measured using an automated Coulter analyzer, and plasma fibrinogen levels were determined by the reference Clauss method on an ACL TOP Family IL 550 coagulometer. The relationships between ROTEM parameters and laboratory values were assessed using Pearson correlation coefficients.

Results:

During the study period (November 2022 to June 2025), a total of 212 ROTEM tests were performed, of which 97 met the inclusion criteria of having simultaneous platelet counts and plasma fibrinogen measurements. The mean age of the included patients was 22 years, with 58% males and 42% females. Most tests were requested for postoperative cardiac surgery patients and for those experiencing hemorrhagic shock in the intensive care setting. FIBTEM parameters (A5, A10, and MCF) showed very strong correlations with plasma fibrinogen levels ($r = 0.86, 0.86, 0.85$; $p < 0.0001$), confirming their reliability in detecting hypofibrinogenemia. EXTEM parameters (A5, A10, MCF) exhibited moderate correlations with platelet counts ($r = 0.54–0.57$), but the correlation for EXTEM MCF improved in severe thrombocytopenia (<50 × 10?/L; $r = 0.64$), indicating greater sensitivity in this subgroup. Clinically, 64% of patients with simultaneous reductions in both EXTEM and FIBTEM clot firmness were diagnosed with thrombocytopenia.

Conclusion:

These findings indicate that FIBTEM is a robust predictor of fibrinogen deficiency, while EXTEM provides complementary information on platelet status, particularly in severe thrombocytopenia or when interpreted alongside FIBTEM.

PO72

Early Rotem measurements guide timely hemostatic interventions: correlation of A5 and A10 with MCF

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Unlike conventional coagulation tests, ROTEM provides a rapid, global assessment of hemostatic function within 5–15 minutes, guiding immediate clinical decision-making in bleeding scenarios. Early ROTEM parameters, specifically A5 and A10, have been reported to strongly reflect overall coagulation status, offering the potential to predict hemostatic competence well before traditional clot measurement. This capability is especially important in high-risk clinical contexts, such as trauma resuscitation, cardiac surgery, and liver transplantation, where timely correction of coagulopathy is vital to patient outcomes. Recognizing the potential of early ROTEM parameters to provide rapid insight into coagulation status, this study aimed to evaluate their correlation with MCF across EXTEM, INTEM, and FIBTEM assays in our experience.

Methods:

A retrospective observational study was conducted on patients who underwent ROTEM testing between November 2022 and June 2025 at our institution. The analysis included EXTEM, INTEM, and FIBTEM assays performed to evaluate coagulation status in various clinical settings. For each assay, early ROTEM parameters—A5 (clot amplitude at 5 minutes) and A10 (clot amplitude at 10 minutes)—along with Maximum Clot Firmness (MCF), were systematically extracted from thromboelastometric tracings. All tests were performed according to manufacturer recommendations under standardized laboratory conditions. Pearson correlation coefficients (r) were calculated to determine the strength of association between A5, A10, and MCF for each assay. Correlations were interpreted as very strong when $r > 0.9$.

Results:

A total of 212 patients were included in the analysis, comprising 123 adults (57%) and 89 pediatric patients (43%), with ages ranging from 5 days to 99 years (mean age 26 years). The sex ratio (M/F) was 1.4, corresponding to 58% males and 42% females. ROTEM testing was primarily indicated for the monitoring of cardiovascular surgeries, both adult and pediatric, followed by requests from the Anesthesiology and Intensive Care Unit, mainly for the management of hemorrhagic shock. Across all assays, early ROTEM parameters (A5 and A10) demonstrated a very strong positive correlation with MCF. In EXTEM and INTEM assays, correlation coefficients for A5 and A10 versus MCF consistently exceeded 0.95, reflecting near-linear relationships. The highest correlation was observed in the FIBTEM assay, where both A5 and A10 demonstrated near-perfect concordance with MCF ($r > 0.98$). When stratified by age, both adult and pediatric subgroups exhibited highly consistent correlation profiles between early ROTEM parameters and MCF, indicating that the predictive reliability of A5 and A10 is independent of patient age.

Conclusion:

Early ROTEM parameters A5 and A10 show an excellent correlation with MCF across all assays, confirming their reliability as rapid predictors of clot strength. Their use enables early, targeted hemostatic intervention and supports the implementation of fast, goal-directed transfusion strategies. Integrating these parameters into clinical protocols can markedly improve the timeliness and precision of coagulation management, particularly in cardiac surgery, trauma, and massive bleeding contexts.

PO73

Challenges of anticoagulation in an obese polytrauma patient on antifungals: a case report

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Background/introduction:

Anticoagulation in polytrauma patients can be particularly challenging, especially in the setting of obesity, multiple surgeries, and infectious complications requiring therapies with potential pharmacological interactions.

Material and methods/Case report:

A 34-year-old male with grade III obesity (119 kg), type 2 non–insulin-treated diabetes mellitus, and dyslipidemia was admitted in March 2025 to a tertiary hospital after a motorcycle accident with polytrauma. He underwent multiple surgeries and required prolonged hospitalization (>3 months), followed by 3 weeks of rehabilitation.

He was on prophylactic enoxaparin, dose-adjusted for obesity (60 mg once daily), with periodic peak Anti-Xa monitoring showing values between 0.2–0.5 IU/mL. Renal function remained normal.

Nonetheless, on March 20, about two weeks after starting prophylaxis, he developed left lower limb edema. Doppler ultrasound confirmed a left femoral/popliteal deep vein thrombosis (DVT), related to immobilization and the presence of a left femoral central venous catheter (CVC). The CVC was relocated to the right subclavian vein, and postural drainage plus therapeutic anticoagulation with unfractionated heparin (UFH), adjusted by aPTT, were initiated.

