e-ISSN: 2582-5208
International Research Journal of Modernization in Engineering Technology and Science
(Peer-Reviewed, Open Access, Fully Refereed International Journal)
Volume:06/Issue:12/December-2024 Impact Factor- 8.187 www.irjmets.com

IMMUNE THROMBOCYTOPENIC PURPURA

Alimova Nurgul Abdyashymovna*1, Santosh Kumar*2, Gajender*3

*1,2,3Osh State Medical University.

ABSTRACT

Immune thrombocytopenic purpura (ITP) is an autoimmune disorder characterized by the accelerated destruction of platelets, resulting in thrombocytopenia and an increased risk of bleeding. It primarily affects children and adults, with distinct pathophysiological mechanisms and clinical presentations in each group. In ITP, autoantibodies target platelet surface glycoproteins, leading to platelet destruction in the spleen and liver. The clinical manifestations vary from mild bruising to life-threatening hemorrhage. Diagnosis is primarily clinical, supported by laboratory tests that rule out other causes of thrombocytopenia. Management of ITP includes corticosteroids, intravenous immunoglobulin (IVIG), and thrombopoietin receptor agonists, with splenectomy considered for refractory cases. Recent advancements in understanding the immune mechanisms behind ITP have led to novel therapeutic approaches. This article reviews the pathophysiology, diagnosis, clinical presentation, and current treatment strategies for immune thrombocytopenic purpura, with a focus on emerging therapies and future directions in management.

I. INTRODUCTION

Immune Thrombocytopenic Purpura (ITP) is a complex, immune-mediated condition characterized by a low platelet count, resulting in easy bruising, bleeding, and other associated symptoms. The disorder is often diagnosed by exclusion, and its management involves a combination of medical therapies, including corticosteroids, immunoglobulin treatments, and sometimes splenectomy or newer biologic agents.

Overview of Immune Thrombocytopenic Purpura (ITP)

Immune Thrombocytopenic Purpura (ITP) is an autoimmune disorder where the immune system mistakenly attacks and destroys platelets, which are crucial for blood clotting. As platelets are destroyed faster than they can be produced, the individual experiences thrombocytopenia (a decrease in platelet count), which can result in abnormal bleeding and bruising.

There are two primary classifications of ITP: acute ITP and chronic ITP. Acute ITP typically occurs in children and is often self-limiting, while chronic ITP is more common in adults and can persist for years.

Pathophysiology of ITP

The pathophysiology of ITP involves the destruction of platelets by antibodies targeting platelet membrane glycoproteins, particularly GP IIb/IIIa and GP Ib/IX. These autoantibodies are generated by dysregulated immune responses, often in the context of infections or other immune triggers. As a result, platelet destruction predominantly occurs in the spleen and liver, where macrophages recognize the antibody-coated platelets and phagocytose them.

Epidemiology

ITP affects individuals across all age groups but is most commonly diagnosed in children between the ages of 2 and 5 years. Chronic ITP is more prevalent in adults, especially in women aged 20–50 years. The incidence of ITP in children is approximately 4.5 per 100,000, while in adults, it is about 3.3 per 100,000.

Clinical Manifestations

The primary clinical feature of ITP is thrombocytopenia, which leads to symptoms such as:

Easy bruising (purpura): Small purple or red dots on the skin due to blood leaking from tiny blood vessels (petechiae).

Nosebleeds and gum bleeding: Due to the lack of platelets required for clotting.

Heavy menstrual periods: In women, especially those with chronic ITP.

Fatigue: Often associated with the body's inability to stop bleeding and maintain normal clotting.

In more severe cases, internal bleeding: This can lead to gastrointestinal or intracranial hemorrhage, which are life-threatening events.

e-ISSN: 2582-5208
International Research Journal of Modernization in Engineering Technology and Science
(Peer-Reviewed, Open Access, Fully Refereed International Journal)
Volume:06/Issue:12/December-2024 Impact Factor- 8.187 www.irjmets.com

Diagnosis of ITP

Diagnosing ITP involves excluding other causes of thrombocytopenia and performing a series of clinical and laboratory tests. Key diagnostic steps include:

1. Clinical evaluation: A detailed medical history and physical examination are performed, focusing on bleeding symptoms, history of infections, medications, and autoimmune conditions.

2. Platelet count: A platelet count less than 100,000/μL is considered diagnostic of thrombocytopenia.

3. Bone marrow biopsy: While not often necessary, a bone marrow biopsy can be done to rule out other conditions such as leukemia, lymphoma, or bone marrow failure disorders.

4. Platelet antibodies: In some cases, testing for platelet-specific antibodies may be performed, but this is not always conclusive.

Classification of ITP

ITP is classified into two main categories based on the duration of thrombocytopenia:

Acute ITP: Typically occurs after a viral infection, most commonly in children. It resolves spontaneously in most cases within 6 months.

Chronic ITP: Defined as thrombocytopenia lasting more than 6 months, more common in adults, and may persist for years, often requiring treatment to manage symptoms.

