

Immune Thrombocytopenia (ITP)

Post-Transfusion Purpur

Drug-Induced Immune Thrombocytopenia

Description:

These immune-mediated thrombocytopenias are driven by antibodies directed against platelet surface antigens, such as glycoprotein (GP) Ib/IX or GP IIb/IIIa.

Typical patients present with isolated thrombocytopenia; red blood cell counts, white blood cell counts, and serum chemistry panels remain unremarkable. ITP can develop idiopathically or secondary to infections, transfusions, concurrent autoimmune conditions, hematologic malignancies, or as an adverse drug reaction.

Primary ITP remains a diagnosis of exclusion; there is no definitive diagnostic test. While the detection of anti-platelet antibodies confirms the clinical suspicion of ITP, the absence of detectable antibodies does not exclude the diagnosis.

The juvenile form of ITP frequently manifests following viral infections and presents with profound thrombocytopenia (<2 G/L). In contrast, chronic ITP is more prevalent among elderly patients and often exhibits only mildly reduced platelet counts.

First-line therapy for ITP primarily involves corticosteroids, supplemented by high-dose intravenous immunoglobulin (IVIG) infusions in cases of life-threatening hemorrhage. Second-line treatment employs thrombopoietin receptor agonists (TPO-RAs, such as romiplostim, eltrombopag, avatrombopag, etc.). Immunosuppression with rituximab may also be effective. Other agents (fostamatinib, efgartigimod, ianalumab) or splenectomy represent further therapeutic options.

For comprehensive details on ITP, please refer to the relevant medical literature and the current Onkopedia guidelines:

(<https://www.onkopedia.com/de/onkopedia/guidelines/immunthrombozytopenie-ityp/@@guideline/html/index.html>).

Post-transfusion purpura can manifest approximately 1–2 weeks following the administration of blood products (packed red blood cells or platelet concentrates), precipitating severe thrombocytopenia with a pronounced bleeding diathesis. Because standard therapeutic modalities are limited (corticosteroids, IVIG, TPO-RAs, etc., are typically ineffective), there is a substantial risk for intracranial hemorrhage. The thrombocytopenia usually persists for 2–3 weeks, after which platelet counts generally recover spontaneously. The elevated bleeding risk during this phase may potentially be mitigated by the administration of tranexamic acid, desmopressin, or recombinant factor VIIa (NovoSeven®) (all used off-label).

References:

Thomas L, Labor und Diagnose, 2023, Release 5: <https://www.labor-und-diagnose.de/index.html>

Parameterkatalog des Klinischen Instituts für Labormedizin, Med.Univ.Wien und AKH Wien:

<https://www.akhwien.at/default.aspx?pid=3982>

Leistungsverzeichnis der Klinischen Chemie, Univ.Klinikum Ulm:

<https://www.uniklinik-ulm.de/zentrale-einrichtung-klinische-chemie/leistungsverzeichnis.html>