Immune Thrombocytopenic Purpura (ITP)

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High School Scholars Day at Loyola University

July 25, 2025

Objectives

- Review epidemiology and presentation of ITP
- Understand the mechanisms and pathophysiology of ITP
- Understand different treatment modalities of ITP

Background

- **ITP is an immune disorder marked by low platelet count due to:**
  - Destruction of platelets
  - Impairment in platelet production
- **Can be acute or chronic, occurs in all ages, and ranges in severity:**
  - Adult cases are more likely to be chronic
- **Patient may present with petechiae or mucocutaneous bleeding**
- **Diagnosis of exclusion**

Methods

- **Literature review through PubMed search**
- **Focus areas:**
  - Pathophysiology
  - Mechanism of Action
  - Clinical Presentation
  - Treatment (Current and Investigational)
  - Prognosis

Etiology

Primary ITP:

- Idiopathic; no identifiable cause

Secondary ITP:

- Is associated with disorders like
  - Viral (Hep C, HIV, H. pylori)
  - Immune (SLE, APS, CVID)
  - Malignancy (CLL, Hodgkin's lymphoma)
  - Others

- **Has higher chances of spontaneous remission**

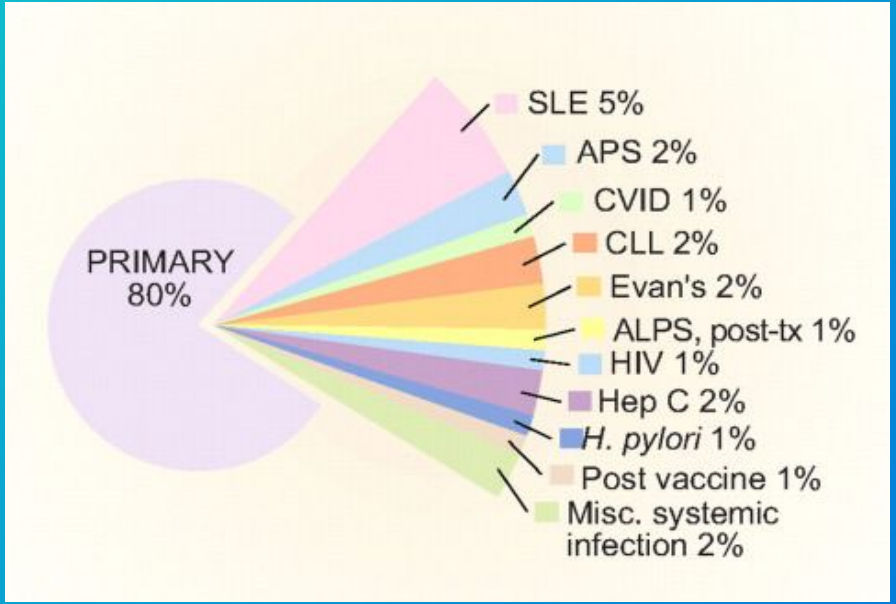


Image 1

Epidemiology

- Rare: 1.1–12.5 cases per 100,000 annually
- Primary ITP is more common in:
  - Women
  - Children ages 1–5
  - Young adults
  - Adults over 60
- Adult cases are more likely to become chronic
- Secondary ITP has a higher age of onset and has no female predominance

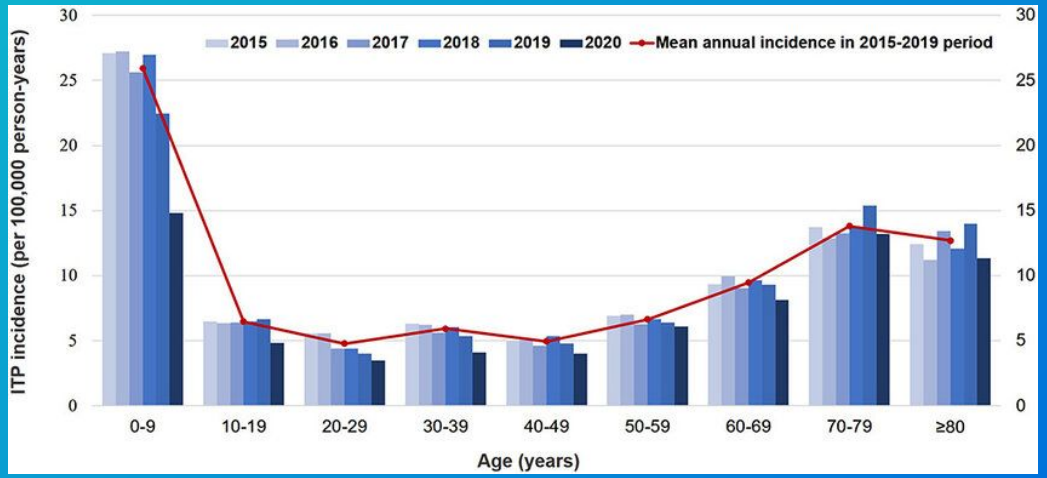


Image 2

Pathophysiology

Autoimmune condition involving IgG antibodies:

