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COVID-19 INDUCED SPLENIC INFARCTION

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ABSTRACT

The Coronavirus (Covid-19) infection has been a global threat over the last 2 years. According to WHO, the Coronavirus Dashboard Tracker at the end of 2021, tracked over 274 million cases of Covid-19 and at least 5.34 million deaths. Despite the aggressive efforts to vaccinate as many people as possible, the cases are still on the rise, especially with the emergence of the new Omicron variant.[1] The Covid -19 infection has been reported to be associated with various venous as well as arterial thromboembolic phenomena.[2,3] This is due to the fact that the infection can trigger hypercoagulable status, due to the release of many inflammatory intermediates that can trigger endotheliitis and increased viscosity. We herein present a case of a 71-year-old gentleman who presented with acute splenic infarct two weeks after being treated in the hospital for COVID-19 pneumonia.

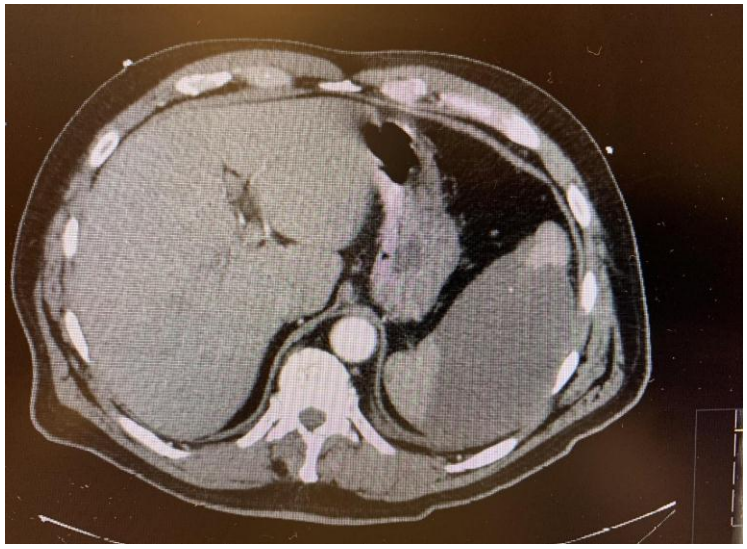
KEYWORDS: COVID-19 infection Splenic infarct Hypercoagulable status.

