An overview on Thrombocytopenia

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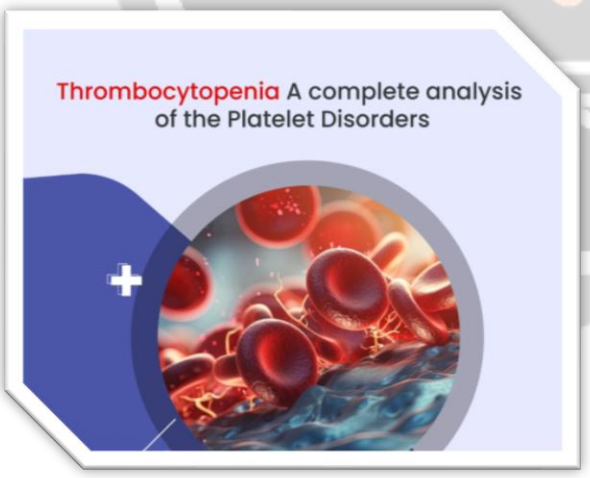
Abstract:
Thrombocytopenia is common in intensive care patients and causes increased morbidity and mortality due to complications such as bleeding and transfusion. This is due to many factors such as platinum depletion, tracking, destruction and consumption. It is important to fully understand this process for correct diagnosis and treatment. Treatment strategies focus on controlling the implant problem by administering injections such as platinum to treat complications such as bleeding. Recent research supports the use of blood transfusion as a treatment option. This review focuses on the different pathophysiological mechanisms of thrombocytopenia encountered in the intensive care unit, examining different platelet transfusion rates, alternatives, and the potential role of thromboelastography (TEG) in guiding the appropriate administration of blood to prevent correction of coagulopathy.

The normal number in adults is between 150,000 and 450,000 per microlitre; More than 450,000/microliter is called thrombocytosis and less than 150,000/microliter is called thrombocytopenia. Different types of thrombocytopenia are defined according to the cause, such as immune thrombocytopenic purpura, heparin-induced thrombocytopenia, thrombocytopenic purpura, and drug-induced thrombocytopenia.

Keywords: Thromboelastographic, Pathophysiology, Pseudo Tharmbocytopenia, Sepsis.

❖ Introduction:



❖ Thrombocytes, also called platelets, are small, colorless particles found in the blood. George Gulliver drew these using a two-lens microscope in 1841, and William Addison compared fibrin tissue in 1842. Lionel Beale was the first to publish platinum drawings in 1864. Thrombocytopenia occurs in adults when the platelet count falls below the normal range of 150,000 to 450,000 per microlitre. Platelets play an important role in clot formation to stop bleeding in injuries, maintain blood immunity and promote clot formation. These important cellular components are produced in bone, the spongy organ within bone that contains cells that develop red blood cells, white blood cells, and platelets. Thrombopoietin regulates platelet production. The half-life of platelets in a healthy body is 10 days. When the platelet count drops below 100,000/microlitre, bleeding slows down. Aplastic anemia and leukemia, blood disorders associated with decreased platelet production, contribute to thrombocytopenia.

Thrombocytopenia severity is categorized into three stages based on platelet count: mild (100,000 to 150,000/microliter), moderate (50,000 to 100,000/microliter), and severe (< 50,000/microliter). Conversely, when the platelet count exceeds 450,000/microliter, it is termed thrombocytosis. There are two types: primary thrombocytosis, caused by hematopoietic cell alterations, and secondary thrombocytosis, caused by external factors like chronic inflammation, cancer, and iron deficiencies. Primary immune thrombocytopenia is an

organ-specific autoimmune condition marked by a decreased platelet count in the peripheral blood. Symptoms encompass fatigue and the presence of dry or wet purpura. While some patients may experience mild symptoms or none at all, severe and life-threatening bleeding can occur. Despite advancements, diagnosing ITP still relies on excluding any detectable underlying cause for thrombocytopenia during investigation.

❖ **History**:

Gathering a comprehensive history is crucial for pinpointing the cause of thrombocytopenia. Patients with platelets exceeding 50,000/mL typically exhibit few symptoms, while those with platelets below 20,000/mL are more prone to spontaneous bleeding.

