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Andes pediatr. 2021;92(6):917-923
DOI: 10.32641/andespediatr.v92i6.3360

CLINICAL CASE

Acute pancreatitis associated with SARS-CoV-2 infection in a pediatric patient

Pancreatitis aguda asociada a infección por SARS-CoV-2 en un paciente pediátrico

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Received: October 9, 2020; Approved: April 4, 2021

What do we know about the subject matter of this study?

Although SARS-CoV-2 infection presents diverse possible manifestations, the description in the literature of pancreatic involvement associated with this infection is limited.

What does this study contribute to what is already known?

A new case of acute pancreatitis with temporal association with SARS-CoV-2 infection where the most frequent etiologies were excluded and therefore forewarn of this manifestation and possible causal relationship.

Abstract

The recent discovery of SARS-CoV-2 and the disease COVID-19 which affects different organs and systems, mainly the respiratory one, representing a new challenge for physicians. Pancreatic affection is barely described, with only a few cases reported in the literature. **Objective:** to communicate a case of acute pancreatitis associated with SARS-CoV-2 infection, to contribute to the knowledge of this new virus and its possible forms of presentation. **Clinical Case**: An eleven-year-old male adolescent, with no history of contact with people confirmed or suspected of COVID-19, was admitted to the hospital with a 3-day history of periumbilical and epigastric abdominal pain, vomiting, and absence of stools, with no other symptoms. The laboratory tests showed increased pancreatic enzymes. Computed tomography was compatible with acute edematous pancreatitis, without signs of biliary pathology, diagnosing acute pancreatitis, at the same time that SARS-CoV-2 was isolated in the respiratory tract. Other possible differential diagnoses and history of epidemiological contact were ruled out. The patient was managed in the Critical Patient Unit with support measures. He evolved favorably, without respiratory symptoms. **Conclusion**: SARS-CoV-2 infection can be associated with atypical presentations, including acute pancreatitis. The physiopathological mechanism of pancreatic damage is not yet clear. Physicians should be aware of the COVID-19 involvement of other systems, beyond the respiratory one.

Keywords:
Pancreatitis;
Coronavirus;
SARS-CoV-2;
COVID-19;
Coronavirus Infections;
Pediatrics

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How to cite this article: Andes pediatr. 2021;92(6):917-923. DOI: 10.32641/andespediatr.v92i6.3360

Introduction

In December 2019, the first case of SARS-CoV-2 coronavirus was detected in Wuhan, China, and according to data provided by the World Health Organization (WHO), it is responsible for one of the largest pandemics of the last century, with more than 122 million people infected and more than 2,700,000 deaths until March 21, 2021[1]. In Chile, until March 25, 2021, there have been 1,125,521 cases of COVID-19 (approximate rate 5,622 cases per 100,000 inhabitants), where 7.03% were children under 15 years of age[2].

Worldwide, researchers are trying to reveal as much information as possible about this new disease. Although in adult patients it mainly affects the respiratory system, fever (35-71% cases), cough (37-57%), and dyspnea (6.5-28.6%) have been reported in children as well as gastrointestinal manifestations with 18% frequency on average. Gastrointestinal manifestations in the pediatric population include abdominal pain (6.7-7.7%), diarrhea (8.8-21.4%), nausea, and/or vomiting (6.4-10.4%), among other less frequent manifestations such as jaundice or elevated transaminases levels[3-6]. In Chile, according to the epidemiological report "Epidemiological description of children and adolescents with COVID-19 Chile (epidemiological week 10 to 44, 2020)", up to November 1, 2020, of the 57,375 children with COVID-19, 9.5% presented diarrhea and 7.6% abdominal pain[7].

A systematic review that included 43 studies with 18,246 patients describes diarrhea as the most common gastrointestinal symptom (11.5%), followed by nausea (6.3%) and abdominal pain (2.3%). Of those with gastrointestinal symptoms who had their transaminases levels measured, 31.4% showed a slight increase in aspartate aminotransferase and alanine aminotransferase[8].

