International Journal of Clinical Science and Medical Research
ISSN(print): 2770-5803, ISSN(online): 2770-582X
Volume 06 Issue 01 January 2026
DOI: https://doi.org/10.55677/IJCSMR/V6I1-02/2026, Impact Factor: 8.005
Page No : 05-09

Ischaemic Sclerosing Cholangitis Post COVID-19: A Case Report

H.H. Abakar[1*], H. Guenim[2], D. Labarriere[3], JP. Lagasse[4]
[1,2,3,4]Gastroenterology Department, Orléans University Hospital, Orléans, France.

ABSTRACT

Coronavirus disease 2019 (COVID-19) primarily presents with respiratory symptoms. However, it is increasingly recognized as a multisystem condition involving extra-pulmonary complications, including hepatic involvement.

Objective: We report a case of hepatic injury in the form of ischemic sclerosing cholangitis secondary to SARS-CoV-2 infection.

Case report: A 71-year-old female patient was admitted to the intensive care unit for two months due to COVID-19 viral pneumonia complicated by acute respiratory distress. During the course of her hospitalization, she developed abnormal liver tests suggestive of cholestasis. A diagnosis of post-COVID-19 ischemic cholangitis was established. She was treated with ursodeoxycholic acid combined with bezafibrate. The clinical course was unfavorable, marked by the development of secondary cirrhosis complicated by edema-ascites decompensation. Given her age, liver transplantation was not considered.

Conclusion: Post-COVID-19 ischemic cholangitis is a rare but serious complication. It should be considered in the presence of persistent cholestasis following severe COVID-19 pneumonia.

Published Online: January 05, 2026

KEYWORDS:
COVID-19 pneumonia, Cholestasis, Ischemic cholangitis.

INTRODUCTION

Coronavirus disease 2019 (COVID-19), caused by SARS-CoV-2, mainly presents with respiratory symptoms. However, it is increasingly recognized as a multisystem disorder, involving extrapulmonary complications, particularly at the hepatic level.

Liver involvement related to COVID-19 typically manifests, in the early stages of the disease, as a hepatocellular injury often associated with a minor elevation in liver enzymes. However, in more advanced stages, some patients may develop severe cholestatic liver injury. We report a case of hepatic involvement in the form of ischaemic sclerosing cholangitis secondary to SARS-CoV-2 infection.

MEDICAL CASE REPORT

This concerns a 71-year-old female patient, hypertensive and diabetic under treatment, followed since 2017 for a low-grade non-Hodgkin lymphoma initially treated with rituximab and bendamustine, then rituximab and chloraminophene, in complete hematologic remission. She was hospitalized in intensive care for 2 months for a COVID-19 viral pneumonia associated with septic shock and paroxysmal atrial fibrillation. Her respiratory status deteriorated rapidly, requiring invasive ventilation just hours after admission, which had to be maintained for 42 days.

During her hospitalization, she initially developed an increase in gamma-GT to 246 U/L with no other liver-function abnormalities (initial liver panel was normal), then one month later a progressive icteric cholestasis appeared: alkaline phosphatase 259 U/L (N < 120), gamma-GT 328 U/L (N < 36), with bilirubin 29 µmol/L (predominantly conjugated) and a mild transaminasaemia (< 2× the upper limit of normal). Three months later there was a marked increase with alkaline phosphatase 1429 U, gamma-GT
1699 U, bilirubin 47 µmol/L, AST 40 U/L, ALT 27 U/L. The etiological work-up found no viral cause (neither HBV nor HCV), no signs of autoimmunity.

Abdomino-pelvic CT scan showed no morphological argument for chronic liver disease, no focal hepatic lesion, no portal thrombosis.

Corresponding Author: H.H. Abakar

Cite this Article: H.H. Abakar, H. Guenim, D. Labarriere, JP. Lagasse (2026). Ischaemic Sclerosing Cholangitis Post COVID-19: A Case Report. International Journal of Clinical Science and Medical Research, 6(1), 05-09. https://doi.org/10.55677/IJCSMR/V6I1-02/2026

MRCP (magnetic resonance cholangiography) found a pattern compatible with sclerosing cholangitis, showing upstream dilatations and narrowed/stented intra-hepatic bile ducts mainly in the right liver lobe without nodular lesion on contrast injection.

Ileo-colonoscopy, searching for a chronic inflammatory bowel disease, was unremarkable.