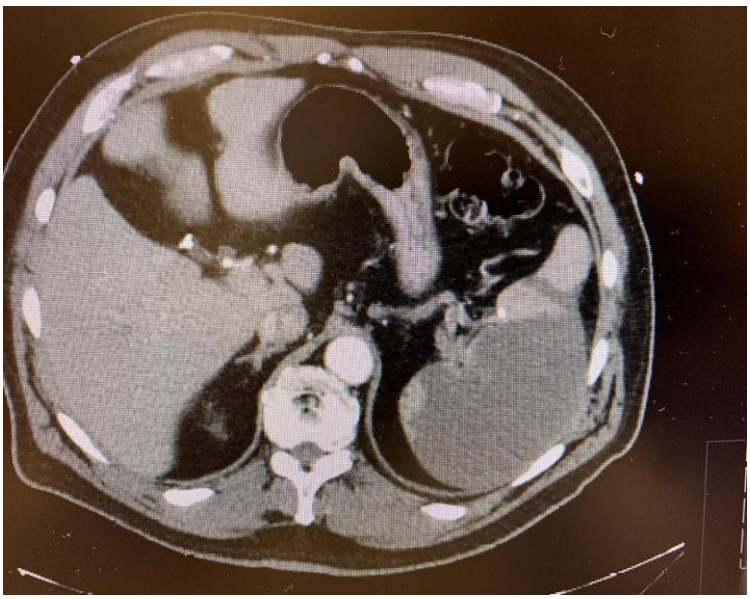
CASE REPORT

A 72-year-old Caucasian gentleman with a past medical history of Hypertension, Type 2 Diabetes mellitus, and Morbid obesity with body mass index (BMI) of 36 was recently admitted with COVID-19 Pneumonia and Acute hypoxic Respiratory failure requiring oxygen supplementation. He received Remdesivir and Decadron as part of his management. He improved clinically and was discharged after 5 days of hospitalization without requiring oxygen at home. During his stay in the hospital, he received VTE (venous thromboembolism) prophylaxis with Enoxaparin at 0.5 mg/kg subcutaneously every twelve hours. He presented 10 days after discharge with pleuritic pain involving the left chest and left upper quadrant of the abdomen. A CTA (Computed Tomography Angiography) was obtained to rule out pulmonary embolism. The scan revealed bilateral pulmonary infiltrates which were expected with his recent history of pneumonia. The CTA also revealed a partial thrombus in the celiac trunk with the splenic artery showing subtotal occlusion with large splenic infarct. There was no evidence of any cardiac involvement with a normal echocardiogram without any thrombi. Coagulation studies were obtained promptly as part of workup with the results in days ahead as follows. Antithrombin III Antigen was 87% (normal range 80-100%). Antithrombin Activity 89% (normal range 80-130%). Protein C Activity 90% (normal range 70-150%). Protein S Activity 104% (normal range 65-160%). Factor V Leiden Mutation negative. Partial Plasma Time (PTT) 29.1 seconds (normal range 23.8-33,8 seconds). Prothrombin Time (PT) 13.4 seconds (normal range 11.7-14.3). Antiphospholipid Antibody was negative.

The patient was admitted to the hospital. Surgery and hematology evaluated the patient. The recommendation was to treat patient with anticoagulation and observe closely.

He was put on a therapeutic dose of Heparin infusion. His symptoms improved significantly with improving pain and no evidence of any fever or leukocytosis. He was discharged on day 6 of hospitalization on treatment dose of Apixaban. Patient remained stable at 3 and 6 months follow up at the clinic.





DISCUSSION

COVID-19 pandemic has created a significant impact around the world. In the United States, the cases have exceeded 50 million, with around 800,000 deaths. COVID-19 has been associated with a multitude of complications ranging from pneumonia and respiratory failure, sepsis,[4,5] renal failure, neurological disorders to other hematological diseases including venous as well as arterial thrombo-embolic disorders.[6,7] There have been a number of reported cases of aortic thrombosis complicating COVID-19 infections including mural thrombi and mesenteric occlusion.[8] The SARS-COV-2 virus attacks the body by attaching its spike S glycoprotein to the Angiotensin Converting Enzyme 2 thus inhibiting the mitochondrial function in the host cell including the endothelium of the lungs and blood vessels.[9] This leads to endotheliitis and activation of coagulation cascade with the release of factor VIII, fibrinogen and lupus anticoagulants.[10] In addition, there is an increased number of prothrombotic microparticles released that can increase the viscosity of the blood.[11] All of the above lead to a hypercoagulable state. This risk tends to increase more in the ICU patients who tend to have a more prolonged course of immobilization. In a number of large studies involving ICU patients hospitalized with COVID complications, the incidence of VTE (venous thromboembolism) ranged from 6.2 to 13.6 percent.[12,13,14] Those are relatively better numbers than the initial studies done in the beginning of the pandemic. There are a number of reported cases of Limb ischemia, Strokes, Myocardial Infarction as well as Aortoiliac occlusions. Splenic Infarct, although rare, can be underdiagnosed especially if the patient has mild symptoms. Without utilizing radiology procedures like CT computed tomography, the diagnosis can be missed.[15]

There are a number of possible mechanisms contributing to Splenic Infarct. One of the possibilities is most likely due to hypercoagulable state with increased levels of Factor VIII, Factor X, as well as Fibrinogen leading to endotheliitis and occlusion of the blood vessels of the spleen which is highly vascular. Also, the humoral B response to the infection increasing leukocytic reaction can ultimately stagnate in the spleen causing congestion and infarct.

Due to the increased risk of thromboembolic complications, there are new guidelines which are recommended by multiple medical societies in the United States including The American College of Cardiology, American Society of Hematology, NIH (National Institute of Health) and many other reputable societies. It is recommended that VTE pharmacologic prophylaxis is recommended for all COVID -19 hospitalized patients.[16] Also, it is recommended that patients with elevated VTE risk, length of stay in hospital more than 3 days with low risk of bleeding should receive VTE prophylaxis for 30 days with Rivaroxaban or Enoxaparin, both being the drugs of choice. There still seems to be a great need for more extensive studies to evaluate the efficacies of different anticoagulants in preventing and treating thromboembolic complications ofCOVID-19 infection.

CONCLUSION

COVID-19 infection can be associated with multiple complications. One of these are thromboembolic events affecting arterial as well as venous circulatory systems. By giving prophylactic anticoagulation to hospitalized patients we can decrease the severity of such complications if not prevent them. Early diagnosis and treatment of such events can change mortality and morbidity in this patient population.

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