

Disease	Cause	Notes	Treatment
Immune thrombocytopenic Purpura	IgG Antibodies against platelet antigens (GP IIb/IIIa)	<ul style="list-style-type: none"> - Most common in children and adults - Large platelets on blood smear - low platelets count - Increased megakaryocytes number - Normal PT & PTT - Enlarged B-cells follicles and increased plasma cells 	<ol style="list-style-type: none"> 1- Self-limiting in children 2- Corticosteroids may help in children but adults will recover then have a relapse* 3. Splenectomy
Heparin-Induced Thrombocytopenia	Heparin-induced IgG antibodies against platelet's cell membrane protein → platelet factor 4	<ul style="list-style-type: none"> - More common with unfractionated Heparin (high molecular weight) - Paradoxical thrombocytopenia and thrombosis 	
Thrombotic Microangiopathies [mainly in TTP and HUS]	<p>TTP→ a defect in ADAMTS3 that cleaves <u>vWF precursor</u> to the smaller form (the large precursor binds a large number of platelets forming thrombi)</p> <p>HUS → <i>infection</i> by enterohemorrhagic E.coli (O157:H7) which produce <u>shiga toxin</u> that reaches the kidneys and causes vessels damage and microthrombi</p>	<p>Symptoms → fever, thrombocytopenia, microangiopathies, hemolytic anemia, renal failure [more prominent in HUS], CNS damage [only in TTP].</p> <ul style="list-style-type: none"> - HUS → more common in children - Normal PTT/ PT - Low platelets - On blood smear → schistocytes 	plasmapheresis and corticosteroids
von Willebrand Disease	-Mainly autosomal dominant defect causes reduction in vWF* - Type I →	<ul style="list-style-type: none"> - vWF causes platelets' adhesion so when deficient we will have superficial bleeding - vWF carries factor VIII in the plasma so in severe cases we will 	Desmopressin (ADH analog)

	<p><i>decreased vWF</i></p> <ul style="list-style-type: none"> - Type IIa → <i>decreased vWF precursor</i> (high molecular weight) - Type IIb → <i>normal vWF precursors number but decreased half-life</i> (similar to TTP without microthrombi) 	<p>have factor VIII deficiency</p> <ul style="list-style-type: none"> - high PTT in cases of VIII absence - abnormal ristocetin test 	
Hemophilia A	<p><i>x-linked deficiency in factor VIII</i> (more common in males)</p>	<ul style="list-style-type: none"> - 30% of cases have no family history - Mild deficiency causes excessive bleeding after trauma - Symptoms appear after marked decrease in factor levels → <u>deep tissue bleeding</u> but <i>no petechiae</i> - Severe deficiency may cause death - High PTT corrected by mixing study * 	Recombinant factor VIII
Hemophilia B [christmas disease]	<p><i>x-linked deficiency in factor IX</i></p>	<ul style="list-style-type: none"> - similar to hemophilia A but less common - decreased factor IX levels 	
Disseminated Intravascular Coagulation	<p><i>Increased release of tissue factor</i></p>	<ul style="list-style-type: none"> - Secondary to: <ol style="list-style-type: none"> 1. complicated labour 2. Cancer → Acute Promyelocytic leukemia T epithelial tumours that secrete mucin [activate coagulation] 3. Snake venom 4. Sepsis and Viremia 5. Inflammation 6. Trauma - high PT/PTT - Blood smear → schistocytes, thrombocytopenia 	<p>- D-dimer is the best screening test *</p>

*Information from pathoma in red

* vWF → when the subendothelium is exposed due to injury, vWF binds gplb (CD42b) on platelets and promotes their adhesion

* Mixing Study → to determine whether we have autoantibodies or true deficiency by adding the supposedly missing factors → corrected?deficiency, not corrected? autoantibodies

* D-dimer → results from *fibrin* splitting