Disseminated Intravascular Coagulation (DIC)

Clinical Cases Applicability: Postpartum hemorrhage**,** HELLP Syndrome**,** Placental abruption**,** Fetal demise**,** Amniotic fluid embolism

Learning Objectives:
1) Understand the basic principles of normal hemostasis
2) Understand which clotting factors are measured in commonly ordered coagulation labs 3) Understand the pathophysiology of DIC

Clinical Presentation: Bleeding, hypotension out of proportion to bleeding, and coagulopathy

How does a clot form? (See figure 1)
Tissue factor at the wound site + VIIa → activates X and IX to generate small amounts of thrombin → activates factors V, VIII, XI and platelets, which leads to amplification of thrombin **How does a clot stop forming?**
1)Antithrombin inhibits thrombin 2)Activated protein C block activation of factors V and VIII 3)Tissue factor pathway inhibitor blocks activation of factor VII
How is a clot removed (fibrinolytic system)?
Fibrin & tissue plasminogen activator bind plasminogen → plasmin → cleaves fibrin → releases FDP (fibrin degradation products)

What clotting factors are measured when you order aPTT? PT?
-aPTT: "Intrinsic" pathway; all coagulation factors except VII and tissue factor
-PT: "Extrinsic" pathway; tissue factor, VII, V, X, II (prothrombin), fibrinogen
-factor VII, IX, X, II, protein C&S are vitamin K dependent → altered by warfarin

What happens in DIC? See figure 2
Coagulation and fibrinolysis become abnormally activated within the vasculature
Endothelial damage → activation of coagulation cascade → extensive thrombi in microvasculature and larger vessels→consumption of factors, platelets and anticoagulant factors (S, C, antithrombin)→ consumption greater than production "consumptive coagulopathy"→fibrinolysis→large amount of FDP interferes with fibrin clot formation and platelet aggregation→tissue/organ damage from reduced perfusion, thrombosis and/or bleeding
Pathophysiology in pregnancy:
Decidual cells lining the vascular bed of the placenta strongly express tissue factor (similar to endothelial cells); TF released at sites of decidual trauma→coagulation cascade activation
-Abruption, fetal demise: Release of procoagulants due to significant injury/necrosis of fetoplacental tissue
-AFE: Amniotic fluid rich in procoagulants and anticoagulants
-Preeclampsia, HELLP: Contribute to endothelial cell damage
-Hemorrhage leading to shock: severe tissue hypoxia proposed to result in release of TF from damaged cells

What are laboratory findings that are consistent with DIC?
↓Platelets, hemolytic changes in peripheral blood smear (anemia & schistocytes), ↓fibrinogen (normal fibrinogen levels range from 373 -619 mg/dL in 3rd trimester), ↑D-dimer (FDP), and ↑ PT and aPTT.

How do you treat DIC?
Focused on correcting the underlying cause, maintenance of maternal volume status, and replacement of blood products: platelets, fresh frozen plasma (contains all coagulation factors), cryoprecipitate (smaller volume than FFP, rich in von Willebrand factor, VIII, XIII, fibrinogen)