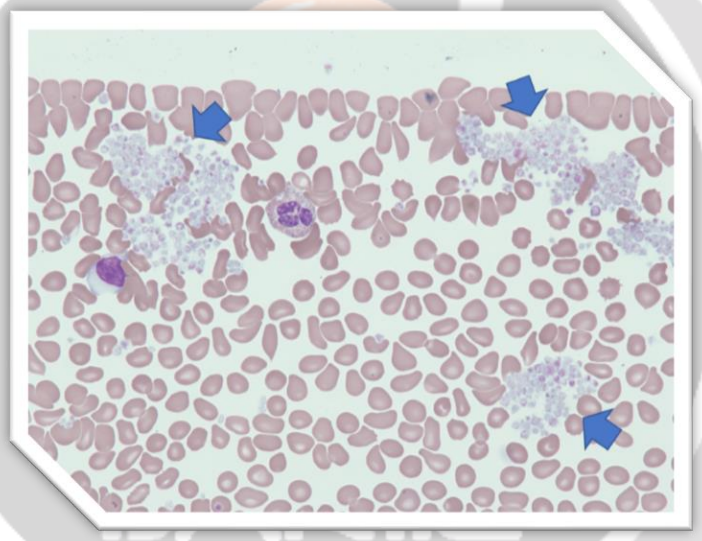
Inquire about prior blood count tests and baseline platelet counts, focusing on recent drops in platelet count. Assess the history of bleeding, including petechiae, haemorrhagic bleeding, gingival bleeding, and epistaxis. Explore potential exposures and symptoms of infections, considering viral, bacterial, and rickettsial origins. Evaluate HIV infection risk and inquire about travel to malaria, dengue, and Ebola-endemic areas.

Conduct a diet history to identify nutritional deficiencies and inquire about conditions such as SLE, RA, bariatric surgery, and blood transfusions. Review the medication list, including over-the-counter drugs, quinine-containing beverages, and herbal teas. For hospitalized patients, investigate exposure to heparin products. Check for a family history of thrombocytopenia or bleeding disorders.

In pregnant patients, inquire about headaches, visual symptoms, abdominal pain, and flu-like symptoms, as these may indicate preeclampsia/HELLP syndrome.

❖ **Types**:

1. **Pseudo thrombocytopenia**



Pseudo thrombocytopenia can result in falsely low platelet counts due to platelet clumping during automated counting, often caused by EDTA-dependent antibodies. This can be confirmed by repeating the count with alternative anticoagulants like citrate, heparin, or oxalate. EDTA-independent pseudo thrombocytopenia may occur due to autoantibodies, like cold agglutinins. Giant platelets and platelet satellitism can also lead to inaccurately low platelet counts. Confirming thrombocytopenia through a peripheral smear examination is crucial before further investigation or treatment.

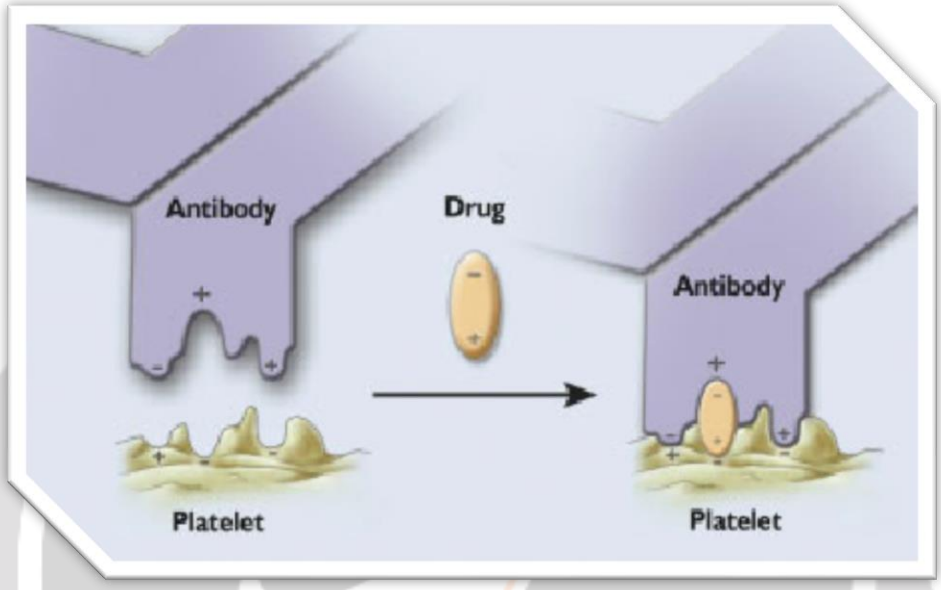
The use of GP2b3a inhibitors, such as abciximab, eptifibatide, and tirofiban, can cause pseudo thrombocytopenia in both EDTA and citrate. It's essential to rule out pseudo thrombocytopenia before diagnosing real thrombocytopenia from these inhibitors and considering any discontinuation. In patients undergoing percutaneous intervention, where these inhibitors are crucial to prevent in-stent thrombosis, careful assessment is necessary. If true thrombocytopenia is confirmed, switching to direct thrombin inhibitors like bivalirudin or lepirudin is recommended. Platelet transfusion is advised for severe bleeding or counts below 10,000/μL. Heparin-induced thrombocytopenia (HIT) should also be ruled out in PCI patients using heparin, and if confirmed, managed with DTI agents, avoiding platelet transfusions. Unlike GP2b3a inhibitors associated with bleeding, HIT is linked to thrombosis.

