

Title:

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Authors:

Irene Chivato Martín-Falquina, Sara García-Morán, Manuel Alfonso Jiménez Moreno

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Acute pancreatitis in SARS-CoV-2 infection. Beyond respiratory distress

Irene Chivato Martín-Falquina, Sara García-Morán and Manuel Alfonso Jiménez

Moreno

Department of Digestive Diseases. Hospital Universitario de Burgos. Burgos, Spain

Correspondence: Irene Chivato Martín-Falquina

e-mail: ichivatomf@saludcastillayleon.es

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Dear Editor,

We read with great interest the two letters published in November (1,2) regarding

SARS-CoV-2 infection and acute pancreatitis (AP). We report our only case of AP

related to such infection.

Case report

A 55-year-old male, with no past medical history or toxic habits was admitted to the

Intensive Care Unit (ICU) in March 2020, presenting respiratory insufficiency and a

confirmed SARS-CoV-2 infection. He was treated with hydroxychloroquine, lopinavir,

azithromycin and methylprednisolone, according to the valid protocol at that time.

After removing such drugs, laboratory test showed hyperamylasemia. A computed

tomography was performed, revealing signs of acute interstitial edematous

pancreatitis, with peripancreatic collections of up to 6 cm. Laboratory tests excluded

autoimmunity, hypertriglyceridemia and hypercalcemia as etiological agents.

Abdominal ultrasound and endoscopic ultrasound (EUS) showed no irregularity in the

gallbladder or bile ducts, or anatomical abnormalities of the pancreas. EUS-guided



cystogastrostomy drainage was performed using lumen opposing stent. The patient was discharged and was asymptomatic. After having reasonably dismissed other etiologies, we considered that the SARS-CoV-2 infection was related to AP.

Discussion

There are only few articles about AP caused by SARS-CoV-2. In a 52 COVID-19 patient series, 17 % developed pancreatic damage. In another cohort with 67 patients, pancreatic injury was described in 2 % of the cohort (4). Previous studies have shown that SARS-CoV-2 binds angiotensin-converting enzyme 2 receptors to enter, replicate inside and destroy pancreatic cells (2). Pancreatic insult is also related to an incommensurate immune response to the viral antigen (3). Regarding a thrombotic origin proposed by Cienfuegos (2), our patient was previously anticoagulated. Despite being an atypical manifestation of SARS-CoV-2 infection, we believe AP must be taken into consideration in patients with abdominal symptoms. Larger series are needed to determine predisposing factors to develop pancreatic injury.

References

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