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Acute Pancreatitis: an Unusual Complication of COVID 19 - about a Case Report

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Abstract **Case Report**

Covid 19 is an emerging infectious disease, responsible for severe acute respiratory syndrome. It has an essentially respiratory tropism, but this does not prevent it from causing a series of complications affecting several organs. Acute pancreatitis is an acute inflammation of the pancreas caused mainly by bile duct obstruction and alcohol abuse. In this work, we report a rare and unusual complication of this virus affecting the pancreas in a 38-year-old woman who developed acute pancreatitis associated with SARS-CoV-2 infection. We would also discuss the causal link between SARS-CoV-2 and acute pancreatitis, as well as their different diagnostic, epidemiological and therapeutic aspects.

Keywords: Acute pancreatitis; Covid 19; Infection etiology, Case report.

I. INTRODUCTION

Covid 19 is an emerging infectious disease, responsible for severe acute respiratory syndrome, caused by a virus called SARS COV 2, which belongs to the coronavirus family. This disease firstly reported in China in December 2019, could be declared as a 21st century pandemic because of its high contagiousness [1].

SARS-CoV-2 has an essentially respiratory tropism, but this does not prevent it from causing a series of complications, affecting several organs. Pneumonia, respiratory failure, renal failure and sepsis are the most frequent complications, but also disseminated intravascular coagulation, pneumothorax, myocarditis and rhabdomyolysis [2].

In this work, we report a rare and unusual complication of this virus affecting the pancreas in a 38-year-old woman who developed acute pancreatitis associated with SARS-CoV-2 infection and was admitted to the medical intensive care unit of the Mohamed V military training hospital in Rabat.

Through this clinical case and a review of the literature, we would also discuss the causal link between SARS-CoV-2 and acute pancreatitis, as well as their different diagnostic, epidemiological and therapeutic aspects.

II. CASE REPORT

A 38 year old woman, unvaccinated against SARS-COV-2, her comorbidities include chronic hypertension under unspecified treatment and which was complicated by nephropathy at an advanced renal failure, not yet dialysed, with iron deficiency anaemia. The patient had no history of biliary disease, pancreatic disease including diabetes. She denied any dependence on alcohol, medication or any symptoms pointing to severe acute respiratory syndrome.

She presented to the emergency department with early and intermittent postprandial emesis of food, which had been occurring abruptly for a week without pain, fever or other significant signs.

The initial clinical examination revealed that the patient was in good condition, conscious, with good spatio-temporal orientation, anicteric, apyretic. Respiratory, hemodynamic and neurological functions were stable. Her BMI was normal. Abdominal examination found a soft abdomen with normal hepatosplenic volumes, with no evidence of portal hypertension or hepatocellular failure. The rest of the physical examination findings were unremarkable.

Initial laboratory findings were relevant for high lipase (>3000 IU/l) which was greater than 38 times the upper limit of normal, collapsed haemoglobin at 5.8 g/dl, hyperleukocytosis at 9800 cells/µl with 92% neutrophils, C-reactive protein at 57 mg/l, renal failure with glomerular filtration rate at 2 ml/min/1. 73m², hyponatremia, hypochloremia with metabolic acidosis.

Table 1: Initial biological findings of the patient

	Result	Unit	Normal range
Leucocytes	9.8	$X10^3$/µL	4 - 10
Neutrophils	92.6	%	42 - 77
Lymphocytes	2.7	%	20 - 45
Erythrocytes	2.26	$X10^6$/µL	3.9 – 5.5
Hemoglobin	5.8	g/dL	12 - 16
PCV (Hematocrit)	16.3	%	36 - 47
MCV	72	fL	82 - 98
MCH	25.8	Pg	27 - 33
MCMH	35.9	g/dL	23 - 36
Platelets	145	$X10^3$/µL	150 - 450
Lipase	>3000	U/L	<78
AST	20	U/L	<35
ALT	14	U/L	<40
GGT	19	U/L	<32
PCR	57.3	mg/L	<5
Na^+	116	mmol/L	135 - 145
K^+	3.6	mmol/L	3.7 – 5.3
Cl^-	88	mmol/L	95 - 110
HCO_3^-	9	mmol/L	21 - 28
Proteins	78	g/L	64 - 83
Urea	2.67	g/L	0.15 – 0.38
Creatinine	174	mg/L	6 - 13
GFR	2	ml/min/1.73m^2	>60
Troponin	12	ng/L	2 - 16

Given the epidemiological situation, a rapid antigen test on a nasopharyngeal swab for SARS-CoV-2 was performed and found to be positive, the result was confirmed by a polymerase chain reaction (PCR) test.