So far, there are few cases reported in the literature on acute pancreatitis associated with SARS-CoV-2 infection in adults and the pediatric population[9-14]. A great structural similarity between SARS-CoV-2 and SARS-CoV has been described, which has led to the assumption that this new virus infects cells using angiotensin-converting enzyme 2 (ACE-2) as a receptor[15].

Although the pulmonary location of this receptor is widely known, clinical studies have demonstrated the expression of ACE-2 in other tissues, including the islets of Langerhans, making the pancreas a hypothetical site of possible infection and damage[16-18]. Recent reports describe that SARS-CoV-2 uses the ACE-2 receptor to enter the cell in synergy with the transmembrane serine protease 2 (TMPRSS2) located on the surface of host cells. Specifically, viral glycoprotein S is divided by the TMPRSS2, facilitating viral activation, which represents one of the key host factors for SARS-CoV-2 pathogenicity[19-20].

The objective of this article is to report a case of acute pancreatitis associated with SARS-CoV-2 infection in order to contribute to scientific knowledge about this new virus and its possible forms of presentation, seeking to alert non-specialist, specialist, and subspecialist physicians of this potential presentation, to facilitate its detection and early management.

Clinical Case

An 11-year-old male adolescent with history of chronic constipation, overweight, and full vaccination scheme according to the Chilean national immunization program, with no history of contact with people confirmed or suspected of having COVID-19, nor travel abroad. He consulted the emergency department of the *Hospital Doctor Exequiel González Cortés* (HEGC) in May 2020 with a 3-day history of colicky, periumbilical, and epigastric pain, associated with food refusal and frequent vomiting (approximately 10 episodes per day), in addition to the absence of bowel movements for 2 days. He did not report fever in the days before hospitalization, nor respiratory symptoms.

On admission to the emergency department, the patient presented low-grade fever (37.7°C axillary), hypertension (149/90 mmHg, > p99 for age, height, and sex), without respiratory distress, well perfused and hydrated, and the abdominal examination showed a distended and tender abdomen, without Blumberg's sign.

An abdominal X-ray (standing anteroposterior and lateral decubitus) and rectal examination were performed, both with normal results. The patient was evaluated by the surgery team, who considered that he did not present an acute surgical abdomen, ruling out a possible intestinal obstruction. However, due to the intensity and characteristics of the abdominal pain, laboratory tests were performed, highlighting elevated C-reactive protein (CRP) of 61 mg/L [normal value (NV) < 5], leukocytosis 15,030/uL with polymorphonuclear predominance (84.6%), lymphopenia (5.6%, absolute value 842/uL), amylase 1,100 U/L (NV 28-100), lipase 1,054 U/L (NV 13-60), total bilirubin 1.3 gr/dL (34% direct), and hypoprothrombinemia (prothrombin time 57%). The rest of the liver tests were within normal ranges, normal lipid profile (triglycerides 71 mg/dL), and normal blood calcium levels.

An abdomen and pelvis CT scan with contrast was requested (figure 1), which showed the pancreas with a slight general increase in volume and loss of acinar pattern, with a prominent main pancreatic duct (diameter 3 mm), compatible with acute interstitial edematous

pancreatitis without signs of biliary disease. According to the criteria of the International Study Group of Pediatric Pancreatitis (INSPPIRE)[21], the diagnosis of acute pancreatitis was established by meeting the three criteria (abdominal pain, elevation of serum lipase and/or amylase ≥ 3 times the normal limit, and compatible imaging findings).

The patient was hospitalized in the Critical Patient Unit (CPU) for monitoring, where, according to hospital admission protocol, a nasopharyngeal swab sample was collected to detect SARS-CoV-2 by reverse transcription-polymerase chain reaction (RT-PCR). In the CPU, the patient developed a low-grade fever, hypertension, severe abdominal pain that required continuous infusion pump with fentanyl for a day, and prolonged fasting (> 72 hrs.) due to oral intolerance. On the third day of hospitalization, it was reported a positive SARS-CoV-2 PCR test result, with monitoring tests showing a progressive decrease in pancreatic enzymes and an increase in CRP (table 1).

