Section: Internal Medicine

Original Research Article

# THE ITP TRISKELION: THREE CASES ILLUSTRATING THE CLINICAL HETEROGENEITY OF IMMUNE THROMBOCYTOPENIA

**Hema K[1], Rajashekar V[1], Akshay Kumar[1], Mukil.S[2], Senthil Priyan[2], Harsha Vardhanan T[3], Kapil R Alex[3], T.S.Santhi[4]**

[1]Junior Resident, Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, India
[2]Assistant Professor, Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, India
[3]Medical Student, Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, India
[4]Professor, Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, India

*Received* : 02/10/2026
*Received in revised form* : 26/10/2026
*Accepted* : 09/11/2026

*Keywords:*
*ITP.*

Corresponding Author:
**Dr. Harsha Vardhanan T,**
Email: harshavardhanan216@gmail.com

DOI: 10.47009/jamp.2026.8.6.16

Source of Support: Nil,
Conflict of Interest: None declared

*Int J Acad Med Pharm*
2026; 8 (6); 87-92

## ABSTRACT

Immune thrombocytopenia (ITP) is an acquired autoimmune disorder characterised by isolated thrombocytopenia resulting from immune-mediated platelet destruction and impaired platelet production in the absence of other identifiable causes. Although typically presenting with mucocutaneous bleeding, ITP demonstrates significant clinical heterogeneity and may occur as a primary disorder or secondary to systemic diseases. We report a case series of three patients with varied presentations of ITP encountered at a tertiary care centre, including primary ITP with severe mucosal bleeding, ITP secondary to systemic lupus erythematosus, and an atypical presentation associated with neurological manifestations. These cases highlight the diverse clinical spectrum of ITP and underscore the importance of early recognition, thorough evaluation for secondary causes, and timely immunomodulatory therapy, particularly in atypical presentations requiring a multidisciplinary approach to optimise outcomes.

## INTRODUCTION

Immune thrombocytopenia (ITP) is an acquired autoimmune disorder characterised by immune-mediated destruction of platelets and impaired platelet production, resulting in an increased risk of bleeding.[1]

Autoantibodies directed against platelet surface glycoproteins, particularly glycoprotein IIb/IIIa and glycoprotein Ib/IX, play a central role in disease pathogenesis by promoting premature platelet clearance and suppressing megakaryocyte function.[2] ITP may occur as a primary disorder or secondary to a wide range of underlying conditions, including autoimmune diseases, infections, lymphoproliferative disorders, and drug exposure.[1]

The annual incidence of ITP ranges from approximately 1.1 to 12.5 cases per 100,000 population, with a trimodal age distribution involving young children, young adults, and individuals older than 60 years.[2]

The disease demonstrates a slight female predominance, particularly during the reproductive years.[2] Clinically, ITP most commonly presents with mucocutaneous bleeding manifestations such as petechiae, purpura, ecchymoses, epistaxis, and gingival bleeding, while severe hemorrhagic complications, including gastrointestinal and intracranial bleeding, are uncommon. In addition to bleeding manifestations, patients may present with constitutional symptoms, fatigue, thrombotic complications, or features of an underlying systemic illness, reflecting the broad clinical spectrum of the disease.[3]

Given its diverse etiologies and variable clinical manifestations, ITP frequently poses a diagnostic challenge in clinical practice. Distinguishing primary ITP from secondary causes requires a systematic evaluation integrating clinical assessment, laboratory investigations, and, in selected cases, bone marrow examination. Early recognition is essential, as the disease may range from isolated mucocutaneous bleeding to life-threatening multisystem involvement. This case series presents three clinically distinct cases of ITP admitted to the Institute of Internal Medicine, Rajiv Gandhi Government General Hospital, highlighting the remarkable heterogeneity of disease presentation and the importance of a multidisciplinary approach in diagnosis and management.


ISSN (O): 2687-5365; ISSN (P): 2753-6556

## CASE PRESENTATION

**Case 1**

A 65- year- old male with a known history of poorly controlled type 2 diabetes mellitus presented with three days of bleeding gums and ten days of generalised fatigue. There was no history of fever, recent drug intake, hematuria, melena at presentation, or similar illness in family members. The patient had a history of chronic alcohol consumption for 10 years and smoking for 20 years.

