DIC (disseminated intravascular coagulation): Several clinical conditions can initiate DIC including shock, sepsis, cancer, and obstetric complications. DIC may cause the following:

- Formation of thrombi within the microvasculature and ischemic damage to tissue and organs
- Consumption of fibrinogen, platelets, and other factors
- Activation of the fibrinolytic system and generation of fibrin degradation products

The outcome can be uncontrolled bleeding and hemorrhage. Platelet transfusion is contraindicated because it complicates the condition by contributing to thrombosis.

HIT (heparin-induced thrombocytopenia): HIT is induced by heparin and may cause activation and damage to the platelets and the endothelium and susceptibility to thrombosis. Type I is non-immune with mild thrombocytopenia after heparin exposure and resolves within a few minutes to days. Type II is immune-stimulated and may lead to life-threatening complications. Discontinuation of heparin therapy results in recovery but a small percentage (0.9%) may develop thrombosis or an unexplained decrease in platelet count.

ITP (idiopathic thrombocytopenic purpura): ITP results from autoantibodies directed against platelet antigens. Acute ITP is commonly seen in children, often after a viral infection. Chronic ITP is most frequent in adults, mostly females, and may last for months to years. Treatment consists of steroids, intravenous immunoglobulin, RhIG, and sometimes splenectomy. Since the antibody in ITP has broad reactivity against platelets, platelet transfusions should be avoided except in emergencies.

TTP (thrombotic thrombocytopenic purpura), indistinguishable from HUS (hemolytic-uremic syndrome), occurs suddenly and spontaneously and without apparent cause. TTP/HUS are acute, potentially fatal disorders characterized by severe thrombocytopenia, fragmented red blood cells, hemolysis, acute renal failure, fever, and organ damage, including CNS confusion and coma, jaundice, thrombotic microangiopathy, and proteinuria.

The treatment of choice for TTP is a daily plasma exchange, a therapeutic plasma exchange (TPE). Multiple relapses that require plasma exchange may occur. The conditions described as TTP/HUS are both multisystem disorders, in which platelet/fibrin thrombi occlude the microcirculation which explains why platelet transfusions are avoided. Patients presenting with fulminant TTP usually have platelet counts below 20,000/µL and lactic dehydrogenase (LDH) levels above 1000 IU/mL, resulting from systemic ischemia and hemolysis. The peripheral blood smear characteristically shows increased number of schistocytes. Evidence for disseminated intravascular coagulation is generally absent.

TTP usually develops without obvious cause, although episodes may occur after infections, pregnancy, or use of some common drugs such as ticlopidine, or clopidogrel. HUS is typically a disease of children associated with enterohemorrhagic *Escherichia coli*.

HELLP (hemolysis, elevated liver enzymes, low platelets) is a rather obscure life-threatening syndrome of hemolysis, elevated liver enzymes, and low platelets occurring in the prenatal or postpartum period of pregnancy.

Often a patient who develops HELLP syndrome has already been diagnosed with pregnancy-induced high blood pressure or pre-eclampsia (high blood pressure and proteinuria). Other symptoms include marked onset of headaches, blurred vision, malaise, nausea, vomiting, upper abdominal pain and tingling in the extremities.