

Splenic Infarction Associated with COVID-19 In Pediatrics: Case Report

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INTRODUCTION

SARS-CoV-2 disease is a recently emerged infection that caused a global pandemic, declared by the World Health Organization in March 2020. It had an impact on pediatric health, highlighting the relationship between inflammation, hypercoagulability, and vascular thrombosis. We present the case of a 10-year-old female patient who experienced splenic infarction associated with Covid-19, successfully treated with open splenectomy and low molecular weight heparin anticoagulation. Coagulation abnormalities in Covid-19 patients have been described since the early cases observed in Wuhan, China, but there is limited information on their presence in pediatrics. The unusual case we present could represent a manifestation of these alterations.

CLINICAL CASE

This involves a 10-year-old female school student with no reported history of chronic diseases, previous surgeries, or traumatic events. She presented with a respiratory illness along with her entire family four weeks prior to her admission, and all of them tested positive for SARS-CoV-2 antigen.

She sought consultation at the pediatric emergency service, brought in by her mother, who reported eight days of evolution with colicky abdominal pain, non-radiating, accompanied by fever, nausea, and vomiting.

In the physical examination, the patient had a temperature of 38.5°C, a heart rate of 115 beats per minute, a respiratory rate of 32 breaths per minute, and oxygen saturation of 97%. No adventitious sounds were auscultated on chest examination. The abdomen was flat, with hypoactive peristalsis, soft, depressible, painful to medium and deep palpation in the left hypochondrium, a very painful spleen pole, the rest of the abdomen without pain, and no signs of peritoneal irritation were observed.

In complementary studies, the following results were observed: hemoglobin 11.4 g/dL, hematocrit 34.7%, white blood cells 20,690 thousand/ μ L, neutrophils 18,170 thousand/ μ L, monocytes 1.41 thousand/ μ L, platelets 608 thousand/ μ L, glucose 90 mg/dL, urea 23.90 mg/dL,

creatinine 0.4 mg/dL, total bilirubin 0.26 mg/dL, direct bilirubin 0.14 mg/dL, indirect bilirubin 0.12 mg/dL, GGT 27 U/L, lipase 28.6 U/L, AST 14 U/L, ALT 9 U/L, alkaline phosphatase 154 U/L, chloride 101.3 mmol/l, potassium 4.3 mmol/l, sodium 136 mmol/l, calcium 8.9 mmol/l, LDH 383 U/L, lipase 28.6 U/L, amylase 55 U/L, CRP 14.8 mg/dL, PT 19.1 sec, APTT 20.2 sec, fibrinogen 470 g/dL, D-dimer 7.77 mg/L. SARS-CoV-2 antibody test with positive IgG. The general urine examination shows negative leukocyte esterase, 25 protein, negative hemoglobin, 0-3 leukocytes per field, few epithelial cells, moderate bacteria, and moderate mucin filament.

Abdominal ultrasound reported findings of splenic infarction, minimal homolateral pleural effusion to the splenic infarction, and free fluid in the pelvic cavity. She was evaluated by Hematology, who requested a transfusion of 2 units of fresh frozen plasma. The patient was admitted to the ward for hemodynamic preparation, and four days later, an open splenectomy was performed with surgical findings of a spleen with splenomegaly adhered to the omentum, costal wall, pancreas, stomach, and suspensory ligaments. (Figure 1).



Figure 1: Spleen with areas of extensive ischemia and necrosis.

The pathology report showed intense nonspecific acute and chronic inflammation (abundant polymorphonuclear cells and lymphocytes), areas with extensive ischemia and necrotic zones (Figure 2), recent hemorrhages, especially subcapsular, and intense nonspecific chronic vasculitis (Figure 3).

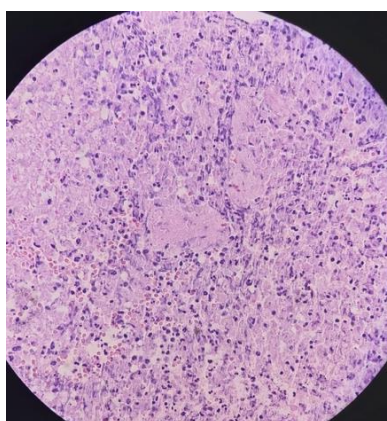


Figure 2: Spleen with areas of extensive ischemia and necrotic zones.