On examination, the patient was conscious and oriented. His pulse rate was 73 beats/min, blood pressure was 120/80 mmHg, respiratory rate was 19 breaths/min, and oxygen saturation was 98% on room air. Gingival bleeding and oral ulcers were present. There was no pallor, icterus, lymphadenopathy, or hepatosplenomegaly. Cardiovascular and respiratory examinations were unremarkable except for bilateral basal crepitations. HIV serology was non- reactive, ultrasound of the abdomen showed no significant abnormality, and ECG revealed normal sinus rhythm. Initial evaluation revealed severe thrombocytopenia, with platelet counts as low as 2 × $10^3$/µl, while haemoglobin and leukocyte counts were relatively preserved. Peripheral smear demonstrated severe thrombocytopenia with dimorphic anaemia and neutrophilic predominance without blast cells. Despite supportive measures, the patient continued to exhibit marked fluctuations in platelet counts, with recurrent severe thrombocytopenia (2 × 10 $^3$/µl on admission, 8 × 10 $^3$/µl during hospitalisation, and 4 × 10 $^3$/µl subsequently). During the hospital stay, the patient developed melena, suggestive of ongoing bleeding. Basic biochemical tests were done as shown in [Table 1].

**Table 1: Laboratory and Diagnostic Investigations of Case 1**

| Investigation | Patient value | Reference range |
|---|---|---|
| Hemoglobin | 9.2–13 g/dL | 13–17 g/dL |
| Total leukocyte count | 5.8–21.4 ×$10^3$/µL | 4–11 ×$10^3$/µL |
| Platelet count (Nadir) | 2×$10^3$/µL | 150–450 ×$10^3$/µL |
| Prothrombin time (PT) | 10.6 s | 11–14 s |
| International normalised ratio (INR) | 0.9 | 0.8–1.2 |
| HbA1c | 8.9% | <5.7% |
| Lactate dehydrogenase (LDH) | 308 U/L | 140–280 U/L |
| Blood urea | 61 mg/dL | 15–45 mg/dL |
| Total protein | 5.7 g/dL | 6.0–8.3 g/dL |
| Serum albumin | 3.1 g/dL | 3.5–5.0 g/dL |

**Legend: PT: Prothrombin time; INR: International normalized ratio; HbA1c: Glycated hemoglobin; LDH: Lactate dehydrogenase.**

Bone marrow aspiration and biopsy revealed trilineage hematopoiesis with erythroid hyperplasia and adequate megakaryocytes, favouring peripheral platelet destruction. No evidence of granuloma, necrosis, or haematological malignancy was identified. Marrow fibrosis was graded as Grade I. Evaluation for secondary causes showed negative HIV and COVID- 19 testing, while ANA by Hep- 2 demonstrated cytoplasmic positivity (3 +). Based on the clinical presentation, the exclusion of secondary causes, and preserved megakaryopoiesis on bone marrow examination, a diagnosis of immune thrombocytopenia was established.

The patient was managed with platelet and blood component transfusions for active bleeding, along with intravenous dexamethasone, intravenous immunoglobulin, and rituximab. Owing to persistent thrombocytopenia, eltrombopag was initiated. Supportive therapy included tranexamic acid, folic acid, vitamin B12 supplementation, and optimisation of glycemic control with insulin and metformin.

**Case 2**

A 39-year-old female, a known case of hypothyroidism on regular treatment, presented with bilateral knee pain and generalised body pain of nine months' duration, intermittent in nature. She also reported easy bruising for one month, with ecchymotic patches over the right thigh, left forearm, and venipuncture sites, along with generalised petechial rash. She additionally complained of heavy menstrual bleeding during the preceding two menstrual cycles. There was no history of epistaxis, oral bleeding, hematuria, melena, hematochezia, headache, or seizures. She denied similar episodes in the past, and family history was unremarkable.

During hospitalization, she developed additional systemic symptoms, including pain and swelling of the small joints of the hands with redness and early morning stiffness, swelling of the knees, elbows, and wrists limiting daily activities, recurrent low-grade fever with chills, painless oral ulcers, hair fall, intermittent red eye, painful lip edema, muscle cramps, numbness and tingling, lethargy, chest pain, exertional breathlessness, throat pain, posterior neck swelling, and recurrent erythematous skin lesions. There was no history suggestive of Raynaud phenomenon, abdominal pain, vomiting, psoriasis, or frothy urine.

