THE HEMATOLOGY INDICA

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NEWSLETTER

Theme : Unraveling the mysteries of coagulation

CLINICAL AND DIAGNOSTIC CHALLENGES IN THE MANAGEMENT OF ANTIPHOSPHOLIPID ANTIBODY SYNDROME

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Introduction:

Antiphospholipid antibody syndrome (APS) is a systemic autoimmune disorder characterized by recurrent thrombosis, pregnancy morbidity, and a wide spectrum of non-thrombotic manifestations associated with persistently positive antiphospholipid antibodies (aPL). It represents the **most common acquired thrombophilia**, accounting for a substantial proportion of venous thromboembolism (VTE) and ischemic stroke in young individuals. Despite increasing recognition, APS continues to pose diagnostic and therapeutic challenges due to the complexity of its clinical spectrum, the absence of a single gold-standard laboratory test, and evolving classification criteria. This review discusses the essential facts, diagnostic pathways, and current controversies, while highlighting updates from the **2023 ACR/EULAR APS classification criteria**.

Epidemiology and Clinical Relevance

APS affects approximately **2–4%** of the general population, although clinically significant disease occurs in a smaller subset. Antiphospholipid antibodies are seen in **15–20% of patients with deep vein thrombosis**, and nearly **one-third of strokes in patients <50 years** may have an underlying aPL positivity. Low-titer, transient anticardiolipin antibodies (aCL) can appear in up to **10% of healthy individuals**, especially following infections. The prevalence of positive aPL increases with age in association with other autoimmune conditions such as **SLE (10–40%)** and **rheumatoid arthritis (20%)**. Asymptomatic aPL-positive individuals have an annual thrombosis risk of **0–4%**, influenced by the antibody profile and coexisting risk factors.

Defining APS: Evolution of Criteria

APS is fundamentally defined as an autoimmune thrombophilia characterized by:

1. **Vascular thrombosis** – arterial, venous, or small-vessel thrombosis.
2. **Pregnancy morbidity** – recurrent early abortions, fetal death, or premature births due to placental insufficiency.
3. **Non-thrombotic manifestations** – thrombocytopenia, livedo reticularis, cardiac valve disease, neurological deficits.
4. **Laboratory evidence** – persistent presence of lupus anticoagulant (LA), anticardiolipin antibodies, or anti-β2 glycoprotein I (β2GPI) antibodies.

The **Revised Sapporo Criteria (2006)**, also known as the Sydney criteria, require at least **one clinical and one laboratory criterion**, with the laboratory test repeated after **12 weeks** to confirm persistence. These criteria have been widely used for both clinical practice and research.

However, several limitations exist:

- Non-classical manifestations (hematologic, neurologic) are not considered.
- IgM isotypes of aCL/β2GPI contribute minimally to thrombosis risk.
- IgA isotypes, now increasingly linked to APS in SLE, are excluded.
- No consideration of primary vs. secondary disease.
- Complement dysregulation, important in catastrophic APS (CAPS), is not included.
- The 12-week interval can be impractical in acute clinical settings.

2023 ACR/EULAR Classification Criteria: What's New?

To address these limitations, the **2023 ACR/EULAR criteria** introduced a more refined, risk-stratified approach. Key features include:

- **Entry criterion:** At least one positive aPL test within **3 years** of an aPL-related clinical event.
- **Weighted scoring:** Six clinical domains and two laboratory domains, with each item assigned points (1–7).
- **Classification threshold:** At least **3 points each** from clinical and laboratory domains.
- **High specificity and sensitivity:** Approximately **99% specificity** compared to 86% for Sapporo; **84% sensitivity** compared to 99% for Sapporo.

These criteria better reflect current pathophysiological understanding, enable precise classification of homogeneous patient groups, and facilitate research in APS.

Lupus Anticoagulant (LA): Central but Complex

Among laboratory tests for APS, **lupusanticoagulant** remains the strongest predictor of thrombosis. It differs from antibody ELISAs by being a **functional assay**, detecting antibodies that interfere with phospholipid-dependent coagulation reactions. Over **50%** of APS patients may exhibit isolated LA positivity. These patients have a markedly increased risk of myocardial infarction, ischemic stroke, and pregnancy morbidity.

Additionally, LA is essential for identifying **triple positivity**—concurrent LA, aCL, and anti-β2GPI positivity—which conveys the **highest risk** for both first and recurrent thrombotic events.

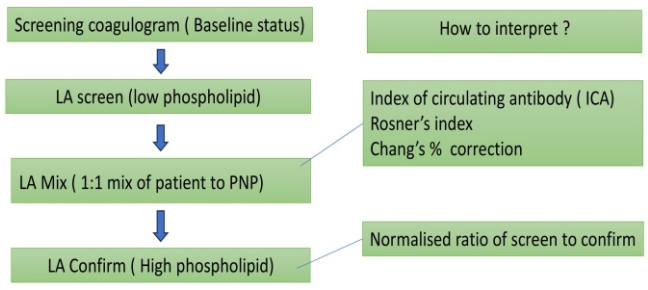
However, LA testing is technically challenging due to numerous confounders, including anticoagulant therapy, acute inflammation, and pregnancy.

Principles of Testing for Lupus Anticoagulant

LA detection follows a systematic **screen–mix–confirm (S-M-C)** algorithm, as endorsed by the ISTH.

1. Screening Tests

Two phospholipid-dependent coagulation assays must be used:



"The art of medicine is long, but life is short." - Hippocrates

INSIDE

- **APTT-based LA screen** – Highly sensitive due to low phospholipid content; begins at the level of Factor XII activation.
- **Dilute Russell's Viper Venom Time (DRVVT)** – More specific; activates Factor X directly, bypassing intrinsic pathway factors.

Using both tests improves diagnostic accuracy, as some LAs prolong only one assay.

2. Mixing Studies

Performed when the screening test is prolonged to distinguish between **factor deficiency** and **inhibitor**.

- Patient plasma is mixed in a 1:1 ratio with pooled normal plasma.
- If clotting time **corrects**, a factor deficiency is likely.
- If it **fails to correct**, a phospholipid-dependent inhibitor such as LA is suspected.

The **Rosner Index** further quantifies correction:

- <12: correction (factor deficiency)
- 12–15: borderline
- >15: presence of inhibitor (e.g., LA)

However, interpretation can be complicated by coexisting factor deficiencies, acute-phase reactants, or strong inhibitors.

3. Confirmatory Tests

Confirmatory assays use the same reagent system but with **excess phospholipids**. Normalization of clotting time confirms **phospholipid dependency** of the inhibitor.

The **screen/confirm ratio** is considered the most robust parameter for LA identification.

Challenges in LA Testing

- Testing should be avoided during acute thrombosis, pregnancy, or inflammatory flare due to false positives/negatives.
- Vitamin K antagonists, direct oral anticoagulants (DOACs), and direct Xa inhibitors significantly interfere with assays.

