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Acute Pancreatitis in a Patient with COVID-19: A Case Report

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ABSTRACT

The global pandemic of the infectious disease coronavirus 2019 (COVID-19) caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) is a predominantly respiratory disease. Gastrointestinal symptoms occur in 15-20% of patients with COVID-19, however, there have not been many case reports of acute pancreatitis in patients with COVID-19. We presented the case of a 28-year-old girl suffering from COVID-19 with acute pancreatitis in the absence of other known etiological risk factors for pancreatitis. Laboratory analysis revealed a marked elevation of lipase and amylase. CT of the abdomen showed an edematous pancreas with diffuse enlargement. She was diagnosed with acute pancreatitis due to COVID-19 after carefully ruling out other causes. She was managed symptomatically, and improvement in her clinical condition was observed and was discharged with outpatient follow-up.

Introduction

A new disease, COVID-19 caused by Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2) has caused a global health crisis, a pandemic, with more than 581.3 million cases worldwide and over 6.4 million deaths [1].

The transmission of respiratory pathogens have been associated with three primary modes known as "contact," "droplet," and "airborne" transmission. The contact transmission can occur directly by physical touch or indirectly **via** fomites containing settled droplets [2].

COVID-19 is well described as a respiratory disease, but recent studies have shown that an increasing number of patients report gastrointestinal manifestations such as diarrhea, nausea, vomiting, and abdominal pain [3]. Despite the fact that knowledge about this virus is added daily, the impact of COVID-19 on the pancreas is still less researched. We presented a patient with COVID-19 who had acute pancreatitis without respiratory symptoms.

Case Presentation

It was about a 28-year-old woman, who was admitted to the clinic for infectious diseases of the University Clinical Center of the Republika Srpska Banja Luka. Three days before admission, she felt a headache, a high temperature, which lasted for one day, epigastric pain with propagation to the back, as well as nausea and vomiting. Comorbidities were excluded, except for obesity [Body Mass Index (BMI): 31.1]. He has an allergy to pollen from before, does not consume alcohol, smokes, uses up to 10 cigarettes a day. He works as an administrative technician. She had surgery

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on her eyelids, was treated for depression a few years ago, used clomipramine tablets, and does not take medication at the time of symptoms. There were no health problems in the family. Upon admission, a Polymerase Chain Reaction (PCR) test was performed, which confirmed the infection with COVID-19. On admission, the patient had a temperature of 37.6C, pulse rate of 78 beats per minute, normal blood pressure (125/70mmHg), no signs of dehydration, anicteric and Oxygen Saturation (SaO2) of 98% on room air. Diffuse abdominal pain and pronounced epigastric pain during abdominal palpation were recorded. There were no other findings in the physical and neurological examination. Levels of alpha amylase, lipase, pancreatic amylase and C reactive protein were elevated on admission. Laboratory analyzes are presented in table 1.

A computerized tomography of the Chest and abdomen (CT) is performed. Computed tomography of the chest showed in the lung parenchyma in the postero- and laterobasal segments bilateral minor zones of GGO (eng. Ground-glass opacity) as part of the COVID-19 infection. On the pleura, thickenings in both lower lobes of no more than 5 mm can be seen as part of scar changes. The heart is not enlarged. There is no fluid in the pericardium. There are no pathological lymphatics in the mediastinum. Conclusion: Co-RADS 5A, early stage disease.

Computed tomography of the abdomen with contrast describes, in conclusion: 1. Enlarged and heterodense head of the pancreas and the uncinate process, most likely in support of acute pancreatitis, but another etiology cannot be ruled out. 2. Intestinal pneumatosis in the wall of the duodenal gyrus accompanied by free fluid around the duodenal ring. 3.

Multiplied reactive lymphatics intra- and retroperitoneally. On admission, antibiotics are included therapeutically, empirically, for possible upgraded bacterial pneumonia. The patient was kept on a diet for acute pancreatitis, with continuous intravenous fluid replacement, proton pump inhibitors, preventive doses of anticoagulant therapy with broad-spectrum antibiotics.