Immune thrombocytopenic purpura (ITP) is characterized by a solitary condition with low platelet levels, typically below 100,000/microL, accompanied by a widespread purpura rash. White blood cell and haemoglobin levels remain within the normal range. Formerly known as idiopathic thrombocytopenic purpura, it is now recognized as immune thrombocytopenic purpura due to IgG-mediated autoantibodies targeting platelet membrane proteins like GP2b3a complex, GP Ib/IIa, and GP VI. The binding of these antibodies to

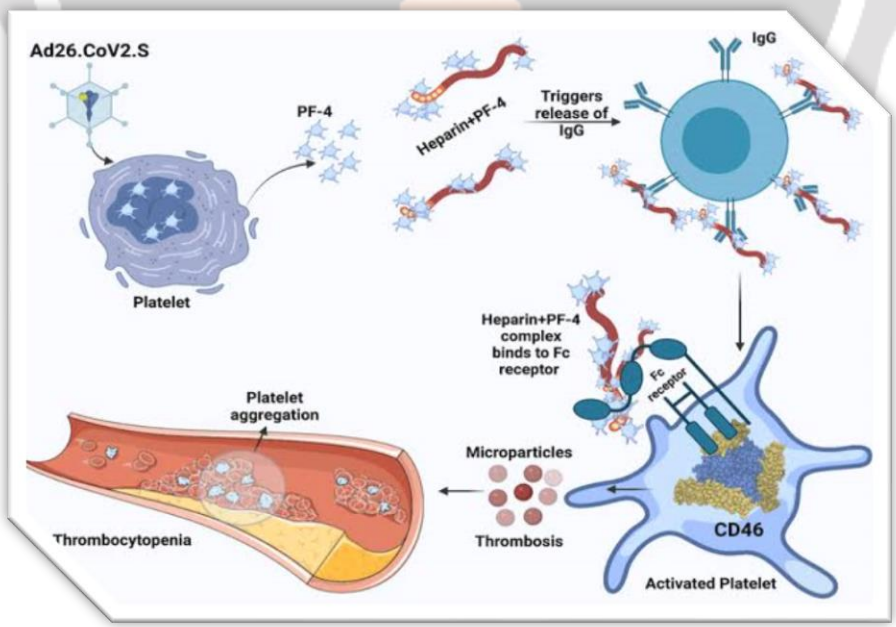
platelets accelerates their clearance by the spleen. Typically triggered by events such as infections or immune alterations, ITP initiates a cascade leading to platelet destruction.

2. Drug-induced thrombocytopenia (DIT):

To attribute drug-induced thrombocytopenia (DIT), specific criteria must be met: thrombocytopenia should follow drug administration, recovery occurs upon discontinuation, alternative causes are ruled out, and re-administration is likely to cause recurrence [11]. DIT can result from immune-mediated destruction or factors like bone marrow suppression. Arnold et al.'s systematic evaluation identified 153 drugs with an increased risk of inducing thrombocytopenia. Definite causes include quinine, quinidine, trimethoprim/sulfamethoxazole, vancomycin, penicillin, rifampin, carbamazepine, ceftriaxone, ibuprofen, mirtazapine, oxaliplatin, suramin, and GP2b3a inhibitors like abciximab, tirofiban, eptifibatide, and heparin [11]. Common mechanisms and contributing drugs for DIT