Only standardized **ELISA** for aCL and β2GPI should be used. Methods like chemiluminescence, magnetic microparticles, or multiplex flow cytometry are not recommended.

Whom to Test and When to Test

LA and aPL testing should be performed **only when clinically indicated**, such as:

- Young patients with unexplained arterial/venous thrombosis
- Recurrent fetal loss or severe preeclampsia
- Thrombosis in unusual sites
- Livedo reticularis, thrombocytopenia, or cardiac valve abnormalities
- Strong family or personal history of thrombosis
- Before discontinuing anticoagulation in unexplained thrombosis

Testing during acute events should be deferred when possible, since acute phase reactants may interfere with results.

Catastrophic Antiphospholipid Syndrome (CAPS)

CAPS is a rare but devastating variant, accounting for <1% of APS cases. It is characterized by:

- **Rapid multiorgan failure**
- **Small-vessel occlusions**
- **Simultaneous or sequential thrombosis in ≥3 organs**
- Frequently triggered by infection, surgery, malignancy, or withdrawal of anticoagulation.

Pathogenesis involves a "thrombotic storm" driven by complement activation, endothelial injury, and cytokine release.

Differential diagnosis of CAPS includes:

- Thrombotic microangiopathies (TTP, HUS)
- DIC
- Sepsis with multiorgan failure
- Heparin-induced thrombocytopenia
- Vasculitides
- Severe preeclampsia/HELLP

Treatment of CAPS

Management must be aggressive and is often lifesaving:

1. **Anticoagulation** – Cornerstone of therapy (usually heparin).
2. **High-dose corticosteroids** – Reduce inflammatory and thrombotic pathways.
3. **Plasma exchange** – Removes antibodies and inflammatory mediators.
4. **IV Immunoglobulin** – Immune modulation in refractory cases.
5. **Rituximab or Eculizumab** – For resistant or complement-mediated disease.
6. **Treatment of underlying triggers** – infections, surgery, etc.

Early recognition and timely treatment significantly reduce mortality.

Non-Thrombotic Manifestations of APS

Although thrombosis and pregnancy morbidity form the core of diagnostic criteria, APS presents with many non-criteria manifestations, including:

- Thrombocytopenia
- Hemolytic anemia
- Livedo reticularis
- Libman–Sacks endocarditis
- Seizures, chorea, cognitive dysfunction
- Nephropathy with small vessel occlusion

These features often provide early clues and may precede overt thrombosis.

Interpretation of Antibody Profiles

The risk of thrombosis varies significantly across antibody combinations:

- **Triple positivity (LA + aCL + β2GPI)** → highest risk
- **Isolated LA positivity** → significant risk
- **Isolated aCL or β2GPI IgM** → minimal risk
- **IgA isotypes** → emerging evidence supports inclusion, especially in SLE

Risk assessment must incorporate other thrombophilic factors such as smoking, hypertension, immobilization, infections, and estrogen exposure.

Therapeutic Approaches in APS

1. Thrombotic APS

Long-term **anticoagulation** remains the mainstay.

- **Vitamin K antagonists (VKA)** such as warfarin remain standard.
- Target INR usually **2–3**; some recommend **3–4** for recurrent arterial events.
- DOACs are **not recommended** in high-risk patients, especially with triple positivity.
- Aspirin may be added in high-risk arterial events.

2. Obstetric APS

Treatment aims to improve placental function:

- Low-dose aspirin
- Prophylactic or therapeutic LMWH
- Close obstetric surveillance

3. Asymptomatic aPL-positive individuals

Management is individualized based on risk profile:

- No routine anticoagulation
- Consider aspirin in high-risk antibody patterns
- Strict control of cardiovascular risks

Future Directions and Challenges

APS remains an area of active investigation. Key challenges include:

- Lack of a single gold-standard diagnostic test
- Variability in laboratory assays
- Limited data on long-term duration and intensity of anticoagulation
- Uncertainty about DOAC use in selected patients
- Need for biomarkers beyond conventional antibodies
- Understanding complementopathies and NETosis in CAPS
- Standardizing IgA aPL testing
- Incorporating non-criteria manifestations into formal criteria

The 2023 ACR/EULAR criteria mark a major step toward better disease stratification but require widespread validation in diverse populations.

Conclusion

APS is a potentially treatable but often underdiagnosed autoimmune thrombophilia with heterogeneous clinical manifestations. Diagnosis requires a high index of suspicion, careful clinical-laboratory correlation, and awareness of the limitations of existing assays. Lupus anticoagulant testing remains central, but interpretation must consider anticoagulation status, acute phase reactions, and assay variability. Catastrophic APS represents a medical emergency requiring prompt and aggressive multimodal therapy.

As knowledge evolves, particularly with insights into complement pathways and newer classification systems, clinicians must remain updated to optimize patient outcomes. Ultimately, individualized risk assessment and judicious use of anticoagulation form the cornerstone of long-term management in APS.

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"Medicine is a science of uncertainty and an art of probability." - William Osler

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Thrombotic Thrombocytopenic Purpura: a review of pathophysiology and laboratory diagnosis

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Introduction

Thrombotic Thrombocytopenic Purpura (TTP) is a fulminant hematological disorder. characterized by systemic microvascular thrombosis, leading to consumptive thrombocytopenia, microangiopathic hemolytic anemia (MAHA), and variable signs of organ ischemia, particularly affecting the brain, heart, and kidneys (1). First described by Eli Moschcowitz in 1924 (2), TTP was historically associated with a pentad of clinical features: MAHA, thrombocytopenia, neurological abnormalities, renal dysfunction, and fever. However, the current diagnostic criteria emphasizes the presence of MAHA and thrombocytopenia without an alternative apparent cause to prompt urgent investigation and management (3).

The seminal discovery in the late 1990s identified severe deficiency of ADAMTS13 (A Disintegrin And Metalloproteinase with ThromboSpondin type 1 repeats, member 13), a von Willebrand factor (VWF)-cleaving metalloprotease, as the central pathogenetic mechanism in most TTP cases (4).

Pathophysiology of TTP

ADAMTS13 is a zinc-containing metalloprotease predominantly synthesized in hepatic stellate cells (7). Its primary known substrate is VWF. VWF is a large multimeric glycoprotein synthesized by endothelial cells and megakaryocytes, playing a crucial role in primary hemostasis (8).

Endothelial cells secrete VWF as unusually large multimers (ULVWF). These ULVWF multimers are highly prothrombotic due to their increased capacity to bind platelets. Under normal physiological conditions, ADAMTS13 cleaves ULVWF multimers within the A2 domain, specifically at the Tyr1605-Met1606 bond. This cleavage process is shear-dependent; high fluid shear stress, as encountered in the microcirculation, unfolds the VWF molecule, exposing the A2 domain and making it accessible to ADAMTS13 (9).

