Central Nervous System Complications of Hemorrhagic and Coagulation Disorders

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KEYWORDS

• Hemorrhagic and coagulation disorders • Neurologic complications • MRI • CT

KEY POINTS

- Hematologic disorders can affect the central nervous system in a variety of ways, producing wide range of neurologic disturbances.
- It is important to identify these complications as early as possible for early intervention and better outcome.
- Cross-sectional imaging, mainly CT and MRI, plays an important role in identifying brain abnormalities and thus helps the clinician in deciding the appropriate course of action and treatment.

INTRODUCTION

Hematologic disorders can affect the central nervous system (CNS) in a variety of ways, producing a wide range of neurologic disturbances. It is important to identify these complications as early as possible for early intervention and better outcome. If untreated some of these complications may have irreversible deficit to fatal outcome. Today cross-sectional imaging, mainly computed tomography (CT) and MRI, plays an important role in identifying brain abnormalities and thus helps the clinician in deciding the appropriate course of action and treatment. This article reviews the basics of hemostasis including the coagulation cascade and the application of basic laboratory evaluation of hematologic function. The standard classification of the hematologic disorders is used to categorize the clotting and bleeding diatheses. The imaging features of various neurologic disorders associated with these clotting and bleeding diatheses are then discussed in detail.

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COAGULATION CASCADE

Normal hemostasis is an elaborate, multifactorial process involving the interaction of multiple elements to produce two main physiologic effects: the maintenance of liquidity of blood and prevention of bleeding after vessel damage. Normal platelet function maintains hemostasis in an undamaged vessel. Active hemostasis, or formation of fibrin in blood vessels, depends on progression through three main processes: (1) vascular, (2) platelet, and (3) coagulation stages of clot formation.[1] The vessel and vascular endothelium play an important primary role in the initiation of clotting. Vascular constriction at the time of injury can be the sole mechanism of preventing minor bleeding. In addition, vascular endothelium damage exposes blood to collagen, fibrinogen, and von Willebrand factor (vWF), which stimulates platelet adhesion. Conversely, intact endothelium is responsible for inhibition of hemostasis through prostacyclin and nitrogen oxide, which inhibit platelet aggregation.[1]

Platelet aggregation contributes to the next phase of hemostasis by binding circulating vWF and collagen released from injured endothelium to its surface receptors. After aggregation and activation, platelets undergo morphologic change forming a plug that releases substances, such as calcium, serotonin, and ADP, which promote further coagulation. Many additional factors, which play a role in coagulation, are released from dense granules secreted by aggregating platelets. These factors include platelet factor 3; β-thromboglobulin; platelet-derived growth factor; thrombospondin; factor V; and plasma proteins, such as fibrinogen and IgG.[1]

The final stage of the clotting process is the coagulation cascade, where a series of coagulation factors become sequentially activated to produce a fibrin clot (Fig. 1). There are two coagulation pathways, which culminate into a final common pathway. The liver produces factors involved in these pathways, therefore liver disease

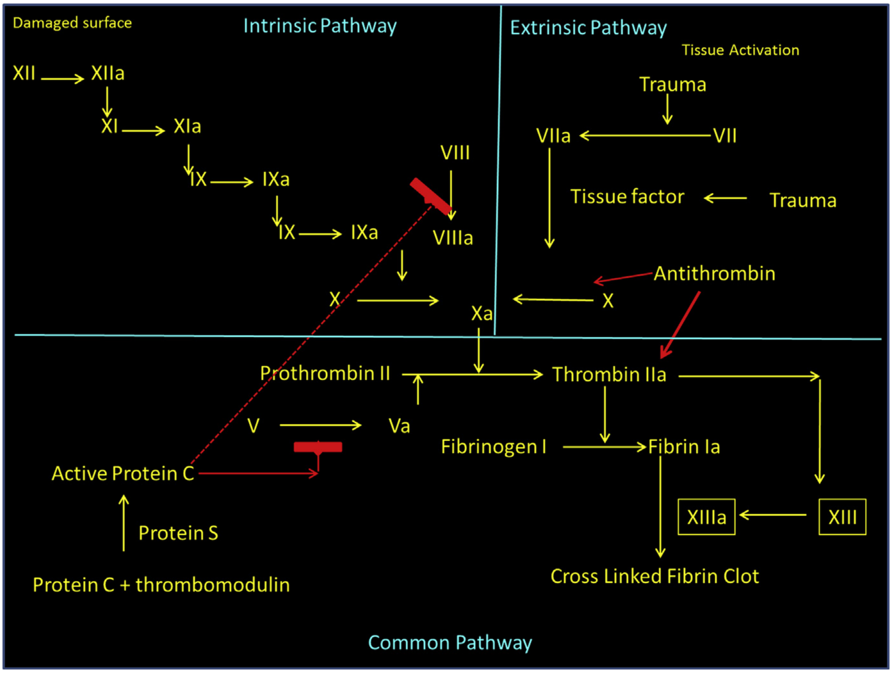

Fig. 1. Clotting cascade.