3. Heparin-induced thrombocytopenia:

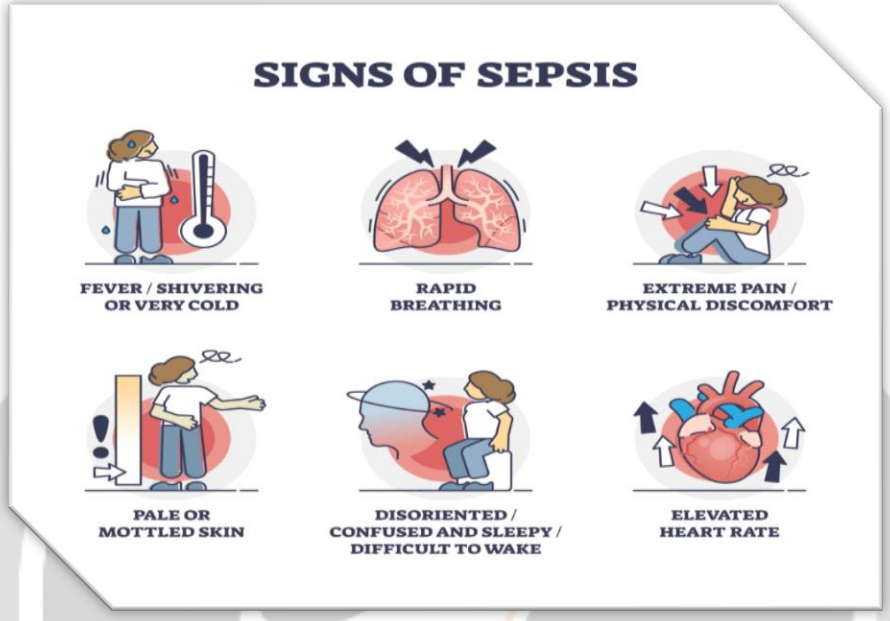


Type 1 HIT is characterized by a transient, non-immune-mediated drop in platelet count within the first few days of heparin initiation, with spontaneous resolution and no complications. In contrast, type 2 HIT, an immune-mediated reaction, leads to a >50% platelet count decreases 5-10 days post-heparin exposure. It's associated with antibodies against platelet factor 4, often resulting in platelet counts below 100x10^9/L. Type 2 HIT may manifest with skin issues at injection sites and systemic symptoms. Prolonged heparin use in type 2 HIT

increases the risk of severe thrombosis, including DVT, PE, and potentially life-threatening arterial occlusions. The 4Ts score aids in identifying patients prone to heparin-induced thrombocytopenia.

4. Sepsis:

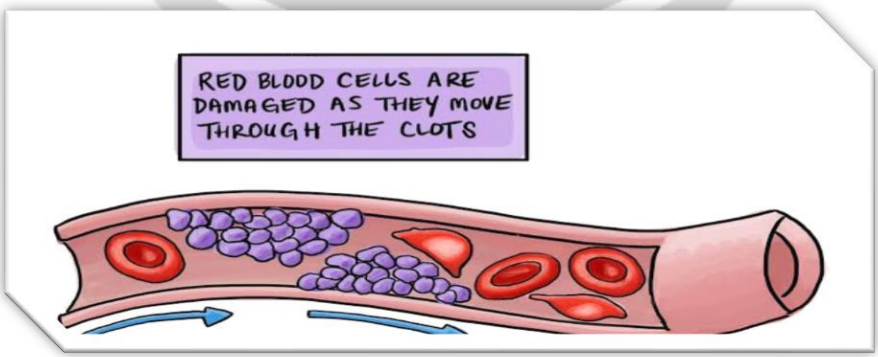
Platelet-related conditions pose distinct challenges in clinical scenarios. Heparin-induced thrombocytopenia (HIT) has two types—type 1, non-immune-mediated and reversible, and type 2, immune-mediated with severe complications. The 4Ts score aids HIT risk assessment. Sepsis, a life-threatening response to infection, often involves thrombocytopenia. Coagulopathy in sepsis arises from a dysregulated haemostatic system, with platelet activation and aggregation contributing to microvascular dysfunction. Thrombocytopenia's multifactorial nature includes decreased production, increased elimination, and various causes.



A comprehensive review on thrombocytopenia in intensive care patients emphasizes its association with morbidity and mortality. Understanding mechanisms, diagnostic approaches, and management strategies, including platelet transfusions, is crucial for optimal care.