Consequences of Severe ADAMTS13 deficiency

In TTP, severe deficiency of ADAMTS13 activity (<10% of normal) disrupts this critical regulatory mechanism (7). The inability to cleave ULVWF leads to its persistence and accumulation on endothelial surfaces and in circulation. These hyper-adhesive ULVWF multimers spontaneously bind to platelets, particularly under the high shear stress conditions prevalent in arterioles and capillaries, initiating the formation of platelet-rich microthrombi (9). These microthrombi are characteristic of TTP and are composed primarily of VWF and platelets.

The microthrombi cause partial or complete occlusion of small vessels in various organs, leading to MAHA, consumptive thrombocytopenia and organ ischaemia and dysfunction (10,11). Reduced blood flow due to microvascular occlusion leads to tissue hypoxia and organ damage. The brain, heart, kidneys, pancreas, and adrenal glands are commonly affected, manifesting as neurological symptoms (headache, confusion, seizures, focal deficits), cardiac involvement (arrhythmias, myocardial infarction), renal impairment (proteinuria, hematuria) and abdominal pain (11) (Figure 1).

Types of TTP and mechanisms of ADAMTS13 deficiency

Immune-mediated TTP (iTTP): It is the most common form, particularly in adults, and results from the development of autoantibodies (primarily IgG, IgM and IgA rarely) directed against ADAMTS13 (12). These autoantibodies can inhibit ADAMTS13 activity or accelerate its clearance.

The triggers for autoantibody production in iTTP are not always clear but can be associated with various factors, including infections (e.g., HIV, influenza), drugs (e.g., ticlopidine, clopidogrel, quinine etc), pregnancy, autoimmune disorders (13). Female sex and African ancestry are recognized risk factors, and certain HLA class II alleles (e.g., HLA-DRB1*11) have been associated with an increased susceptibility to developing iTTP.

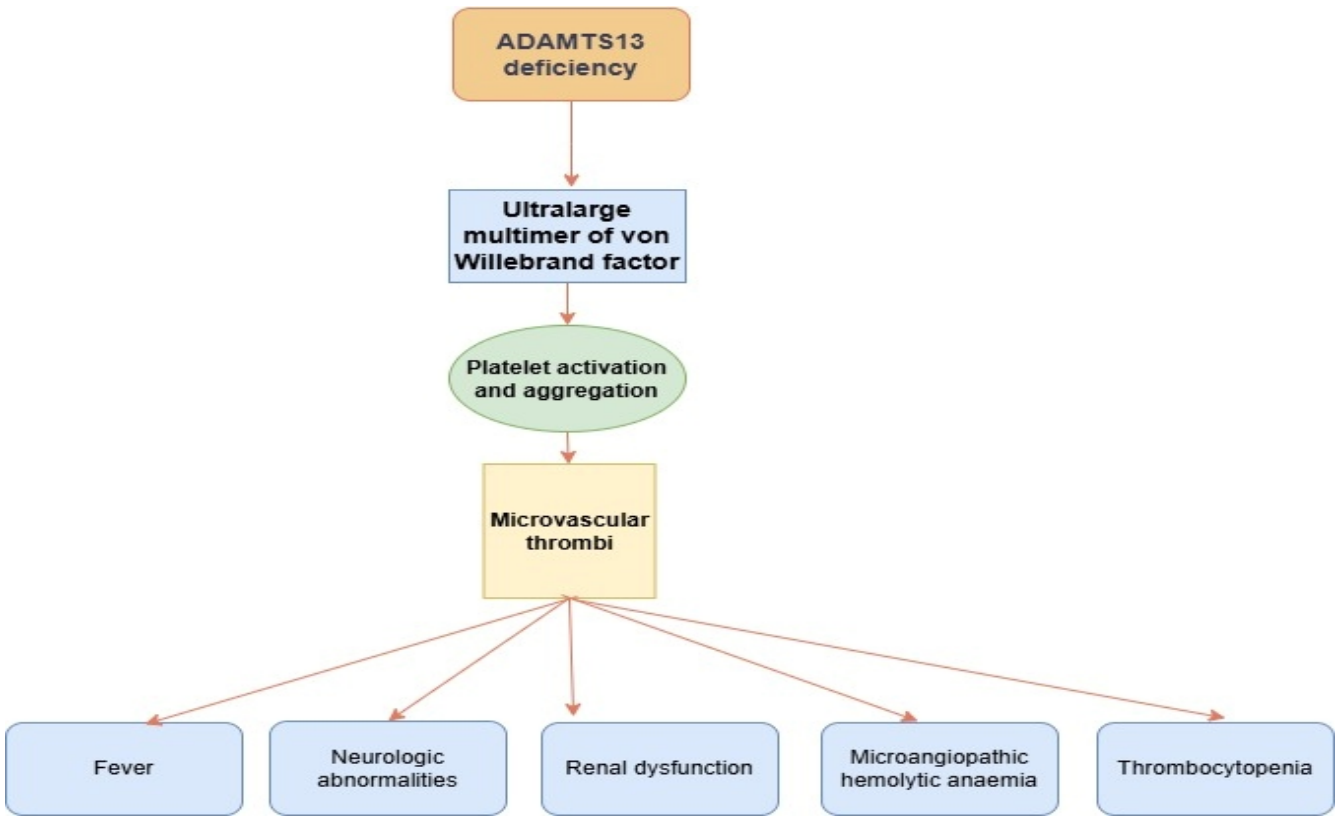
Congenital TTP (cTTP) / Upshaw-Schülman-syndrome cTTP is a rare autosomal recessive disorder caused by biallelic mutations in the *ADAMTS13* gene (14). Over 200 mutations have been identified, leading to severely reduced synthesis or secretion of functional ADAMTS13 protein. Patients with cTTP typically have lifelong severe ADAMTS13 deficiency (usually <5-10% activity) and an absence of ADAMTS13 inhibitors (15).

The clinical presentation of cTTP is heterogeneous. Some individuals present in the neonatal period are with severe jaundice, thrombocytopenia, and MAHA, while others may remain asymptomatic until childhood or even adulthood, with acute episodes often precipitated by triggers such as infections, surgery, or pregnancy (14).

Laboratory diagnosis of TTP

A prompt and accurate laboratory diagnosis is crucial for initiating life-saving treatment in TTP. The diagnostic process involves initial screening tests to identify MAHA and thrombocytopenia, followed by specific assays to confirm severe ADAMTS13 deficiency (16-18).

- **Complete blood Count (CBC) and Peripheral blood smear (PBS):**
  - **Thrombocytopenia:** Typically severe (platelet count <30 x 109/L), although TTP can occur with higher platelet counts.
  - **Schistocytes:** Their presence on the PBS is a hallmark of MAHA. A significant number (e.g., >1% of red cells) is usually expected (19).