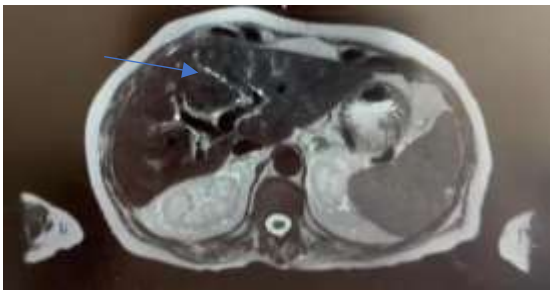
Serum IgG4 levels were within normal range, and HIV serology was negative. FibroScan revealed increased hepatic stiffness at 23.5 kPa, though this finding was interpreted with caution given the presence of cholestasis.

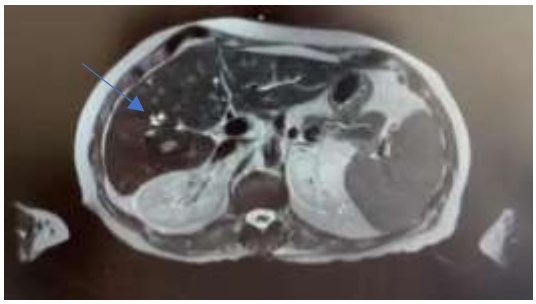
Upon review of the MRCP (Images 1, 2), the findings were considered not suggestive of primary sclerosing cholangitis. A thorough review of all therapeutic agents administered during the patient's ICU stay, compared with data reported in the literature, did not identify any drug known to be associated with cholangiopathy. The final diagnosis retained was postICU ischemic cholangitis. Treatment with ursodeoxycholic acid at 20 mg/kg/day was initiated, along with cholestyramine to manage the onset of pruritus.

The subsequent course was initially marked by persistent cholestasis with disabling pruritus. Follow-up MRCP (Images 3, 4) revealed worsening of tight, stepwise strictures affecting the intrahepatic bile ducts and the upper biliary confluence, sparing the main bile duct. It also showed dilatation of the intrahepatic bile ducts and marked enlargement of lymphatic vessels, suggesting ischemic cholangitis. Addition of a PPAR agonist (bezafibrate) was then proposed.

Over a three-year follow-up, the disease progressed to secondary cirrhosis with edematousascitic decompensation unresponsive to diuretics, requiring repeated ascitic drainages.

Considering the patient's age, liver transplantation was not deemed an appropriate option.





Figures 1 and 2: MR cholangiography slices (2020) showing dilatations of the intra-hepatic bile ducts (arrows)

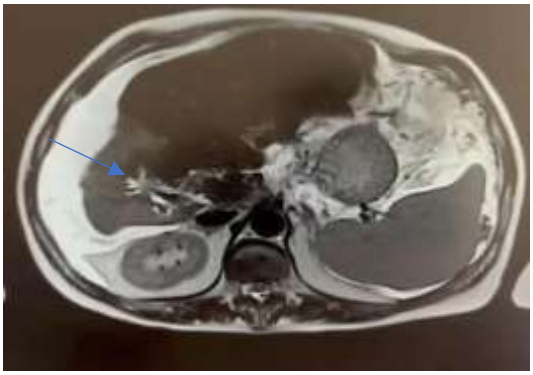


Figure 3: MR-cholangiography slice (2025): persistent dilatation of the right intra -hepatic bile ducts with a dysmorphic liver associated with signs of portal hypertension.

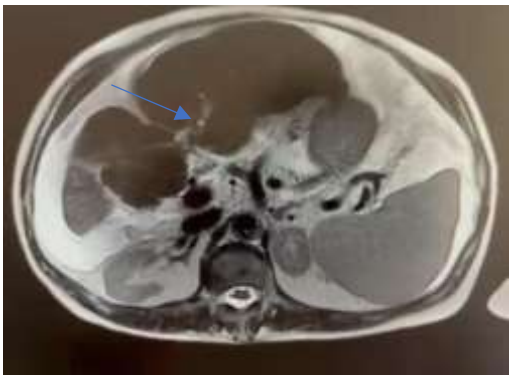


Figure 4: MR cholangiography slice (2025): a very tight stricture of the confluence of the intra-hepatic bile ducts.

DISCUSSION

Considered a variant of secondary sclerosing cholangitis in critically ill patients, ischemic cholangitis associated with SARS-CoV-2 infection (post-COVID-19 ISC) is an emerging disease. It occurs particularly in patients who have undergone prolonged management in intensive care units [1–7]. It is characterized by ischemic lesions of the bile ducts caused by respiratory failure, systemic hypotension, or vasopressor administration [1,8].