The diagnosis of acute pancreatitis associated with severe acute respiratory syndrome was retained and the patient was transferred to the intensive care unit for multidisciplinary management. She was neurologically, hemodynamically and respiratorily stable with an oxygen saturation of 96% on room air and no typical Covid 19 symptoms, while cardiac electrocardiogram showed sinus tachycardia at 101 bpm with left atrioventricular failure.

In agreement with the gastroenterologist and the nephrologist, an initial conservative treatment was started which included rehydration, fasting, analgesics and sodium supplementation. For her severe anaemia, she received 2 bags of packed red blood cells transfused with 20 mg of Furosemide in between and 20 mg at the end, which improved her haemoglobin from 5.8 to 7.2 g/dl.

As part of the etiological work-up and to assess the severity of the acute pancreatitis, an abdominal CT scan with contrast injection was performed, which showed an enlarged pancreas with loss of lobulation of its contours, associated with infiltration of the peripancreatic fat and the presence of a few sub-centimetric latero-aortic lymph nodes, the largest of which measured 8 mm.

The result of the abdominal CT confirmed our diagnosis of acute pancreatitis by classifying it as stage C according to Balthazar's classification. However, the gallbladder and bile ducts were without abnormalities while the renal damage was remarkable.

The patient underwent a parallel abdominal ultrasound which revealed a picture in favour of bilateral chronic kidney disease and the work-up was completed with calcium, triglycerides, alkaline phosphatase, gamma-glutamyltransferase and total bilirubin, all of which were within normal ranges.

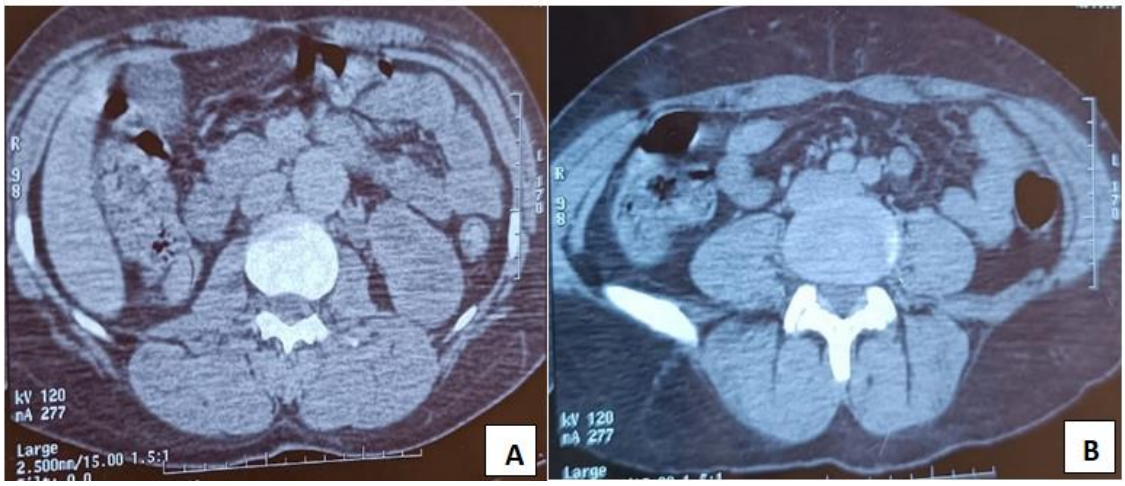

Figure 1 (A and B): Abdominal CT scan with contrast injection schowing an enlarged pancreas with loss of lobulation of its contours, associated with infiltration of the peripancreatic fat

Assessment of respiratory involvement following SARS-CoV-2 infection was performed by chest CT scan which showed the presence of lesions suggestive of Covid 19 presenting as diffuse ground glass with right posterobasal Crazy paving estimated to be severe in extent between 50-75%, associated with diffuse centrilobular micronodules. According to the CORADS classification scale for thoracic CT, this finding was classified as CORADS 6.