On examination, the patient was conscious and oriented. Her pulse rate was 80 beats/min, blood pressure was 110/80 mmHg, respiratory rate was 16 breaths/min, oxygen saturation was 98% on room air, and she was afebrile. Petechiae and ecchymotic patches were present over the extremities [as shown in Figure 1]. There was no cyanosis, icterus, clubbing, pedal oedema, or peripheral lymphadenopathy. Cardiovascular examination revealed normal heart sounds without murmurs. Respiratory examination showed bilateral normal


ISSN (O): 2687-5365; ISSN (P): 2753-6556

vesicular breath sounds. Abdominal examination was unremarkable, with no organomegaly.



**Figure 1: Ecchymotic Patches in extremities**

Ophthalmological evaluation was normal. Cardiology evaluation for chest pain and exertional dyspnea revealed no significant abnormality. Echocardiography showed normal left ventricular systolic function without regional wall motion abnormalities, valvular lesions, pulmonary hypertension, pericardial effusion, or intracardiac thrombus. Ultrasonography of the abdomen was also unremarkable.

Initial laboratory evaluation showed bicytopenia with severe thrombocytopenia (platelet nadir: 11 $\times 10^3/\mu L$) and anaemia (haemoglobin nadir: 6.2 g/dL). Peripheral smear revealed normocytic normochromic anaemia with neutrophilic leukocytosis and thrombocytopenia. Reticulocyte index was 0.811. In view of bicytopenia, a differential diagnosis of Evans syndrome and immune thrombocytopenia was considered. Autoimmune workup showed strongly positive antinuclear antibody (homogeneous pattern, 3+), markedly elevated anti-double-stranded DNA antibody levels, and reduced complement levels (C3 and C4), supporting a diagnosis of systemic lupus erythematosus. Anti-HCV, HIV, and HBsAg were negative. Although bone marrow examination was advised, the patient declined the procedure.

The relevant laboratory investigations are summarised in [Table 2].

**Table 2: Laboratory and Diagnostic Investigations of Case 2**

| Investigation | Patient value | Reference range |
|---|---|---|
| Hemoglobin | 6.2 g/dL | 12–15 g/dL |
| Total leukocyte count | 7.9 $\times 10^3/\mu L$ | 4–11 $\times 10^3/\mu L$ |
| Platelet count (nadir) | 11 $\times 10^3/\mu L$ | 150–450 $\times 10^3/\mu L$ |
| Anti-dsDNA antibody | 654.4 IU/mL | <30 IU/mL |
| Complement C3 | 0.619 g/L | 0.9–1.8 g/L |
| Complement C4 | 0.052 g/L | 0.1–0.4 g/L |
| 24-hour urine protein | 1228 mg/day | <150 mg/day |
| C-reactive protein | 34.1 mg/L | <5 mg/L |
| PT/INR | 12.74 s / 0.43 | 11–14 s / 0.8–1.2 |

**Legend: anti-dsDNA: Anti-double-stranded deoxyribonucleic acid antibody; PT: Prothrombin time; INR: International normalized ratio; CRP: C-reactive protein.**

Based on the clinical picture of severe thrombocytopenia with mucocutaneous bleeding, bicytopenia, positive autoimmune markers, hypocomplementemia, and multisystem involvement, a diagnosis of immune thrombocytopenia secondary to systemic lupus erythematosus was made.

The patient was treated with intravenous dexamethasone along with supportive therapy, including folic acid, methylcobalamin, calcium supplementation, multivitamins, and proton pump inhibitor therapy. Hydroxychloroquine and levothyroxine were continued. She also received physiotherapy for musculoskeletal symptoms. Serial monitoring showed gradual haematological improvement, with platelet count rising to 51 $\times 10^3/\mu L$ by discharge. She was discharged with advice for regular follow-up under rheumatology and haematology services.