A German study conducted by Christoph R. Werner et al. in 2020 reported an incidence of 7.8 % (5 out of 64) confirmed cases of CSI-post COVID-19 among ventilated patients with ICU stays longer than 20 days [9].

In January 2021, Roth et al. reported three similar cases. Faruqui et al. published a case series in 2020 of 12 patients

with persistent cholestasis after severe COVID-19, consistent with a diagnosis of CSI [10,11].

The pathophysiology of post-COVID-19 ischemic sclerosing cholangitis (post-COVID-19 ISC) appears to be multifactorial, involving several interconnected mechanisms. It partly results from direct cholangiocyte injury caused by ischemia or hypoxia due to respiratory distress, but also from bile toxicity resulting from altered bile composition, or even direct viral infection. Severe hypotension observed in ICU patients can induce ischemic injury of the bile ducts and contribute to the pathogenesis by altering hepatobiliary transporters. Moreover, sepsis and microcirculatory disturbances are also implicated in this functional alteration [12].

Additionally, hepatic injury associated with SARS-CoV-2 has been widely reported throughout the pandemic. Numerous studies have highlighted elevated liver enzymes from the early stages of infection, as well as parenchymal liver damage. These findings are explained by overexpression of the angiotensin-converting enzyme 2 (ACE2) receptor, particularly in cholangiocytes, which facilitates direct cellular destruction by the virus [13–15].

Furthermore, microvascular changes, including hepatic steatosis and intrahepatic thrombosis, resulting from the thrombogenic properties of the virus, also contribute to parenchymal lesions [14,15]. Finally, Kaltschmidt et al. showed that SARS-CoV-2 can replicate in the liver parenchyma and be secreted into the bile ducts. This replication, combined with platelet activation and inflammatory lesions of the parenchyma, appears to cause direct damage to the biliary system. Biliary necrosis, accompanied by the development of bile duct stenosis, is a determining factor in the onset of sclerosing cholangitis [16].

The main clinical manifestations of post-COVID-19 ischemic sclerosing cholangitis

(CSI-post COVID-19) are jaundice and pruritus, with a biological cholestasis characterised by elevated serum alkaline phosphatase, gamma-glutamyl transferase and bilirubin levels. They typically occur at a later stage of the disease, generally after recovery from acute respiratory illness. The average time between initial infection and diagnosis of CSI-post COVID-19 is reported to range between 90 and 118 days [8,3]. Among other systematically reported findings are male predilection, a median age over 50 years [17,18], and comorbidities such as hypertension or diabetes mellitus [19,8,3,20].

Although definitive criteria have not been established, the generally accepted definition of CSI-post COVID-19 includes the presence of severe cholestasis, as well as biliary-tract abnormalities on imaging or pathology that were not documented prior to COVID-19 [17,19,21].

The imaging findings of ischemic sclerosing cholangitis post-COVID-19 (CSI-post COVID-19) strongly resemble those seen in primary sclerosing cholangitis (PSC) and other secondary forms of sclerosing cholangitis. As in those conditions, one classically observes multifocal strictures of the intra-hepatic bile ducts, producing the characteristic “beaded” appearance [18,19,21]. These strictures may sometimes occur without upstream dilatation**,** making detection difficult on standard CT. Thus, magnetic resonance cholangiopancreatography (MRCP) is the diagnostic modality of reference, as it better visualises these abnormalities.

Imaging also reveals thickening of the bile-duct walls, often associated with contrast enhancement, as well as biliary stasis. Notably, the ductal abnormalities seen in post-COVID-19 cholangiopathy appear almost exclusively in the intra-hepatic bile ducts, with the extra-hepatic ducts generally spared. This particular topographic pattern was confirmed in a study of 17 patients, in which all patients had intra-hepatic involvement and only 5.9 % had associated extra-hepatic duct involvement [18]. This distinctive distribution may help differentiate post-COVID-19 ischemic sclerosing cholangitis (CSI-post COVID-19) from primary sclerosing cholangitis (PSC), which typically affects both biliary territories [22].

Other authors have reported additional radiological abnormalities including the presence of gallstones, biliary sludge in the ducts or gallbladder, as well as hepatic abscesses [2,23]. Furthermore, morphological changes of the liver such as hepatomegaly or a cirrhotic appearance have been observed in nearly 20 % of cases [18].

