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Phlegmonous gastritis in a patient with chemotherapy-induced neutropenia

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Abstract: Phlegmonous gastritis is a rare bacterial infection of the gastric wall that affects immunocompromised patients and has a high mortality rate despite adequate treatment. Various risk factors that compromise the integrity of the gastric mucosa have been described. Symptoms are generally nonspecific and may present as an acute abdomen, making imaging studies especially important. Treatment requires appropriate antibiotic therapy and, occasionally, surgery. The available literature on patients with oncohematological diseases or undergoing chemotherapy who develop phlegmonous gastritis is limited. We present the case of a patient with relapsed acute lymphoblastic leukemia who developed phlegmonous gastritis during the period of neutropenia following chemotherapy.

Key words: phlegmonous gastritis; neutropenia; chemotherapy; acute leukemia

1 Introduction

Phlegmonous gastritis is a bacterial infection characterized by purulent inflammation of all layers of the gastric wall, which can be localized or diffuse. It is a rare disease that affects immunocompromised patients and has a high mortality rate of around 50%. Its pathophysiology is not entirely clear, but risk factors such as some prior degree of mucosal injury, chronic alcoholism, neoplasia, and other immunocompromised states are recognized. Symptoms are usually nonspecific, including abdominal pain, nausea, vomiting, fever, and occasionally an acute abdomen [1-4]. Computed tomography, upper gastrointestinal endoscopy, and endoscopic ultrasound are highly relevant for diagnosis. Treatment requires early recognition for the timely initiation of antibiotic therapy, and sometimes surgical intervention is necessary [5-7].

2 Clinical case

A 38-year-old woman diagnosed with Philadelphia chromosome-negative B-cell acute lymphoblastic leukemia was incompletely treated with the GATLA induction chemotherapy protocol and had to discontinue the last cycle due to pulmonary aspergillosis. Subsequent outpatient check-ups showed negative residual disease and she was awaiting completion of antibiotic treatment before resuming chemotherapy.

The patient presented with a deteriorating general condition and bone pain. Physical examination revealed no significant findings. A peripheral blood smear was performed on an outpatient basis, revealing 30% blasts. The decision was made to admit the patient to reassess systemic involvement and administer rescue therapy.

Upon admission, a CT scan of the skull, chest, abdomen, and pelvis (Figure 1) revealed no significant pathological

findings, and an echocardiogram showed good cardiac function with no valvular abnormalities. The patient was started on chemotherapy with the FLAG-IDA regimen (fludarabine 50 mg/day, days 1 to 4; cytarabine 3.4 g/day, days 1 to 4; idarubicin 20 mg/day, days 2 to 4; plus filgrastim 300 ug daily, from day 0 until polymorphonuclear cells recovered).

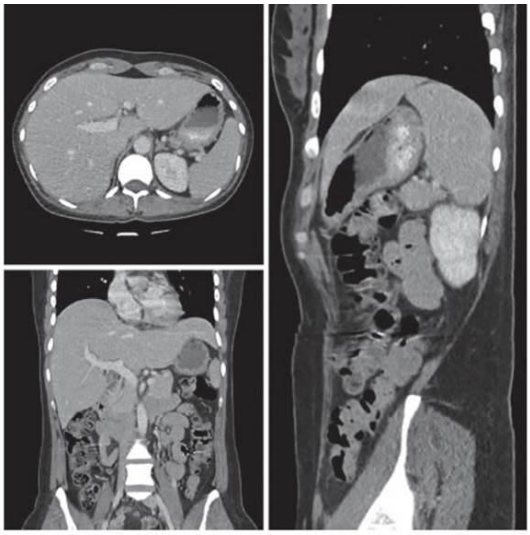

Figure 1. Admission CT scan, abdominal and pelvic sections, without pathological findings

On day 11 of hospitalization, day +6 from the start of chemotherapy, already in the post-chemotherapy aplasia phase, the patient developed abdominal pain predominantly in the epigastrium, associated with fever and vomiting. Hematomesis subsequently developed, with no signs of hemodynamic instability. Supportive care with blood product transfusions and omeprazole was initiated, along with broad-spectrum antibiotic treatment because infectious involvement could not be ruled out. Furthermore, the decision was made to transfer the patient to the Critical Care Unit due to the rapid progression of symptoms.