- IgG targets platelet glycoproteins → cleared by spleen/liver
- IgG binds to megakaryocytes → impairs platelet production

Dysfunction of:

- T helper (Th) cells: Stimulate IgG production
- Regulatory T (Treg) cells: Suppress autoimmunity

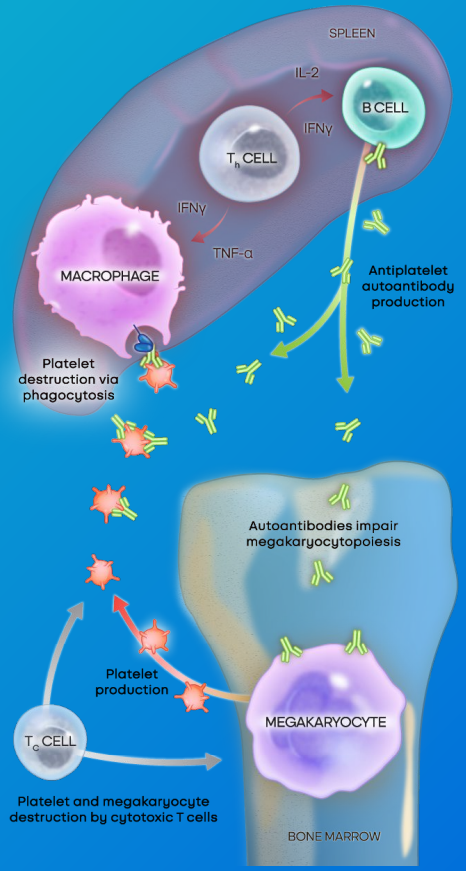


Image 3

Pathophysiology

Normally:

- Th → B cells → IgG → pathogens
- Treg controls IgG production

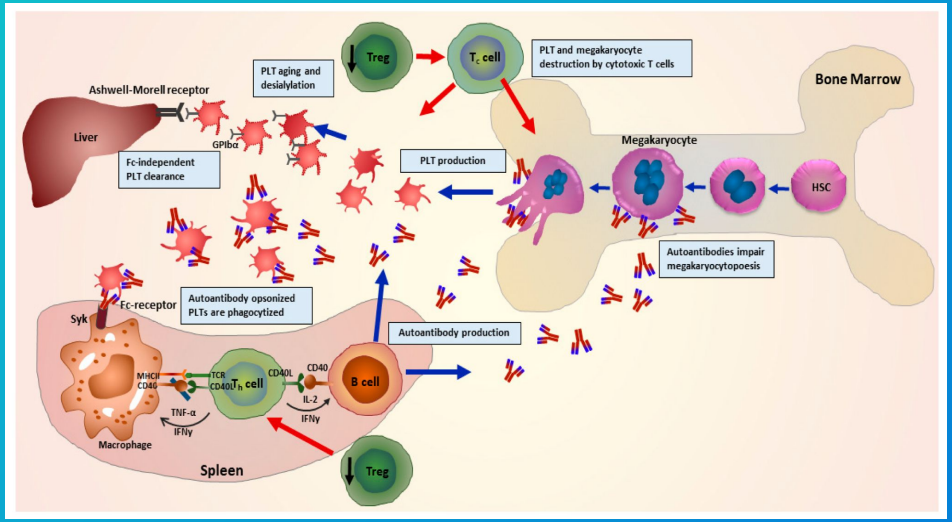


Image 4

In ITP:

- Immune tolerance breaks down → IgG attaches to body's own cells
- Treg cells are impaired → unchecked harmful IgG production
- Leads to destruction of platelets and megakaryocytes → thrombocytopenia

Clinical Features - Symptoms and Signs

- **Petechiae:**
  - 60-100%
  - Most common cutaneous manifestation; often first sign in children and adults
- **Purpura:**
  - 40-80%
  - Typically accompanies petechiae in moderate thrombocytopenia
- **Mucosal Bleeding:**
  - 30-65%
  - Includes epistaxis, gum bleeding, and oral blood blisters; severity correlates with platelet count