Four days later (March 24), UFH was stopped for plastic surgery. Preoperatively, the aPTT was 34.1s. On the following day (March 25), the patient developed a right upper limb DVT (subclavian vein), associated with the new CVC and perioperative status.

During hospitalization, several infectious complications occurred: right lower limb cellulitis (due to an exposed fracture), Enterococcus faecalis bacteremia, and Candida parapsilosis fungemia related to CVC use, requiring debridement, antibiotics, and prolonged antifungal therapy (anidulafungin 100 mg/day IV). At discharge (July 9, 2025), the patient transitioned to oral fluconazole 400 mg/day and Rivaroxaban 10 mg/day for extended anticoagulation, after completing 3 months of therapeutic anticoagulation.

Given concomitant Fluconazole (a CYP3A4 and P-gp inhibitor), an Immunohematology consultation was requested for DOAC monitoring, due to the risk of drug accumulation from impaired metabolism/elimination. Surprisingly, Rivaroxaban levels were undetectable. Therapy was switched to Apixaban 2.5 mg twice daily, considered more suitable for obese patients. A repeat measurement showed a quantifiable trough level of 48 ng/mL, without signs of accumulation. The peak measurement was disregarded due to blood collection occurring only <2 hours after the trough.

Results/Discussion:

CVC placement in patients with high thrombotic risk should always raise concern for clot formation. Moreover, reduced-dose Rivaroxaban as extended therapy in an obese patient under fluconazole raises concerns regarding anticoagulation efficacy. The Immunohematology consultation proved crucial, as laboratory monitoring revealed the need for therapeutic switch.

Conclusions:

This case highlights that in clinical scenarios where anticoagulation efficacy may be compromised (due to factors such as obesity or drug interactions) individualized management and laboratory monitoring are essential to ensure a more adequate therapy

PO75

Performance of routine coagulation assays in predicting Apixaban level in patient with atrial fibrillation

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Background:

Apixaban is currently used as standard therapy for patients with atrial fibrillation. Although routine coagulation tests such as prothrombin time (PT) and activated partial thromboplastin time (aPTT) are commonly performed in emergency department laboratory evaluations, their ability to predict specific apixaban plasma concentrations remains unclear. Objective: To assess the correlation between routine coagulation assays and apixaban activity measured by chromogenic anti-Xa assay.

Material and methods:

A prospective observational study enrolling patients with non-valvular atrial fibrillation receiving apixaban was conducted. Blood samples were collected at both trough and peak plasma concentrations to measure apixaban activity using a chromogenic anti-Factor Xa assay specifically calibrated for the drug. PT and aPTT were measured simultaneously on an ACL TOP analyzer (Instrumentation Laboratory, IL) using the corresponding HemosIL reagents.Patients were stratified into three groups according to anti-Factor Xa levels: residual (<80 ng/mL), therapeutic (80–200 ng/mL) and supratherapeutic (>200 ng/mL).

Results:

Among the 40 patients with non-valvular atrial fibrillation receiving apixaban, the mean anti-Xa activity was 220 ± 18 ng/mL at peak and 118 ± 21 ng/mL at trough. At peak concentration, the mean PT and aPTT were 52 ± 3.9% and 34.5 ± 0.87 s, respectively. At trough, they were 66 ± 5.8% and 33 ± 0.8 s, respectively. A prolongation of PT and/or aPTT was observed in 75% of patients at peak concentrations, whereas 37% (15 patients) had normal routine coagulation test results at trough concentrations. Based on anti-Xa activity, 9 patients (22.5%) had residual concentrations, 17 (42.5%) were within the therapeutic range and 14 (35%) had supratherapeutic levels. Among patients in the residual group, 5 (55%) showed normal PT and aPTT values corresponding to a sensitivity of 56% and a specificity of 90% for identifying residual apixaban concentrations.In the therapeutic range group, 14 patients (82%) had prolonged PT and/or aPTT. In the supratherapeutic group, all patients (100%) exhibited prolonged PT and/or aPTT results corresponding to a negative predictive value of 100% with a specificity of 31% and a sensitivity of 100%.The correlation analysis demonstrated a strong positive correlation between anti-Factor Xa activity and PT ($r = 0.70$, $p < 0.05$) and a moderate positive correlation with aPTT ($r = 0.47$, $p < 0.05$). Regarding the therapeutic group, the correlation remained moderate for PT ($r = 0.61$, $p < 0.05$) and weaker for aPTT ($r = 0.38$, $p = 0.09$). In the subgroup with residual apixaban levels, PT and aPTT showed a strong correlation with anti-Factor Xa activity ($r = 0.78$, $p < 0.05$; and $r = 0.60$, $p < 0.05$, respectively). Among patients with supratherapeutic levels, the correlation between anti-Xa activity and PT was moderate ($r = 0.65$, $p < 0.05$) and stronger for aPTT ($r = 0.87$, $p < 0.05$).

Conclusions:

Prothrombin time demonstrated a stronger and more consistent correlation with anti-Factor Xa activity compared with aPTT. Normal PT and aPTT values may help exclude supratherapeutic concentrations. however, both assays remain insufficient for accurately estimating drug levels, particularly within the therapeutic range or for ruling out clinically non-relevant concentrations.

PO77

Prevalence and clinical significance of anti- ß2 glycoprotein I antibodies in kidney transplant recipients

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Background:

Anti-phospholipids antibodies, particularly anti-β2 glycoprotein I (anti-β2GPI), are associated with thrombotic events and immune mediated complications. Their frequency appears to be increased among kidney transplant patients suggesting a potential role in adverse post-transplant outcomes such as thrombosis or graft rejection. **Objectives:** This study aimed to determine the prevalence of anti-β2GPI antibodies (IgG and IgA isotypes) before and after transplantation and to evaluate their impact on the occurrence of acute rejection and graft thrombosis.

