### **Post-COVID-19 Cholestatic** hepatitis

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#### Introduction:

- In addition to the pulmonary consequences, the incidence of liver injury is being reported.
- o understanding the hepatotropism of SARS-COV-2 is of paramount importance as it can lead to a more severe COVID-19 course.

### **Case Presentation:**

A 49-year-old lady with a history of seizure disorder, migraines, hypertension, and recent six-week ICU admission for COVID illness requiring mechanical ventilation (ECMO), presented to our gastroenterology clinic for a post-discharge follow-up. During admission, she consistently had an abnormal elevation of liver enzymes (Peak ALP 2000, AST, ALT 300, Bilirubin 2.5). MRCP was normal, and a liver biopsy showed intralobular bile duct injury showing cytologic atypia, periductal edema, periductal-intraductal neutrophilic/ lymphocytic inflammation, focal mild hepatocellular cholestasis, Kupffer cell hyperplasia. She had no history of chronic liver disease, and LFT was normal at baseline. Post-discharge LFT showed alkaline phosphatase of 1514 with total bilirubin 1.4. Infective (Hepatitis panel), Inflammatory/ Autoimmune (ANA, ASMA, AMA, P-ANC, Anti-SLA/LP, Anti ds-DNA, ALKM-1, and ALKM-3) workup was negative. Repeat imaging with USG and MRI was unremarkable. She received a course of prednisone after discharge with no improvement in LFT; hence it was tapered off. She is currently asymptomatic six months after acute illness. However, She continues to have persistently elevated liver enzymes with a gradual downtrend, the latest being ALP 490, AST 86, ALT 95, and T. Bilirubin 0.8.

# Post-COVID-19 Cholestatic Hepatitis. Do we know it all?

# DISCUSSION!

• Only six cases reported to date.

Largely Asymptomatic.

• Persistently elevated liver biochemistry with unclear etiology despite extensive workup. • A confluence of SSC-CIP (Secondary Cholangitis of Critically III Patients) and direct hepatic injury from COVID-19 is the suspected pathophysiology. ACE2 is expressed primarily in cholangiocytes, which has been suggested as the entry point for hepatobiliary damage.

 SARS-CoV-2 DNA has been isolated from postmortem livers, and electron microscopy has demonstrated viral particles within hepatocytes with viral cytopathic effects. • More research is warranted regarding this syndrome because it has been implicated in ongoing liver damage severe enough to warrant liver transplantation. • Ursodeoxycholic acid and obeticholic acid can improve symptoms of cholestatic liver disease.



**Questions? Contact Information:** 

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## **Histology:**





### **References:**

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