Tests performed according to the local protocol for patients with COVID-19 presented altered results indicating elevated D-dimer (7,504 ng/mL; NV < 400), and normal fibrinogen and LDH levels. Initial CT scan was reviewed, and, on suspicion of necrotizing pancreatitis, blood cultures were collected, and empirical intravenous antibiotic therapy was started with amikacin (15 mg/kg/day) and metronidazole (30 mg/kg/day) in addition to thromboprophylaxis with subcutaneous enoxaparin (1 mg/kg/day) due to elevated D-dimer.

On the sixth day of evolution of the clinical picture, he presented fever (peak 38.2°C) and low procalcitonin levels (0.1 ng/mL), without recurrence of fever in the following days. On the same day, an abdominal ultrasound was performed that showed no alteration of the biliary tract and a control CT scan of the chest, abdomen, and pelvis showed a persistent increase in pancreatic volume, without areas of necrosis or collections, greater dilation of the pancreatic duct compared with the previous image (5 vs 3 mm), and atelectasis condensations in posterior lung segments, without alteration of the lung parenchyma, compatible with COVID-19.

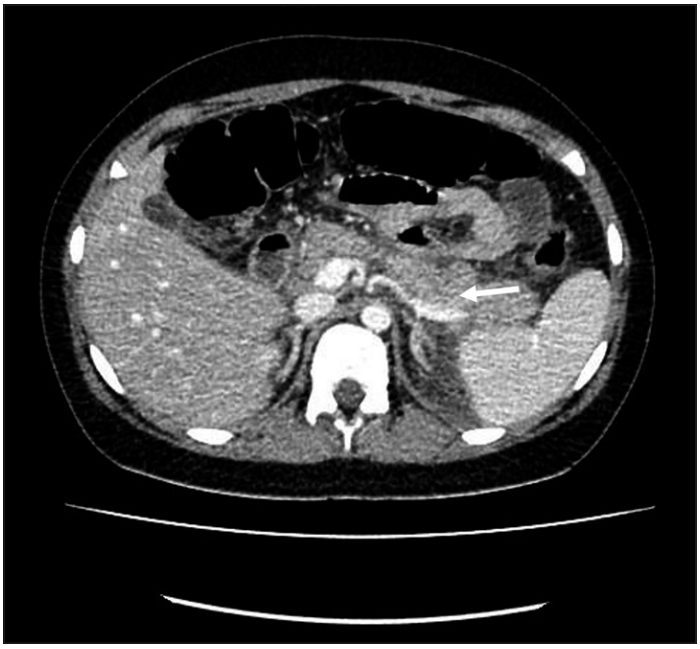

Figure 1. Computed tomography scan of the abdomen and pelvis with contrast corresponding to the first day of hospitalization, compatible with acute interstitial edematous pancreatitis. White arrow: enlarged pancreatic parenchyma with loss of acinar pattern.

To continue with the differential diagnosis of other possible causes of pancreatitis, during the twelfth day of evolution, a cholangiopancreatography was performed, which showed a pancreatic duct of normal diameter, without signs of cholelithiasis or choledocholithiasis. In addition, serological tests for cytomegalovirus (CMV) presented negative IgM and positive IgG that was interpreted as an old infection; anti-VCA IgM for Epstein Barr virus (EBV) was negative, and the respiratory pathogens molecular panel by nasopharyngeal swab was also negative. Likewise, there was no microbiological development in the blood cultures collected before starting antimicrobial therapy.

