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Idiopathic Thrombocytopenic Purpura : A Review and Case Report

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ABSTRACT

Immune thrombocytopenia (ITP) is a diverse condition in which managing and predicting patient responses to therapy poses a problem. It can develop in healthy people, including kids and young adults, after a viral infection and significantly impair a patient's quality of life. Thrombopoietin receptor agonists (TPO-RAs), spleen tyrosine kinase (Syk) inhibitors, or immunological suppression (corticosteroids and intravenous immunoglobulins) are the mainstays of ITP treatment. This article provides a quick overview of the occurrence, aetiology, classification, and several therapy approaches based on recent research. We also present a case report on ITP and summarize the key points in the diagnosis and management of it. In order to improve the disease prognosis and lower the incidence or frequency of refractoriness, a deeper understanding of ITP is needed in order to offer patients with individualized therapy alternatives.

Keywords: Immune thrombocytopenia, Thrombopoietin receptor agonists, corticosteroids, Syk inhibitors, immunoglobulins.

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INTRODUCTION

Idiopathic Thrombocytopenic Purpura [ITP] is a disorder of no recognized cause characterized by low platelet count (<1,50,000 cells/μL) in peripheral blood which increases the risk of bleeding and partial clot formation[1,2]. Because of its immune-mediated rapid platelet destruction and decreased platelet synthesis, it is often referred to as immunological thrombocytopenic purpura [3]. Autoantibodies against immunoglobulin G (IgG) cause circulating platelets to become sensitized, resulting in purpura, hemorrhagic episodes, and low platelet counts[4]. Risk of bleeding depends on platelets level, with counts <50000 cells/μL indicating a risk of dangerous bleeding from trauma and counts <20000 cells/μL indicating a risk of spontaneous bleeding [2].

METHOD

Method of Search: Research on ITP and articles collected through various database searches as medscape, pubmed, lexicomp.

Types of ITP are:

Primary ITP: Occurs without an underlying cause and can be further classified based on timing and persistence of symptoms as shown in Table 1. Secondary ITP: Occurs from an underlying cause, by drug responses, autoimmune disorders such as Systemic Lupus Erythematosus (SLE), Chronic Lymphocytic Leukaemia (CLL), Human Immunodeficiency Virus (HIV) [4].

Table 1: Types of Primary ITP [4]

Types of Primary ITP	Timing and persistence of symptoms
Newly diagnosed ITP	0-3 months from diagnosis
Persistent ITP	3-12 months from diagnosis
Chronic ITP	> 12 months from diagnosis
Refractory ITP	Cases that do not resolve with splenectomy

Epidemiology:

The study conducted primarily in Europe showed that ITP occurs in up to 6.4 per 100,000 children and 3.3 per 100,000 adults per year. Chronic ITP affects around 20% of children, with a larger risk if they are over 10 years old or have platelet count of 20000 cells/μL at presentation. Approximately 37-50% of children with chronic ITP experience remission within 4 years of diagnosis [5]. The prevalence of ITP in men and women is approximately the same after 65 years. Patients with ITP are at higher risk of thromboembolism due to various factors such as the residual platelets, though few, are often hyperactive and pro-thrombotic, the underlying systemic inflammation contributes to endothelial dysfunction and a pro-coagulant state, sometimes compounded by the presence of anti-phospholipid antibodies, and crucially several ITP

treatments, including thrombopoietin receptor agonists, splenectomy, and corticosteroids, can independently increase thrombotic risk, further complicated by the common presence of conventional cardiovascular risk factors in ITP patients [6]. This necessitates a careful, individualized approach in ITP management, balancing the risks of both hemorrhage and thrombosis. Secondary ITP accounts for around 20% of ITP diagnosis. An epidemiological investigation in France reported an 18% incidence of secondary ITP [7]. ITP affects 50-100 new cases per million people annually, with children making up half of the total [2]. Patients with isolated ITP and a positive antinuclear antibody [ANA] have higher risk of developing systemic autoimmune disorders like SLE [8].

Etiology:

ITP majorly caused by decreased bone marrow growth or platelet breakdown in the peripheral blood circulation [1]. Other causative factors include:

1. Low platelet count and geriatrics are associated with increased risk of bleeding [7].
2. Viral infections that occur due to immune-mediated platelet death, with or without megakaryocyte damage such as Dengue fever, hepatitis B and C, influenza, herpes, cytomegalovirus, HIV, parvovirus, mumps, and other viral infections are common causes of thrombocytopenia.
3. Drug-induced thrombocytopenia results from heparin, quinine, sulfonamides, ampicillin, vancomycin, piperacillin, acetaminophen, ibuprofen, naprones, cimetidine, and glycoprotein IIb/IIIa inhibitors.
4. Also Iron deficiency anemia causing thrombocytopenia has been reported in a study [9].