Methods:

A bicentric case-control study was conducted including renal transplant patients presenting with graft thrombosis (group 1, n=22) or rejection (group 2, n=20) compared to a control group (group 3, n=28) of renal transplant recipients without complications matched for age and sex.

Results:

The three groups were comparable in terms of demographic characteristics, clinical data, and pre-transplant conditions. In the pre-transplantation setting, anti-β2GPI IgG antibodies were detected in 11 patients: 5 from group 1, 5 from group 2 and 1 control. Only one patient from group 2 tested positive for anti-β2GPI IgA antibodies.

After transplantation, anti-β2GPI IgG antibodies were positive in 3, 4, and 2 patients from groups 1, 2, and 3, respectively. Anti-β2GPI IgA were detected in 2 patients from group 2 and 3 patients in the control group. Pre-transplantation anti-β2GPI IgG prevalence was significantly higher in the rejection group than in the control group (p=0.013). After transplantation, this frequency was significantly higher in patients who experienced thrombosis or rejection) than in patients without complications (p=0.038 and p=0.007, respectively). Neither pre- nor post-transplantation anti-β2GPI IgA antibody frequencies were associated with thrombosis or rejection.

Conclusions:

Anti-β2GPI IgG antibodies appear to be involved in renal graft rejection and thrombosis. Pre-transplant screening for these antibodies could help identify at-risk patients and improve the prevention of post-transplant complications.

PO78

Simultaneous onset of stroke and STEMI: diagnostic and therapeutic dilemmas in emergency medicine

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Introduction:

Debate continues over whether post-stroke cardiac dysfunction stems from pre-existing vascular comorbidity, is stroke-induced, or from both. 10–20% of acute stroke patients experiencing severe cardiac events, even without prior heart disease. Cardio-cerebral infarction is an uncommon but serious overlap of acute heart and brain ischemia. Its complex nature and poor outcomes remain challenging due to the absence of unified diagnostic or therapeutic guidelines. We present a clinical case where stroke and cardiac pathology were diagnosed simultaneously.

Patient and methods:

Chart review of the digital patient records.

Case description:

A 74-year-old woman was admitted to the ICU in August 2024 with ST-segment elevations in leads II, III, and aVF within the thrombolysis window from Emergency room. She was conscious, oriented, and pain-free. She experienced before the event head pressure, headache, dizziness, balance loss, inability to stand, and dyspnoea during yoga exercises. Her history includes hypertension, dyslipidaemia, mixed-type asthma, anxiety disorder and sleep apnoea. Medicines: Perindoprilum, Amlodipinum, Indapamidum, Rosuvastatin and Metoprolol.

Since the patient's medical history and the ECG findings were inconsistent, in ICU a CT scan of the brain before routine thrombolysis was performed, as the symptoms and the patient's complaints suggested a neurological pathology.

10.08 CT: Occlusion of the distal vertebral arteries and the origin of the basilar artery. Approximately 50% stenosis of the left internal carotid artery.

Neurologists joint opinion:

Occlusion of the distal segments of the vertebral arteries has not, at this point, resulted in neurological symptomatology. The patient shows no signs of acute cerebral infarction, NIHSS 0.

Multidisciplinary consultation (neurologist, internal medicin, radiologist and anesthesiologist) first found no contraindications for thrombolysis.

Concerns:

- efficacy of thrombolysis on potentially chronic cerebral thrombi and the risk of intracerebral haemorrhage.
- which protocol should be applied for thrombolysis — the cardiac protocol or the cerebral protocol.

Consultation with the tertiary hospital cardiologist, the decision was made to omit thrombolysis and patient was transferred for cardiological intervention.

Patient underwent PCI for distal RCA occlusion; LAD pre-occlusion with collaterals was also noted. The procedure was complicated by hypotension, loss of consciousness, and brief AV block. Subsequently, the patient developed altered mental status and dysarthric speech.

11.08 MRI revealed acute ischaemic lesions in multiple vascular territories, suggesting embolic stroke (NIHSS 4). Initial rehabilitation showed truncal ataxia and broad-based gait with high-frame assistance. By discharge, mobility improved, though supervision remained essential.

Conclusion:

Although insights into pathogenesis may provide answers in the future, during emergencies, viewing pathologies as separate entities can hinder effective decision-making. Acute myocardial

infarction and acute ischaemic stroke are both critical emergencies, and their coexistence markedly worsens prognosis.

PO81

The role of coagulation factors VIII, IX, and XI in pregnancy loss: a case–control study

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Background:

Elevated levels of coagulation factors VIII, IX, and/or XI, combined with the physiological hypercoagulable state of pregnancy, may contribute to early or late pregnancy loss when a comprehensive etiological work-up reveals no abnormalities. The aim of our study was to investigate the association between elevated levels of factors VIII, IX, and XI and pregnancy loss.

Material and methods:

A comparative case-control study was conducted between January 2022 and June 2023 in the hematology laboratory. Patients with at least one early pregnancy loss (EPL) and/or one late pregnancy loss (LPL) who had undergone blood testing for hereditary and/or acquired thrombophilia were included. Coagulation factor assays (VIII, IX, and XI) were performed using a one-step chronometric method on the ACL TOP 500®/IL automated system. Clinical data were collected from patients' medical records. Statistical analysis was performed using SPSS software.

Results:

A total of 60 patients and 60 age-matched controls were included. The median age was 36 years. Ten patients (16.7%) had a personal history of venous or arterial thromboembolic disease. Pregnancy losses were distributed as follows: a single EPL in 30 patients (50%), a single LPL in 9 patients (15%), and recurrent pregnancy loss (RPL) in 21 patients (35%).