"The best way to understand disease is to study the patient, not the disease." - William Osler

- **Markers of hemolysis:** Like lactate dehydrogenase, unconjugated bilirubin, and reticulocyte count will be elevated (20).

Coagulation Studies:

- **Prothrombin Time (PT), Activated Partial Thromboplastin Time (aPTT):** Typically normal or near normal. This helps differentiate TTP from DIC, where these are usually prolonged (21).
- **Fibrinogen:** Usually normal or even elevated (as an acute phase reactant).
- **D-dimer:** May be mildly to moderately elevated but generally not to the levels seen in florid DIC (21).

Confirmatory Testing: ADAMTS13 Assays

Demonstration of severe ADAMTS13 deficiency is the cornerstone for confirming the diagnosis of TTP (5,32). Blood samples for ADAMTS13 testing should ideally be drawn *before* the initiation of plasma exchange (PEX) or administration of plasma products. However, treatment should *not* be delayed while awaiting ADAMTS13 results if clinical suspicion is high.

- **ADAMTS13 Activity Assay:**
  - Measures the functional ability of ADAMTS13 to cleave its VWF substrate.
  - Severe deficiency, typically defined as activity <10% of normal (or <10 IU/dL), is diagnostic of TTP in the appropriate clinical context (22,23).
  - Various methodologies are available, including:
    - **Immunological methods with functional readout:** ELISA-based assays using full-length VWF or VWF fragments (e.g., VWF73, a recombinant 73-amino acid VWF fragment) as substrate, followed by detection of cleavage products (24).
    - **Fluorescence Resonance Energy Transfer (FRET) assays:** Utilize a synthetic VWF peptide substrate labelled with a FRET pair (e.g., FRETS-VWF73). Cleavage separates the pair, resulting in a measurable fluorescent signal (25).
    - **Chromogenic assays:** Often automated, measuring cleavage of a VWF fragment (26).
- **ADAMTS13 Inhibitor (Autoantibody) Testing:**
  - Performed if ADAMTS13 activity is severely deficient, to differentiate between iTTP and cTTP.
  - **Functional Inhibitor assays (e.g., Bethesda assay or mixing studies):** Patient plasma is mixed with normal plasma (source of ADAMTS13), and residual ADAMTS13 activity is measured. A significant reduction in activity indicates the presence of a functional inhibitor. Results are often reported in Bethesda Units (BU) (27).
  - **Anti-ADAMTS13 antibody ELISA:** Directly detects IgG (and sometimes IgA or IgM) autoantibodies binding to recombinant ADAMTS13. Provides a quantitative measure of antibody levels.
- **ADAMTS13 Antigen Levels:**
  - Measures the concentration of ADAMTS13 protein, typically by ELISA.
  - In iTTP, antigen levels can be low or normal. In cTTP, antigen levels are usually very low or undetectable. Activity and inhibitor assays are generally more informative for acute diagnosis and classification.
- ***ADAMTS13* Gene Mutation Analysis:**
  - Indicated for patients with confirmed severe ADAMTS13 deficiency when:
    - No ADAMTS13 inhibitor is detected.
    - Clinical presentation is suggestive of cTTP (e.g., neonatal onset, childhood presentation, positive family history, recurrent episodes without detectable inhibitors after initial presentation).
    - To confirm the diagnosis of cTTP and for genetic counseling .
  - Involves sequencing the *ADAMTS13* gene to identify pathogenic mutations (28).

Clinical Scoring Systems

To aid in early risk assessment and decision-making before ADAMTS13 results are available, clinical scoring systems have been developed to predict the probability of severe ADAMTS13 deficiency. The commonly used scores are plasmic score and French score. These scores can help clinicians prioritize urgent plasma exchange for high-risk patients but should not replace ADAMTS13 testing (29,30)

Differential Diagnosis

TTP must be differentiated from other TMAs and conditions causing MAHA and thrombocytopenia, as their management differs significantly.

- **Hemolytic Uremic Syndrome (HUS):**
  - **Typical HUS (STEC-HUS):** Often follows diarrheal illness caused by Shiga toxin-producing *Escherichia coli* (STEC) or *Shigella dysenteriae*. Renal failure is typically more prominent than in TTP. ADAMTS13 activity is normal or only mildly reduced (>10-20%) (31).
  - **Atypical HUS (aHUS):** A rare, complement-mediated disorder characterized by genetic or acquired defects in complement regulatory proteins. Severe renal involvement is common. ADAMTS13 activity is normal or mildly reduced (32).
- **Disseminated Intravascular Coagulation (DIC):** Usually triggered by sepsis, trauma, malignancy, or obstetric complications, characterized by widespread microvascular thrombosis and paradoxical bleeding. Laboratory findings include prolonged PT and aPTT, low fibrinogen, markedly elevated D-dimer. Schistocytes may be present but ADAMTS13 activity is generally not severely deficient (33).
- **HELLP Syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets):** Distinguished by significant liver enzyme elevation in third trimester pf pregnancy. ADAMTS13 activity can be moderately reduced in some cases but is not typically <10% (34).

Monitoring During Treatment and Remission

Laboratory parameters are crucial for monitoring response to therapy (PEX, immunosuppressants like corticosteroids and rituximab, and caplacizumab) and detecting relapses:

- Daily monitoring of platelet count, LDH, haemoglobin, and peripheral blood smear for schistocytes during the acute phase.
- Periodic measurement of ADAMTS13 activity and inhibitor titers during and after treatment can help assess the immunological response, guide tapering of immunosuppression, and predict the risk of relapse (35-37).

Conclusion

TTP is a formidable clinical entity rooted in severe ADAMTS13 deficiency, leading to systemic microvascular thrombosis with potentially devastating consequences. Laboratory diagnosis hinges on the prompt recognition of MAHA and thrombocytopenia, followed by confirmatory assays demonstrating severe ADAMTS13 deficiency and, in iTTP, the presence of inhibitors. The availability of ADAMTS13 testing has transformed the diagnostic landscape, allowing for more precise differentiation from other TMAs and guiding targeted therapies.

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Role of Novel anti-Hemophilia agents in Hemophilia A/B

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Hemophilia A (HA) and B are inherited bleeding disorders caused by a deficiency of factor VIII or factor IX, respectively. The administration of recombinant or purified factor remains to be the standard of care. However, this treatment strategy results in a high economic and personal burden to patients, further exacerbated by the development of inhibitors. Apart from the standard factor concentrates, there are extended-half-life (EHL) FVIII and FIX products that offer twice a week {EHL FVIII (half-life [$t_{1/2}$] 1.5-fold of FVIII)} or once a week administration {EHL FIX ($t_{1/2}$ 2.5- to 5-fold of FIX)} frequency.