Pathophysiology:

This involves various molecular and cellular mechanisms for ITP Pathogenesis as below:

Humoral Response and Antiplatelet antibodies:

The involvement of the humoral reaction against platelets has been known since the 1950s. In a study it was observed that ITP patient's plasma resulted in thrombocytopenia when infused into healthy volunteers due to transfer of autoantibodies. It was also shown that IgG was responsible for this impact, as it binds to several platelet glycoproteins, including GPIIb/IIIa, GPIb/IX [10]. Platelets with bound autoantibodies are identified by phagocytes bearing Fcγ receptors, resulting in increased antibody-mediated platelet phagocytosis and death largely in the spleen [11]. The spleen is considered as the site of aggressive autoimmune response that results in the growth of germinal centres, mutant formation and high affinity antiplatelet antibody secreting plasma cells. Some of these plasma cells move to the bone marrow and can dwell there for a long

time. Although IgM antiplatelet antibodies have been detected, the majority of antibodies are IgG, seen in 77% of the patients with somatic hypermutation in variable Immunoglobulin genes. Splenic T follicular helper cells (TFHs) are involved in the selection of autoreactive B-cell clones in germinal centres [10]. These autoantibodies bind to megakaryocytes, inhibiting their maturation or causing their demise, while thrombopoietin (TPO) is unable to normalize platelet count [11].

Peripheral destruction of platelets:

Occurs in the blood, spleen, liver due to abnormal bone marrow formation caused by an immunological reaction to megakaryocytes and low TPO levels due to complement-dependent cytotoxicity. Antibodies attach to platelet glycoprotein, activate the normal complement system and form the Membrane Attack Complex (MAC), which causes platelet lysis [10]. Complement activation causes C3b deposition on platelets, promoting phagocytosis by macrophages in the spleen which is mediated by the ligation of antiplatelet antibodies to FCγR and C3b to CR1. Platelet desialylation is an FCγR-independent route of platelet death, that removes sialic acids, sugars on platelet surfaces. GPIb/IX antibodies cause platelet desialylation by recruiting neuraminidase-1 to the plasma membrane. Desialylated platelets are identified by the Ashwell-Morell receptor expressed on hepatocytes, resulting in their removal from circulation and the generation of TPO, a significant growth factor of megakaryocytes. During ITP, monocytes exhibit increased expression of FCγR I and FCγRIIa/FCγRIIb ratio, leading to enhanced phagocytic potential [12].

T-cell Dysregulation:

Due to breakdown in self-tolerance of platelets, the antigen presenting cell process is activated, which presents platelet autoantigens to autoreactive T cells. This triggers a cascade of events including stimulation of autoantibody production, cytotoxic T Lymphocytes (CTL) activation and proliferation, abnormal number and function of regulatory T cells (Tregs), production of abnormal T helper cells and abnormal T-cell anergy [12]. T-helper cells recognize peptide antigens and associate them with MHC complexes on APC [13]. Tregs maintain immunological tolerance by controlling B-cell and T-cell-mediated autoimmunity and inducing a tolerogenic state through interactions with dendritic cells. A loss of peripheral tolerance is caused by the suppression of regulatory T cell activity, which is indicated by a decrease in their production and/or function. The severity of ITP is related to the level of Treg abnormalities. Multiple T helper populations (Th1/Th17/Th22/Tregs) contribute to the pathogenesis of ITP, with proinflammatory Th17 cells expressing the CD154 surface receptor and producing IL-21, which promotes the development of

autoreactive B-cells and generation of antiplatelet antibodies. elevated levels of Th1/Th2 cells could increase severity of chronic ITP [12]. CTL also affect megakaryocytes as evidenced by their increased recruitment in the bone marrow which is triggered by fractalkine, a chemokine that binds to CX3CR1 produced by CTL and their potential to interfere with platelet synthesis [10].

Impaired thrombopoiesis:

Megakaryocytes have been shown to have an innate defect in platelet generation and a reduced ability to create proplatelets [12].

Clinical manifestations include Petechiae, Purpura, Bruises, Bleeding gums, Blood in urine, Heavy menstrual periods, Hematoma, Hemorrhage, Heavy nose bleeds, Fatigue, Anaemia and Iron deficiency, Prolonged bleeding from cuts, Enlarged spleen.

