Covid-19 Associated Coagulopathy

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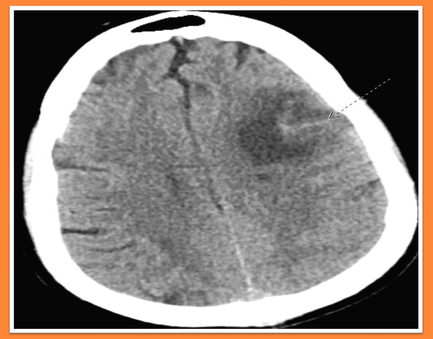
Introduction

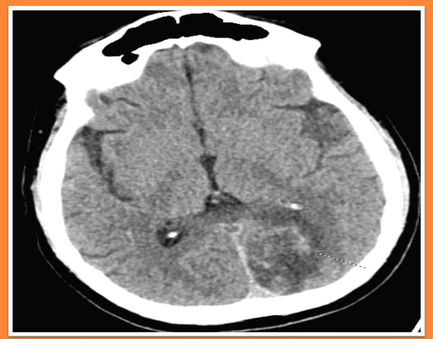
Coronavirus Disease 2019 (COVID-19) is a novel coronavirus that has not been previously identified. On February 11, 2020 the WHO officially named the virus and on March 11, 2020 they characterized the virus as a pandemic. As of January 2021, the virus has affected roughly 100 million people world-wide and close to 25 million in the United States alone. Individuals infected with COVID-19 may have several varied and complex coagulopathies that are not completely understood. Potential pathways that have been proposed to explain this coagulopathy range from disruption of Virchow's triad to the intrinsic and extrinsic coagulation pathways. This thromboinflammation can, in turn, result in ischemic events such as ulcers, myocardial infarction and, in the case of this presentation, strokes and deep venous thrombosis.

Case Description

We present a 45 year old male who presented for shortness of breath and syncope who was subsequently found to be positive for COVID-19. The patient developed respiratory failure requiring intubation and mechanical ventilation. A CT Brain demonstrated multiple acute infarctions in multiple vascular territories. Patient then developed bilateral deep venous thrombosis involving the femoral and popliteal veins. Due to his inflammatory markers being extremely elevated (D-Dimer >30.00, Ferritin 3,219) and clinical sequelae, he was started on therapeutic anticoagulation. The patient subsequently developed hemorrhagic conversion of his strokes, developed dysphagia, and required insertion of IVC filters in place of anticoagulation. Our patient presented at a max-mod assist functional level with multiple medical complications. After an intensive IRF course, the patient was discharged 10 days later at a supervision level. COVID-19 associated hypercoagulability is becoming a complicated phenomenon that is quickly occupying rehabilitation hospitals.

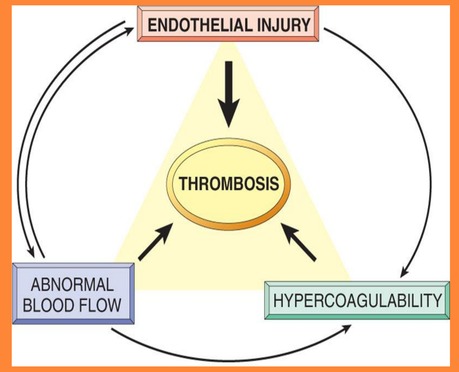
Hemorrhagic Conversion



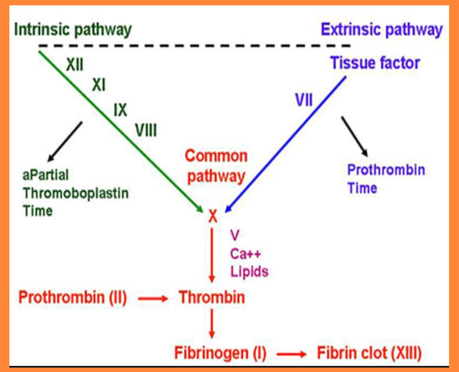


COVID-19 associated coagulopathy has been closely compared with Disseminated Intravascular Coagulation (DIC) based on laboratory data. However, the main clinical distinction between COVID-19 and DIC coagulopathies is that COVID-19 presents with thrombosis and DIC presents with bleeding. Interestingly, autopsies performed on post-mortem COVID-19 patients demonstrate deep venous thrombosis, pulmonary embolism, micro-emboli in the alveoli, endotheliatis, limb ischemia, and increased angiogenesis. LMW heparin is known to reduce the risk of VTE. VTE prophylaxis dosing has been associated with decreased mortality in a retrospective study of 449 individuals with COVID-19. However, in some patients – such as ours – the risk of thrombotic events is so high that there is indication for therapeutic dose anticoagulation. As often happens, there is a risk of hemorrhagic conversion. Due to the multi-factorial nature of COVID-19 associated coagulopathies, our patient failed anti-coagulation therapy and required placement of IVC filters secondary to hemorrhagic conversion.

Virchow's Triad



Coagulation Cascade



Results and Discussion

There is evidence of direct invasion causing injury to the endothelial cells caused by SARS-CoV-2. Other mediators of endothelial injury are thought to be due to a cytokine storm induced by Interleukin-6 as well as other inflammatory mediators. Tocilizumab is an immunosuppressant that is actively being used and researched for its role as an IL-6 inhibitor to block this exact process. In addition to Endothelial Injury, hyperviscosity of the blood plasma has been well documented in COVID-19 patients. This leads to a hypercoagulable state similar to that of Waldenstrom macroglobulinemia. Very elevated levels of D-dimer have been observed in COVID patients and correlate with disease severity. While intubated in the ICU, our patient had a D-dimer level above 30.00 mg/L FEU. His D-Dimer showed a slow downtrend over the following months, however was still elevated at 4.09 mg/L FEU at the end of his hospitalization in rehabilitation.

A portion of patients diagnosed with COVID-19 will inevitably have complicated hospital courses developing critical illness myopathies and in the case of our patient, vascular phenomenon. Rehabilitation hospitals are already quickly becoming an ideal destination for these patients who are decompensated and still require medical intervention and oversight. In the case of our patient, we hope to shed light on how complex the management of these patients can be and the difference that a short stay in a rehabilitation hospital can make in their recovery.

References

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