The median levels of factors VIII, IX, and XI were 73%, 90.5%, and 87.9%, respectively. Increased levels of factors VIII, IX, and XI were observed in 10%, 6.7%, and 8.3% of cases, respectively, with mean elevated levels of 226.5%, 243.4%, and 257.6%.

The patient group had significantly higher median levels of factors VIII, IX, and XI compared with controls ($p<0.001$). The subgroup of patients with RPL also showed significantly higher levels of these factors than controls ($p<0.001$). The frequency of elevated factor VIII was significantly greater in the patient group ($p=0.027$).

A statistically significant association was also found between levels of coagulation factors VIII, IX, and XI (categorized according to control tertiles) and pregnancy loss ($p<0.001$).

Conclusions:

Our study demonstrated a statistically significant association between pregnancy loss and elevated levels of coagulation factors VIII, IX, and XI. These factors may therefore represent independent risk factors for obstetric complications. Their assessment could be integrated into second-line thrombophilia screening or even considered in antepartum thromboprophylaxis strategies. Large-scale prospective case-control studies are warranted to further clarify these associations.

PO84

Frequency and Clinical Profile of Hereditary Thrombophilia: Factor V Leiden and G20210A Mutations

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Background:

Hereditary muta ons of factor V Leiden and factor II (G20210A) represent the most frequent causes of gene c thrombophilia and cons tute a major risk factor for venous thrombosis. This study aimed to evaluate their prevalence and clinical characteristics in our population. Material and methods: A retrospec ve study conducted over 14 years (2010–2024), including 534 pa ents tested for factor V Leiden and G20210A muta ons. Collected data included age, sex, thromboembolic events, affected vascular territory, obstetric history, and pre-transplant indica ons. Gene c screening was performed by PCR–RFLP on genomic DNA extracted from peripheral blood, using HindIII and MnlI restric on enzymes to detect Factor V Leiden and Prothrombin G20210A (Factor II) mutations, respectively.

Results:

A total of 534 pa ents were included, the majority origina ng from the neurology department (33.3%), followed by internal medicine (15.2%) and pediatrics (14.6%). Mean pa ent age was 34 years [range: 1–82], with a sex ra o of 0.85. The venous system was the most affected territory (44.2%). Recurrent thromboembolic events occurred in 8.2% of pa ents, and 6.4% had a history of obstetric complica ons. The factor V Leiden muta on was found in 13.7% of pa ents, mostly heterozygous (11.2%), affec ng 8.2% of males (mean age 35 years, [13–56]). These pa ents mainly presented with cerebral arterial thromboses, with 5.6% experiencing recurrent events and 0.5% repor ng obstetric history. The factor II (G20210A) muta on was observed in 5.1% of pa ents, mostly heterozygous (2.9%), affec ng 1.7% of females (mean age 32 years, [8–63]). Thromboses were equally distributed between venous and arterial territories (1.45% each). Venous thromboses were predominantly located in cerebral veins and deep veins (DVT), whereas arterial thromboses mainly involved cerebral circula on. Recurrent events occurred in 0.96% of cases, and no obstetric history was reported. Finally, 2% of pa ents presented double heterozygosity for factor V Leiden and factor II mutations.

Conclusions:

Our 14-year experience demonstrates that factor V Leiden and prothrombin G20210A mutations are prevalent hereditary risk factors for thromboembolic events in our population. Their significant association with both venous and arterial thromboses, particularly in young patients and those with recurrent events, highlights the importance of targeted genetic screening.

PO86

aPTT and PT-derived clot waveform parameters as biomarkers of venous thrombosis: a bicentric case–control study

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Background:
Venous thrombosis results from an imbalance between procoagulant and anticoagulant mechanisms. Routine coagulation tests, such as activated partial thromboplastin time (aPTT) and prothrombin time (PT), measure clotting time but do not fully capture clot formation dynamics. Clot Waveform Analysis (CWA), using first and second derivative curves of aPTT and PT, provides additional insights into coagulation kinetics. This study aimed to evaluate the value of PT and aPTT derivative analysis in patients with venous thrombosis compared with healthy participants.

Materials and methods:
This bicentric case–control study included patients with venous thrombosis from Charles Nicolle Hospital (Group 1, n=30) and La Rabta Hospital, Tunis (Group 2, n=50), compared to age- and sex-matched healthy controls (n=50), over a one-year period. PT and aPTT were measured, and clot waveform derivatives (Max1, Max2, Min2) were obtained using the ACL TOP analyser. Data were processed in Microsoft Excel for statistical analysis.
Exclusion criteria: samples with incomplete or missing historical data.

Results:
The mean age of patients was 41 years (range 9–70) for Group 1 and 39 years (range 1–67) for Group 2, compared with 33 years (range 22–62) for healthy participants. Female-to-male ratios were 1.64, 1.38, and 2.57, respectively.
Mean PT values were 14.62 s (Group 1), 12.50 s (Group 2), and 12.36 s (controls). Mean aPTT values were 32.6 s (Group 1), 29.6 s (Group 2), and 30.64 s (controls). Prolonged aPTT was observed in 17.2% of patients in Group 1 (n=5) and 4% in Group 2 (n=2), while reduced PT was observed in 20.7% of patients in Group 1 (n=6).
Mean peaks of the first and second derivatives of aPTT (Max1, Max2, Min2) and PT (Max1) were higher in patients with venous thrombosis. For aPTT: Max1 values were 289.2 ±132.2, 223.8 ±110.6, and 170.7 ±38.1 mAbs/s, Max2 values were 1015.1 ±449.9, 751.7 ±350.4, and 605.2 ±135.9 mAbs/s², Min2 values were -500.5 ±185.1, -327.9 ±176.2, and -280.5 ±70.6 mAbs/s² for Groups 1, 2, and controls, respectively. PT Max1 peaks were 408.1 ±221.1, 339.0 ±166.3, and 267.8 ±56.4 mAbs/s for the same groups.
In Group 1, 63.3% of patients (n=19) were receiving anticoagulants at the time of analysis. Among these patients, aPTT derivative peaks were lower compared with untreated patients (Max1: 259.1 ±99.8 vs 316.3 ±155.1 mAbs/s; Max2: 889.8 ±370.6 vs 1142.5 ±503.9 mAbs/s²), as were PT derivative peaks (Max1: 350.9 ±158.6 vs 468.7 ±256.7 mAbs/s).

