Acute liver failure in asymptomatic COVID-19

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Introduction

Coronavirus disease 2019 (COVID-19) has been studied extensively since its emergence with considerable focus on the secondary hypercoagulable state and high incidence of thromboembolic events. While cases of pulmonary embolism (PE) have been widespread and numerous in severe COVID-19, there are very few cases of marked thromboembolic events in patients with mild symptoms. Furthermore, cases of thromboembolic sequelae in young age groups have not been described. We present the first case of bilateral pulmonary embolism and acute liver failure without findings of severe COVID-19.

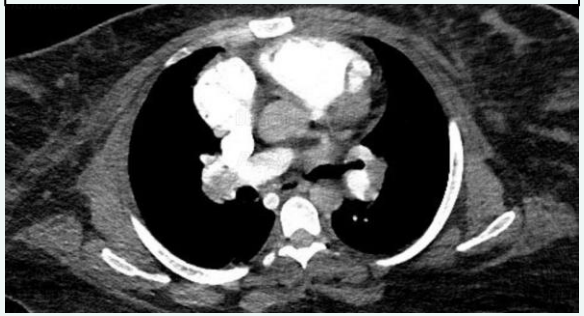
Case Report

A 30-year-old G5P5 African American female with a history of bipolar disorder 1 and polysubstance abuse presented from home via EMS with witnessed tonic-clonic seizures for the previous 3 days.

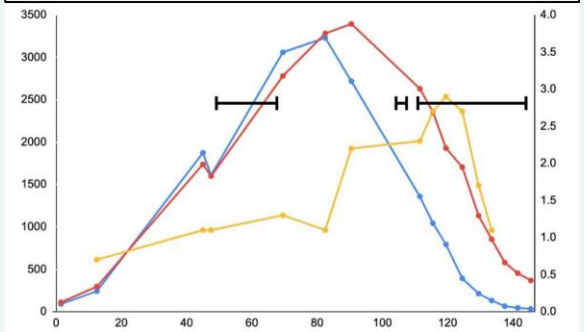
On admission, she had a mild transaminitis. Within 24 hours her liver function tests (LFTs) showed an AST 1874, ALT 1738, and LDH 2837. A urine drug screen was obtained and was positive for phencyclidine. Ultrasound abdomen showed hepatomegaly and cholelithiasis, and questionable portal vein thrombosis. She was started on a heparin drip. CT abdomen pelvis with IV contrast showed thickening of gallbladder with a large gallstone and no abnormalities noted in any vessels. While WBC count was only 10.9, she was started on piperacillin-tazobactam and metronidazole in hope to treat a possible underlying cholecystitis. Her liver enzymes increased to AST 3060, ALT 2681, LDH 4835 and platelets dropped from 179k to 33k. At this point her COVID-19 PCR resulted to be positive. Heparin was held and platelets were ordered. She remained hemodynamically stable on room air.

She was then transferred to a tertiary center where CT chest showed bilateral saddle PE with right heart strain, confirmed by transthoracic echocardiogram. She was treated with N-acetylcysteine, vitamin K, platelets, and cryoprecipitate. She was started on a bivalirudin drip, later transitioned to fondaparinux. HIT assay, ADAMTS13, and human chorionic gonadotropin were negative. Her liver enzymes trended down with anti-coagulation suggesting a pathology of shock liver.

Imaging



Lab Studies



Timeline of liver enzyme and total bilirubin measurement in a patient with hypoxic liver injury. The red colored line represents AST, the blue line represents ALT, and the yellow line represents bilirubin. The blue and red lines are represented by the left y-axis, whereas the yellow line is represented by the right y-axis. The first horizontal bold line represents IV heparin; the second bold line represents IV bivalrudin and the third bold line represents IV fondaparinox.

Discussion

Severe COVID-19 is defined as a SpO2 <94% on room air at sea level, a respiratory rate of >30 breaths/min, PaO_2/FiO_2 < 300 mm Hg, or lung infiltrates >50%. The incidence of PE in COVID-19 is reported as 2.6 - 8.9% for hospitalized patients and diagnosed in up to one third of critically ill patients. Our patient's lack of typical symptoms and stable vital signs made PE an unlikely clinical diagnosis. As the timeline suggests, the pulmonary embolism took an indolent course to present itself later as the inciting factor for hypoxic injury to the liver. At first consideration, this patient matches the typical presentation for acute liver failure: previously healthy, age in the 30s, hepatic dysfunction, coagulopathy, and markedly elevated liver chemistries. However, it was not until the patient received a CT of her chest that hypoxic liver injury was diagnosed. When acute liver injury is present in setting of hypercoagulable conditions, hypoxic hepatitis or ischemic hepatitis should be considered. While asymptomatic COVID-19 is typically not considered to be a hypercoagulable state, the possibility of thromboembolic event should not be overlooked.

Conclusion

Submassive pulmonary emboli in patients without respiratory symptoms or chest discomfort is rare. However, the suggestion that secondary hypercoagulability due to COVID-19 causing drastic subclinical and potentially life-threatening scenarios is concerning. This case provides an example of the insidious nature of COVID-19 and adds caution to the broad range of associated symptoms.

References

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