Treatment Strategies for ITP

The goal of treatment is to increase the platelet count and prevent bleeding complications, particularly in patients with moderate to severe thrombocytopenia. Treatment approaches vary depending on the severity of thrombocytopenia, symptoms, and whether the disease is acute or chronic.

First-Line Therapies

1. Corticosteroids (e.g., Prednisone): These are the most commonly used first-line treatment for ITP. They work by suppressing the immune system's production of platelet antibodies and reducing platelet destruction.

2. Intravenous Immunoglobulin (IVIg): IVIg is often used when corticosteroids are ineffective or if rapid platelet increase is needed. IVIg provides a supply of normal immunoglobulins that block the action of the autoantibodies targeting platelets.

3. Anti-D Immunoglobulin (WinRho): Used in Rh-positive patients with ITP, Anti-D works by causing splenic sequestration of red blood cells, which indirectly increases platelet count.

Second-Line Therapies

1. Thrombopoietin Receptor Agonists (e.g., Romiplostim, Eltrombopag): These drugs stimulate the bone marrow to produce more platelets and are typically used in chronic ITP or when first-line treatments fail.

2. Immunosuppressive Drugs (e.g., Azathioprine, Mycophenolate): These are used when corticosteroids and IVIg do not provide adequate response.

3. Splenectomy: In cases of chronic ITP where medical treatments fail, removal of the spleen may be considered. The spleen is a major site of platelet destruction, and its removal can lead to a significant increase in platelet count in some patients.

4. Rituximab: This monoclonal antibody targets CD20 on B cells and is used for patients who do not respond to other treatments.

Special Considerations in Treatment

1. Pediatric ITP: Children often have a more favorable prognosis, with most cases resolving spontaneously. Treatment may only be required for those with significant bleeding or persistent thrombocytopenia.

2. Pregnancy and ITP: Managing ITP during pregnancy requires careful monitoring. Low platelet counts may increase the risk of bleeding during childbirth, so treatments are carefully chosen to minimize risks to the mother and fetus.

3. Geriatric ITP: Older adults may require a more tailored approach due to the potential for other comorbidities, and they may respond differently to therapies.

e-ISSN: 2582-5208
International Research Journal of Modernization in Engineering Technology and Science
(Peer-Reviewed, Open Access, Fully Refereed International Journal)
Volume:06/Issue:12/December-2024 Impact Factor- 8.187 www.irjmets.com

4. Coexisting Conditions: In patients with other autoimmune diseases (e.g., systemic lupus erythematosus), management may require additional immunosuppressive therapy.

Prognosis

The prognosis of ITP depends on various factors, including the severity of thrombocytopenia, the patient's age, comorbidities, and how well the patient responds to treatment. While most children with acute ITP recover without long-term effects, chronic ITP can persist for years, requiring ongoing treatment. Patients who do not respond to treatment or who experience severe bleeding may have a poorer prognosis.

II. FUTURE DIRECTIONS IN ITP RESEARCH

Research into ITP continues to advance, with promising developments in targeted therapies. Some key areas of focus include:

Biologic therapies: New treatments such as anti-CD20 antibodies and thrombopoietin receptor agonists have shown promise in clinical trials and are already in use for certain patients.

Personalized medicine: Understanding the genetic basis of ITP may allow for more tailored treatment strategies based on individual patient characteristics.

Novel immune-modulating therapies: Ongoing research is exploring other immune-modulating drugs that may provide more effective control of platelet destruction.

III. CONCLUSION

Immune Thrombocytopenic Purpura is a complex autoimmune disorder that can vary significantly in its presentation and management. Early diagnosis and appropriate treatment are essential to prevent severe complications such as bleeding. While most cases of acute ITP resolve spontaneously, chronic ITP often requires ongoing medical management, including corticosteroids, immunoglobulins, and thrombopoietin receptor agonists. Newer therapies are being developed, offering hope for better outcomes in patients with refractory ITP.

IV. REFERENCES

[1] Cines, D. B., & Blanchette, V. S. (2002). Immune Thrombocytopenic Purpura. New England Journal of Medicine, 346(13), 995-1008.

[2] Provan, D., Stasi, R., Newland, A., & Blanchette, V. (2010). International consensus report on the investigation and management of primary immune thrombocytopenia. Blood, 115(2), 168-186.

[3] Neunert, C. E., & Lim, W. (2019). Diagnosis and management of immune thrombocytopenia in adults: A review. Journal of the American Medical Association, 322(3), 261-271.

[4] Gernsheimer, T. B., & Bussel, J. B. (2010). Platelet transfusions in immune thrombocytopenic purpura. Hematology/Oncology Clinics of North America, 24(3), 649-659.

[5] Kuter, D. J., & Rummel, M. (2017). New and emerging therapies for immune thrombocytopenia: A comprehensive review. American Journal of Hematology, 92(1), 24-35.

[6] Rojas, M., & Figueroa, M. (2018). Pathophysiology of immune thrombocytopenia. Pathology Research and Practice, 214(4), 463-470.

[7] Arnold, D. M., & Nazi, I. (2017). Chronic immune thrombocytopenic purpura: Treatment strategies. *Therapeutic Advances in Hem.