**Case 3**

A 62-year-old female with no known comorbidities presented with bilateral lower limb weakness and loss of vision in the right eye of one week's duration. Fifteen days before admission, she had developed shortness of breath and low-grade intermittent fever for which she was empirically initiated on anti-tubercular therapy at a peripheral centre. Following initiation of therapy, she developed abdominal pain, vomiting, generalised pruritus, diffuse skin rash, and acute urinary retention. Subsequently, she developed an acute onset of paraparesis and was referred to our centre for further evaluation.

On examination, the patient was drowsy but arousable and obeyed simple commands. Her pulse rate was 112 beats/min, blood pressure was 100/70 mmHg, respiratory rate was 26 breaths/min, and oxygen saturation was 98% on room air. Neurological examination revealed flaccid paraparesis with preserved upper limb power, bilateral extensor plantar responses, sensory impairment below the T10 dermatome, and bladder involvement requiring catheterisation. Ophthalmological examination revealed markedly reduced vision in the right eye with a relative afferent pupillary defect, suggestive of optic neuritis. Generalised purpuric and ecchymotic lesions were present over the body.

Magnetic resonance imaging of the spine demonstrated longitudinally extensive transverse myelitis involving the D2-D11 vertebral levels without contrast enhancement. Magnetic resonance imaging of the brain revealed a right cerebellar


ISSN (O): 2687-5365; ISSN (P): 2753-6556

haemorrhage with associated subarachnoid haemorrhage. Magnetic resonance angiography showed no evidence of arterial stenosis or occlusion. In view of the coexistence of longitudinally extensive transverse myelitis and optic neuritis, neuromyelitis optica spectrum disorder was considered; however, both aquaporin-4 and myelin oligodendrocyte glycoprotein antibodies were negative. Autoimmune workup revealed weakly positive antinuclear antibody, while ANCA and lupus anticoagulant were negative. Initial laboratory evaluation revealed severe thrombocytopenia with a platelet nadir of 3 $\times 10^3/\mu L$, anaemia, transaminitis, and elevated inflammatory markers. Peripheral smear demonstrated dimorphic anaemia with neutrophilic leukocytosis and thrombocytopenia. Cerebrospinal fluid analysis was unremarkable, and the direct Coombs test was negative. CT thorax demonstrated sequelae of old treated pulmonary tuberculosis with bronchiectatic changes.

The relevant laboratory investigations are summarised in [Table 3].

**Table 3: Laboratory Investigations of Case 3**

| Investigation | Patient value | Reference range |
|---|---|---|
| Hemoglobin | 9.6 g/dL | 12–15 g/dL |
| Total leukocyte count | 9.3 $\times 10^3/\mu L$ | 4–11 $\times 10^3/\mu L$ |
| Platelet count (nadir) | 3 $\times 10^3/\mu L$ | 150–450 $\times 10^3/\mu L$ |
| Lactate dehydrogenase (LDH) | 448 U/L | 140–280 U/L |
| C-reactive protein (CRP) | 14.58 mg/L | <5 mg/L |
| Aspartate aminotransferase (AST) | 65.87 U/L | 10–40 U/L |
| Alanine aminotransferase (ALT) | 72 U/L | 7–56 U/L |
| Total protein | 5.4 g/dL | 6.0–8.3 g/dL |
| Serum albumin | 2.4 g/dL | 3.5–5.0 g/dL |
| Blood urea | 40 mg/dL | 15–45 mg/dL |
| Serum creatinine | 0.4 mg/dL | 0.6–1.2 mg/dL |
| Random blood glucose | 214 mg/dL | 70–140 mg/dL |
| Reticulocyte count | 2.5% | 0.5–2.5% |

**Legend: LDH: Lactate dehydrogenase; CRP: C-reactive protein; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase.**

During hospitalisation, the patient developed hypotension with severe left ventricular systolic dysfunction requiring inotropic support. Echocardiography performed subsequently demonstrated recovery of left ventricular systolic function with preserved ejection fraction.

Based on the presence of severe thrombocytopenia associated with extensive purpura, intracranial haemorrhage, and exclusion of alternative etiologies, a diagnosis of immune thrombocytopenia with atypical neurological manifestations was considered. The patient was treated with high-dose intravenous methylprednisolone (1 g/day) and intravenous immunoglobulin. She additionally received platelet and packed red blood cell transfusions, inotropic support, broad-spectrum antibiotics, and supportive care. Following treatment, platelet counts improved significantly to 327 $\times 10^3/\mu L$. She was subsequently transitioned to oral corticosteroids along with calcium, folic acid, vitamin B12 supplementation, and physiotherapy.

