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Letter to the Editor

Pancreas and Coronavirus Disease-2019

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Severe acute respiratory syndrome coronavirus 2 (SARS-COV-2) virus affects many other organs than lung and upper airways. Gastrointestinal system appears to be affected both in the upper and in the lower portions. Moreover, hepatic, myocardial, and renal involvements are reported. Furthermore, pancreas can be affected in coronavirus disease-2019 (COVID-19) and the presence of SARS-COV-2 in the pancreatic tissue has been confirmed during autopsy.[1]

This is related to the presence of angiotensin-converting enzyme type 2 (ACE-2) receptor localized both in the exocrine and endocrine parts of pancreas. Thus, Yang et al[2] and colleagues reported frequent hyperglycemia in SARS-COV infections that can be related to damage of pancreatic islets (through ACE-2 binding).

Following, I presented a brief review of the few papers described the potential relationship between COVID-19 and pancreas.[3-8]

Liu et al[3] and colleagues confirmed that the possible pancreatic damage of SARS-COV-2 infection may be related to ACE-2 expression and assessed that expression of this receptor is higher in the pancreas than in the lung according to ribonucleic acid (RNA) analysis. One hundred twenty-one (121) COVID-19 positive patients of their case series reported high-levels of amylase and lipase according to the severity of the disease; but pancreatic necrosis was not seen at imaging in the study. Moreover, the authors pointed drug-induced pancreatic damage due to non-steroidal anti-inflammatory drugs (NSAIDs) and/or glucocorticoids as possible confounding factor. It was also underlined that the prolonged effect on pancreas could cause a worsening of systemic inflammation and acute respiratory distress syndrome (ARDS) and could evolve in chronic pancreatitis. I agree with the authors that, on one hand, pancreatic damage can contribute to the "cytokines' storm" during COVID-19 from activation of complement system (as in acute pancreatitis) and subsequent worsening of ARDS; on the other hand, chronic pancreatic damage can follow the same mechanisms of fibrosis observed for the lung.

Wang et al[4] and colleagues discovered that 17% of 52 COVID-19 patients had a pancreatic injury (defined as increased blood level of amylases and lipases): for me, this is a controversial finding.

Commenting on this finding, De-Madaria et al[5] explained that increased amylases and lipases in COVID-19 patients can be associated to acidosis, renal failure and gastroenteritis, especially if imaging is negative for pancreatic injury. And, I completely agree that hyperamylasemia and hyperlipasemia do not mean pancreatitis in absence of symptoms and imaging correlation.

Mukherjee et al[6] concluded that, considering the available data, COVID-19-associated pancreatic dysfunction may exist and can be responsible of atypical "pancreatitis-like" clinical presentation of the disease. However, the authors did not report any personal cases and based their conclusions on the previous findings that I reported above.

Hadi et al[7] described two out of three family members diagnosed with acute pancreatitis associated with COVID-19 excluding many other potential confounding factors.

Patel et al[8] reported that gastrointestinal symptoms, included abdominal pain in 2.2% of patients, occurred later than respiratory symptoms without a clear physiopathological mechanism. I think that late onset is at the base of the doubts of COVID-19 related pancreatitis.

The American National Pancreas Foundation, on its website, assessed that, on one hand, acute pancreatitis can worsen the immune response to COVID-19 and that, on the other hand, chronic pancreatitis-associated diabetes represents a bad prognostic factor in COVID-19 patients.

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Recently (during April 2020), we reported four cases of hyperamylasemia associated to hyperlipasemia in COVID-19 patients in Niguarda Hospital, Milan, Italy. Three cases resolved spontaneously in a mean of 15-days and no abdominal imaging was performed; one evolved in walled-off pancreatic necrosis (WOPN) treated by endoscopic ultrasound-guided transgastric drainage.

We can conclude that the expression of ACE-2 in pancreatic tissue make the pancreas a target organ for SARS-COV-2 and the virus can damage both exocrine and endocrine function of the gland. Increase of amylases and lipases can be caused by many confounding factors in COVID-19 patients; so, imaging and symptomsare important to evaluate the true involvement. Considering that abdominal pain is one of the known symptoms associated with SARS-COV-2 infection we must consider acute pancreatitis as a main cause especially in case of diffuse or upper abdominal pain.

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