Table 1. Summary of laboratory test results during hospitalization

	Days of hospitalization					
Test (Unit of measurement, normal value)	1	2	3	4	5	6
C reactive protein (mg/L, ≤ 5)	61.7	107.9	190.5	203.9	177.4	127
White blod count (/uL, 4,500 – 13,500)	15030	10630	11820	12320	-	-
Amylase (U/L, 28 - 100)	1100	467	230	147	120	115
Lipase (U/L, 13 - 60)	1054	444	203	226	88.6	124.8
D-Dimer (ng/mL, ≤ 500)	-	-	7504	5658	2829	3928

From the fifth day of hospitalization, the patient presented no fever, with improvement in oral tolerance, without abdominal pain or respiratory symptoms, or requiring oxygen at any time, completing seven days of empirical antibiotic therapy.

After hospital discharge, he was in good general condition, with episodes of mild abdominal pain that resolved with paracetamol. He was in follow-up by telephone regularly and was evaluated in the gastroenterology polyclinic 14 days after discharge, presenting normal physical examination, decreased pancreatic enzymes, and a D-dimer within normal ranges.

Discussion

We present a case of acute pancreatitis in an adolescent associated with SARS-CoV-2, with no other etiological agents identified. It is not clear if SARS-CoV-2 was the cause of pancreatitis or only a temporary association; if so, the diagnosis would be idiopathic pancreatitis and concomitant asymptomatic SARS-CoV-2 infection since the patient had no other clinical manifestations. However, in approximately 8-10% of cases, viruses can be a causative agent of pancreatitis[22]. Regarding the etiology of pancreatitis, several possible causes were ruled out, such as biliary disease, hypercalcemia, hypertriglyceridemia, hypotension, alcohol consumption, drugs use, medications, trauma, and other viral infections, therefore, it could be said that SARS-CoV-2 infection plays a role as a possible causative agent of pancreatitis.

Chao et al and Siegel et al have reported that the most useful indicator for the diagnosis of acute pancreatitis is the dilatation of the pancreatic duct with sensitivity in pediatrics by ultrasound between 78-92%, with a high positive (86-91%) and negative (75-84%) predictive value (23, 24). Therefore, considering that in our clinical case cholangiopancreatography ruled out obstructive etiology, the dilatation and subsequent normalization of the diameter of the pancreatic duct observed could be attributed to the natural course of acute pancreatitis.

Previous studies have described a TMPRSS2-associated ACE-2 binding as one of the main routes of entry of SARS-CoV-2 into the cell[15,18-20]. Likewise, some authors describe the presence of ACE-2 in different tissues other than the lung[16-18]. In pancreatic histological sections, ACE-2 protein has been found in the microvasculature of exocrine tissue and islets and a subset of pancreatic ducts, while TMPRSS2 protein has been detected exclusively in ductal cells[25], therefore, SARS-CoV-2 could affect pancreatic tissue.

There is a report in the literature that describes the isolation of SARS-CoV-2 by PCR in pancreatic tissue, but it is unknown if the mechanism of SARS-CoV-2 damage could have a direct cytopathic effect on the pancreas or mediated by inflammatory and/or immunological processes[26]. Likewise, we did not find studies that associate SARS-CoV-2 viral load with the severity of pancreatic alteration. More studies are needed to clarify the pathophysiological relationship between SARS-CoV-2 and pancreatic damage.

In the context that SARS-CoV-2 could be associated with acute pancreatitis, so far, few studies have reported a relationship between them. The paper by Wang F et al. in Wuhan included 52 patients with COVID-19, of whom 17% had pancreatic alteration defined as elevated serum amylase levels[12]. In a retrospective cohort by Mcnabb-Baltar J et al., with 71 patients, 12.1% developed hyperamylasemia and only 2 patients (2.8%) exceeded 3 times the maximum normal limit, however, the authors conclude that none corresponded to acute pancreatitis *per se*[27].

The aforementioned studies have exclusively included adult patients with COVID-19. At the time of this publication, few cases of pancreatitis associated with COVID-19 have been reported in children. The first published case was a 7-year-old girl who, after being discharged with a diagnosis of pancreatitis, without respiratory symptoms, consulted again two weeks later due to recurrence of gastrointestinal symptoms with elevated pancreatic enzymes, which, due to suspicious contact, was tested and found positive for SARS-CoV-2[13].