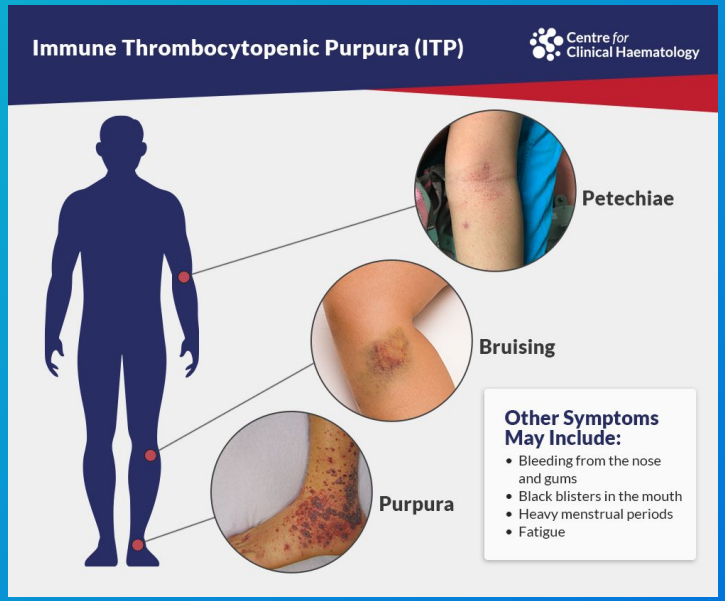


Image 5

Clinical Features - Symptoms and Signs

- **Fatigue:**
  - 30-40%
  - Common in adults, possibly underreported; often chronic
- **Menorrhagia:**
  - 20-40% in women
  - One of the most frequent presentations in menstruating females
- **Hematuria:**
  - 5-10%
  - Less common; may indicate severe thrombocytopenia
- **Hematoma:**
  - <5%
  - Usually in more severe cases or after trauma

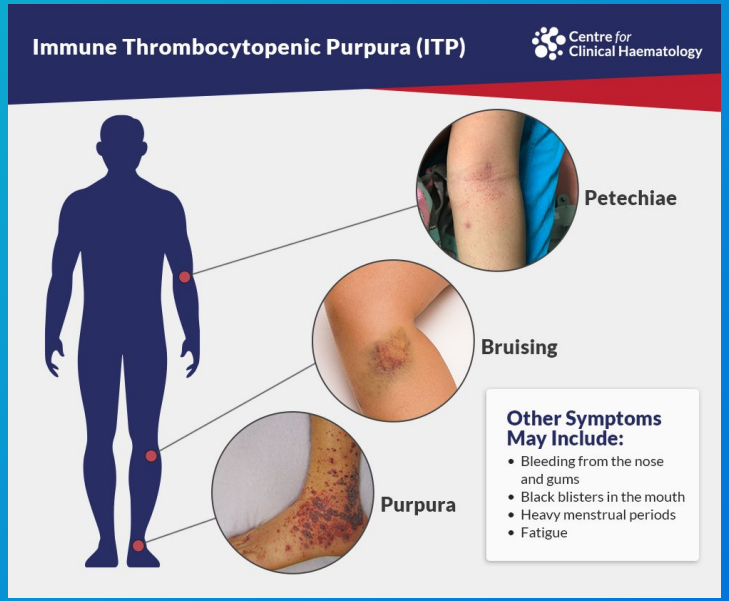


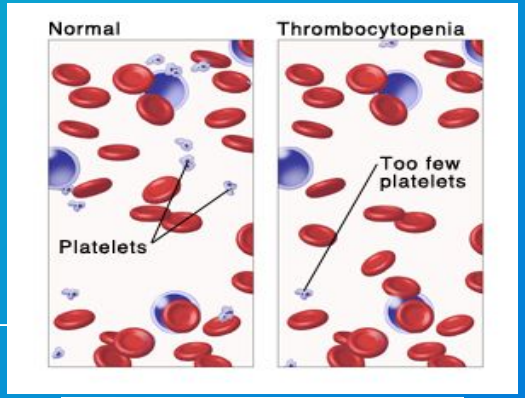
Image 5

Clinical Features - Investigations

All Patients:

- **CBC (Complete Blood Count):**
  - Low platelet count (often <100,000/µL), WBCs and RBCs are typically normal
  - Initial test in any patient with unexplained bleeding or bruising
  - If isolated thrombocytopenia is found with normal WBCs and RBCs – suspect ITP
- **Peripheral Blood Smear:**
  - Microscopic review of blood to assess cell shape, size, and distribution
  - Helps rule out other causes of low platelets (e.g., clumping, schistocytes from TTP)
  - Low platelets, but normal in appearance
  - No platelet clumping (rules out pseudothrombocytopenia)
  - No schistocytes, blasts, or abnormal cells (helps exclude TTP, leukemia, etc.)

