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Letter to Editor

Hypercoagulability States in COVID-19 Patients and Cutaneous Findings as Its Marker

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Patients diagnosed with COVID-19 have a higher risk for thrombosis, which is the term used to denote an occlusion or blood clot that occurs in a vein [1-4]. These, in turn, can lead to detrimental consequences including motor impairments, as well as increased morbidity and mortality rates. Therefore, it is vital to assess routine laboratory and cutaneous findings to prevent any ensuing organ damage.

German physician Rudolf Virchow identified some of the primary factors that may increase an individual's susceptibility to developing thrombophilia [1-5]. Virchow's triad comprises endothelial vessel wall injury, stasis or slowing down of the blood, and finally, hypercoagulability [2-4]. These three factors can be exacerbated by many components, including an individual's genetic predisposition, or acquired factors like COVID-19, smoking, or hypertension [1,3,4]. Their contribution to hypercoagulability is discussed next.

Endothelial injury can lead to turbulences in blood vessels, subsequently altering blood flow dynamics. If left unchanged, an inflammatory response is mounted via the complement system. Activation of the complement system results in an increased concentration of proinflammatory cytokines, thus promoting a hypercoagulable state [1,2]. As discussed above, stasis, which is used to describe immobilization of blood flow can result from prolonged stillness and directly increases hypercoagulability in patients [1,2]. Finally, prothrombic factors from the coagulation cascade system of our body can be altered, thus leading to a hypercoagulable state [1,2]. The correlation of Virchow's triad as it relates specifically to COVID-19 patients will be discussed in detail below.

Studies have shown that endothelial cell injury can occur as a direct response to the COVID-19 virus [1-4]. The damage created by the virus leads to activation of the complement-mediated system factors C5b-9, which subsequently leads to hypercoagulability [2,3]. In addition, endothelial injury can also result from the catheterization and intubation procedures implemented in severe COVID-19 cases, which can cause physical damage to blood vessels. Additionally, COVID-19 patients, especially those necessitating hospitalization and ICU care, are at an increased risk of thrombosis due to their prolonged immobility. This largely sedentary state directly impacts blood flow dynamics, for it promotes the genesis of stasis. Thirdly, the elevation of prothrombic factors such as fibrinogen and D-dimmer levels have been reported in COVID-19 patients, which directly lead to prolongation of Prothrombin Time (PT) and activated Partial Thromboplastin Time (aPTT) [1-3].

Routine monitoring of blood labs, including Complete Blood Count (CBC), platelet count, coagulation studies (PT and aPTT), fibrinogen, and D-dimer levels should be assessed to evaluate the coagulability state of COVID-19 patients. Moreover, given the fact that a hypercoagulable state can also manifest as a dermatological condition, physicians can also use this as a metric to diagnose hypercoagulability in patients [5]. Some of the most common cutaneous manifestations of hypercoagulability include purpura, livedo reticularis, livedo vasculopathy, chronic venous ulcers, and superficial venous thrombosis [4,5]. The clinical manifestation of Deep Vein Thrombosis (DVT), Pulmonary Embolism (PE), stroke, limb ischemia, bleeding, and any associated dermatological findings urge us to investigate the relationship between COVID-19 and increased susceptibility to bleeding and thrombosis [1-3]. This way, physicians can predict hypercoagulability state severity in COVID-19 patients and be better equipped to assess the overall risk of morbidity and mortality.

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