On the histopathological level, post-COVID-19 ischemic sclerosing cholangitis is characterized by typical cholangiocyte lesions. These include degenerative alterations such as cytoplasmic vacuolization, necrosis and apoptosis. A ductular reaction is frequently observed, marked by variable proliferation of bile ducts, accompanied by a light to moderate mixed inflammatory infiltrate within the portal tracts. Portal and septal fibrosis are also noted, as well as concentric periductal fibrosis characteristic of the chronic evolution of bile-duct injury.

Microvascular changes add to this picture, particularly signs of microangiopathy. This manifests as endothelial edema associated with luminal narrowing of the hepatic arteries, as well as endophlebitis of the portal veins [2,24,25]. These alterations suggest an ischemic mechanism involved in the development of liver lesions. In this context, Tsutsumi et al. reported a correlation between elevated fibrinogen levels and liver injury in COVID-19 patients, concluding that hepatic dysfunction could result from microvascular thrombosis [26].

The study by Shih et al., which to date represents the largest series of liver biopsies (seven cases), confirmed these characteristic lesions of post-COVID-19 cholangiopathy [27]. In contrast, the work of Esposito et al., conducted on patients with secondary sclerosing cholangitis not related to COVID-19, did not find similar results, which supports the hypothesis of a distinct pathological phenotype specific to post-COVID-19 SSC [28]. In our case, a liver biopsy was not performed following a collegial decision.

Treatment options for post-COVID-19 SSC remain limited and unstandardized. Most patients reported in the literature received ursodeoxycholic acid (UDCA). Similarly to the management of patients with primary sclerosing cholangitis, UDCA could protect cholangiocytes from inflammation [29]. At the same time, endoscopic retrograde cholangiopancreatography (ERCP) has been suggested as a way of improving prognosis, particularly in patients with biliary obstruction [30,31]. However, the benefits of ERCP in this context remain unclear, and this invasive procedure carries significant risks, including pancreatitis and bacterial cholangitis.

Although some patients with post-COVID-19 SSC recover, many eventually progress to biliary cirrhosis or liver failure, requiring liver transplantation [17, 21].

Liver transplantation is the only curative option for advanced cases of post-COVID-19 SSC. Several studies have reported that up to 22% of patients with ischemic sclerosing cholangitis require transplantation [1,2,24,32,33]. In another series, 50% of the 12 patients with postCOVID-19 SSC either underwent liver transplantation or were listed for it [3].

Without transplantation, the prognosis remains very poor: only 40.0% of patients were alive one year after the onset of post-COVID-19 SSC. Leonhardt et al. [5] reported mortality rates of 60% during the first 12 months and 83% over a 24-month period. In the case of our patient, although the disease progressed to decompensated cirrhosis with ascites and edema, her age precluded consideration of liver transplantation.

CONCLUSION

Post-COVID-19 SSC is a rare but serious complication. Any prolonged cholestasis following severe COVID-19 pneumonia should raise suspicion for this diagnosis. Imaging with MRCP and/or ERCP, along with histopathological examination, are reliable and effective methods for diagnosis. Although observations have shown that UDCA can improve liver function tests, liver transplantation appears to be a salvage treatment in severe cases. The prognosis for these patients is often poor, with a high risk of complications in the absence of liver transplantation, as illustrated by the case of our patient.