Image 6



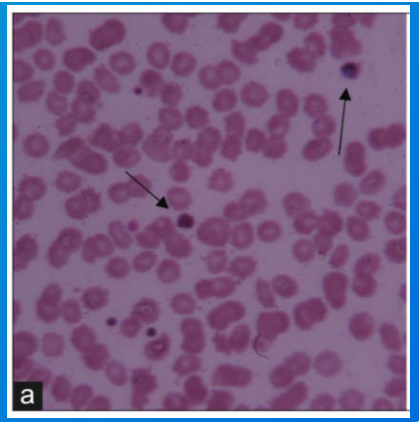


Image 7

Clinical Features - Investigations

Secondary:

- **Bone Marrow Biopsy:**
  - Assesses bone marrow cellularity and megakaryocytes (platelet precursors)
  - In ITP, normal or increased megakaryocytes suggest peripheral destruction
  - Normal to increased megakaryocytes (large platelet-producing cells)
  - Otherwise normal marrow cellularity
  - No dysplasia or infiltration (rules out leukemia, MDS, etc.)
  - Not routinely done in typical ITP
  - Reserved for: Atypical presentation (e.g., pancytopenia, abnormal smear), Age >60, Non-responders to therapy, Before splenectomy
- **HIV and Hepatitis C Screening**

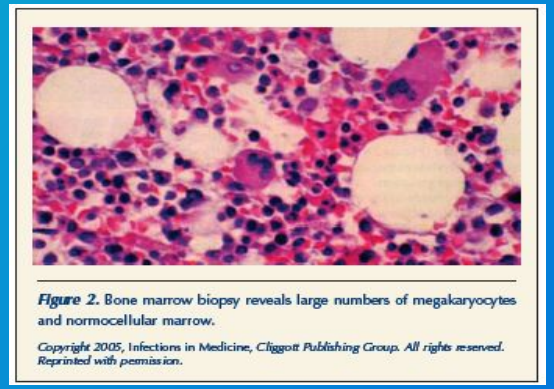


Figure 2. Bone marrow biopsy reveals large numbers of megakaryocytes and normocellular marrow.

Image 8

Clinical Features - Investigations

Secondary:

- **Autoimmune Markers:**
  - Screens for underlying autoimmune diseases such as SLE (Systemic Lupus Erythematosus), which can present as secondary ITP
  - High-titer ANA or positive anti-dsDNA → points toward SLE
  - Helps differentiate primary ITP from autoimmune-related thrombocytopenia

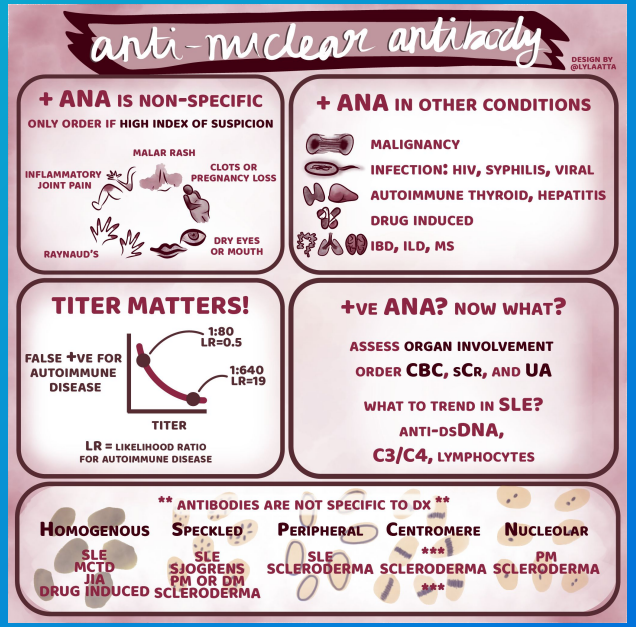


Image 9

Clinical Features - Investigations

Test	What It Checks	What You See	When to Use
CBC	Blood cell counts	Isolated ↓platelets; normal WBCs & RBCs	First-line for any bleeding/bruising patient
Peripheral Smear	Cell appearance	↓Platelets, normal shape; no clumping, no blasts, no schistocytes	Always after abnormal CBC
Bone Marrow	Megakaryocyte activity, Bone marrow health	↑Megakaryocytes; otherwise normal marrow	Atypical cases, elderly, non-responders
HIV/HCV Screen	Viral infections	Positive or negative viral markers (serology/RNA)	All new ITP cases, esp. with risk factors
Autoimmune Panel	Autoimmune causes (like lupus)	Positive ANA ± anti-dsDNA = autoimmune process	If systemic signs or young female