However, inhibitor development remains a significant complication in the treatment of patients with HA and HB, resulting in bleeding episodes despite factor therapy. This phenomenon occurs with an incidence of 25% to 30% in HA (more common in severe HA than in non-severe HA) [Witmer C et al] and 3% to 5% in HB and even higher in those harbouring the null mutations [Dolan G et al]. Even with attempted immune tolerance induction and immunosuppression, inhibitors recur in up to 30% of HA and 20% of HB patients [Dolan G et al , Antun A et al, Santoro C et al]. Traditionally, hemostatic control in patients with HA/HB that develop inhibitors is achieved with the help of bypassing agents (BPA) such as activated prothrombin complex concentrate (cPCC) or recombinant factor VII (rVII). In retrospective analyses, both these agents have been found to have similar efficacy and side effect profiles. Additional challenge with these agents is that their efficacy cannot be monitored using the standard lab assays with a reported failure rate ranging from 7-11.6% and thrombosis rates between 4-6.5% [Ma AD et al]. Thus, alternative strategies to improve thrombin generation resulting in hemostatic control with less failure rates and better side effect profile have been the focus of research. Among these, 2 broad categories are factor mimetics and rebalancing agents.

FACTOR MIMETICS OR SUBSTITUTION THERAPY

These molecules behave as factor but without actually being the factor itself. This includes Emicizumab (Hemlibra), a humanized, bispecific, monoclonal immunoglobulin G4 antibody that binds to activated FIX (FIXa) and FX, thereby performing the function of FVIII by bringing FIXa and FX into close enough proximity to facilitate FX activation. It was approved for use in the United States in 2017, Europe and Japan in 2018 , and subsequently to India in 2019. It offers advantage of less frequent and flexible dosing, as the half-life is 26.8 days.

Across the HAVEN trials i.e HAVEN1-7, Emicizumab showed excellent results with markedly reduced Annual Bleeding Rates (ABRs) thereby improving target joint health without any major side effects in patients with HA with or without inhibitors [Callaghan MU et al]. Additionally, it is administered subcutaneously, which reduces the need for venous access making it an appealing option for pediatric patients who may have difficulty with frequent intravenous infusions. Furthermore, the extended dosing intervals (weekly, bi-weekly, or every four weeks) further enhance convenience and can improve adherence to prophylactic regimens.

How to prescribe Emicizumab (HEMLIBRA)?

The loading dose is 3mg/kg/week for 4 weeks followed by maintenance dose (1.5mg/kg/week OR 3mg/kg every 2 weeks OR 6 mg/kg every 4 weeks).

Real world data on the efficacy and side effects of Emicizumab is increasing. Various studies from low middle income countries like India have reported the efficacy even at a lower dose.

What to do if patient has breakthrough bleeding while on emicizumab prophylaxis?

While on emicizumab prophylaxis, breakthrough bleeding must be treated with clotting factor concentrates (CFC) in patients with Hemophilia A without inhibitors and rFVII in patients of Hemophilia A with inhibitors. Concomitant use of emicizumab and aPCC should be avoided as there is a risk of thromboses and thrombotic microangiopathy.

Is development of antidrug antibodies (ADAs) after emicizumab administration a concern?

Emicizumab is a very effective drug with minimal immunogenicity. In the HAVEN clinical trials, 0.75% (3/398) of subjects developed ADAs with neutralizing potential including 2 children in the HAVEN 2 pediatric trial. A total of 5 cases have been reported to have ADA of neutralizing potential till date. Their presence should be suspected if patient presents with unexpected bleeding, along with prolonged aPTT. It has also been observed that patients harbouring ADAs to emicizumab were identified in the first year following initiation of treatment (first 25 weeks of emicizumab exposure (4 out of 5 cases)) with the exception of the case from Japan. Furthermore, the majority of patients that developed ADAs were those with FVIII inhibitors suggesting that immune system in patients with FVIII inhibitors are primed to make antibodies against therapeutic proteins. Thus, it is desirable to ensure close follow-up after initiating emicizumab, especially in the first 6 months for unexpected bleeding while on emicizumab prophylaxis [Kizilocak H et al].

Is there any other factor mimetic being investigated?

Among the next-generation activated FVIII mimetic bispecific antibody developed by Novo Nordisk, Denmark is Mim8 (Denecimig), which is under phase 3 investigation for subcutaneous management of Hemophilia A with or without FVIII inhibitors. A fixed dose approach as per body weight range and patient chosen dosing frequency, has been used in phase 3 trials thereby reducing the need for dose calculations and potentially reducing treatment wastage. [Ostergaard H et al]

REBALANCING AGENTS

The hemostatic system comprises of pro-hemostatic proteins and their inhibitors that work in harmony to ensure that there is neither excessive bleeding nor excessive clotting. In Hemophilia, reduced amounts of pro-hemostatic proteins such as FVIII or FIX and relatively increased amounts of coagulation inhibitors, result in excessive bleeding. Thus, the rebalancing approach improve hemostasis not by increasing factor levels or mimicking their function but rather by reducing or inhibiting the natural inhibitors of coagulation i.e., antithrombin, tissue factor pathway inhibitor (TFPI), protein C or protein S.

Among the rebalancing agents, Fitusiran reduces the amount of antithrombin in the circulation, while concizumab, marstacimab, and MG1113 reduce the circulating pool of TFPI. Serpin PC depletes the pool of activated protein C (APC), while several other molecules reduce the pool of protein S, which is the cofactor for APC. All these agents are administered subcutaneously.

What is the advantage of rebalancing agents over factor mimetics?

Notably, they have the ability to achieve hemostatic control in patients with both Hemophilia A and B with or without inhibitors.

Is there any safety concern?

An important concern with rebalancing agents revolves around safety. Can one get the balance right? i.e., in order to achieve hemostatic control and reduce bleeding, the molecule should not overcorrect and result in a significant risk for thrombosis. The data emerging from the clinical trials suggests that thrombosis is indeed a risk when using these agents; however, for most of the products, the benefit to risk ratio has favoured continued development. Notably, one anti-TFPI molecule, befovacimab (Bayer, Whippany, NewJersey) led to significant thrombosis concern that the development was discontinued. [Mancuso et al]

Figure1: Summary of Novel therapeutic approaches in management of Hemophilia

*In conjunction with the Fitusiran (Qfitlia) approval, the FDA also approved the Siemens Healthineers' INNOVANCE anti-thrombin assay as a companion diagnostic to measure AT levels.

There are several other molecules that are still in phase 3 trials, but have not been approved.

What is the dosing schedule for Marstacimab?

Marstacimab-hncq is given as once weekly subcutaneous injection for prophylaxis. The loading dose is 300mg followed by 150mg once every week.

How to treat breakthrough bleeding while on Marstacimab prophylaxis?

