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Atypical COVID–19 presentation with Budd-Chiari syndrome leading to an outbreak in the emergency department

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1. Introduction

The SARS-CoV-2 virus infection causes a broad spectrum of presentation, ranging from asymptomatic or mild upper airway symptoms to acute respiratory failure [1]. The recognition of a vascular involvement associated with a hypercoagulable state allows atypical clinical presentation with any vascular territory involvement [2,3]. We describe an atypical presentation of coronavirus disease 2019 (COVID–19) with Budd-Chiari syndrome due to hepatic vein thrombosis without respiratory symptoms during the early phase that causes an emergency department outbreak because this patient was not initially placed into a private room.

2. Case report

A 50-years-old woman was admitted to the emergency department with severe right upper quadrant abdominal pain for 6 days associated with nausea and vomiting. She observed both jaundice and unmeasured fever 2 days before hospitalization. The patient has a previous asthma diagnosis and uses an inhalator corticoid. She denied pregnancy, oral contraceptive use, and alcohol consumption. On physical examination, we observed jaundice and pain during palpation of the right upper quadrant abdominal. Her vital signals on presentation showed a blood pressure of 110/70 mmHg, a pulse of 95 beats per minute, respiratory rate of 20 cycles per minute, and arterial oxygen saturation of 96% on room air. Initially, she denied any respiratory symptoms. The general laboratory tests are shown in Table 1. Abdominal ultrasound showed hepatomegaly and enlarged spleen, without biliary tract obstruction and ascites. Firstly, the differential diagnosis was performed among some infectious or autoimmune diseases. However, the laboratory tests for infectious and autoimmune diseases were negative. Table 1

On the third day of hospitalization, the patient started with a sore throat and a runny nose. At this moment, she was placed into a private room, and the real time polymerase chain ration (RT-PCR) of the oropharyngeal swab for the SARS-CoV-2 virus was positive. At this time, a second blood test was performed, and the tests did not confirm this diagnosis, except for the presence of low levels of anti-cardiolipin IgM. Table 1 The patient presented a good clinical evolution with normalization of the liver biochemical tests. A new MRI was performed 3 months later and showed complete resolution of the left suprahepatic vein thrombosis, and the repeated anti-cardiolipin IgM was negative. This patient's clinical presentation was compatible with a Budd-Chiari syndrome, and since no other predisposing factor was found, we believe that this event was related to SARS-CoV–2 virus infection. Initially, because of this atypical presentation, this patient stayed out of a private room. After this, twenty healthcare workers of the emergency department were confirmed for SARS-CoV-2 virus infection in the next 2 weeks. We hypothesized that this patient could have been the index case of this hospital outbreak.
Thrombosis in COVID-19 patients can occur in a variety of vascular territories, including the portal vein, suprahepatic vein, mesenteric vein, etc. This can be a consequence of activated coagulation, endotheliopathy, up-regulated innate and adaptive immunity, and the activated complement system. These events associated with COVID-19 seem to be related to in situ thrombosis caused by multifaceted mechanisms including activated coagulation, endotheliopathy, up-regulated innate and adaptive immunity, and the activated complement system.

Post-mortem liver biopsies from 48 patients who died from severe COVID-19 disease confirm that liver failure is not a main finding. These findings ruled out antiphospholipid syndrome diagnosis. There are descriptions of transitory anti-cardiolipin antibodies positivity in SARS-CoV-2 infected patients [13].

These events associated with COVID-19 seem to be related to in situ thrombosis caused by multifaceted mechanisms including activated coagulation, endotheliopathy, up-regulated innate and adaptive immunity, and the activated complement system [2].

In our case, the patient underwent an extensive investigation, and we did not find another cause for this thrombotic presentation beyond the acute SARS-CoV-2 virus infection. Only low titers of anti-cardiolipin IgM antibody were observed during the acute phase; however, this repeated test was negative after 3 months. These findings ruled out antiphospholipid syndrome diagnosis. There are descriptions of transitory anti-cardiolipin antibodies positivity in SARS-CoV-2 infected patients [13].

Post-mortem liver biopsies from 48 patients who died from severe COVID-19 disease confirm that liver failure is not a main concern and this organ is not the target of significant inflammatory damage. On the other hand, the findings are highly suggestive for marked derangement of intrahepatic blood vessel network secondary to systemic changes induced by the virus [14].

The emergency physician needs to recognize these atypical manifestations and place patients with similar presentations immediately into the private room while waiting for RT-PCR for SARS-CoV-2 virus results to avoid emergency department outbreaks.
in patients with undefined abdominal pain and elevated liver biochemical tests in the department emergency during COVID-19 pandemic.

Declaration of competing interest

The authors declare no conflict of interest.

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