Treatments/Therapies for ITP: Key Principles

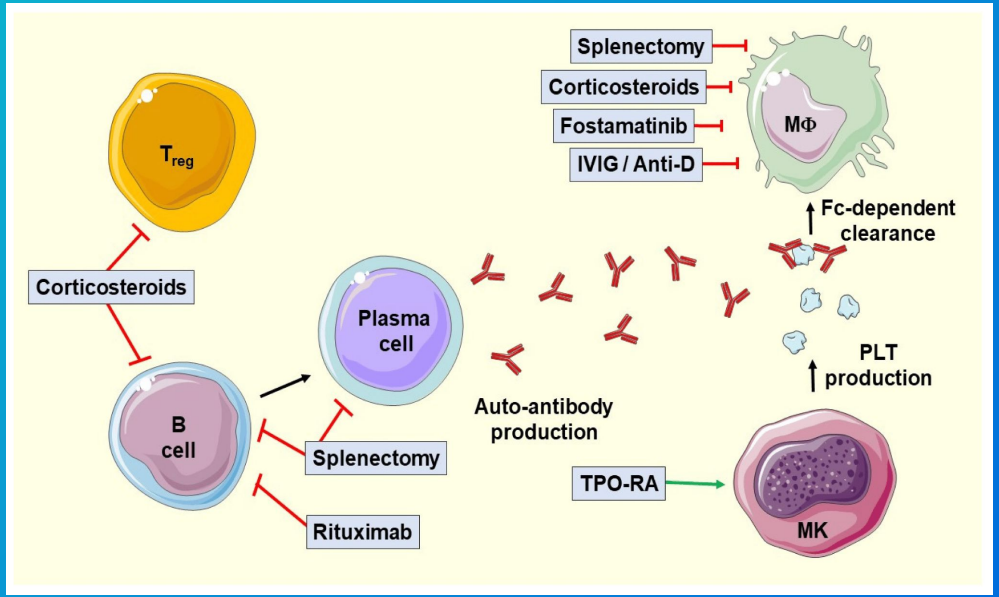
Goal: Prevent bleeding and maintain a safe platelet count, not necessarily normalize it

Approach: Balance between reducing platelet destruction and stimulating platelet production

Therapy choice depends on:

- Severity of thrombocytopenia
- Bleeding risk
- Chronicity (acute vs chronic)
- Age, comorbidities, patient preference

Image 10



Treatments - Current Therapies/Treatments for ITP

First-line Therapies:

- **IVIG (Intravenous Immunoglobulin):**
  - Blocks Fc receptors on splenic macrophages → protects antibody-tagged platelets
  - Platelet count <10,000/uL
  - Pre-surgery or high bleeding risk
- **Corticosteroids (Prednisone, Dexamethasone):**
  - Suppresses autoimmune response and antibody production
  - First-line in newly diagnosed ITP
- **Anti-D Immunoglobulin:**
  - Induces mild hemolysis → diverts immune destruction away from platelets
  - Causes transient hemolysis that distracts the immune system, leading to temporary preservation of platelets
  - Less effective in adults than children and contraindicated in Rh-negative or splenectomized patients

Image 11

First-Line Therapies/Treatments

Therapy	Mechanism	Effect	When to Use	When to Not Use
IVIG (Intravenous Immunoglobulin)	Blocks Fc receptors on splenic macrophages → protects antibody-tagged platelets	Rapid but temporary increase in platelet count	-Emergency bleeding -Platelet count <10,000/uL -Pre-surgery or high bleeding risk	-Not ideal for long-term management -Avoid if cost or IV access is a concern
Corticosteroids (Prednisone, Dexamethasone)	Suppresses autoimmune response and antibody production	Increases platelets in most within 3–7 days	-First-line in newly diagnosed ITP -Often combined with IVIG for severe cases	-Avoid long-term use due to side effects (e.g., diabetes, osteoporosis, infection risk)
Anti-D Immunoglobulin	Induces mild hemolysis → diverts immune destruction away from platelets	Transient hemolysis that distracts immune system; short-lived (7–21 days)	-Rh-positive, non-splenectomized patients only -Alternative to IVIG in specific populations	-Do NOT use in Rh-negative or splenectomized patients -Use caution in adults