The patient developed mixed shock (distributive-hypovolemic), requiring vasoactive agents and daily transfusions of red blood cells and platelets. A CT scan of the abdomen and pelvis showed exclusive gastric involvement, with marked homogeneous thickening of the entire gastric wall (Figure 2), which, comparatively, was not present in the previous study, associated with reticulation of the perigastric fat, and a small amount of free fluid. An upper videoendoscopy was also performed, which showed diffuse involvement of the entire gastric mucosa, erosions, petechial lesions with a subepithelial hemorrhagic appearance, thickening of the folds, and purulent contents in the cavity. The clinical picture and imaging findings were consistent with a diagnosis of phlegmonous gastritis.

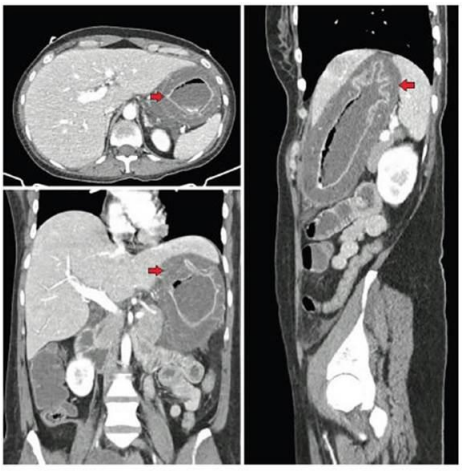

Figure 2. Posterior CT scan of the abdomen and pelvis showing diffuse thickening of the entire gastric wall (arrows)

The patient presented a good initial response and clinical improvement after 48 hours of treatment, regaining oral tolerance. Due to this improvement and the lack of other associated complications, it was decided to continue with conservative management and postpone surgery. Daily follow-up with X-rays was performed to monitor progress, which did not reveal pneumoperitoneum. Escherichia coli and Klebsiella pneumoniae were isolated in blood cultures without acquired resistance.

On day 16 of hospitalization, the patient developed a fever, so new cultures were taken, the antibiotic regimen was expanded to cover multidrug-resistant organisms, and a new CT scan was performed. The images showed persistent thickening of the gastric wall, and secondary complications requiring surgical intervention were ruled out. Carbapenemase-producing K. pneumoniae was subsequently isolated in blood cultures, and targeted antibiotic treatment was initiated promptly.

On the 18th day of hospitalization, the patient presented a new episode of massive upper gastrointestinal bleeding, with hypovolemic shock refractory to therapeutic measures and died.

Surrogate informed consent was obtained from a direct relative, who stated that the protection of personal data is respected according to Law 25.326 - Habeas Data.

3 Discussion

Phlegmonous gastritis is a rare inflammatory-infectious disease whose pathophysiology is not fully understood. A pathophysiology similar to that of neutropenic enteropathy has been proposed, describing a combination of mucosal damage from chemotherapy agents and invasion of the intestinal wall by microorganisms, which triggers the inflammatory process [7-11]. It has been categorized into three types: type 1 or primary, which appears to be the most common, is observed in patients with mucosal damage due to peptic ulcers or gastric cancer; type 2 or secondary, is observed secondary to systemic infections such as endocarditis or local infections of the biliary tract or liver abscesses; and type 3 or idiopathic, which occurs in immunocompromised patients in whom no clear associated factor is found [2,5].

Notwithstanding its etiology, the signs and symptoms are shared, with the sudden onset of fever, epigastric abdominal pain, nausea, hematemesis and sometimes vomiting with purulent contents, which although considered pathognomonic is extremely rare. Since its manifestations are little distinguishable from other causes of acute abdomen, it is difficult to identify clinically. The findings in tomography and videoendoscopy are also common in different etiologies: localized or diffuse thickening of the gastric wall, mucosal irritation, edema, erosions and ulcers, with hemorrhages and purulent contents [9,12]. The main causative agents that have been described in the different reports and series of cases are Streptococcus spp., followed by Staphylococcus spp. and Escherichia coli along with other enterobacteria [2-4,6].

Treatment consists of supportive measures, gastric rest, parenteral nutrition, and early antibiotic administration. Surgery should be considered in refractory cases and in cases of complications such as localized abscess formation or gastric perforation. If possible, partial resection is preferred over total gastrectomy due to its high morbidity and mortality. Regardless, mortality is high despite adequate treatment; the percentages reported in different reports are heterogeneous, close to 50% for the diffuse form treated with antibiotics and substantially lower for the localized form. In some reports, mortality is higher for patients who required surgical intervention [7,9]. There are no studies comparing conservative management versus aggressive management with early surgical intervention.

Phlegmonous gastritis, or necrotizing neutropenic transmural gastritis, is a phenomenon that deserves to be differentiated from neutropenic enterocolitis, given its exclusive involvement of the stomach. Recognition of this rare disease is imperative, and we believe it should be included as a differential diagnosis in any neutropenic patient with epigastric pain.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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