## DISCUSSION

Immune thrombocytopenia (ITP) is an acquired autoimmune disorder characterised by immune-mediated peripheral platelet destruction and impaired platelet production.[4] The pathogenesis of ITP is complex and involves the generation of autoantibodies directed predominantly against platelet surface glycoproteins, particularly GPIIb/IIIa and GPIb/IX, resulting in accelerated platelet clearance by the reticuloendothelial system, primarily in the spleen.[1,5] In addition, autoreactive T cells contribute to platelet destruction and may directly impair megakaryocyte maturation and thrombopoiesis within the bone marrow.[5] Consequently, ITP is now recognised as a disorder involving both increased platelet destruction and inadequate platelet production.[6]

ITP remains a diagnosis of exclusion, necessitating careful evaluation to distinguish it from numerous conditions that may present with thrombocytopenia or other cytopenias.[6] Differential diagnoses include pseudothrombocytopenia, thrombotic microangiopathies, bone marrow disorders such as myelodysplastic syndrome and aplastic anaemia, drug-induced thrombocytopenia, infections, and secondary autoimmune disorders.[7] Clinical assessment supported by peripheral smear examination, relevant serological investigations, and bone marrow evaluation in selected cases is therefore essential to establish the diagnosis and identify potentially reversible secondary causes.[6,7]

In the present series, three cases of ITP encompassing diverse clinical phenotypes were analysed [Table 4], highlighting the marked heterogeneity of the disorder. While the first case represented classical primary ITP with severe mucocutaneous bleeding, the second case illustrated secondary ITP occurring in association with systemic lupus erythematosus, and the third case demonstrated an unusual presentation with extensive neurological involvement, including longitudinally extensive transverse myelitis, optic neuritis, and intracranial


ISSN (O): 2687-5365; ISSN (P): 2753-6556

haemorrhage. Although some patients exhibited typical bleeding manifestations, others posed significant diagnostic challenges, emphasising the importance of integrating clinical, laboratory, radiological, and immunological findings during evaluation.

**Table 4: Comparing the presentations and features of the three cases of ITP**

| Case | Etiology | Clinical Presentation | Key Diagnostic Clue | Management | Special Points |
|---|---|---|---|---|---|
| Case 1 | Primary ITP | Gingival bleeding, oral ulcers, fatigue, and melena | Severe isolated thrombocytopenia with preserved megakaryopoiesis on bone marrow examination and exclusion of secondary causes | Platelet and blood component transfusions, IV dexamethasone, IVIG, rituximab, eltrombopag, and supportive therapy | Classical severe primary ITP with recurrent profound thrombocytopenia requiring multimodal therapy |
| Case 2 | Secondary ITP associated with systemic lupus erythematosus (SLE) | Easy bruising, petechiae, menorrhagia, polyarthritis, and mucocutaneous manifestations | Positive ANA (3+), elevated anti-dsDNA antibodies, low complement levels, and bicytopenia suggestive of underlying SLE. | IV dexamethasone, hydroxychloroquine, levothyroxine, hematinic supplementation, physiotherapy, and supportive care | ITP was the initial manifestation leading to the diagnosis of underlying SLE |
| Case 3 | Primary ITP with atypical neurological manifestations | Acute paraparesis, right optic neuritis, purpura, altered sensorium, and intracranial haemorrhage | Severe thrombocytopenia associated with LETM, optic neuritis, cerebellar haemorrhage. | IV methylprednisolone, IVIG, blood product transfusions, inotropic support, antibiotics, and intensive supportive care | Rare presentation of ITP with LETM, optic neuritis, and intracranial haemorrhage mimicking a demyelinating disorder |

The first case represents a classical yet severe presentation of primary ITP, manifesting with profound thrombocytopenia, gingival bleeding, and subsequent melena. Mucocutaneous bleeding is among the commonest manifestations of ITP; however, spontaneous oral haemorrhage may occasionally be the presenting feature and necessitates urgent evaluation and treatment.[8] Similar presentations with severe oral bleeding as the initial manifestation of ITP have been previously reported, highlighting the importance of early recognition to prevent catastrophic hemorrhagic complications.[9] Owing to persistent severe thrombocytopenia despite first-line therapy, eltrombopag, a thrombopoietin receptor agonist, was initiated.[10] Eltrombopag has emerged as an effective second-line agent in refractory or persistent ITP, promoting platelet production and achieving sustained platelet responses while reducing bleeding risk.[10]