Diagnosis: The identification of ITP is based on the diagnosis of exclusion such as:

1. Low platelets is the major clinical haematological identification apart from severe bleeding [2].
2. Family history of thrombocytopenia, comorbidities, bleeding history, and duration of thrombocytopenia [14].
3. Presence of antiplatelet antibodies in the blood [2].
4. Prothrombin time and activated partial thromboplastin time assessment required to schedule invasive procedures or assess the risk of bleeding.
5. Individuals who experience recurring epigastric pain suggestive of a peptic ulcer should be checked for Helicobacter pylori infection, which could be probable cause of ITP [4].
6. Bone marrow examination to be performed as a precautionary test for ITP diagnosis in patients who are above 60 years, patients who do not respond to treatment, have relapse of ITP after remission and in patients before splenectomy. This must include aspiration, biopsy, flow cytometry, cytogenetic and molecular biology studies. Any rise in megakaryocytes may determine ITP [2,14].

Treatment

1. The therapeutic goal is to minimize bleeding and increase platelet count to 20000 - 30000 cells/µL while ensuring patient safety [13,15].
2. For patients with no major bleeding but platelet count of less than 30000 cells/µL, a haematology consultation is mandated as treatment is multifactorial and individualized[15].
3. Hospital admission recommended for patients with a platelet count less than 20000 cells/µL with active bleeding or on anticoagulant therapy and require close monitoring[16].
4. Pharmacological therapy involves use of i) Splenic tyrosine kinase (Syk) inhibitors, ii) Thrombopoietin receptor agonists (TPO-RA) [17]. Fostamatinib a Syk inhibitor prevents

platelet disintegration by blocking FcγR-mediated destruction of opsonized platelets. It affects the clearance of antibody-coated platelets by the monocyte-macrophage system. Used in treatment for primary ITP with failed conventional treatments. Given as oral medication with doses ranging from 100 mg twice a day to 150 mg twice daily depending on platelet counts[18]. TPO RA stimulates production of platelets. They do not have resemblance to natural thrombopoietin, therefore any antibodies directed against it will not interact with the patient's native thrombopoietin. It was discovered that around one-third of patients on TPO-RAs could be gradually weaned off it, while maintaining a safe and normal platelet count that did not require treatment [19,13]. It has been linked to higher platelet counts, fewer bleeding incidents, and a low requirement for rescue or supplementary therapies [20]. Example: Romiplostim, is a large peptide molecule that binds to the extracellular part of the TPO receptor in the same way that native TPO does [13]. Initial dose 1 mcg/kg SC weekly given at lowest dose to achieve and maintain a platelet count ≥50 x 10^9/L as necessary to reduce the risk for bleeding.

5. ITP patients with severe bleeding are treated with following regimens as: i) Platelet transfusions to be effective in life-threatening conditions associated with intracranial haemorrhage (ICH) [16]. ii) Corticosteroids, considered as first line therapy for primary ITP, with either prednisone (0.5-2 mg/kg daily for a tapering course lasting 4-8 weeks) or dexamethasone (40 mg daily for 4 days for 1-4 repeating cycles) [18]. iii) Intravenous Immunoglobulin G (IVIgG) used either alone or in conjunction with steroids for quick platelet response. It can be administered at a dose of 400 mg/kg/day for 5 days or 1 g/kg/day for 2 days. A randomized trial reported that using IVIgG as a single dose (1 gm/kg) were 67% more susceptible to have an improvement in platelet count by day 4 while those who received a lower initial dose (0.5 mg/kg), showed only 21% improvement [15,18].
6. Second-line therapy for ITP in drug resistant individuals include dapsone, danazol, azathioprine, cyclosporine, mycophenolate mofetil, vinca alkaloids, and cyclophosphamide [18].
7. Haemostatic medicines as tranexamic acid (TXA) (0.5-3 gm/day) and epsilon aminocaproic acid (EACA) (2-24 gm/day) given to reduce bleeding.

Patients with ITP should also be advised to avoid alcohol and nonsteroidal anti-inflammatory medicines, as these can increase the risk of bleeding [15].

8. In emergency conditions, a combination of early treatments, such as IV corticosteroids, IVIG, and anti-fibrinolytic drugs should be used [16].

CASE REPORT:

