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CASE REPORT | LIVER

Alcoholic Hepatitis and COVID-19: The Question of Steroids

Sara Zelman, MD[1], Erik Holzwanger, MD[1], Raza Malik, MD[1], and Aaron Dickstein, MD[1]

[1]*Department of Gastroenterology and Hepatology, Tufts Medical Center, Boston, MA*

ABSTRACT

Severe acute respiratory syndrome coronavirus 2/novel coronavirus-19 (COVID-19) has rapidly become a global pandemic since the first cases from Wuhan, China, were reported in December 2019. The pandemic has made it more challenging to treat various gastrointestinal disorders, including acute alcoholic hepatitis (AH). One of the mainstays of treatment for severe AH involves corticosteroids (mainly prednisolone). A concern when treating with prednisolone is the worsening of underlying infection. There may be an additional risk in treating COVID-19–infected patients. We present a case of a patient with severe acute AH and concomitant COVID-19 infection who did well with corticosteroid therapy without evidence for worsening infection.

INTRODUCTION

Novel coronavirus-19 (COVID-19) has created new challenges for gastroenterologists when managing common gastroenterological disorders.[1] In particular, the decision to initiate steroids in acute alcoholic hepatitis (AH), which is always challenging in normal times, is now a more difficult decision given concern for worsening infection. This is compounded in patients with COVID-19 because there are early data suggesting worsened outcomes with steroids in COVID-19–infected patients without AH.[2] AH is a syndrome with a constellation of symptoms that can range from mild symptoms to fulminant liver failure.[3,4] Several models have been created to assess the severity of AH. One of these models is the Maddrey discriminant function (MDF), which is useful for estimating short-term mortality and determining which patients may benefit from corticosteroids.[3] Patients with an MDF score of 32 or higher have a 28-day mortality of approximately 65%.[2] Studies suggest that the use of corticosteroids in this population may reduce the risk of death within 28 days of treatment; however, no long-term mortality benefit exists.[5,6] How these patients respond to steroids while also having COVID-19 infection is an open question. We present a case of a patient with severe acute AH and COVID-19 infection who responded well to corticosteroid therapy without adverse outcomes related to the infection.

CASE REPORT

A 57-year-old homeless man with chronic alcohol abuse and alcoholic cirrhosis with known portal hypertension presented with a dry cough, worsening fatigue, and melena for 1 week. Leading up to his presentation, he had been drinking 1 pint of vodka daily. On presentation, he had normal hemodynamics and his oxygen saturation was 98% on room air. A rectal examination did not reveal melena or hematochezia. Laboratory data were notable for a normal blood urea nitrogen/creatinine, hemoglobin/hematocrit of 6.4 g/dL/21.6%, platelets of 54 K/μL, total bilirubin/direct bilirubin 10.3 mg/dL/6.0 mg/dL, alkaline phosphatase 162 IU/L, aspartate aminotransferase 260 IU/L, alanine aminotransferase 53 IU/L, and international normalized ratio (INR) 1.9. Viral hepatitis panel was unremarkable. COVID-19 polymerase chain reaction test was positive. Chest x-ray revealed bibasilar opacities. Right upper quadrant ultrasound with Doppler revealed cirrhotic morphology of the liver and no portal vein thrombosis. MDF was 58.6. Model For End-Stage Liver Disease score was 22. He was started on a continuous octreotide infusion, intravenous pantoprazole, and intravenous ceftriaxone given concern for gastrointestinal bleed. The patient became acutely hypoxic with an oxygen saturation of 80% on room air at the initiation of the blood transfusion, which was believed to be secondary to COVID-19 infection. He was admitted to the intensive care unit for close monitoring, but did not require intubation.

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Correspondence: Sara Zelman, MD (szelman@tuftsmedicalcenter.org).

Endoscopic evaluation was deferred given COVID-19 infection, recent EGD without concerning lesions, and lack of overt bleeding. He was not initiated on treatment for COVID-19. On hospital day 3, he became encephalopathic and was started on lactulose and rifaximin with improvement. At the same time, he began having worsening kidney function and was given a working diagnosis of hepatorenal syndrome, which improved with albumin. He also had worsening coagulopathy with INR 3.4 and significant worsening of his total bilirubin to 27.9 mg/dL. He continued to be COVID-19 positive on repeat testing.

His MDF increased to 155, and he was started on prednisolone 40 mg daily. Seven days into therapy, total bilirubin down trended to 21.8 and kidney function improved. A Lille score was calculated to be 0.980. Although the Lille score suggested the patient may be a corticosteroid nonresponder, his improved mental status, decreased total bilirubin, and improved kidney function suggested he was responding to treatment and led to the decision to continue prednisolone for 28 days total. His respiratory status remained stable-to-improved, and he did not require supplemental oxygen during the remainder of his hospital course.

DISCUSSION

There are several contraindications to corticosteroid therapy for acute AH including active infection due to risk of worsening sepsis, and gastrointestinal bleeding, as steroids can lead to gastrointestinal mucosal damage with subsequent bleeding .[7] The COVID-19 pandemic has created a potential new challenge in treating AH. We expect that the incidence of AH will increase from increased alcohol intake due to psychosocial factors spurred on by the pandemic.[8] For these patients, gastroenterologists will have to weigh the risk/benefits of corticosteroid therapy in the setting of COVID-19 positivity.

The current recommendation is that corticosteroids should generally be avoided in viral infections because they may delay viral clearance, thus prolonging disease course.[2,9–11] Previous studies that have evaluated corticosteroids in this setting have shown that severe AH carries an increased risk of infection. In particular, nonresponders of corticosteroid therapy seem to be at greater risk.[12] Thus, use of the Lille score, especially in the COVID-19–positive patients, may be even more important to prevent worsened infections. Fortunately, in our case, the patient did well despite being a nonresponder per the Lille score.

Basing corticosteroid initiation purely on MDF will also create an obstacle because hepatocellular injury and cholestatic injury are known consequences of COVID-19, and may cause an elevated score not solely related to inflammation from alcohol.[13,14] Many medications used to help treat the virus may also cause hepatotoxicity.[13,14] In our case, severe AH seemed to be more influential on mortality than active COVID-19 infection. This in turn led us to initiate and continue corticosteroid therapy, albeit with a questionable Lille score, due to significant symptomatic improvement and decreased total bilirubin.

We present this case to add to the growing body of COVID-19 literature. We show that in a COVID-19–positive patient with AH, prednisolone therapy can be safely used because we did not find any adverse effects on his infection status. Based on our case, COVID-19 positivity may not be an absolute contraindication to treatment. Larger prospective studies will need to evaluate the safety profile of corticosteroids in acute AH and COVID-19 infection.

DISCLOSURES

Author contributions: All authors contributed equally to this manuscript. A. Dickstein is the guarantor.

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