REFERENCES

1. Edwards, K.; Allison, M.; Ghuman, S. Secondary sclerosing cholangitis in critically ill patients: A rare disease precipitated by severe SARS-CoV-2 infection. BMJ Case Rep. 2020, 13, e237984. [CrossRef] [PubMed] 12.
2. Roth, N.C.; Kim, A.; Vitkovski, T.; Xia, J.; Ramirez, G.; Bernstein, D.; Crawford, J.M. Post-COVID19 Cholangiopathy: A Novel Entity. Am. J. Gastroenterol. 2021, 116, 1077–1082. [CrossRef] [PubMed]
3. Faruqui, S.; Okoli, F.C.; Olsen, S.K.; Feldman, D.M.; Kalia, H.S.; Park, J.S.; Stanca, C.M.; Figueroa Diaz, V.; Yuan, S.; Dagher, N.N.; et al. Cholangiopathy After Severe COVID-19: Clinical Features and Prognostic Implications. Am. J. Gastroenterol. 2021, 116, 1414–1425. [CrossRef] [PubMed]
4. Arnstadt, B.; Zillinger, C.; Treitl, M.; Allescher, H.D. Corona again? SSC after a severe COVIDdisease. Z. Gastroenterol. 2021, 59, 1304–1308. [CrossRef]
5. Leonhardt, S.; Jurgensen, C.; Frohme, J.; Grajecki, D.; Adler, A.; Sigal, M.; Leonhardt, J.; Voll, J.M.; Kruse, J.M.; Korner, R.; et al. Hepatobiliary long-term consequences of COVID-19: Dramatically increased rate of secondary sclerosing cholangitis in critically ill COVID-19 patients. Hepatol. Int. 2023, 17, 1610–1625. [CrossRef]
6. Hartl, L.; Haslinger, K.; Angerer, M.; Semmler, G.; Schneeweiss-Gleixner, M.; Jachs, M.; Simbrunner, B.; Bauer, D.J.M.; Eigenbauer, E.; Strassl, R.; et al. Progressive cholestasis and associated sclerosing cholangitis are frequent complications of COVID-19 in patients with chronic liver disease. Hepatology 2022, 76, 1563–1575. [CrossRef] [PubMed]
7. Butikofer, S.; Lenggenhager, D.; Wendel Garcia, P.D.; Maggio, E.M.; Haberecker, M.; Reiner, C.S.; Brullmann, G.; Buehler, P.K.; Gubler, C.; Mullhaupt, B.; et al. Secondary sclerosing cholangitis as cause of persistent jaundice in patients with severe COVID-19. Liver Int. 2021, 41, 2404–2417. [CrossRef] [PubMed]
8. Hunyady P, Streller L, Rüther DF, Groba SR, Bettinger D, Fitting D, et al. Secondary sclerosing cholangitis following coronavirus disease 2019 (COVID-19): a multicenter retrospective study. Clin Infect Dis 2023;76:e179-e187
9. Christoph R. Werner, Stefano Fusco, Nisar P. Malek, Dörte Wichmann , Katharina Kienzle Incidence of Secondary Sclerosing Cholangitis in Hospitalized Long COVID-19 Patients: A Retrospective Single Center Study
10. Faruqui, S.; Okoli, F.; Olsen, S.; Feldman, D.M.; Kalia, H.S.; Kim, S. Bile duct injury and severe cholestasis in patients recovering from severe COVID-19: A novel entity of COVID-associated cholangiopathy. AASLD Digit. Exp. 2020, 42. Available online: https://www.natap.org/2020/AASLD/AASLD_136.htm (accessed on 16 August 2024).
11. Henrie, J.; Gerard, L.; Declerfayt, C.; Lejeune, A.; Baldin, P.; Robert, A.; Laterre, P.-F.; Hantson, P. Profile of liver cholestatic biomarkers following prolonged ketamine administration in patients with

COVID-19. BMC Anesthesiol. 2023, 23, 44. [CrossRef]