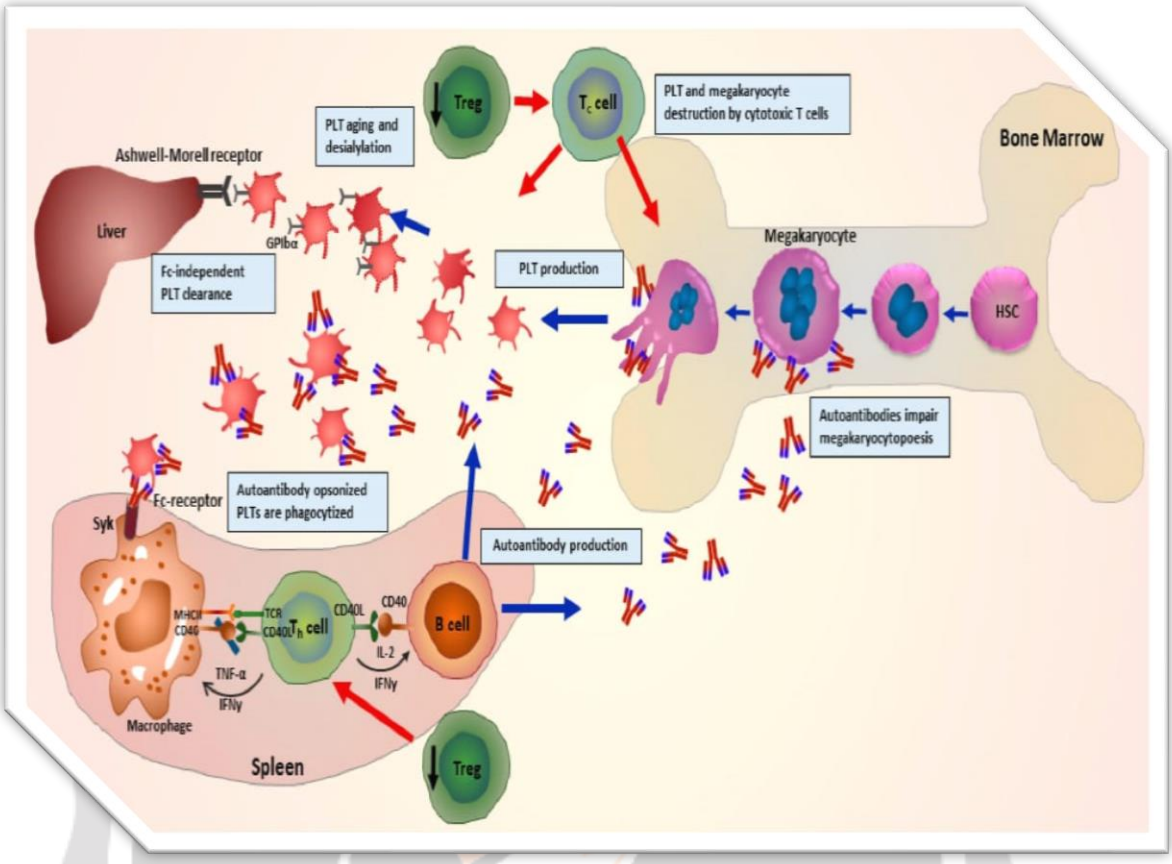
5. Thrombotic microangiopathies:

Thrombotic microangiopathies constitute a group of disorders marked by microangiopathic haemolytic anaemia, thrombocytopenia, and microthrombi causing ischemic tissue injury. The central feature is microangiopathic haemolytic anaemia, reflecting red blood cell destruction within microvasculature, coupled with thrombocytopenia from platelet activation and consumption. Thrombotic thrombocytopenic purpura (TTP) and haemolytic uremic syndrome (HUS) represent primary forms, and regardless of the cause, timely intervention is imperative as thrombotic microangiopathy is a hematologic emergency.



❖ **Pathophysiology:**

Seventy years have passed since the influential Harrington-Hollingsworth experiment, revealing that infusing plasma from ITP patients into healthy volunteers led to significant thrombocytopenia. Current understanding points to B and T cell defects as a key element in ITP pathophysiology (Figure 1). Compelling evidence suggests that platelet autoimmunity arises from a breakdown in self-tolerance mechanisms. The details of these mechanisms will be explored.



Malfunctions in antigen presenting cells (APC), crucial for initiating IgG responses in ITP, involve T helper cells recognizing peptide antigens on APCs, including dendritic cells (DC), macrophages, and sometimes, B cells. Recent studies even propose platelets and megakaryocytes as potential APCs. Notably, DCs, powerful professional APCs, exhibit impaired function in ITP, with observations of reduced DC-associated indoleamine 2,3-dioxygenase 1 (IDO1) impacting regulatory T cell (Treg) differentiation. Macrophages, primarily responsible for splenic platelet destruction, may, during inflammation, intensify antigen presentation. This dual role could contribute to an autoantigen feedback loop in ITP, warranting consideration for immunosuppressive therapy targeting APC defects.

❖ **Pathophysiological Mechanism:**

The primary mechanisms leading to thrombocytopenia encompass pseudo thrombocytopenia, haemodilution, reduced platelet production, heightened platelet consumption, increased sequestration, and platelet destruction. In ICU settings, common causes include drug-induced thrombocytopenia (especially due to heparin and glycoprotein IIb/IIIa inhibitors), alcohol, antibiotics, sepsis, major bleeding, and microangiopathic haemolytic anemias like TTP, HUS, and DIC. Understanding these diverse mechanisms is crucial for appropriate management. For example, major bleeding-induced thrombocytopenia may require platelet transfusion, whereas TTP and HUS contraindicate platelet transfusion, favoring plasmapheresis. Specific conditions, such as HITT, demand prompt diagnosis, heparin cessation, and alternative anticoagulant initiation. Reduced platelet production can stem from bone marrow suppression, infections, malignancies, or nutritional deficiencies. ICU patients often exhibit a combination of increased destruction and consumption, as seen in sepsis and DIC, influenced by various mechanisms like complement activation, coagulation markers, hem phagocytosis, ADAMTS13 depletion, and histone release.