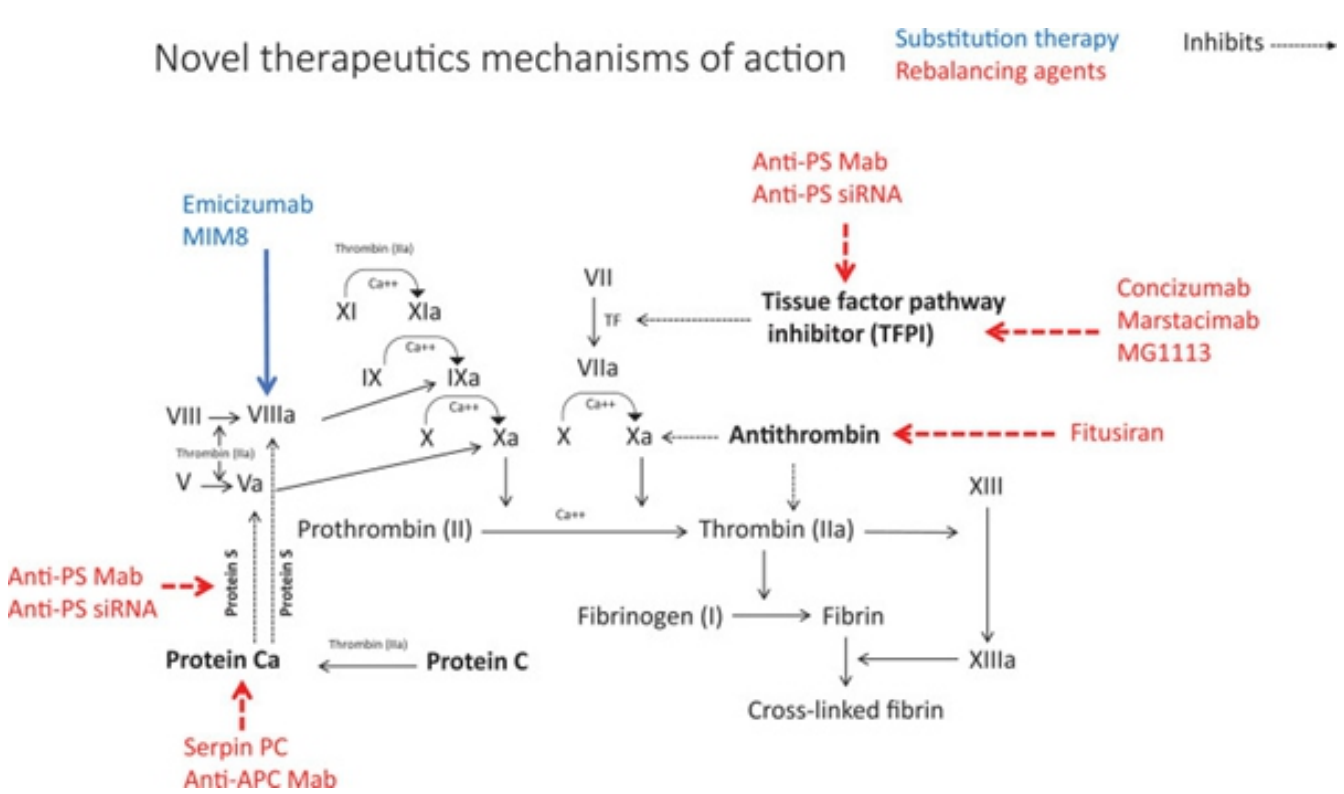
Factor VIII and factor IX products can be administered for the treatment of breakthrough bleeds. Do not use additional doses of Marstacimab to treat breakthrough bleeds

Are there any side effects?

The most common side effects are injection site reactions (itching, swelling, hardening, redness, bruising, pain at the injection site), headache, and itching.

What is the dosing schedule for Concizumab?

Loading: Day 1- 1mg/kg subcutaneous followed by



Which rebalancing agent is FDA approved for use in patients with Hemophilia?

"Pathology is the foundation upon which the science of medicine is built." - William Boyd

Rebalancing Agent	Approved Age	Mechanism of action	Countries with approval	Approved for
Marstacimab-hncq (Hympavzi, Pfizer)	Adults and Children ≥12 years age	• Anti-tissue factor pathway Inhibitor (TFPI) antibody • Once weekly administration	FDA approved in October, 2024 [United States] November, 2024 [Europian Union]	Hemophilia A/B without inhibitors
Concizumab (Alhemo, NovoNordisk)	Adults and Children ≥12 years age	• Tissue factor pathway inhibitor (TFPI) antagonist • Once daily subcutaneous administration	FDA approved on 20 December 2024 [United States] – with inhibitors 31 July 2025 – #extended to Hemophilia A/B without inhibitors December 2024 [Europian Union] July 2025 – #extended to Hemophilia A/B without inhibitors **February 2025 [India]**	Hemophilia A/B with inhibitors
***Fitusiran** (Qfitlia, Sanofi)	Adults and Children ≥12 years age	• Antithrombin antagonist. • Subcutaneous monthly administration	FDA approved on 28 March 2025 [United States]	Hemophilia A/B with or without inhibitors

0.2mg/kg [Day 2 onwards] once daily for 4weeks.
Maintenance: Doses need to individualized based on concizumab plasma concentration (after 4weeks of starting concizumab but no later then 8 weeks from start of Concizumab) administration)
*It is important to maintain concizumab plasma concentration above 200 ng/mL to decrease the risk of bleeding episodes. If concizumab-mtci plasma concentration remains <200 ng/mL at two consecutive measurements, treatment benefit versus potential risk of bleeding should be considered.

How to treat bleeding episodes while on Concizumab?

No dose adjustment is required in the case of breakthrough bleeds.

Are there any side effects?

Few non-fatal thromboembolic events were reported in the Explorer 7 trial [Matsushita *et al*]. However, these cases occurred in patients with multiple risk factors for thromboembolism. Other side effects include hypersensitivity reactions and increased lab values of Fibrin D-dimer and Prothrombin Fragment 1.2.

What is the current evidence supporting use of fitusiran (QFITLIA)?

Fitusiran has demonstrated clinical efficacy in the ATLAS-INH (inhibitor patients) [Young G et al] and ATLAS A/B (noninhibitor patients) [Srivastava A *et al*] trials where encouraging results with clear superiority of fitusiran over on-demand treatment by bypassing agents was observed. The comparison of fitusiran as prophylactic agent vs prophylaxis with bypassing agents in inhibitor positive patients and clotting factor concentrates in those without inhibitors in the ATLAS-PPX study demonstrated effectiveness of fitusiran [Kenet G *et al*].

What is the dosing schedule of Fitusiran? How to modify doses of Fitusiran?

The starting dose is 50 mg subcutaneous every 2 months.