Conclusion:
Clot Waveform Analysis (CWA) constitute a promising approach to refine coagulation assessment. By providing dynamic information on clot formation, it may contribute to a better understanding of hypercoagulable states. Larger studies are, however, needed to confirm these findings and clarify the diagnostic value of CWA.

PO87

Clinical evaluation of routine coagulation tests for estimating rivaroxaban activity in atrial fibrillation patients

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Background:
Rivaroxaban is currently recommended as first-line therapy for non-valvular atrial fibrillation (NVAF). Despite its predictable pharmacological profile, assessment of anticoagulant activity may be required in specific clinical situations. Moreover, intra- and inter-individual variability in rivaroxaban concentrations has been reported in the literature. Therefore, the development of reliable tools capable of estimating its anticoagulant effect is warranted, particularly in emergency settings such as hemorrhage or urgent surgical intervention.

Aim:
This study aimed to investigate the relationship between rivaroxaban anti-Xa activity and routine coagulation parameters (prothrombin time (PT) and activated partial thromboplastin time (aPTT)) measured at trough and peak concentrations.

Methods:
This prospective observational study enrolled patients with NVAF receiving rivaroxaban. Blood samples were collected at both trough and peak concentrations to assess rivaroxaban activity using a chromogenic anti-Factor Xa assay specifically calibrated for the drug. PT and aPTT were measured simultaneously. All coagulation assays were performed on an ACL TOP analyzer (Instrumentation Laboratory, IL) using the corresponding HemosIL reagents. Patients were categorized into three groups according to their anti-Factor Xa levels: residual (<125 ng/mL), therapeutic (125–375 ng/mL), and supratherapeutic (>375 ng/mL).

Resultats:
A total of 35 patients were included. The mean peak and trough concentrations of rivaroxaban were 188 ± 23 ng/mL and 90 ± 12 ng/mL, respectively. At peak concentration, the mean PT and aPTT were 58 ± 9 % and 34.8 ± 0.8 s, respectively. At trough concentrations they were 70 ± 3.7 % and 32.7 ± 7.8 s, respectively. Prolongation of PT and/or aPTT was observed in 85% of patients at peak concentrations whereas 21% of patients had normal routine coagulation tests at trough concentrations. Based on anti-Xa levels, 13 (37%) patients were classified in the residual group, 17 (48%) in the therapeutic group and 5 (14%) in the supratherapeutic group. In the residual group, 5 (38%) patients had normal PT and aPTT values. All patients in both the therapeutic and supratherapeutic groups exhibited prolonged PT and/or aPTT values. Thus, Prolongation of PT and/or aPTT demonstrated a sensitivity of 100% and a specificity of 17% for detecting supratherapeutic rivaroxaban levels with a negative predictive value of 100%. A strong positive correlation was found between anti-Factor Xa activity and both PT and aPTT) (r = 0.8 ; $p < 0.05$). In the therapeutic group, the association with PT and aPTT was moderate (r = 0.57, $p < 0.05$; r = 0.49, $p < 0.05$, respectively). Among individuals with residual rivaroxaban activity, correlation with anti-Xa activity was excellent with PT (r = 0.87, $p < 0.05$) and moderate with aPTT (r = 0.48, $p < 0.05$). In the supratherapeutic group, PT maintained a moderate correlation (r = 0.56, $p < 0.05$) while the association with aPTT was attenuated (r = 0.30, $p <$ 0.05).

Conclusion:
Prothrombin time demonstrates a more consistent association with rivaroxaban anti-Xa activity than aPTT. However, neither test can reliably estimate therapeutic or residual plasma concentrations. Drug-specific calibrated anti-Xa assays remain the gold standard for assessing rivaroxaban level particularly in emergency or perioperative clinical decision-making.

PO88

Clinical profile of HIT and correlation with the 4T score

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Background:
Heparin-induced thrombocytopenia (HIT) is a potentially severe immune-mediated complication, and early recognition is crucial to prevent associated thromboembolic events. The 4T score is the reference tool for assessing the pretest probability of HIT. However, the correlation between the 4T score and the positivity of confirmatory immunological tests remains debated. This study aimed to describe the clinical and biological characteristics of patients with suspected HIT and to evaluate the concordance between the 4T score and anti-PF4 antibody positivity.

Matériels et méthodes:
We conducted a retrospective study over a 4-year period (January 2022 – September 2025), including all patients with suspected heparin-induced thrombocytopenia (HIT) for whom anti-PF4 antibodies were measured. The 4T score was systematically calculated to assess the pretest probability of HIT. Screening for anti-PF4/heparin antibodies was performed using an immunoturbidimetric assay (HemosIL® HIT-Ab, ACL TOP analyzer, Werfen®). A result ≥1?**U/mL** was considered positive.