❖ **Etiology:**

1. Drugs:

- **Antiplatelet agents**: Abciximab, Eptifibatide, Tirofiban.
- **Anticoagulants:** Heparin (including Heparin-induced thrombocytopenia)
- **Analgesics:** Acetaminophen.
- **NSAIDs:** Ibuprofen, Naproxen, Amiodarone.
- **Antibiotics:** Cimetidine, Piperacillin, Vancomycin.
- **Anti-seizure medications:** Carbamazepine.

2. Diseases and Disorders:

- Alcoholism and Alcohol use disorder.
- Autoimmune diseases: Systemic lupus erythematosus, Rheumatoid arthritis, Henoch-schonlein purpura (small blood vessels).
- Splenomegaly.
- Bone marrow diseases: Myelodysplastic syndromes, certain lymphomas, Leukaemia, and Aplastic anemia.

3. Chemical Exposures:

- Arsenic.
- Benzene.
- Pesticides.

4. Microorganisms:

- Chickenpox virus.
- Hepatitis C.
- Cytomegalovirus.
- Epstein-Barr virus.
- Human immunodeficiency virus.
- Parvovirus.

5. Rare Conditions Causing Blood Clots:

- Thrombotic thrombocytopenic purpura (TTP).
- Disseminated Intravascular Coagulation (DIC).

❖ **Symptoms**:

- Bleeding gums.
- Blood in the stool (black), urine (hematuria), or vomit.
- Red or pink urine.
- Heavy menstrual periods.
- Rectal bleeding.

❖ **Causes:**

1. Primary Immune Thrombocytopenia (Primary ITP):

An autoimmune condition where antibodies target platelets, leading to their destruction.

2. Drug-Induced Immune Thrombocytopenia:

- Heparin-induced thrombocytopenia (HIT): Anti-platelet antibodies activate platelets, causing thrombosis.
- Other drugs: Quinine, sulphonamides, ampicillin, vancomycin, piperacillin, acetaminophen, ibuprofen, naproxen, cimetidine, glycoprotein IIb/IIIa inhibitors, and various over-the-counter remedies, supplements, foods, and beverages.

3. Drug-Induced Non-Immune Thrombocytopenia:

Some medications, like valproic acid, daptomycin, and linezolid, suppress platelet production.

4. Infections:

- Viral infections: HIV, hepatitis C, Epstein-Barr virus, parvovirus, mumps, varicella, rubella, Zika virus.
- Bacterial and parasitic infections: Sepsis, Helicobacter pylori, leptospirosis, brucellosis, anaplasmosis, malaria, babesiosis.

5. Other Causes:

- Hypersplenism due to chronic liver disease.
- Chronic alcohol abuse.
- Nutrient deficiencies: Folate, vitamin B12, copper.
- Autoimmune disorders: Systemic lupus erythematosus, rheumatoid arthritis with secondary ITP.
- Pregnancy-related thrombocytopenia: Gestational thrombocytopenia, preeclampsia, HELLP syndrome.

❖ **Other Causes**:

Additional factors contributing to thrombocytopenia include:

1. Myelodysplasia

2. Malignancy:

- Cancer associated with chronic disseminated intravascular coagulation (DIC).
- Cancer causing marrow suppression, including leukaemia, lymphoma, and solid tumors.

3. Paroxysmal Nocturnal Hemoglobinuria (PNH)

4. Thrombotic Microangiopathy (TMA)

5. Thrombotic Thrombocytopenic Purpura (TTP):

Manifested by fever, renal failure, thrombocytopenia, and microangiopathic haemolytic anaemia with or without neurologic manifestations.

6. Haemolytic Uremic Syndrome (HUS):

Caused by Shiga toxin-producing organisms (E. coli and Shigella), often observed in children.

7. Drug-Induced TMA:

Triggered by substances like quinine and specific chemotherapy agents.

8. Antiphospholipid Antibody Syndrome

9. Aplastic Anemia

10. Inherited Thrombocytopenia:

More common in children and rare in adults.

11. Other Rare Syndromes:

- Von Willebrand disease type 2.
- Alport syndrome.
- Wiskott-Aldrich syndrome.
- Fanconi syndrome.
- Thrombocytopenia-absent radius syndrome.
- Bernard–Soulier syndrome.
- May-Hegglin anomaly.

❖ **Diagnosis:**

1. Physical Examination:

Conduct a thorough examination, including scrutiny of the skin and potential bleeding sites, along with assessing the liver, spleen, and lymph nodes. Thrombocytopenia-related bleeding typically presents as petechiae, nonpalpable purpura, and ecchymosis. Dry purpura refers to skin-related purpura, while wet purpura involves mucosal purpura.

Inspect for hepatomegaly and splenomegaly, indicators found in conditions such as lymphoma, chronic liver disease, and various hematologic disorders. Enlarged lymph nodes may signify infections, autoimmune disorders, lymphoma, or other malignancies.