Concizumab Level*	Dose
<200 nanogram (ng)/mL	Increase to 0.25 mg/kg SC once daily
200-4000 ng/mL	Continue 0.2 mg/kg SC once daily
>4000 ng/mL	Decrease to 0.15 mg/kg SC once daily

The dose and/or interval is adjusted if needed to maintain antithrombin (AT) activity between 15-35%.
If last dose administered was 50 mg every 2 months and AT <15%, reduce dose to 20 mg every 2 months, if AT 15-35%, continue 50 mg every 2 months, if AT >35% after 6 months, increase to 50 mg every month. If last dose administered was 20 mg every 2 months and AT <15%, reduce to 10 mg every 2 months, if AT 15-35%, continue 20 mg every 2 months , if AT >35% after 6 months, increase to 20 mg every month. If last dose administered was 10 mg every 2 months and AT <15%, discontinue fitusiran, if AT 15-35%, continue 10 mg every 2 months , if AT >35% after 6 months, increase to 10 mg every month.

How to treat breakthrough bleeding while on fitusiran prophylaxis?

If bleeding occurs ≤7 days after initiating, patient's prior dosing regimen of clotting factor concentrates CFCor BPA prophylaxis should be used. If bleeding occurs >7 days after initiating, reduced dose and frequency of CFC/BPA to minimize thrombotic events should be used.

Are there any side effects with fitusiran?

Notably, there is <5% risk of thrombotic events with fitusiran as suggested by the phase 3 clinical trials. Other adverse events includes increased alanine aminotransferase levels (although not >3 times the upper limit of normal). Lastly, cholelithiasis seems to also be an emerging concern with this agent, although again the benefits outweigh the risks.
Thus, to conclude factor mimetic and rebalancing therapies that have reached a sufficiently advanced stage in clinical trials, a reduction in ABRs has indeed been noted. However, this efficacy has been shown with prophylaxis only and not as on-demand therapy i.e., for control of acute bleed, including emicizumab. Furthermore, as of now among the novel agents, Emicizumab is only FDA approved molecule available in India that offers improvement in quality of life by reducing annual bleeding rates, improving joint health, reduction of social and economic burden in patients with Hemophilia A with or without inhibitors.

Mild and moderate bleeds	Additional treatment with FVIII or FIX or bypassing agents (e.g., rFVIIa or aPCC) • Lowest-approved dose and the dose interval is recommended • For aPCC, a maximum dose of 100 units/kg body weight within 24 hours is recommended
Severe bleeds	Dosing instructions provided in the approved labeling for the specific product based on clinical judgement

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■■

"Diseases desperate grown/By desperate appliance are relieved/Or not at all." - William Shakespeare

THE HEMATOLOGY INDICA
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NEWSLETTER
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National Conference & Workshop on Flow Cytometry in Hematopathology

Organized by Department of Pathology, ESIC Medical College & Hospital, Faridabad
Under the Aegis of ISHBT and ICH | 25–26 April 2025

It was a proud moment for the Department of Pathology, ESIC Medical College and Hospital, Faridabad, to successfully host a **two-day National Conference & Workshop on "Flow Cytometry in Hematopathology"** on **25th–26th April 2025**. This landmark event was the **first of its kind pan ESIC institutions in India**, and also a pioneering initiative in the **Haryana and NCR region**.

The event was organized under the esteemed aegis of the **Indian Society of Hematology and Blood Transfusion (ISHBT)** and the **Indian College of Hematology (ICH)** — apex bodies committed to the advancement of hematology research, education, and patient care in India.

Esteemed Guests & Keynote Sessions

The event was graced by **Dr. H.P. Pati**, Dean, ICH, and a renowned figure in the field of Hematology, who served as the **Chief Guest**. With his extensive experience, including leadership roles across national hematology bodies and former professorship at AIIMS New Delhi, he delivered an enlightening **keynote address on "Flow Cytometry in Hematopathology."**

Dr. Renu Saxena, was **Guest of Honor** for this event. She is a Director, Department of Pathology, Laboratory Medicine, and Head, Hemato-pathology at Medanta - The Medicity, Gurgaon, and former Professor and Head at AIIMS New Delhi. She is an eminent hematopathologist who brought tremendous value to the occasion with her vast expertise and inspiring presence. Her words were a true motivation for young medical professionals.

Organizing Leadership

The success of the conference was made possible under the guidance of **Dr. A. K. Pandey**, Dean, and **Dr. Sandeep Kumar**, Medical Superintendent, whose leadership has continually steered the institution toward academic excellence.

The **Organizing Chairpersons** of the event were:

- **Brig. Dr. Tathagata Chatterjee**, President-Elect, ISHBT, a distinguished academician and leader, whose vision and mentorship were instrumental in shaping the event.
- **Dr. Mukta Pujani**, Head of the Department of Pathology, whose meticulous planning and dedication ensured smooth execution of the entire event.

The conference was efficiently coordinated by **Organizing Secretary Dr. Dipti Sidam**, along with **Dr. Shilpi More, Treasurer** and the **entire Pathology team**, whose tireless efforts made this a memorable academic celebration.

Academic Highlights

The conference featured:

The Organizing Committee

- **Distinguished national speakers** from Delhi/NCR and other regions.
- **Six interactive workshops**, including **live gating sessions**, offering practical insights into flow cytometry applications in **Benign Hematopathology and Hemato-oncology**.
- Participation of **nearly 200 delegates** from across the country, reflecting the pan-India impact of the event.
- Award of **8 CME credit hours** by the Haryana Medical Council, acknowledging the academic depth of the program.

The overwhelming participation and engagement reaffirmed the need for such academic endeavors.

Closing Note

We express heartfelt gratitude to all the speakers, delegates, and organizing members whose enthusiasm, dedication, and scholarly contributions made this conference an overwhelming success. We remain committed to fostering academic excellence and innovation in the field of Hematopathology through such future initiatives.

Lymphoma Update 2025

Lymphoma Update 2025 was organised by the Department of Pathology, Lady Hardinge Medical College & Associated Hospitals, New Delhi, on 26th July .

The event was formally inaugurated by Dr. Sunita Sharma (DGHS), Dr. Anju Seth (Principal, LHMC), and Dr. Kiran Agarwal (Head, Department of Pathology, LHMC), along with the organising team. Dr. Shailaja Shukla served as the Organising Chairperson, and Dr. Vandana Puri as the Organising Secretary of the event. The update witnessed an overwhelming response with over **200 registrations,** reflecting the academic enthusiasm and interest in the subject. A significant highlight of the event was the launch of the book "**Recent Advances in Hematology-5**", edited by Dr. Vandana Puri, Dr. Jasmita Dass, and Dr. Tathagata Chatterjee, marking an important milestone in the field of hematopathology.