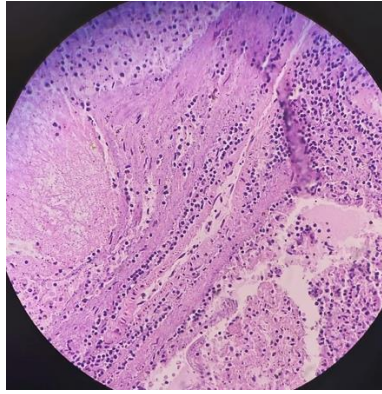


Figure 3: Intense nonspecific chronic vasculitis.

After her recovery, she continued management for a month with subcutaneous enoxaparin 20 milligrams, along with follow-up outpatient visits with hematology and pediatric surgery.

DISCUSSION

Currently, it is reported that infants can develop SARS-CoV-2 associated with an inflammatory condition called Pediatric Multisystem Inflammatory Syndrome (MIS-C), which generally arises weeks after acquiring the viral infection (1).

Vascular thrombosis in pediatrics occurs between 1.2% and 26%, generated by inflammatory processes that lead to endothelitis with alterations in microvascular structures and the formation of clots (2,3).

The presence of a virus can lead to dysfunctional endothelium, which becomes proadhesive and procoagulant, causing endothelitis with the accumulation of inflammatory cells and increasing cell death. This results in severe deterioration of systemic microcirculatory function in different vascular beds and its consequent clinical effects (4,5,6).

The lung does not seem to be the primary affected organ. The pediatric multisystem inflammatory syndrome temporarily associated with SARS-CoV-2 can affect the gastrointestinal tract, with the most common findings being inflammation of the terminal ileum and mild ascites. Hepatomegaly, splenomegaly, infarctions of solid abdominal organs (liver, spleen, pancreas, kidneys), perivesicular/periportal edema, increased echogenicity of the renal cortex, and lymphadenopathy are also common findings (7).

Risk factors include age ≥ 12 years, mechanical ventilation, deep venous approach, presence of multisystem inflammatory syndrome, obesity, and cancer (8).

The CDC defines the syndrome of systemic acute inflammatory response as a person under 21 years of age who presents with fever ($> 38.0^{\circ}\text{C}$ for ≥ 24 h or subjective report of fever lasting ≥ 24 h), laboratory evidence of inflammation (including, among others, one or more of the following elevated elements: PCR, erythrocyte sedimentation rate, fibrinogen, procalcitonin, D-dimer, ferritin, lactate dehydrogenase, interleukin 6, neutrophils. Reduced lymphocytes and albumin levels) and evidence of clinically severe disease requiring hospitalization, with multisystem organ involvement (>2) (cardiac, renal, respiratory, hematological,

gastrointestinal, dermatological, or neurological), without probable alternative diagnoses, positive for current or recent SARS-CoV-2 infection by RT-PCR or serology, or antigen test; or exposure to COVID-19 within 4 weeks before the onset of symptoms (9).

The diagnosis is supported by specific analyses:

Particularly D-dimer (increased levels are used as a precision marker for diagnosis, prognosis, and progression) (10), prothrombin time, and partial thromboplastin time (their prolonged duration is associated with increased coagulation and worse prognosis in patients with vascular thrombosis, although they may be normal) (11), von Willebrand factor (some authors consider it a marker of endothelial injury and a predictor of severity) (12,13), quantification of coagulation factors (mainly altered due to consumption in coagulopathies), fibrinogen (increases in vascular thrombosis), ultrasounds with dopplers, and tomographies (10,12).

The patient had a history of COVID-19 four weeks before admission. In the laboratory tests, prolonged prothrombin and partial thromboplastin times, elevated PCR, increased fibrinogen, and elevated D-dimer were found. Abdominal ultrasound revealed splenic infarction and minimal homolateral pleural effusion to the splenic infarction, leading to the decision to perform an open splenectomy.

Currently, therapeutic criteria for anticoagulant treatment have not been unified, so each country has its protocols according to its expert groups (1,2). In this case, management was carried out for a month with low molecular weight heparin, and follow-up with hematology was provided after hospital discharge.

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