Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Correspondence

COVID-19-related thrombotic microangiopathy in a cirrhotic patient

A 56 year-old cirrhotic patient presented in the emergency department on March 12th 2020 with melena, haematuria and petechiae; he reported fever in the previous two weeks.

He had an history of hepatitis C virus infection with sustained virological response to direct acting antivirals in 2015. In 2016, he developed a non-neoplastic spleno-porto-mesenteric thrombosis with cavernomatous transformation and was started on fondaparinux 5 mg/day. At the last outpatient visit on February 2020, his Child-Pugh score was B8 and the MELD score was 14, haemoglobin level was 13 g/dL and the platelet count was 36,000 × 10^9/L. The most recent upper endoscopy showed small oesophageal varices without red signs and mild hypertensive gastropathy.

At admission, a rhino-pharyngeal swab was performed which tested positive for SARS-CoV-2 infection and a chest CT showed bilateral interstitial pneumonia. The patient presented mild dyspnoea with a respiratory rate of 22 and a SpO2 of 95% on room air. Platelet count was 1000 × 10^9/L and haemoglobin level was 8.5 g/dL; over the first 48 h haemoglobin dropped to a minimum level of 5.5 g/dL. Laboratory tests were consistent with thrombotic microangiopathy: LDH 400 IU/L, D-dimer 10,5 mcg/mL. Fondaparinux was stopped. The patient required a total amount of 14 units of erythrocytes and 19 units of buffy-coat platelets. Antiplatelet indirect antibodies were found to be negative while anti-HLA class I antibodies were positive. Dexamethasone and intravenous immunoglobulins were used but proved ineffective.

At day seven, the patient experienced acute liver decompensation with large ascites, and acute kidney injury-hepatorenal syndrome type I, requiring treatment with albumin and terlipressin infusion. At day 20, RF swab was repeated and tested negative for SARS-CoV-2 infection and the result was confirmed on two other swabs, 48 h apart. After viral clearance, laboratory tests and clinical conditions progressively improved. Platelet count increased and stabilized around 10–15,000/10^9/L, haemoglobin level exceeded 8 g/dL without further transfusion requirement. Haemorrhagic manifestations also resolved. The Child-Pugh score gradually improved from C11 to B8, and the MELD score from 22 to 16.

The novel coronavirus infection (now classified as SARS-CoV-2), first identified in December 2019 in Wuhan, has contributed to significant mortality in different countries [1]. This case suggests that SARS-CoV-2 can trigger severe thrombotic microangiopathy which can end up with uncontrolled bleeding, due to endothelial lung damage resulting in microthrombi formation and platelet consumption. The pathogenesis of thrombocytopenia could also recognise other mechanisms [2,3]:

a. development of autoantibodies or immune complexes;
b. direct infection of hematopoietic stem / progenitor cells: following virus infection, the cytokine storm destroys bone marrow cells and leads to a decrease in platelet production.

The clinical consequences are to be feared in patients with cirrhosis due to the concomitant reduction of thrombopoietin and presence of hypersplenism.

Bacterial infections are a recognized precipitant factor of acute decompensation and/or acute-on-chronic liver failure in patients with cirrhosis. This case suggests that SARS-CoV-2 infection could be a causal factor precipitating liver decompensation.

Declaration of Competing Interest

None

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