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Figure 1

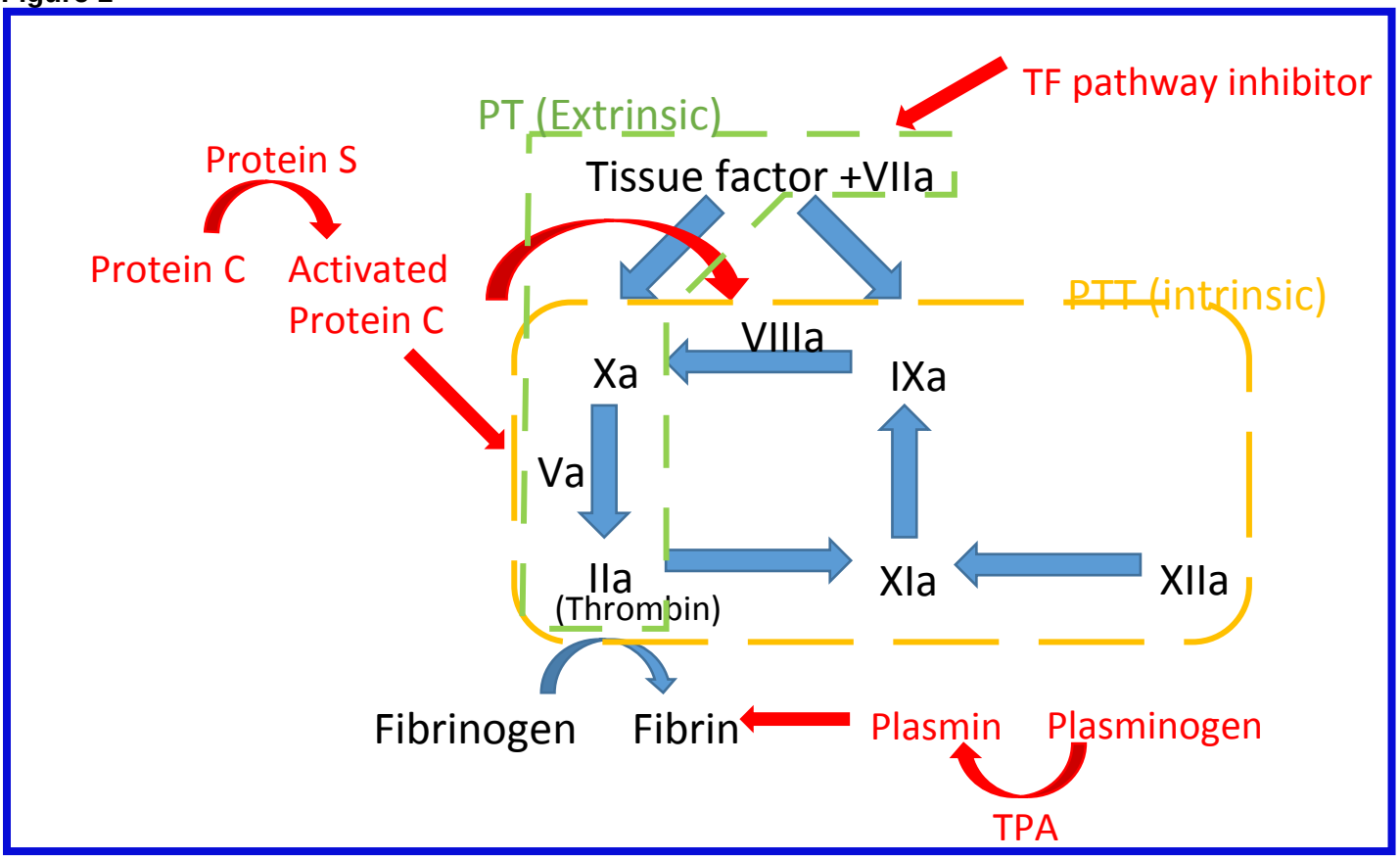
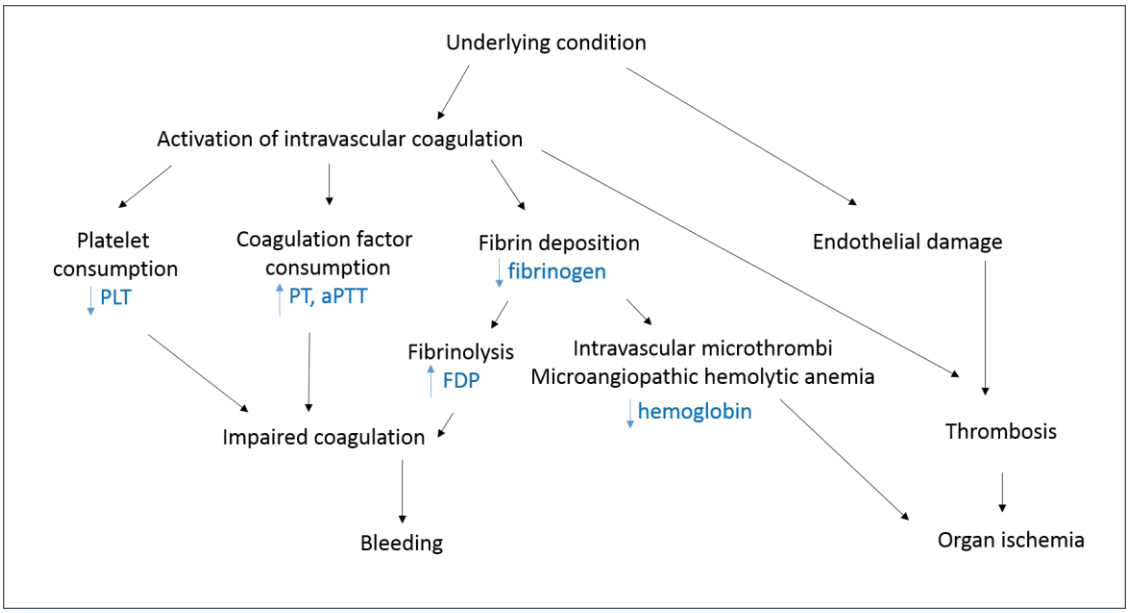


Figure 2



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