During hospitalization, she had no respiratory signs or symptoms, nor did she need oxygen supplementation. Repeated, control radiographic and CT findings of the chest did not show any focal consolidations. The patient's appetite eventually improved and his diet improved. Other causes of acute pancreatitis such as drugs, trauma, and hypotension were excluded, and the patient was discharged after 10 days of hospitalization. After the patient leaves the hospital, a magnetic resonance imaging of the abdomen and a Magnetic Resonance Cholangiopancreatography (MRCP) are performed: conclusion: the findings are within physiological limits.

Esophagogastroduodenoscopy speaks in favor of gastroesophageal reflux and chronic gastritis with individual erosions. Under the further supervision of an endocrinologist, she has lost 12 kg of body weight since being discharged from the hospital, and is eating according to the nutritionist's advice, along with proton pump inhibitors and Creon tablets.

Discussion

The spike glycoprotein of the SARS-CoV-2 virus binds to the Angiotensin-Converting Enzyme 2 (ACE2) receptor

Table 1: Laboratory analyses.				
Laboratory findings	Upon admission	After 48 hours	Upon discharge	1 month after discharge
Leukocytes (mm ³)	13.8	11.56	4.72	5.5
Platelets (mm³)	325.000	238.000	264.000	288.000
Hemoglobin (g/dL)	137	15.8	125	129
Alanine aminotransferases (U/L)	17	123	11	8.1
Aspartate aminotransferase(U/L)	10	33	22	10.8
Total bilirubin (mg/dL)	3.6	5.8	4,5	
Direct bilirubin (mg/dL)	4.6	2.2	1.7	
Alkaline phosphatases (U/L)	41	37	38	
Urea (mg/dL)	3.1		2.5	3.2
Creatinine (mg/dL)	59		58	80
Alpha-amylase (U/L)	514	56	66	42
Pancreatic amylase	416	36	41	17
C-reactive protein (mg/L)	13.0	139.6	14.3	3.2
Lipases (U/L)	907	45	65	24
Potassium	4.7	4.3		
Cholesterol (mmol/L)			3.3	4.69
Triglycerides (mmol/L)	1.6		1.6	2.12
Blood glucose	6.1	5.3	5.3	4.3

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on human cells, which enables the entry of SARS-CoV-2 into the target cell [4]. ACE2 receptors are present on the surface of respiratory tract cells, lung alveoli, heart, blood vessels, liver, kidney, digestive tract, exocrine pancreas and islet cells [4,5]. This is exactly why COVID-19 has such a diverse symptomatology that differs from other "ordinary" respiratory infections. The exocrine part of the pancreas is responsible for the production and activation of enzymes that participate in the metabolism of proteins, carbohydrates and fats. The endocrine part of the pancreas consists of islets of Langerhans – clusters of several types of cells that produce insulin, glucagon, somatostatin and pancreatic polypeptide. In people who suffer from diabetes and are infected with a virus (beta cell infection), sugar metabolism may deteriorate [5].

According to current guidelines, the diagnosis of acute pancreatitis requires at least two of the three following signs: (1) abdominal pain, (2) amylase or lipase >3 times the upper limit of normal, and (3) characteristic findings of acute pancreatitis on abdominal imaging (contrast improved CT, ultrasound, magnetic resonance) [6]. The exact mechanism by which viruses cause pancreatitis is not known and each virus can cause pancreatitis through a different mechanism. These mechanisms include viral replication in pancreatic acinar cells resulting in protease leakage and activation, along with cholangiopathy and ampullary edema [7]. Some patients develop symptoms of COVID-19 and abdominal pain at the onset of infection, and others develop acute pancreatitis a few days after being diagnosed with COVID-19. Our patient developed acute pancreatitis without respiratory complications at the onset of infection.