The second case illustrates secondary ITP occurring in association with systemic lupus erythematosus (SLE), an important autoimmune cause of immune-mediated thrombocytopenia. Although most patients present with primary ITP, approximately 3-15% of patients initially diagnosed with isolated ITP subsequently develop SLE.[11] Large cohort studies have demonstrated that patients with ITP possess a significantly increased risk of future SLE development, particularly females and those with positive ANA serology.[11,12] A recent systematic review involving over 14,000 ITP patients reported ANA positivity in 17.5% of cases, with ANA-positive individuals exhibiting a 26-fold higher risk of developing SLE.[13] The presence of bicytopenia, strongly positive ANA and anti-dsDNA, hypocomplementemia, polyarthritis, oral ulcers, alopecia, and proteinuria in our patient strongly supported an underlying lupus-associated etiology.

Management of SLE-associated ITP largely parallels that of primary ITP, with corticosteroids remaining the cornerstone of initial therapy, while IVIG is reserved for severe thrombocytopenia, active bleeding, or situations requiring rapid platelet elevation.[14] Early institution of immunosuppressive therapy in our patient resulted in progressive platelet recovery, emphasising the importance of recognising autoimmune evolution in patients presenting with apparent isolated thrombocytopenia.

The third case represents an exceptionally rare and diagnostically challenging presentation of ITP associated with longitudinally extensive transverse myelitis (LETM) and optic neuritis. The patient presented with acute paraparesis, bladder dysfunction, and rapidly progressive visual loss, all characteristic features of LETM with optic nerve involvement. Similar diagnostic challenges have been reported in the literature, where neurological manifestations were initially masked by concomitant systemic abnormalities and infectious differentials, delaying definitive diagnosis and treatment.[15] MRI demonstrated a long-segment dorsal cord lesion extending from D2 to D11 along with right optic neuritis, while NMO and MOG antibodies were negative, suggesting an underlying immune-mediated process.

Prompt immunotherapy is essential in LETM and optic neuritis to prevent irreversible neurological injury. Current recommendations advocate early initiation of high-dose intravenous corticosteroids, with plasma exchange reserved for severe or refractory cases.[8] Accordingly, our patient was treated with intravenous methylprednisolone and


ISSN (O): 2687-5365; ISSN (P): 2753-6556

IVIG, resulting in stabilisation of both neurological and haematological parameters.

This case series highlights the broad clinical spectrum of immune thrombocytopenia, encompassing classical primary disease, secondary autoimmune thrombocytopenia, and rare neurological manifestations. It emphasises that similar haematological presentations may arise from diverse underlying etiologies, while atypical and overlapping features frequently complicate diagnosis. A structured and comprehensive evaluation is therefore essential to distinguish primary from secondary causes and to facilitate timely institution of appropriate therapy. The occurrence of uncommon associations such as SLE-associated ITP and LETM with optic neuritis further supports the concept that ITP is often a manifestation of complex systemic immune dysregulation rather than merely an isolated haematological disorder.

## CONCLUSION

Immune thrombocytopenia exhibits a broad and heterogeneous clinical spectrum, extending beyond isolated thrombocytopenia to include secondary autoimmune associations and rare extrahematological manifestations. This case series highlights the diagnostic challenges posed by atypical presentations and underscores the importance of a comprehensive and systematic evaluation to distinguish primary from secondary causes. Recognition of overlapping autoimmune features and uncommon complications is essential, as early diagnosis and timely institution of appropriate immunomodulatory therapy can significantly improve patient outcomes and prevent life-threatening complications.