The scientific programme began with registration,

Dr Tathagata addressing the audience

followed by a slide session with mentors, setting the tone for an interactive and enriching day. Dr. Sumeet Gujral delivered the first talk on *"Introduction to Lymph Nodes and Approach to Lymphoma"*, chaired by Dr. Vijay Kumar and Dr. Shailaja Shukla. Dr. Tathagata Chatterjee followed with an insightful session on *"Flow Cytometry Demystified: A Tool for Lymphoid Neoplasms"*, moderated by Dr. Monica Sharma and Dr. Sarika Singh. After the inauguration and high tea, Dr. Saumya Ranjan Mallick presented *"A Systematic Approach to B-cell and T-cell Lymphomas"*, chaired by Dr. Kiran Agarwal and Dr. Mrinalini Kotru.

The academic sessions continued with

Dignitaries launching "Recent Advances in Hematology-5", (L–R) Dr. Shailaja Shukla, Dr. Vandana Puri, Dr. Kiran Agarwal (HOD Pathology LHMC), Dr. H. P. Pati (Dean, ICH), Dr. Sunita Sharma (DGHS), Dr. Tathagata Chatterjee (President, ISHBT), Dr. Sumit Gujral, Dr. Jasmita Dass and Dr. Anju Seth (Principal, LHMC).

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"The greatest doctor is also a philosopher." - Galen

Dr. Balamurugan T discussing *"Case-Based Approach to B-cell Lymphoma"*, chaired by Dr. Jyoti Kotwal and Dr. Smita Singh. Dr. H.P. Pati elaborated on *"Lymphoma: A Pathologist's Primer"*, with Dr. Shilpi Agarwal and Dr. Tathagata Chatterjee as session chairs. Dr. Shailaja Shukla then delivered an engaging lecture on *"Hodgkin Lymphoma: Classic, Nodular, and Everything Between"*, chaired by Dr. Prerna Chadha and Dr. P. L. Jyotsna. Another highlight of the programme was the panel discussion moderated by Dr. Mukul Agarwal and Dr. Jasmita Dass, focusing on *"What Does a Clinician Want from a Pathologist for Lymphoma Diagnosis?"*. Esteemed panelists included Dr. Richa Chauhan, Dr. Prerna Arora, Dr. Narender Tejwani, Dr. Prerna Chadha, Dr. Kavita Gaur, Dr. Vandana Puri and Dr. Mukesh Dhankar.The afternoon session included another interactive slide session, followed by a hands-on workshop for residents and participants, providing practical exposure to diagnostic nuances in lymphoma pathology. The update was graced by distinguished faculty, including Dr. Sunita Sharma, Dr. Sumeet Gujral, Dr. Tathagata Chatterjee, Dr Kiran Agarwal, Dr. Saumya Ranjan Mallick, Dr. Balamurugan T, Dr. H.P. Pati, Dr. Shailaja Shukla, Dr. Mukul Agarwal, Dr. Jasmita Dass, Dr. Richa Chauhan, Dr. Prerna Arora, Dr. Narender Tejwani, Dr. Prerna Chadha, Dr. Kavita Gaur, Dr. Mukesh Dhankar, Dr. Vandana Puri, Dr. Geetika Sharma, Dr. Jyoti Garg, Dr. Neha Suman, Dr. Shivali Sehgal, and Dr. Gaurav Gahlot. Chairpersons included Dr. Vijay Kumar, Dr. Monica Sharma, Dr. Sarika Singh, Dr. Kiran Agarwal, Dr. Mrinalini Kotru, Dr. Jyoti Kotwal, Dr. Smita Singh, Dr. Shilpi Agarwal, and Dr. P.L. Jyotsna.

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CME and Workshop on the basics of Hemostasis and Coagulation

In the ever evolving and dynamic field of Hemostasis, the Hematology Section of the Department of Pathology of Amrita Institute of Medical Sciences and Research Centre (Faridabad) successfully conducted one and a half-day **"CME and Workshop on the basics of Hemostasis and Coagulation"** under the esteemed aegis of Indian Society of Hematology and Blood Transfusion (ISHBT), Indian College of Hematology (ICH) and Regional Society of Practicing Pathologist – IAPM (Haryana Chapter). The event was granted 7 credit hours by Haryana Medical Council.

The one-and-a-half-day event brought together eminent experts, clinicians, and young researchers, creating a vibrant academic platform for sharing knowledge and practical skills in the field of coagulation and hemostasis. The scientific program featured insightful lectures on fundamental and advanced concepts of hemostasis, bleeding disorders, diagnostic updates in APLA syndrome, anticoagulant monitoring, and quality control in coagulation laboratories by Dr. Jasmina Ahaluwalia (PGI Chandigarh), Brig. Dr. Tathagata Chatterjee (ESIC Hospital, Faridabad), Dr. Pravas Mishra (Amrita Hospital, Faridabad), Dr Jasmita Dass (AIIMS, New Delhi), Dr Anil Handoo (BLK – MAX, New Delhi) & Dr Ruchi Gupta (SGPGIMS, Lucknow), Dr. Sarika Singh (MAMC, New Delhi), Dr. Vandana Puri (LHMC, New Delhi), Dr. Monika Gupta (PGIMS, Rohtak) and Prof. Mrinalini Kotru (UCMS New Delhi).

The event commenced with the lamp lighting ceremony by Dr. Sanjeev Singh, Medical Director of Amrita Hospital, Faridabad along with Principal, Dr. Sarmila Chandra (President, ISHBT), Dr. Sunita Singh (President, IAPM, Haryana Chapter) and Prof. K. R. Rathi, Head, Department of Pathology. Day 1 of the CME set the stage for an enriching academic experience with a series of insightful lectures delivered by distinguished experts in hematology and coagulation science. The academic sessions were further enriched by a quiz competition for postgraduate students, moderated by Brig. Dr. Tathagata Chatterjee, which witnessed active participation and enthusiastic engagement. The day concluded with a high-level panel discussion involving pathologists and clinicians, focusing on the comparative utility of global versus conventional hemostatic assays and their clinical significance, thereby bridging theoretical knowledge with real-world clinical applications.

The second day of the CME was dedicated to an immersive wet lab experience conducted in the state-of-the-art laboratory at Amrita Hospital. Under the coordination of Dr. Kusum Gupta (Assistant Professor, Pathology), participants received hands-on training on advanced coagulation analyzers provided by Werfen. Each delegate was guided through detailed demonstrations addressing pre-analytical and analytical considerations, followed by practical exercises in mixing studies directly on the analyzer. The CME & Workshop received outstanding feedback from delegates and faculty for its well-structured scientific agenda, thoughtfully designed laboratory exposure, and highly engaging interactive sessions.

Organising committee with the delegates

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Dr. A. K. Tripathi (Central)	Zonal Council Members
Dr. Avinash Pophali (West)	Zonal Council Members

"Every disease is a conversation between the body and the mind." - Christian B. Anfinsen