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COAGULOPATHY AND THROMBOTIC COMPLICATIONS IN POST COVID SYNDROME

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Coronavirus disease 2019 (COVID-19) is associated with a distinct coagulopathy characterized by a marked prothrombotic state. Key laboratory features include elevated D-dimer and fibrinogen levels, endothelial activation, platelet hyperreactivity, and a proinflammatory cytokine milieu. These alterations promote excessive thrombin generation while impairing fibrinolysis, shifting hemostasis toward thrombosis.

Clinically, this coagulopathy manifests as an increased risk of venous and arterial thromboembolic events. Venous thromboembolism (VTE) occurs in approximately 15–20% of hospitalized patients and up to 30–35% of those in intensive care, with even higher detection rates when systematic screening is applied. Pulmonary embolism and deep vein thrombosis are the most common complications, although catheter-related thrombosis and arterial events such as stroke and limb ischemia have also been reported. Disease severity correlates strongly with thrombotic risk, and elevated D-dimer levels are consistently associated with poor outcomes and increased mortality.

The pathophysiology of COVID-19-associated coagulopathy is multifactorial, involving systemic inflammation, endothelial injury, platelet activation, and blood flow stasis. A pronounced inflammatory response ("cytokine storm") increases procoagulant activity, particularly through interleukin-mediated upregulation of tissue factor. Endothelial dysfunction, partly due to viral interaction with ACE2 receptors, further promotes thrombosis via activation of the extrinsic coagulation pathway. This interaction between inflammation and coagulation is described as immunothrombosis. Unlike classical disseminated intravascular coagulation, COVID-19 coagulopathy presents with elevated fibrinogen and D-dimer levels, mild coagulation time prolongation, and relatively preserved platelet counts, reflecting a predominantly hypercoagulable state.

A representative clinical case highlights these challenges. A 71-year-old man with COVID-19 pneumonia developed atrial fibrillation and acute respiratory distress syndrome within 10 days of illness onset. Therapeutic anticoagulation was initiated upon admission; however, by day 20, a gluteal hematoma occurred, necessitating de-escalation to prophylactic dosing. Shortly thereafter, the patient exhibited thromboelastographic hypercoagulability, worsening dyspnea, and oxygen desaturation (SpO_2 82%), indicating ongoing thrombotic risk despite bleeding complications. Therapeutic low-molecular-weight heparin was reintroduced for two weeks, followed by transition to oral anticoagulants upon discharge. This case illustrates the delicate balance between thrombosis and bleeding in COVID-19 management.

Management of COVID-19-associated coagulopathy requires individualized risk assessment and vigilant monitoring. Anticoagulation strategies vary from prophylactic to therapeutic dosing, though optimal regimens remain under investigation. In the post-COVID phase, persistent hypercoagulability may contribute to ongoing morbidity, underscoring the need for long-term follow-up and tailored anticoagulant strategies.

In conclusion, COVID-19-associated coagulopathy is a complex prothrombotic condition that may persist beyond the acute phase and contribute to post-COVID syndrome. The interaction between inflammation, endothelial dysfunction, and coagulation abnormalities drives both acute and long-term thrombotic risk. Further research is needed to optimize risk stratification and guide effective anticoagulation strategies in the post-COVID setting.