## REFERENCES


1. Zainal A, Salama A, Alweis R. Immune thrombocytopenic purpura. Journal of Community Hospital Internal Medicine Perspectives. 2019;9(1):59–61. doi:10.1080/20009666.2019.1565884.
2. Martínez-Carballeira D, Bernardo Á, Caro A, Soto I, Gutiérrez L. Pathophysiology, Clinical Manifestations and Diagnosis of Immune Thrombocytopenia: Contextualization from a Historical Perspective. Hematology Reports. 2024;16(2):204–219. doi:10.3390/hematolrep16020021
3. McMillan R. The Pathogenesis of Chronic Immune Thrombocytopenic Purpura. Seminars in Hematology. 2007;44:S3–S11. (Immune-Mediated Thrombocytopenias: Focus on Chronic Immune Thrombocytopenic Purpura). doi:10.1053/j.seminhematol.2007.11.002
4. Full article: Immune thrombocytopenic purpura. [accessed 2026 June 29]. https://www.tandfonline.com/doi/full/10.1080/20009666.2019.1565884#abstract
5. Cooper N, Ghanima W. Immune Thrombocytopenia Solomon CG, editor. New England Journal of Medicine. 2019;381(10):945–955. doi:10.1056/NEJMcp1810479
6. Zufferey A, Kapur R, Semple JW. Pathogenesis and Therapeutic Mechanisms in Immune Thrombocytopenia (ITP). Journal of Clinical Medicine. 2017;6(2):16. doi:10.3390/jcm6020016
7. Immune Thrombocytopenic Purpura — The Changing Therapeutic Landscape | New England Journal of Medicine. [accessed 2026 June 29]. https://www.nejm.org/doi/abs/10.1056/NEJMp068169
8. Provan D, Semple JW. Recent advances in the mechanisms and treatment of immune thrombocytopenia. eBioMedicine. 2022 [accessed 2026 June 29];76. https://www.thelancet.com/journals/ebiom/article/PIIS2352-3964(22)00009-3/fulltext. doi:10.1016/j.ebiom.2022.103820
9. Sugiura T, Yamamoto K, Murakami K, Horita S, Matsusue Y, Nakashima C, Kirita T. Immune Thrombocytopenic Purpura Detected with Oral Hemorrhage: a Case Report. Journal of Dentistry. 2018;19(2):159.
10. Ferreira IJMCF, Sousa F, Vasco EM, Areia ALF de A, Moura JPAS, Carda J, Ribeiro L. Severe immune thrombocytopenia in pregnancy treated with Eltrombopag – A case report. Journal of Gynecology Obstetrics and Human Reproduction. 2018;47(8):405–408. doi:10.1016/j.jogoh.2018.06.010
11. Bashyal KP, Shah S, Ghimire C, Balmuri S, Chaudhary P, Karki S, Poudel AK, Pokharel A, Devarkonda V, Hayat S. Primary Immune Thrombocytopenic Purpura (ITP) and ITP Associated with Systemic Lupus Erythematosus: A Review of Clinical Characteristics and Treatment Modalities. International Journal of Rheumatology. 2024;2024(1):6650921. doi:10.1155/2024/6650921
12. Roussotte M, Gerfaud-Valentin M, Hot A, Audia S, Bonnotte B, Thibault T, Lobbes H, Le Guenno G, Goulabchand R, Cathebras P, et al. Immune thrombocytopenia with clinical significance in systemic lupus erythematosus: a retrospective cohort study of 90 patients. Rheumatology. 2022;61(9):3627–3639. doi:10.1093/rheumatology/keab925
13. Pamuk ON, Ali SM, Hasni S. Development of systemic lupus erythematosus in patients with immune thrombocytopenic purpura: A systematic meta-analysis. Autoimmunity Reviews. 2023;22(4):103297. doi:10.1016/j.autrev.2023.103297
14. Rungjirajittranon T, Owattanapanich W. A serious thrombotic event in a patient with immune thrombocytopenia requiring intravenous immunoglobulin: a case report. Journal of Medical Case Reports. 2019;13(1):25. doi:10.1186/s13256-018-1955-x
15. De Silva NS, Jayasinghe RP, Ginneliya DC, Mohideen MS. Coexistence of neuromyelitis optica spectrum disorder and Evans syndrome, complicated by cytomegalovirus colitis, and candidaemia: a therapeutic dilemma. Asian Journal of Internal Medicine. 2026;5(1):126–131. doi:10.4038/ajim.v5i1.389.




ISSN (O): 2687-5365; ISSN (P): 2753-6556