2. Evaluation:

Evaluate isolated thrombocytopenia by obtaining a comprehensive set of tests, including CBC, peripheral blood smear, HIV, and HCV screenings.

Repeat CBC to confirm thrombocytopenia. Anemia and thrombocytopenia may occur in infections, DIC, sepsis, thrombotic microangiopathy, and autoimmune disorders like Felty syndrome.

Leucocytosis and thrombocytopenia can manifest in infection, malignancy, and chronic inflammatory conditions.

Pancytopenia is characteristic of myelodysplastic syndromes.

For patients with symptoms or signs of autoimmune disorders like SLE or APS, obtain anti-nuclear antibodies and antiphospholipid antibodies, respectively.

In cases of thrombosis, consider heparin-induced thrombocytopenia (platelet factor 4 antibodies), APS (antiphospholipid antibodies), DIC, and PNH (PT, aPTT, fibrinogen, LDH).

Check liver enzymes and coagulation tests in patients with liver disease.

3. Bone Marrow Examination:

Perform a bone marrow biopsy when the cause of thrombocytopenia is unclear or when a hematologic disorder is suspected.

Normal or elevated megakaryocytes in bone marrow may indicate conditions with increased platelet destruction.

Reduced megakaryocytes, along with an overall cell reduction, are observed in aplastic anemia.

In SLE, a severe reduction or absence of megakaryocytes is attributed to an autoantibody against the thrombopoietin receptor.

Megaloblastic changes in RBCs and granulocytes occur in vitamin B12, folate, and copper deficiency. Dysplastic cells are present in myelodysplasia.

4. Thrombocytopenia in Pregnancy:

Thrombocytopenia in pregnancy, the second most prevalent hematological abnormality following anemia, occurs in 6.6% to 11.6% of pregnancies. Understanding its diverse causes and appropriate management is crucial for ensuring a safe pregnancy and favorable outcomes.

Table1010 [29] outlines various causes of thrombocytopenia in pregnancy. Gestational thrombocytopenia, an incidental form accounting for 70% to 80% of cases, is most common in the second to

late third trimester, presenting with low to moderate thrombocytopenia. Severe cases require further investigation to rule out alternative causes and typically resolve one to two months post-delivery.

Immune thrombocytopenic purpura (ITP) is the second most common cause, characterized by platelet counts below 100 x 10^9/L early in pregnancy, declining as gestation progresses. Treatment options for platelet counts less than 30 x 10^9/L or symptomatic bleeding include prednisone, intravenous immunoglobulin (IvIg), a combination of steroids plus IvIg, azathioprine for refractory cases, and second-trimester splenectomy for those unresponsive to initial therapies. Many non-pregnant ITP treatments are avoided due to potential toxicities. For epidural anesthesia, platelet counts of at least 80-100 x 10^9/L are recommended, while 50 x 10^9/L is advised for cesarean sections.

❖ **Treatment**:

Platelet transfusions alternatives and additions:

1. **Antifibrinolytic Therapy: Tranexamic Acid (TXA):**

Tranexamic acid (TXA), an antifibrinolytic agent, has proven effective in reducing bleeding risk and transfusion requirements during surgery. It also decreases mortality risk in trauma patients and shows benefits in individuals with thrombocytopenia associated with MDS and mucous membrane bleeding. Caution is advised for patients with ischemic heart disease and haematuria.

Recommended by various guidelines, including the National Institute for Health and Clinical Excellence (NICE), Cochrane review (Wardrop et al., 2013), and British Committee for Standards in Hematology (BCSH) guidelines [25]. The CRASH-2 trial in 2013 demonstrated that when administered within one hour, TXA significantly reduces mortality in bleeding trauma patients by about one-third, without increasing side effects. However, its efficacy diminishes after three hours post-injury.

The CRASH-3 trial in 2019 explored the impact of TXA on traumatic brain injury (TBI) patients with mild to moderate head injuries (Glasgow coma score (GCS) 13-15) and evidence of intracranial bleeding on initial CT head. TXA, when used within three hours of injury, was found to reduce mortality secondary to head injuries by minimizing intracranial bleeding [26].

Contrarily, the HALT-IT trial in 2020 concluded that tranexamic acid is not recommended for use in gastrointestinal bleeding and may lead to increased venous thromboembolic complications, such as DVT and PE.

2. **Desmopressin (DDAVP):**

Desmopressin, a synthetic analog of vasopressin, acts by stimulating the release of factor VIII from endothelial stores and enhancing von Willebrand factor activity, thereby supporting coagulation. Extensive research has explored the application of desmopressin in various scenarios, including trauma patients on aspirin, perioperative bleeding situations, individuals with uremia, and those with von Willebrand disease.