12. Leonhardt S, Veltzke-Schlieker W, Adler A, Schott E, Eurich D, Faber W, Neuhaus P, Seehofer D. Secondary Sclerosing Cholangitis in Critically Ill Patients: Clinical Presentation, Cholangiographic Features, Natural History, and Outcome: A Series of 16 Cases. Medicine (Baltimore) 2015; 94: e2188 [PMID: 26656347 DOI: 10.1097/MD.0000000000002188]
13. Sharma A, Jaiswal P, Kerakhan Y, Saravanan L, Murtaza Z, Zergham A, Honganur NS, Akbar A, Deol A, Francis B, Patel S, Mehta D, Jaiswal R, Singh J, Patel U, Malik P. Liver disease and outcomes among COVID-19 hospitalized patients - A systematic review and meta-analysis.
14. Ann Hepatol 2021; 21: 100273 Chan HL, Kwan AC, To KF, Lai ST, Chan PK, Leung WK, Lee N, Wu A, Sung JJ. Clinical significance of hepatic derangement in severe acute respiratory syndrome. World J Gastroenterol 2005;
15. Zhao B, Ni C, Gao R, Wang Y, Yang L, Wei J, Lv T, Liang J, Zhang Q, Xu W, Xie Y, Wang X, Yuan Z, Zhang R, Lin X. Recapitulation of SARS-CoV-2 infection and cholangiocyte damage with human liver ductal organoids. Protein Cell 2020;
16. Kaltschmidt B, Fitzek ADE, Schaedler J, Förster C, Kaltschmidt C, Hansen T, Steinfurth F, Windmöller BA, Pilger C, Kong C, Singh K, Nierhaus A, Wichmann D, Sperhake J, Püschel K,
17. Huser T, Krüger M, Robson SC, Wilkens L, Schulte Am Esch J. Hepatic Vasculopathy and Regenerative Responses of the Liver in Fatal Cases of COVID-19. Clin Gastroenterol Hepatol 2021;
18. Faruqui S, Shanbhogue K, Jacobson IM. Biliary tract injury in patients with COVID-19: a review of the current literature. Gastroenterol Hepatol
19. Ghafoor S, Germann M, Jüngst C, Müllhaupt B, Reiner CS, Stocker D. Imaging features of COVID-19-associated secondary sclerosing cholangitis on magnetic resonance cholangiopancreatography: a retrospective analysis. Insights Imaging 2022;13:128
20. Yanny B, Alkhero M, Alani M, Stenberg D, Saharan A, Saab S. Post-COVID-19 cholangiopathy: a systematic review. J Clin Exp Hepatol 2023;13:489-499
21. Heucke N, Keitel V. COVID-19-associated cholangiopathy: what is left after the virus has gone? Hepatology 2022;76:1560-1562
22. Veerankutty FH, Sengupta K, Vij M, Rammohan A, Jothimani D, Murali A, et al. Post-COVID-19 cholangiopathy: current understanding and management options. World J Gastrointest Surg 2023;15:788-798
23. Seo N, Kim SY, Lee SS, Byun JH, Kim JH, Kim HJ, et al. Sclerosing cholangitis: clinicopathologic features, imaging spectrum, and systemic approach to differential diagnosis. Korean J Radiol 2016;17:25-38
24. Kulkarni AV, Khelgi A, Sekaran A, Reddy R, Sharma M, Tirumalle S, et al. Post-COVID-19 cholestasis: a case series and review of literature. J Clin Exp Hepatol 2022;12:1580 1590
25. Durazo, F.A.; Nicholas, A.A.; Mahaffey, J.J.; Sova, S.; Evans, J.J.; Trivella, J.P.; Loy, V.; Kim, J.; Zimmerman, M.A.; Hong, J.C. Post-COVID-19 Cholangiopathy—A New Indication for Liver Transplantation: A Case Report. Transplant. Proc. 2021, 53, 1132–1137
26. Ferreira, F.B.; Mourato, M.; Bragança, S.; Paulo, J.B.; Sismeiro, R.; Pereira, A.; Mónica, A.N.; Lourenço, L.C.; Cardoso, M. COVID-19-associated secondary sclerosing cholangitis—A case series of 4 patients. Clin. Res. Hepatol. Gastroenterol. 2022, 46, 102048.
27. Tsutsumi T, Saito M, Nagai H, Yamamoto S, Ikeuchi K, Lim LA, et al. Association of coagulopathy with liver dysfunction in patients with COVID-19. Hepatol Res. 2021;51(2):227– 232.
28. Shih, A.R.; Hatipoglu, D.; Wilechansky, R.; Goiffon, R.; Deshpande, V.; Misdraji, J.; Chung, R.T. Persistent Cholestatic Injury and Secondary Sclerosing Cholangitis in COVID-19 Patients. Arch. Pathol. Lab. Med. 2022, 146, 1184–1193.
29. Esposito, I.; Kubisova, A.; Stiehl, A.; Kulaksiz, H.; Schirmacher, P. Secondary sclerosing cholangitis after intensive care unit treatment: Clues to the histopathological differential diagnosis. Virchows Arch. 2008, 453, 339–345.
30. Aguilar MT, Chascsa DM. Update on emerging treatment options for primary biliary cholangitis. Hepat Med. 2020;12:69–77.
31. Engler S, Elsing C, Flechtenmacher C, et al. Progressive scleros ing cholangitis after septic shock: a new variant of vanishing bile duct disorders. Gut. 2003;52:688–93.
32. Voigtlander T, Negm AA, Schneider AS, et al. Secondary scleros ing cholangitis in critically ill patients: model of end-stage liver disease score and renal function predict outcome. Endoscopy.
33. Keta-Cov research group. Electronic address vmaf, Keta-Cov research g. Intravenous ketamine and progressive cholangiopathy in COVID-19 patients. J Hepatol. 2021;74(5):1243–1244
34. Voigtlander T, Jaeckel E, Lehner F, Manns MP, Lankisch TO. Liver transplantation for critically Ill patients with secondary sclerosing cholangitis: outcome and complications. Liver Transpl. 2015;21(10):1295–1299