Results:
A total of 78 heparin-exposed patients were included, with a mean age of 56 years and a male-to-female ratio of 1:1. Most patients were admitted to the medical intensive care unit (55.7%) or postoperative critical care (7.6%), followed by the surgical ICU (8.9%), nephrology (5.1%), and neurology and internal medicine units (3.8% each). Unfractionated heparin (UFH) was administered in 46 patients (59%), while low-molecular-weight heparin (LMWH) was used in 32 (41%). Thrombocytopenia was observed in 32 patients (40.5%), and thrombotic complications occurred in 21 patients (26.6%). The 4T score was low (0–3) in 10.3% of cases, intermediate (4–5) in 62.8%, and high (6–8) in 26.9%. Anti-PF4 antibody testing was performed in 45 patients (57%), of whom 13 (28.9%) were positive and 32 (71.1%) negative. Among antibody-positive patients, 61.5% had an intermediate 4T score, 23.1% a high score, and 15.4% a low score. The positive predictive value (PPV) of the 4T score was 50%, the negative predictive value (NPV) 41.7%, with a false-negative rate of 11%.

Conclusion:
Thrombocytopenia is often multifactorial in critically ill patients, which can falsely elevate 4T scores. This study highlights the limitations of the 4T score in the ICU and emphasizes the need for adapted or alternative scoring systems, alongside anti-PF4 antibody testing, to improve HIT diagnosis and patient management in the ICU.

PO89

Clinical and Thrombotic Risk Profiles in Patients with HFpEF: Preliminary Insights from a Real-World Cohort

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Background:
Heart Failure with Preserved Ejec on Frac on (HFpEF) represents nearly half of all heart failure cases and is strongly linked to metabolic comorbidi es and systemic inflamma on. Thrombo c risk, par cularly in pa ents with atrial fibrilla on (AF), a history of ischemic events or elevated inflammatory or coagula on markers, plays an important role in prognosis, yet remains poorly understood in real-world HFpEF populations. This study aimed to describe the clinical and thrombotic profiles of HFpEF patients from a real-world cohort.

Materials and Methods:
A retrospec ve observa onal study was conducted including 30 pa ents hospitalized with chronic heart failure since September 2018. Clinical, biochemical and outcome data were collected, including demographics, comorbidi es (hypertension, diabetes, dyslipidemia, obesity, AF, prior thromboembolic events), inflammatory markers (CRP) and use of an thrombo c therapy. To characterize the population and identify thrombotic risk factor prevalence, descriptive analysis was performed.

Results:
The median age was 83 years, with a predominance of females (63%). Hypertension was present in 97% of pa ents, diabetes in 57% and dyslipidemia in 60%. Obesity was documented in 43% of cases. AF was highly prevalent (70%) and 17% had a history of thromboembolic events, such as ischemic stroke. An coagulant therapy was prescribed in 60% of the cohort, mainly in those with AF. Despite this, in-hospital mortality remained high (90%). Elevated CRP (>5 mg/L) was observed in 52% of tested pa ents, sugges ng an underlying inflammatory component.

Conclusions:
This real-world HFpEF cohort reveals a strong overlap between metabolic and thrombotic risk factors, particularly AF and inflammation-related comorbidities. Although many patients were on anticoagulation, outcomes remained poor, highlighting that thrombotic risk in HFpEF goes beyond AF and traditional factors. These findings highlight the need to define better thrombotic phenotypes and to explore risk-adapted preventive strategies.

PO90

Lupus anticoagulant testing in tunisian laboratories

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Background:

The most recent guidelines on lupus anticoagulant (LA) detection have contributed to more uniformity in the performance and interpretation of LA testing. However, many points remain to reconsider as there are many differences between laboratories in the selection of LA tests, reagents, methodological detail, and results interpretation. **Aims:**

Our work aims to study the variability in LA detection practice in tunisian laboratories for pre, post and analytical aspects of LA detection.

Methods:

A survey questionnaire was formulated according to the ISTH SSC 2019 international survey and covered the same aspects of LA detection in laboratories. The survey was sent to LA tests performers in Tunisian laboratories and respondents were requested to provide their practice and opinions on LA testing.

Results:

The survey was sent to 13 tunisian laboratories that perform LA testing, 9 laboratories responded to the survey. Among the answers there was an agreement on several practices mostly in the preanalatical aspects related to the blood sample and the test principle. Also there was not an important varialbility in test/reagent choice. However a variability was noted in relation to test timing after thrombotic event as 37.5% of practitionners indicated they have no restriction of timing after a thrombotic event, 37.5% decide depending on the clinical situation and 25% defer for at least 12 weeks. For the timing in relation to pregnancy 55.6% agreed on testing at least 6 weeks post pregnancy and 22.2% indicated they have no restriction, yet 22.2% were uncertain about this point.

For anticoagulant treatment there was generally an agreement on choices of test timing for patients on vitamine K antagonists or heparin; however for direct oral anticoagulants (DOAs) 44.4% indicated that they don't accept patients on DOAs and the other answers were variable, 22.2% accept a treatment interruption for 48 hours before testing, 11.1% suggested the pretreatment of the sample with an antidote and 11.1% indicate the test may be performed only with an adapted method.

There was also a difference and an uncertainty among respondents on the definition of cut-off values and interpretation of results in some clinical contexts. For the normalization of clotting times 55.6% of answers indicated that it should be derived using a geometric mean of healthy donors, 33.3% used a pooled normal plasma (PNP) in the same run as patient tests and 11.1% use an arythmetic mean of healthy donors. As for the calculation of normalized ratios 75% agreed on the use of a PNP prepared in-house. The survey also included examples of clinical contexts and they showed a divergnce in decisions and interpretation among answers.