3. **Thromboelastography (TEG):**

Thromboelastography (TEG) is a non-invasive, in vitro diagnostic study that quantitatively assesses the clotting ability of blood. It dynamically captures changes in blood viscoelastic properties during clotting under low sheer stress, presenting results graphically [28]. Typically, citrated blood samples at room temperature are utilized for TEG studies.

Modifications to the classic TEG enhance diagnostic utility. Native blood TEG or non-activated thromboelastometry (NATEM) employs whole, non-citrated blood samples but requires immediate testing. Rapid TEG (r-TEG) activates blood coagulation faster than conventional TEG, using tissue factor instead of kaolin cephalin reagent. It proves valuable in managing massive transfusions for trauma patients.

TEG platelet mapping evaluates platelet aggregation in the presence of adenosine diphosphate or arachidonic acid, predicting antiplatelet activity (e.g., aspirin, clopidogrel). Heparinase-added TEG (hTEG) measures heparin's impact on blood coagulation, aiding in heparin reversal management. Rotational thromboelastometry (RoTEM), a TEG variation, involves an oscillating pin while the cup remains stable.

The primary focus in managing ITP is to prevent bleeding and ensure patient safety by raising platelet counts to 20-30 x 10^9/L. The goal is safety rather than normalizing platelet counts. Traditionally, immune suppression with corticosteroids and other drugs has been a common approach, but it often comes with side effects and questionable benefits. The recent shift in ITP therapy includes second-generation thrombopoietin receptor agonists (TPO-RAs) and fostamatinib, with ongoing clinical development of other agents like neonatal Fc receptor inhibitors, Bruton tyrosine kinase inhibitors, and anti-complement drugs. See Table 1 for a summary of recent treatments for primary ITP.

In autoimmune diseases, achieving lasting remissions with many treatments is often challenging. The focus has shifted towards improving symptoms and overall quality of life, a perspective also adopted in ITP treatment. Current medical practice employs various models, including the infectious disease, oncologic disease, metabolic disease, and transplant rejection models. For decades, ITP has been managed using the transplant rejection model, involving immune system suppression. However, this approach rarely induces remissions, leading to serious adverse effects on morbidity and mortality, ultimately resulting in a poor quality

of life for ITP patients. Despite ITP being considered a "benign" disorder, treatments often offer limited benefits while negatively impacting patients' health-related quality of life.

❖ **Non-pharmacological management options for ITP include**:

1.Surgery:

Splenectomy, the removal of the spleen, is a permanent and effective solution in adults with ITP as it eliminates the primary source of platelet destruction.

2.Vitamin B12:

Found in foods like beef liver and eggs, Vitamin B12 supports the health of blood cells.

3.Vitamin B (Folate):

Foods like peanuts, black-eyed peas, kidney beans, oranges, and orange juices containing Vitamin B help in maintaining blood cells.

4. Iron:

Essential for producing healthy blood cells, iron-rich foods include mussels, pumpkin seeds, lentils, and beef.

5.Vitamin C:

Found in mangoes, pineapple, broccoli, tomatoes, and cauliflower, Vitamin C aids platelet grouping and function while enhancing iron absorption.

6.Plasma exchange:A therapeutic option.

7.Lifestyle modification:

- Avoid activities causing injuries, such as certain sports (boxing, martial arts, football).
- Moderate alcohol intake.
- Use caution with over-the-counter medications like Aspirin and Ibuprofen, which can hinder proper platelet function.

❖ **Complication**:

Severe thrombocytopenia is linked to significant internal bleeding, with brain haemorrhage being a fatal outcome. Heparin-induced thrombosis (HIT) manifests as both arterial and venous thrombosis, leading to deep venous thrombosis, pulmonary embolism, cerebrovascular accidents, and myocardial infarction. Antiphospholipid antibody syndrome is associated with arterial and venous thrombosis, as well as complications such as abortions. In TTP, microvascular thrombosis affects various organ systems, primarily the central nervous system, while HUS primarily impacts the kidneys.

❖ **Prevention**:

Preventing thrombocytopenia is challenging, but steps can be taken to minimize associated health problems:

- Avoid contact with toxic chemicals
- Be cautious of medications known to cause thrombocytopenia
- Minimize the risk of injuries or trauma
- Limit or avoid alcohol consumption.

❖ **Conclusion**:

In conclusion, thrombocytopenia, characterized by a platelet count below the normal range of 150,000/microliter in adults, necessitates a comprehensive approach. This review has covered thrombocytopenia's types, etiology, pathophysiology, diagnosis, pharmacological and non-pharmacological treatments, and prevention. Successful management hinges on understanding the underlying pathophysiological processes. Individuals with thrombocytopenia should adopt preventive measures, and given the condition's complexity, an interprofessional healthcare team is crucial for achieving optimal patient outcomes.

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