The evolution was marked by the resolution of the pulmonary lesions and pancreatitis and the patient was transferred to the gastroenterology department for further management.

III. DISCUSSION

Acute pancreatitis is an acute inflammation of the pancreas, which can vary from a spontaneously resolving mild disease to a severe disease with fatal complications.

There are many etiologies of AP, with bile duct obstruction (40%) and alcohol abuse (30%) being the most common causes [3]. However, idiopathic AP accounts for 15-25% of cases [4]. Despite the rarity of infectious etiology (≤1) [1], AP of viral origin has already been described in the literature, it has been causally linked to various viruses such as mumps, coxsackie B, hepatitis A, B, E, varicella, zoster, CMV, simple herpes or some upper respiratory viruses. Therefore, SARS-CoV-2 related pancreatitis does not seem to be a coincidence [5].

The incidence of this association is not yet clear. However, it has been found that 17% of patients with severe covid 19 have a pancreatic lesion with elevated lipase and amylase, which may conclude that the incidence of these lesions is not very low in this population. While the prevalence of acute pancreatitis in patients with covid 19 is low (0.27%) [6, 7].

After defining acute pancreatitis by the Atlanta criteria, a retrospective cohort study including patients of different race and age admitted to 12 hospitals, showed that acute pancreatitis was idiopathic with a percentage of 69% in SARS-Cov-2 positive patients compared to 21% in non-covid 19 patients who presented gallstones (34%) and alcohol (37%) as the most common etiologies similar to those in the general population [8].

Our patient was diagnosed with stage C severe acute pancreatitis according to the Atlanta diagnostic definition [6], which requires the presence of at least 2 of the following 3 criteria: epigastric abdominal pain consistent with pancreatitis, a serum lipase or amylase level at least three times the upper limit of normal and evidence of acute pancreatitis on CT, MRI or ultrasound. She met only 2 of 3 criteria because she had no suggestive abdominal pain.

As part of the etiologic diagnosis, lithiasis was excluded by abdominal CT scan which showed an alithiasic, thin-walled gallbladder without dilatation of the hepatic or bile ducts. While metabolic origin was excluded by normal triglyceride and calcium levels. In addition, there was no evidence of alcoholism. However, our patient was positive for SARS-CoV-2 confirmed by a thoracic CT scan with severe infection (50-75%) [9].

In the light of the current findings of idiopathic acute pancreatitis associated with covid 19 pneumonia, this suggested that SARS-CoV-2 may be the likely causal agent [10].

SARS-CoV-2 is a new virus, first detected in 2019. It is one of seven coronaviruses known to date that can infect humans (HCoV-OC43, HCoV-HKU1, HCoV-229E, HCoV-NL63, SARS-CoV-1, MERS-Co, SARS-CoV-2).

It is a large, single-stranded, enveloped, positive-sense RNA virus. The viral membrane consists of a bilayer where structural proteins are implanted. Like SARS-CoV-1 and MERS-CoV, SARS-CoV-2 uses angiotensin converting enzyme 2 (ACE2) as a receptor to enter the host cell. Upon entry, the surface proteins (S) are cleaved into two subunits, S1 which ensures the attachment of the virus to its receptor as the essential step in the infectious process, while S2 allows the fusion of the viral and cell membranes [11].

To understand how the pancreas can be a target organ, one study found that the mRNA level of CEA2 was higher in the pancreas compared to the lungs. As well, analysis of single-cell RNA sequencing data showed that its distribution in the pancreas includes both exocrine glands and islets. This indicates that SARS-CoV-2 can bind to its receptor and cause pancreatic damage.

Focusing on CEA2 expression in the pancreas, they experimented on pancreatic cells derived from induced pluripotent stem cells, showing that they allow SARS-CoV-2 entry leading to infection and morphological disturbances. This suggests that the virus can directly infect pancreatic cells which was confirmed by its detection in post mortem pancreatic tissue of covid patients 19 [12].

IV. CONCLUSION

Several clinical cases have been published in the literature associating acute pancreatitis with covid 19, but still currently no evidence to point to an association between these two affections. More research is needed to study the relationship and the clinical, diagnostic and prognostic aspects between COVID-19 infection and acute pancreatitis.

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