Conclusions:

The results of our study show the interest of practitionners in the standardisation of LA diasgnosis practices and illustrate the points of divergence between laboratories.These results will allow the discussion and generation of standardized guidelines and more uniformity and efficiency in LA testing.

PO91

Pre-biopsy hemostatic profile, hypercoagulability, and hemorrhagic risk in renal biopsy: focus on von willebrand factor

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Backround:
Percutaneous renal biopsy (PRB) is an invasive diagnostic procedure associated with a significant risk of hemorrhagic complications. Pre-biopsy assessment typically relies on routine coagulation tests (prothrombin time, activated partial thromboplastin time, platelet count, and fibrinogen), but their ability to accurately predict bleeding risk remains limited. Von Willebrand factor (vWF), often elevated in inflammatory states or during endothelial activation, may influence hemostatic mechanisms; however, its potential role as a predictive marker for post-biopsy bleeding has not been established.
This study aimed to evaluate the pre-biopsy hemostatic profile, including von Willebrand factor, in patients undergoing percutaneous renal biopsy and to determine its predictive value for post-procedural hemorrhagic complications.

Materials and methods:
We conducted a prospective, single-center study including adult patients scheduled for percutaneous renal biopsy at Charles Nicolle Hospital between June and September 2025. Pre-biopsy hemostatic tests—including prothrombin time (PT), activated partial thromboplastin time (aPTT), fibrinogen, platelet count, D-dimers, von Willebrand factor, and factor VIII—were performed on the ACL-TOP analyzer, and data were processed using Excel. Patients were followed clinically and biologically for 48 hours, with complete blood count (CBC) and renal ultrasound at 24 hours, to monitor for hemorrhagic complications.

Results:
The study included 27 patients who underwent percutaneous renal biopsy, with a mean age of 41 ± 14.9 years (range 23–75) and a male-to-female ratio of 1.5. Pre-biopsy hemostatic parameters showed a mean aPTT of 30.3 s, a mean PT of 11.3 s, a mean fibrinogen level of 4.2 g/L (elevated in 48.1?% and decreased in 3.7?% of patients), and mean D-dimers of 3.56 ± 6.12 µg/mL (elevated in 70.4?%). Mean values for von Willebrand factor (VWF) antigen, VWF activity, and factor VIII were 239.0 ± 1.25?%, 161.9 ± 0.57?%, and 236.5 ± 0.84?%, respectively.
Following biopsy, 7 patients (26?%) developed a hematoma, including 3 (11?%) with macroscopic hematuria. Among these seven patients, 6 had at least one elevated pre-biopsy hemostatic parameter, including fibrinogen, D-dimers, von Willebrand factor, or factor VIII.

Conclusion:
Bleeding risk in patients undergoing percutaneous renal biopsy is multifactorial. Even with a hypercoagulable profile, hemorrhagic complications can occur, highlighting the need for global hemostasis testing and inflammatory marker assessment to better guide pre- and post-biopsy management.

PO92

Thromboprophylaxis in medical inpatients: current practices requiring clarification and need for action

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Background:
Venous thromboembolism (VTE) remains a major public health concern worldwide. The practice guidelines periodically developed by the American College of Chest Physicians (ACCP) are widely regarded as the international standard. However, thromboprophylaxis continues to be misused. This study aimed to evaluate the practice of thromboprophylactic measures for VTE in a medical setting.

Material and methods:
We conducted a prospective descriptive study in our Internal Medicine department over a one-month period, from April 28 to May 31, 2025. Data were collected from prescription charts of hospitalized medical patients, using Padua score to assess their risk of venous thromboembolism.

Results:
A total of 100 prospective patients were included (51 men and 49 women), with a mean age of 54.15 years. The most frequently identified risk factors were the use of hormonal or contraceptive treatments (28%) and prolonged bed rest (15%). Among 19 patients eligible for thromboprophylaxis, 15 effectively received treatment, yielding an adherence rate of 79%, whereas 60% of low-risk patients received prophylaxis without formal indication. Overall, 64% of patients benefited from preventive treatment, with predominantly Low Molecular Weight Heparin (98.4%), and the association of physical methods (early mobilization, elastic compression stockings) in 39.1% of cases. These discrepancies are related to the clinician awareness and some non-updated old “false ideas”.

Conclusions:
These findings highlight an overuse of thromboprophylaxis among low-risk patients and an underuse among high-risk patients, reflecting heterogeneity in risk assessment and adherence to recommendations. They emphasize the need to strengthen the systematic use of validated risk scores as decision-making tools, to promote an individualized, balanced prevention strategy in accordance with actualized recommendations, and based on the clinical judgement and the experience of the clinician.

PO93

Obstetrical antiphospholipid syndrome in a Moroccan cohort: a monocenter study

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Background:

Obstetrical antiphospholipid syndrome (OAPS) is a rare but serious autoimmune disease, responsible for recurrent pregnancy complications such as early or late miscarriages, intrauterine fetal death, and preterm delivery, often in association with severe preeclampsia or intrauterine growth restriction. It is defined by the association of these manifestations with the persistent presence, confirmed at least twelve weeks apart, of antiphospholipid antibodies including anticardiolipins, anti-beta-2-glycoprotein I, and lupus anticoagulant. This study aims to describe the clinical and therapeutic profile of patients with OAPS in a Moroccan setting.

Materials and methods:

We conducted a retrospective descriptive and analytical study on twenty patients managed in our Internal Medicine Department over an eight-year period, from January 2016 to December 2024, all of whom fulfilled the 2006 revised Sapporo classification criteria for antiphospholipid syndrome. Data collected included sociodemographic characteristics, obstetrical and thromboembolic history, extra-obstetrical manifestations, immunological profile, therapeutic approaches, and outcomes. We also aim to compare the diagnosis performance of 2006 revised Sapporo criteria and 2023 ACR/EULAR criteria.

