INTRODUCTION

The ongoing COVID-19 infection has demonstrated potential devastating organ damage including direct liver injury; however the long-term complications are limited. We demonstrate a case of post COVID-19 cholangiopathy with prolonged hyperbilirubinemia, partially responsive to ursodiol.

CLINICAL CASE

Patient is a 59-year-old male presented with weakness and COVID-19 infection. His clinical course continued to deteriorate with multi-organ failure. After a prolonged ICU course, patient’s respiratory failure improved, but had a persistent hyperbilirubinemia. Patient underwent ERCP showing a normal-appearing common bile duct with no evidence of any filling defects (Figure 2). He underwent autoimmune workup with positive ASMA with a titer 1:20, but negative ANA and AMA. HIDA scan showed poor clearance and no activity within the biliary tree, suggestive of underlying intrinsic hepatic abnormality. MRCP showed innumerable cystic dilations and areas of narrowing (Figure 1). Liver biopsy showed widespread cholestasis, bridging fibrosis, and iron deposition primarily within Kupffer cells. Repeat ERCP about 100 days later, showed diffuse intrahepatic cholangiopathy in a pattern most consistent with PSC (Figure 3).

Patient was started on ursodiol 250 mg three times a day with a mild improvement of bilirubin levels of about 25%. After a 6-month hospital course, patient was discharged to an acute care facility. Upon discharge, patient remained jaundice with persistent liver enzymes and a bilirubin of 21 mg/dL. Ultimately, he died without a clear cause nearly 10 months from the initial diagnosis.

DISCUSSION

We described a case of an individual who recovered from severe sepsis secondary to COVID-19 developing significant post COVID-19 cholangiopathy. Despite recovery, patient continued to have chronic cholestasis and liver injury. Liver biopsy demonstrated predominant cholangiocyte injury with bridging fibrosis consistent with progression of secondary sclerosing cholangitis (SCC). Based on histopathological changes, it is postulated that damage occurs secondary to direct bile duct ischemia. While no standard therapies exist and only 1 case report showing post COVID-19 liver transplantation, many therapeutic options are limited. However, in our case, ursodiol demonstrated a significant improvement with a plateau of about 25% of the total bilirubin. SCC carries a poor outcome in the general population without a liver transplantation. Early identification and treatment will be vital with the ongoing pandemic as SCC is associated with considerable morbidity and mortality left untreated.

REFERENCES

