Acute Severe Immune Thrombocytopenic Purpura Following Upper Respiratory Infection in a Patient with Psoriasis

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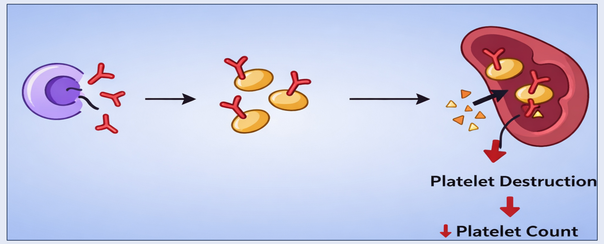
Objectives

- To describe the presentation, diagnostic evaluation, and management of a young adult male with acute severe immune thrombocytopenic purpura (ITP)
- To emphasize the diagnostic role of immature platelet fraction and the effectiveness of corticosteroid therapy in severe ITP.
- To present that autoimmune conditions, such as psoriasis, make ITP more probable.

Introduction

Immune thrombocytopenic purpura is an acquired autoimmune disorder causing isolated thrombocytopenia through immune-mediated platelet destruction and impaired production. ITP is a diagnosis of exclusion, requiring careful assessment to rule out secondary causes like infection, malignancy, thrombotic microangiopathies, liver disease, or medication use. Autoimmune conditions are increasingly linked to hematologic disorders. Psoriasis, a chronic inflammatory autoimmune disease, is associated with systemic immune dysregulation, predisposing patients to other autoimmune phenomena. Recognizing the connection between underlying autoimmune disease and hematologic autoimmunity is important for early recognition and management of severe ITP.



Methods

This is a patient case report based on a patient encounter and hospitalization. A 32-year-old male with a history of psoriasis and URI presented with a new-onset petechial rash and bruising involving the lower extremities and abdomen. Diagnostic evaluation included serial complete blood counts, peripheral smear, immature platelet fraction, coagulation studies, metabolic panel, autoimmune testing, infectious disease screening, and hematology consultation.

Findings

Test	1/14/26 19:48	1/15/26 04:17	1/16/26 03:22
WBC	10.4	11.1 (High)	21.3 (High)
RBC	5.37	5.29	5.02
Hemoglobin	15.5	15.3	14.6
Hematocrit	46.4	44.0	43.0
MCV	86.4	83.2	85.7
MCH	28.9	28.9	29.1
MCHC	33.4	34.8	34.0
RDW-SD	39.2	37.3	39.4
RDW-CV	12.5	12.3	12.7
Platelets	1 (Low)	13 (Low)	40 (Low)
Immature Platelet Fraction	47.6 (High)	11.4 (High)	23.9 (High)
MPV	-	11.4	13.5 (High)

Table 1: Serial CBC demonstrating thrombocytopenia with elevated immature platelet fraction and subsequent platelet recovery with time.

Test	Result
Vitamin B12	536
ANA Screen (IFA)	Negative
RA Screen	Negative
Hepatitis Panel	Non-reactive
HIV Screen	Non-reactive

Table 2: Baseline laboratory evaluation showing normal vitamin B12, negative autoimmune screening, and non-reactive infectious disease panel.

Test	Result
Prothrombin Time (PT)	13.6
INR	1.01
aPTT	28.1
Fibrinogen	370
D-Dimer (Quant)	<0.27

Table 3: Coagulation profile within normal limits, including PT/INR, aPTT, fibrinogen, and D-dimer

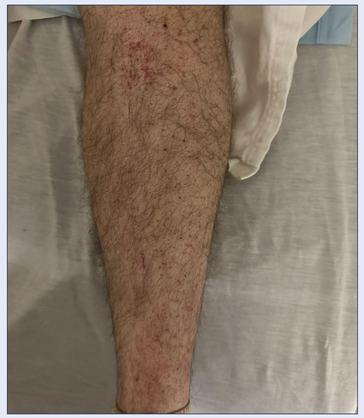


Figure 1: Diffuse petechiae and purpura over the lower leg consistent with cutaneous bleeding from thrombocytopenia.

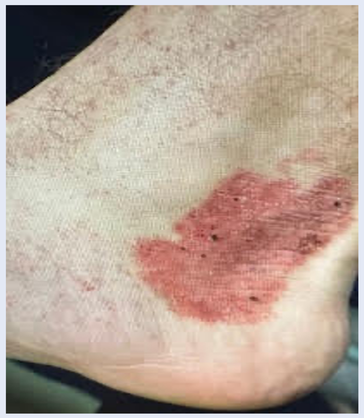


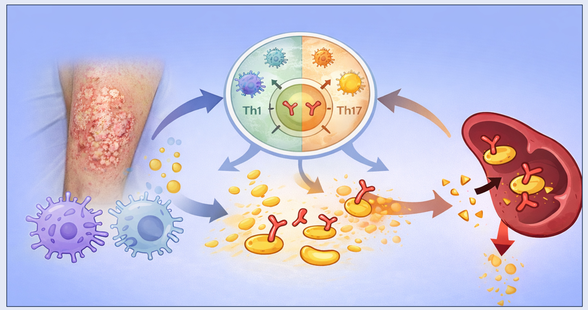
Figure 2: Localized erythematous purpuric plaque over the lateral ankle with superficial excoriations.

Results

The patient was found to have severe isolated thrombocytopenia with a nadir platelet count of 1 $\times 10^3/\mu L$, while hemoglobin and white blood cell counts remained preserved.
Immature platelet fraction was markedly elevated, and peripheral smear demonstrated large platelets, indicating intact bone marrow production and peripheral platelet destruction.
Coagulation studies were normal, and there was no evidence of hemolysis, thrombotic microangiopathy, or liver disease. Infectious and autoimmune evaluations, including HIV, hepatitis, EBV, ANA, and rheumatoid factor, were negative.

Discussion

Psoriasis is driven by the activation of T-cell mediated autoimmune destruction. There is an activation of Th1 and Th17 pathways in addition to the release of TNF-a, IL-23 which lead to inflammation and keratinocyte proliferation.
Similar to psoriasis, ITP also has the activation of Th1, Th17, and TNF-a. This overlap suggests a shared inflammatory milieu making ITP more likely to occur in those with psoriasis.



References