Immune Thrombocytopenia

Iris Ma, MD[a,*], Alexander T. Sandhu, MD, MS[b,c]

KEYWORDS

• Immune thrombocytopenia • Idiopathic thrombocytopenic purpura • Splenectomy
• Thrombopoietin mimetics • Anti-D immunoglobulin

HOSPITAL MEDICINE CLINICS CHECKLIST

1. Immune thrombocytopenia is a diagnosis of exclusion with no confirmatory diagnostic test.
2. Secondary immune thrombocytopenia occurs due to multiple distinct etiologies—including infections, autoimmune disease, and malignancy—but represents less than 20% of adult immune thrombocytopenia cases.
3. Bone marrow biopsy is not necessary for diagnosis but should be pursued in select cases: abnormalities on peripheral smear, abnormal white blood cell or hemoglobin count, or treatment-refractory immune thrombocytopenia.
4. Treatment for immune thrombocytopenia should be initiated in patients with less than 30,000 platelets per microliter or with significant bleeding. First-line treatment for primary ITP is high-dose dexamethasone, intravenous immunoglobulin, or anti-D immunoglobulin. A choice should be made based on rapidity of onset needed, side effect profile, and cost.
5. Immune thrombocytopenia is typically a chronic disease but improves over time in many cases. Splenectomy should be deferred for at least 6 months in favor of treatments with lower long-term morbidity to allow time for disease regression.

DEFINITION

What is immune thrombocytopenia?

Immune thrombocytopenia (ITP) is a syndrome of low platelet count (<100,000 platelets per microliter) caused by autoimmune platelet destruction. It results from the

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[a] California Pacific Medical Center, 2333 Buchanan Street, San Francisco, CA 94115, USA; [b] Veterans Affairs Palo Alto Health Care System, Palo Alto, CA, USA; [c] Department of Medicine, Center for Health Policy and Center for Primary Care and Outcomes Research, Stanford University, 117 Encina Commons, Stanford, CA 94305, USA
* Corresponding author.
E-mail address: MaIH1@sutterhealth.org

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spontaneous or disease-associated development of platelet autoantibodies. In the pediatric population, ITP is most commonly a self-limited postviral phenomenon; however, in adults, it typically results in a chronic disease with multiple remissions and relapses. This review focuses on adult ITP. See **Box 1** for a description of key definitions of ITP disease classifications.[1]

What is the difference between primary and secondary immune thrombocytopenia?

Primary ITP does not have an associated cause. Secondary forms of ITP have many overlapping features with primary ITP but are triggered by various illnesses including viral infections, bacterial infections, rheumatologic disease, and malignancies, particularly hematologic cancers.[2] **Fig. 1** displays the relative frequency of ITP etiologies. The most common secondary ITP disease associations are listed in **Box 2**.[2,3] Distinguishing primary and secondary ITP is important because secondary ITP may improve or resolve after treating the underlying cause. There are many options for treating ITP, and choosing a treatment for secondary ITP that also targets concurrent diseases is preferred.

PATHOPHYSIOLOGY

What is the mechanism of immune thrombocytopenia?

The autoimmune nature of ITP was first elucidated by a hematology fellow and his colleagues who famously performed self-experimentation to investigate the mechanism of the disease.[4] These audacious investigators transfused themselves with the blood from patients afflicted with what was then termed *idiopathic thrombocytopenic purpura*. Lo and behold, the experimenters developed severe symptomatic thrombocytopenia within 60 minutes of blood transfusion. The platelet decrease persisted for about 1 week before resolving. This experiment firmly established the concept that there existed a factor in patients' blood that caused platelet destruction.

Box 1
Definitions

Term	Definition
Thrombocytopenia	<150,000 platelets/microliter
Immune thrombocytopenia	Autoimmune destruction of platelets resulting in blood platelet count <100,000 platelets/microliter
Idiopathic thrombocytopenic purpura	Historic name for ITP now replaced by immune thrombocytopenia
Primary ITP	ITP with no associated trigger
Secondary ITP (disease associated)	ITP with an associated triggering condition such as infection, immune disease, or medication related as shown in **Box 2**
Phases of Disease	
Newly diagnosed ITP	Within 3 mo of first diagnosis
Chronic ITP	Persistent ITP >12 mo
Severe ITP	Significant bleeding symptoms requiring treatment
Response Classification	
Partial response	>30,000 platelets/microliter, doubling of platelet count from nadir, and absence of bleeding
Complete Response	Platelet count >100,000 platelets/microliter and absence of bleeding

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