Here, we discuss a case of ITP following its clinical presentation and treatment regimen. A 48 year old female was admitted at Yashoda Hospital, Hyderabad in the General Medicine ward on 3/9/24. She had complaints of purpura, rashes over lower limb, petechial rash along with headache, epigastric pain and left ankle swelling & pain. This was accompanied by mouth ulcers over tongue, general weakness and hypothyroidism. The laboratory tests reported certain abnormalities as given in Table 2. Laboratory results showed low levels of hemoglobin, Platelet count, Ferritin along with increased level of Prothrombin time which were co-related with the clinical findings of purpura, petechial rash confirming diagnosis of ITP in this patient along with iron deficiency anemia which generally co-occurs due to bleeding disorder. The ITP medication prescription was initiated on Day 3 where IV Pulse Methylprednisolone [IVMP], 1gm, once a day, was given as a short treatment for rapid anti-inflammatory & immunosuppressive effect to stabilize ITP that was continued up-to Day 6. This showed a rapid increase in platelet counts reaching near normal ranges from Day 7 onwards. IVMP also helped in aiding with the patients left ankle swelling and pain that decreased in the consecutive days. However, elevated levels of TLC (11,710 cells/cmm on Day 7 and 17,020 cells/cmm on Day 8) were observed relative to baseline which could be contributed to the use of IVMP high dose. Following which it was changed to per oral formulation of steroid as Tab. Methylprednisolone, 40 mg, once a day on Day 7 and Day 8. Further, dose was established to 60 mg, once a day from Day 9. A thrombopoietin receptor agonists was prescribed such as Tab. Eltrombopag, 10 mg, once a day, from Day 6 onwards and an increased Platelet count could be observed of 1,36,000 cells/µl from 18,000 cells/µl of Day 1. To treat low heamoglobin (Hb) levels contributing to iron deficiency anemia as Inj. Optineuron 1 amp, once a day, was given from Day 1 onwards and improvement in Ferritin and Hb levels were observed of 24 ng/ml & 10.4 g/dL respectively in the consecutive days. For Hypothyroidism, Tab. Levothyroxine, 3.75mg, once a day, was prescribed. The overall medications prescribed shown in Table 3. Hence, in this case study appropriate timely treatment initiation can be seen associated with normalization of pplatelet count, ferritin levels hemoglobin levels enabling the patient recovery in a smooth manner.

Table 2 : Day wise Laboratory Investigational Tests

Tests	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Normal Range
Haemoglobin [gm/dL]	9.90	8.90	9.2	8.9	9.10	9.6	10.4	9.9	11-16.5
Packed Cell Volume [%]	33.5	31.5	32.10	29.6	31.9	33.3	36	33.2	35-50
Total Leukocyte Count [cells/cum]	4430	5260	5000	7770	8940	10900	11710	17020	4000-11000
Platelet count [cells/µL]	18000	30000	13000	12000	30000	72000	136000	170000	100000-400000
Uric acid [mg/dL]	5.7							4.2	2.6-7.2
Ferritin [ng/ml]	9.58	15.7	18.9	20.2	24.5				24-307
Lactate Dehydrogenase [U/L]	329								140-280
Prothrombin time Test- Control-			 17.00 14.10		 20.30 14.10				11-13.5
International Normalized Ratio			1.22		1.47				0.8-1.1
Tri-iodothyronine [ng/dL]						0.85			80-220
Thyroxine [µg/dL]						7.76			5-12

Table 3: Day-wise Medications Prescribed

Medicine Prescribed	Dose	Frequency	D1	D2	D3	D4	D5	D6	D7	D8	D9	D10
Inj. Optineuron	1 amp	OD	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Tab. Levothyroxine	37.5mg	OD		✓	✓	✓	✓	✓	✓	✓	✓	✓
IV. Methylprednisolone	1 gm	OD			✓	✓	✓	✓				
Inj. Diclofenac paracetamol	1 amp	STAT							✓			
Tab. Eltrombopag	50mg	OD 2 hrs BBF							✓	✓	✓	✓
Tab. Gravacal XT (Calcium Citrate, Vitamin D3, Magnesium, and Zinc)	1 tab	BD							✓	✓	✓	✓
Tab. Nimodipine	60 mg	QID							✓	✓	✓	✓
Tab. Prednisolone	40mg	OD							✓	✓		
Inj. Cefoperazone sodium	1.5 gm	BD							✓	✓	✓	✓
Syp. Potassium magnesium citrate	15 ml	BD							✓	✓	✓	✓
Inj. Paracetamol	1 gm	SOS								✓	✓	
Tab. Pantoprazole	40 mg	OD									✓	✓

Inj. Tramadol	50 mg	SOS									✓	✓
Tab. Prednisolone	60 mg	OD									✓	✓
Tab. Paracetamol	1 gm	SOS										✓

[OD: Once a day, BD: Twice a day, QID: Four times a day, SOS: if necessary, STAT: immediately, BBF: Before breakfast, Tab: tablet, Inj: Injection, Syp: Syrup, D1: Day 1]

CONCLUSION:

ITP arises from the sudden death of platelets through intricate mechanisms inside the immune system. The manifestations and indicators of ITP are quite variable and different. The severity of thrombocytopenia and the incidence of bleeding are not consistently connected. Hence, prompt diagnosis, intervention and consistent monitoring can eliminate other potential risks by providing timely management strategy.

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