Results:

The mean age of our patients was 35 years. All patients had a history of miscarriage, with an average of three episodes, mainly early. Intrauterine fetal death occurred in half of the cases and preterm deliveries in one fifth, often linked to severe preeclampsia or eclampsia. Extra-obstetrical manifestations included venous thrombosis, arterial thrombosis, cutaneous involvement such as livedo, as well as neurological and cardiac complications. Lupus anticoagulant was positive only in four patients. Anticardiolipins and anti-beta-2-glycoproteins were found at moderate levels, with frequent multi-positivity in majority of cases. Treatment regimens primarily involved anticoagulation, often requiring bridging with heparin, and included hydroxychloroquine, or immunoglobulins, with overall favorable outcomes despite a persistent risk of recurrence. When applied to our cohort, the 2023 ACR/EULAR criteria demonstrated lower sensitivity compared with the 2006 revised Sapporo criteria, indicating that several patients fulfilling the Sapporo criteria were not captured by the newer classification system.

Conclusion:

Obstetrical antiphospholipid syndrome remains a condition with significant maternal and fetal impact. Early diagnosis based on more pertinent risk stratification with clinic-immunological profile, and a multidisciplinary management are essential to reduce complications and improve their severe prognosis.

Table 1 : Distribution of Clinical and Biological Domains and Scoring According to the 2023 ACR/EULAR Classification Criteria in our Cohort

Clinical and biological domain		2023 ACR/EULAR Scoring	Number of patients presenting with the symptom	Number of patients presenting without the symptom
Obstetric	≥3 Consecutive pre-foetal (<10w) and/or early foetal (10w 0d-15w 6d) deaths	1 point.	12	8
	Foetal death (16w 0d-33w 6d) in the absence of PEC with severe features or PI with severe features		10	10
	PEC with severe features (<34w 0d) or PI with severe features (<34w 0d) with/without foetal death	3 points	2	18
	PEC with severe features (<34w 0d) and PI with severe features (<34w 0d) with/without foetal death	4 points	1	19
Macrovascular	VTE with a high-risk VTE profile	1 point	2	18
	VTE without a high-risk VTE profile	2 points	9	11
	AT with a high-risk CVD profile	2 points	0	20
	AT without a high-risk CVD profil	4 points	3	17
Microvascular	Suspected	2 points.	12	8
	Established	5 points.	0	20
Cardiac valve	Thickening	2 points	2	18
	Vegetation	4 points	1	19
Haematology	Thrombocytopenia	2 points	6	14
Lupus anticoagulant (LAC)	Positive LAC (single-one time)	1 point	10	10
	Positive LAC (persistent)	5 points	10	10
aCL / aβ2GPI antibodies	Moderate or high positive (IgM) (aCL and/or anti-β2 GPI)	1 point	5	15
	Moderate positive (IgG) (aCL and/or anti-β2 GPI)	4 points	10	10
	High positive (IgG) (aCL or anti-β2 GPI)	5 points	0	20
	High positive (IgG) (aCL and anti-β2 GPI)	7 points	1	19
Diagnostic	Number of patients diagnosed with APS according to the both criteria		11 patients	9 patientes

AT : Arterial Thrombosis, **CVD**: Cerebrovascular Disease; **LAC**: Lupus Anticoagulant, **PEC** : Preeclampsia, , **PI**: Placental Insufficiency **VTE**: Venous Thromboembolism,

PO94

Acute management of vascular stroke: experience report of monocentric private expert center in Morocco

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Introduction:

The treatment of Acute Ischemic Stroke (AIS) remains very challenging mainly in Morocco concerning the onset of therapy including thrombolysis, mechanical thrombectomy and bridging. We report here the experience of our expert center analyzing the reasons for a delayed treatment as well as its consequences in terms of clinical outcomes.

Materials and methods:

We performed a retrospective, single-center study between December 2019–December 2024 including all AIS admitted in our neurovascular expert center (n=240). Stratification was performed using various median time criteria from symptom onset to treatment start: group A <4h30, group B 4h30–7h, group C 7h–24h, group D >24h. We analyzed treatment allocation, reperfusion rates, bleeding complications, early mortality, and longitudinal NIHSS (1 week, 1–3–6 months, 1 year), with additional review of organizational and geographical factors.

Results:

Early patients arrival groups A–B were mainly associated with integrated on-site imaging, short distance to the stroke center. Late care groups C–D correlated with off-site imaging (fragmented flow), long distance/inter-hospital transfers, atypical/posterior vascular network and socio-economic constraints. Comorbidities and logistics further skewed patients toward groups C–D. Logically, the earlier was onset of treatment, the better was the result. Early therapy included intravenous thrombolysis (IVT) (± bridging) was low and predominating in groups A–B. Thrombectomy was more associated to group B and the main option in groups C–D remaining effective 7–24h in imagingselected patients. Reperfusion rates were improved mostly in group A, and remained favorable but incomplete in groups B-C, then dropping dramatically in group D. NIHSS longitudinal trajectories confirm this hierarchy with an improved prognosis mostly in group A, remaining favorable but incomplete in group B, with a reduced severity in group C, and a plateau in group D. Morbimortality was directly correlated with delay of onset to treatment. Delays to treatment start were often due to public awareness, the long distance to reach the center, the lack of imaging access (door to imaging) or medical expertise in local facilities (door to needle or door to puncture) and financial issues with limited reimbursement possibilities.

Conclusion:

Treatment timing directly influences AIS prognosis and clinical outcomes. Our data emphasizes the need for action to enlarge medical access to stroke centers with more facilitated in-hospital workflow for specialized acute optimized management