A series of 3 cases was reported in the United States that included an 11-year-old boy, a 15-year-old boy, and a 16-year-old adolescent girl. Like our case, they all presented with epigastric pain and food refusal, but the onset of prodromal symptoms was reported at least 6 days before the diagnosis of pancreatitis. In this series, the similarity of the blood count also stands out, which showed absolute lymphopenia (< 1,500/uL) and leukopenia (< 4,000/uL) in only one of them. All three patients evolved favorably with a hospital stay of 3 to 4 days and had adequate oral tolerance and liver tests and pancreatic enzymes in the process of normalization by the time they were discharged[14]. In contrast, the number of possible etiologies ruled out in our case was significantly higher than in the report, highlighting the dilatation of the pancreatic duct as an element of diagnostic support for acute pancreatitis.

In Northwell, New York, Inamdar Sumant et al studied the epidemiological characteristics of hospitalized patients with and without COVID-19 diagnosed with pancreatitis and found that the cause of pancreatitis was idiopathic in a higher percentage of patients with COVID-19 compared with those with negative SARS-CoV-2 CRP (69% vs 21%, respectively), and a higher proportion of patients of African descent and

Hispanics with COVID-19 (OR 4.48 and 5.07, respectively). There were no differences in mortality or development of necrotizing pancreatitis between those with or without SARS-CoV-2 (28).

Regarding the temporality between SARS-CoV-2 infection and pancreatitis, in this case, it is presumed that it was an acute coronavirus infection and that its only possible manifestation was gastrointestinal and pancreatic involvement since there were no respiratory symptoms. Although it is known that the multisystem inflammatory syndrome in children (MIS-C) associated with COVID-19 can affect multiple systems, including the hematological, urinary, hemodynamic, cardiovascular, and gastrointestinal tract (including pancreas) in up to 92% of patients[29], in this patient, MIS-C was not considered as a diagnosis, mainly due to the absence of persistent fever (according to WHO diagnostic criteria) among other factors, and because this case occurred early in the pandemic, a few days after the first alert report of MIS-C from the Royal College of Physicians (London) in May 2020, and serology for SARS-CoV2 was not yet available in our facility to have more elements to support the differential diagnosis[30,31].

The temporality of SARS-CoV-2 infection and the development of acute pancreatitis has been described to be heterogeneous, and some patients present COVID-19 symptoms and abdominal pain at the onset of infection, however, in other cases have been described that pancreatitis occurs several days after the diagnosis of COVID-19[32].

In this article, we report a case of a male adolescent with clinical symptoms, laboratory, and imaging tests compatible with acute pancreatitis, ruling out various etiologies, except for SARS-CoV-2 viral infection at the time of diagnosis. This possibility requires that physicians consider acute pancreatitis among the possible atypical manifestations of COVID-19, both to increase clinical suspicion and timely management, as well as to increase the number of reports and thus improve the casuistry necessary to determine this possible association.

Conclusion

So far, there is little information available regarding pancreatic involvement associated with SARS-CoV-2 infection in adults and even less in the pediatric population. Future studies could help to clarify the relationship between these phenomena; therefore, it is important to warn health professionals about the possible relationship and the need for considering acute pancreatitis as an atypical presentation of COVID-19.

Ethical Responsibilities

Human Beings and animals protection: Disclosure the authors state that the procedures were followed according to the Declaration of Helsinki and the World Medical Association regarding human experimentation developed for the medical community.

Data confidentiality: The authors state that they have followed the protocols of their Center and Local regulations on the publication of patient data.

Rights to privacy and informed consent: The authors have obtained the informed consent of the patients and/or subjects referred to in the article. This document is in the possession of the correspondence author.

Conflicts of Interest

Authors declare no conflict of interest regarding the present study.

Financial Disclosure

Authors state that no economic support has been associated with the present study.

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