Treatments - Current Therapies/Treatments for ITP

Second-line Therapies:

- **Rituximab (Anti-CD20 Monoclonal Antibody):**
  - Binds to CD20 on B-cells, leading to B-cell depletion → decreases autoantibody production
  - Relapses are common after 1–2 years
  - Preferred in patients avoiding splenectomy
  - Young adults, especially women, respond better
- **Thrombopoietin Receptor Agonists (TPO-RAs) (Eltrombopag – oral, Romiplostim – injectable):**
  - Stimulates the TPO receptor (c-Mpl) on megakaryocytes → increased platelet production
  - Platelet response in 70–90% of chronic ITP patients
  - Patients unresponsive to or relapsing after corticosteroids/rituximab
  - Favorable for patients wishing to avoid surgery

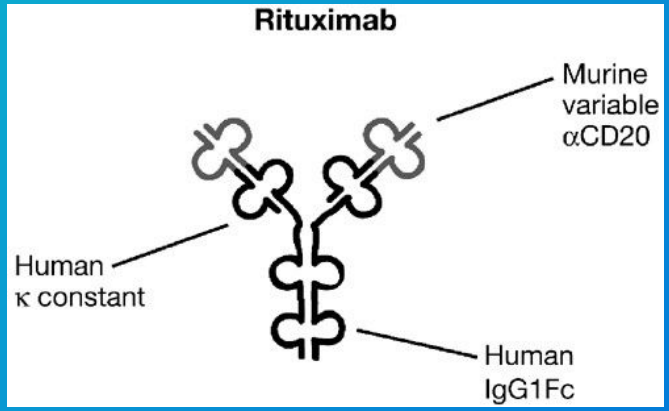


Image 12

Second-Line Therapies/Treatments

Therapy	Mechanism	Effect	When to Use	When to Not Use
Rituximab	Binds CD20 on B-cells → B-cell depletion → decreases autoantibody production	-Increases platelets in 50–60% of refractory cases -Takes 2–8 weeks -Relapses after 1–2 years common	-Steroid-refractory or relapsed ITP -Preferred in patients avoiding splenectomy -Young adults, especially women	-Not ideal for rapid response -Avoid in patients with severe immunosuppression or chronic infections
TPO Receptor Agonists	Stimulates TPO receptor (c-Mpl) on megakaryocytes → ↑ platelet production	-70–90% platelet response in chronic ITP -Effect in 1–2 weeks -Maintained with ongoing use; not curative	-Chronic ITP unresponsive to corticosteroids/rituximab - Surgery-avoidant patients	-Avoid in patients with risk of thrombosis -Monitor liver function with eltrombopag

Treatments - Current Therapies/Treatments for ITP

Third-line Therapies:

- **Immunosuppressants (Azathioprine, Mycophenolate mofetil, Cyclophosphamide, Cyclosporine (off-label use))**
  - Suppress T- and B-cell activity, leading to reduced autoantibody production and decreased immune-mediated platelet destruction
  - Not curative; relapse can occur if therapy is stopped
  - Used when patients fail or cannot tolerate Corticosteroids, Rituximab, TPO receptor agonists, or are not surgical candidates for splenectomy
- **Combination Therapy (For multi-refractory cases):**
  - Targets multiple immune pathways simultaneously, combining mechanisms such as: Decreasing platelet destruction, Suppressing antibody production, Increasing platelet production
  - Useful in life-threatening bleeding or rapid count recovery needs
  - Severe or acute bleeding unresponsive to first-line agents
  - Chronic refractory ITP where monotherapy has failed

Treatments - Current Therapies/Treatments for ITP

Third-line Therapies:

- **Splenectomy:**
  - Removes the primary site of platelet destruction and autoantibody production
  - Risk of post-splenectomy infections and surgical complications
  - Consider after failure of steroids, rituximab, or TPO-RAs
  - Requires vaccination before procedure