Reports from China have described evidence of pancreatic damage, defined as elevated lipase levels, in up to 17% of active cases of COVID-19 [8]. Pancreatic enzymes such as amylase and lipase can be secreted by organs other than the pancreas such as the lungs. Amylases required for starch digestion are mainly secreted by the pancreas and salivary glands, but other organs including normal and diseased lungs [9]. Lipase, which in adults is mainly secreted by the pancreas, is the key enzyme for digesting triglycerides. Lipase is also excreted by the kidneys [9,10]. Also, high serum amylase and lipase activity is reported in conditions such as severe gastroenteritis, diabetes, post-cardiovascular surgery, trauma, burns, and kidney damage due to renal clearance of pancreatic enzymes [10]. Since our patient did not have burns, trauma or surgery, elevated values of amylase and lipase were interpreted as a direct effect of SARS-COV2 on the gastrointestinal tract. An important fact is that the activity and replication of the virus in the intestines can last even after the virus has been cleared from the respiratory tract [10]. Pancreatitis can be a consequence of direct viral invasion, or secondary to hypoxic effects or damage mediated by cytokines, the exact mechanism is difficult to assume at this time [10].

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Pancreatitis can be a consequence of direct viral invasion, or secondary to hypoxic effects or damage mediated by cytokines, the exact mechanism is difficult to assume at this time [3,10]. Severity can be categorized as mild (absence of organ failure or complications), moderately severe (transient organ failure with or without local or systemic complications) and severe (organ failure lasting longer than 48 hours) [6]. The clinical picture of our patient can be classified as mild acute pancreatitis, she was previously healthy, had no history of alcohol or drug abuse, did not take antipyretics that could be the cause of AP. Abdominal CT revealed no signs of gallstones. Furthermore, hypertriglyceridemia as a possible trigger was not considered due to the serum triglyceride level of 1.6 mmol/L. Her improvement with conservative treatment confirmed acute pancreatitis attributable to COVID-19. The patient tolerated well the oral diet, symptomatic, supportive treatment with fluids.

A study investigating the link between COVID-19 infection and the pancreas reported pancreatic damage in 1%-2% and 17% of patients with mild and severe infection, respectively. The authors suggested that pancreatic damage may be exacerbated by systemic inflammation [11]. Elevation of amylase and lipase indicating damage to the pancreas was recorded in 8.5%-17.3% of patients with COVID-19. Higher enzyme levels have been reported in severe patients with COVID-19 [11]. In two autopsy studies, five of 11 (45.5%) and two of eight (25%) cases of focal pancreatitis with hemorrhagic and necrotic changes of the pancreas were found. These changes had no clinical manifestations and were attributed to ischemia and organ damage [12]. The exact pathophysiology of pancreatic damage remains unclear, while the most accepted hypothesis points to pancreatic ischemia [13]. If septicemia progresses to septic shock, not only with COVID-19, but also with other infections, the resulting hypotension and vasodilation reduce blood flow to organs. To protect blood flow to vital organs such as the brain and heart, blood flow to the intestines, superior and inferior mesenteric arteries is reduced as part of a protective mechanism. This is a neurohormonal mechanism that protects vital organs. As a result, blood flow to the liver, pancreas and the entire gastrointestinal system is reduced, causing symptoms such as nausea, vomiting, distension, ileus or diseases such as ischemic hepatitis.

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Amylase, lipase, aspartate aminotransferase and lactate dehydrogenase are released into the bloodstream due to ischemia resulting from reduced blood flow in the pancreas [13]. This damage is mainly caused by hemodynamic deterioration and not by the virus itself.

Conclusion

Although acute pancreatitis is rare in patients with COVID-19, it should be considered in patients with severe epigastric pain and respiratory symptoms, even in mild cases of COVID-19. These patients should be followed up to assess the patient's recovery and/or associated complications, including new-onset diabetes, chronic pancreatitis. The pathophysiological mechanism of increased levels of amylase and lipase in patients positive for COVID-19 seems to have a multifactorial pathogenesis. Additional studies are needed to clarify the causal relationship between SARS-CoV-2 and acute pancreatitis.

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