Third-Line Therapies/Treatments

Therapy	Mechanism	Effect	When to Use	When to Not Use
Immunosuppressants	Suppress T- and B-cell activity → ↓ antibody production and immune-mediated destruction	-Partial/complete response in 40–70% -Takes weeks to months -Not curative; relapse common	-Third-line for multi-refractory ITP -When unable to tolerate other therapies	-Avoid in immunocompromised patients -Risk of infections and long-term toxicity
Combination Therapy	Combines multiple mechanisms (↓ destruction, ↑ production, ↓ antibody production)	-Faster, synergistic response -Sustained improvement in severe cases	-Life-threatening bleeding -Failed monotherapy -Temporizing while awaiting other therapies	-Avoid if individual drugs have significant interaction risks or toxicity
Splenectomy	Removes site of platelet destruction and antibody production	-60–70% long-term remission -Sustained platelet rise -Risk of infection	-Third-line for chronic, refractory ITP -After steroid, rituximab, or TPO-RA failure	-Avoid in children <5 -Patients with high surgical risk -Pre-vaccination required

Treatments - Emerging Therapies/Treatments for ITP

Emerging Therapies:

- **Fostamatinib:**
  - Inhibits SYK (spleen tyrosine kinase) → blocks phagocytosis of antibody-coated platelets by splenic macrophages
  - Especially useful in patients failing TPO-RAs and rituximab
  - FDA-approved for chronic ITP refractory to ≥2 prior therapies
  - Option before or after splenectomy
- **Rilzabrutinib:**
  - Inhibits Bruton's Tyrosine Kinase (BTK) → reduces B-cell signaling and macrophage-mediated platelet destruction
  - Fewer immune-related side effects than rituximab
  - Potential alternative to immunosuppressants or splenectomy

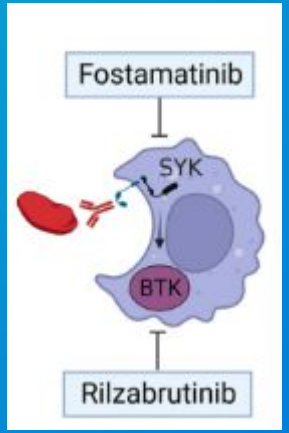


Image 13

Treatments - Emerging Therapies/Treatments for ITP

Emerging Therapies:

- **Efgartigimod:**
  - Inhibits neonatal Fc receptor (FcRn) → enhances clearance of pathogenic IgG antibodies
  - Reduces circulating autoantibody levels
  - Improves platelet counts in patients with autoimmune destruction
  - Investigational for chronic ITP and other antibody-mediated diseases
  - Especially for multi-refractory patients

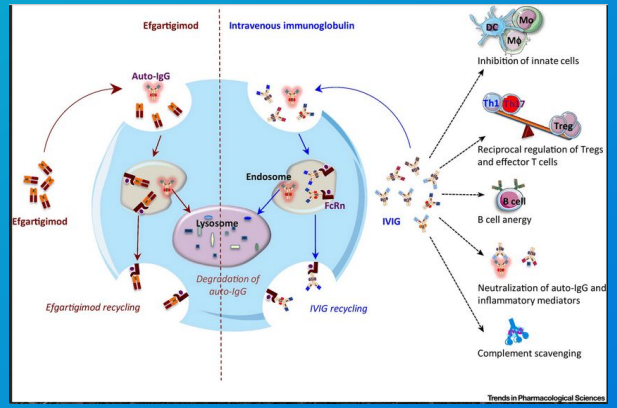


Image 14

Treatments - Emerging Therapies/Treatments for ITP

Emerging Therapies:

- **Rozanolixizumab:**
  - Also inhibits FcRn, reducing IgG lifespan and lowering autoantibody levels
  - Demonstrated platelet count improvement in early clinical trials
  - May provide faster onset than traditional immunosuppressants
  - In clinical development for autoimmune ITP
  - Future option for patients intolerant or resistant to standard therapies

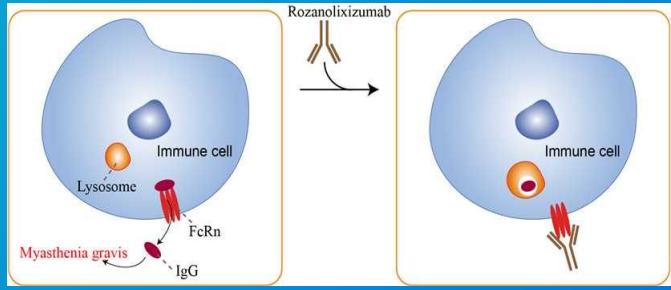


Image 15

Treatments - Emerging Therapies/Treatments for ITP

Emerging Therapies:

- **Veltuzumab:**
  - Humanized anti-CD20 monoclonal antibody (similar to rituximab) → B-cell depletion, but designed for subcutaneous use and improved safety
  - May offer safer profile and better tolerability than rituximab
  - In clinical trials for relapsed/refractory ITP
  - Not yet approved; potential future substitute for rituximab

Emerging Therapies/Treatments

Therapy	Mechanism	Effect	When to Use	When to Not Use
Fostamatinib	Inhibits SYK → blocks phagocytosis of antibody-coated platelets	-30–40% response in refractory cases -Good for TPO-RA/rituximab failures	-Chronic ITP refractory to ≥2 therapies -Before/after splenectomy	-Avoid in uncontrolled hypertension or liver dysfunction
Rilzabrutinib	Inhibits BTK → ↓ B-cell signaling & macrophage-mediated destruction	-Early platelet responses -Fewer immune-related side effects	-Investigational for multi-refractory ITP -Possible alternative to splenectomy	-Not FDA-approved -Avoid outside clinical trial unless compassionate use
Efgartigimod	Inhibits FcRn → clears pathogenic IgG antibodies	-↓ autoantibodies -↑ platelet count in autoimmune disease	-Investigational for chronic ITP -Multi-refractory patients	-Not yet FDA-approved -Avoid using outside trials
Rozanolixizumab	Also inhibits FcRn → reduces IgG lifespan	-Improves platelet counts in early trials -Faster onset	-Clinical development for ITP -Alternative for standard therapy intolerance	-Experimental use only -Monitor for infections or adverse immune effects
Veltuzumab	Anti-CD20 mAb (like rituximab) designed for SC use with better safety	-Promising in relapsed autoimmune cytopenias	-In clinical trials -Potential rituximab substitute	-Not yet approved -Use limited to research settings

Prognosis

Children:

- ~80% recover spontaneously within 6 months
- Rarely require long-term treatment
- Low risk of major bleeding

Adults:

- 60–70% develop chronic ITP
- Platelet counts fluctuate → need for ongoing monitoring
- Worse prognosis in older adults and women >60

Complications

Severe Hemorrhage:

- Occurs in 5–10% of adults
- Intracranial hemorrhage is rare (<1%) but life-threatening
- Highest risk: Platelet count <10,000/uL

Post-Splenectomy Infections:

- Risk of OPSI (overwhelming post-splenectomy infection) <1%
- Vulnerable to encapsulated organisms: Strep. pneumoniae, H. influenzae, Neisseria meningitidis
- Preventable with vaccinations and possible lifelong prophylactic antibiotics

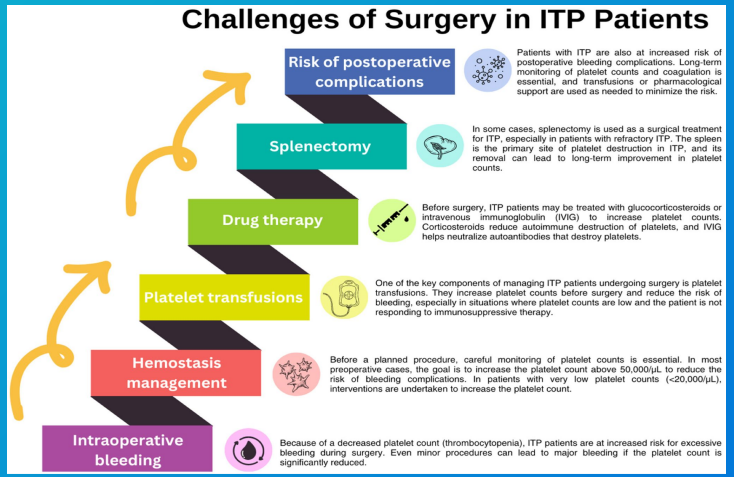


Image 16

Conclusions

- **Future Direction:**
  - Advancements in ITP research have shifted the therapeutic focus from symptomatic treatment to targeted, immune-based approaches
  - Novel therapeutics, including FcRn inhibitors, BTK inhibitors, and other monoclonal antibodies, continue to expand the treatment landscape
  - Novel agents like BTK and SYK inhibitors offer new hope, especially for refractory cases
  - Future directions include:
    - ***Precision Medicine:***
      - Tailoring therapy based on patient-specific immunological profiles and genetic markers (5, 7).
    - ***Long-Term Outcome Studies:***
      - Ongoing trials aim to evaluate the durability, safety, and cost-effectiveness of novel agents (5).
    - ***Biomarker Development:***
      - Identifying predictive markers to guide therapy response and risk stratification (7).

Acknowledgements

We would like to thank Dr. Nahar for mentoring us and would also like to thank Loyola University and GTF for this opportunity!

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THANK YOU