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CLINICAL CASE

Necrotizing gastritis as a rare complication in a pediatric patient with acute lymphoid leukemia

Gastritis necrotizante como complicación infrecuente en un paciente pediátrico con leucemia linfoide aguda

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

Necrotizing gastritis is a rare condition with an unknown prevalence. The diagnosis is often incidental, and the prognosis is unfavorable.

What does this study contribute to what is already known?

We present a clinical case of necrotizing gastritis to emphasize the importance of timely clinical suspicion and early initiation of treatment, which are key factors for a favorable outcome in this rare complication. It should be noted that patients with risk factors such as immunosuppression and the use of cytotoxic drugs should be mindful of this condition.

Abstract

Necrotizing gastritis is an infrequent entity with unknown prevalence, the diagnosis is often incidental during exploratory laparotomy or autopsies of patients with acute abdomen. **Objective:** To present a clinical case of necrotizing gastritis, a rare entity that should be taken into account in the context of immunocompromised patients with associated risk factors. **Clinical Case:** 7-year-old male schoolboy diagnosed with T-precursor acute lymphoid leukemia, finishing induction chemotherapy cycle with PETHEMA 2013 protocol. He presented 12 days of symptoms characterized by epigastric abdominal pain and vomiting, initially acute pancreatitis was suspected, ruled out by normal pancreatic enzymes and abdominal computed tomography. Due to suspicion of acid peptic disease associated with steroids, treatment with proton pump inhibitors and prokinetics was started. Consi-

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dering dyspepsia with alarm signs, such as progression of neutropenia, increased C-reactive protein and clinical deterioration, esophagogastroduodenoscopy (EGD) was performed, compatible with necrotizing gastritis, confirmed by histopathology. He received pharmacological management, zero regimen and parenteral support, and progressive improvement was evidenced in imaging controls. After fasting for 30 days, enteral nutrition was started, well tolerated, with ambulatory follow-up. After improvement, chemotherapy plan was completed, highlighting complete remission, without complications after 2 years. **Conclusion:** Necrotizing gastritis is a rare entity, in the case described the risk factors were immunocompromise, previous management with corticosteroids and cytotoxic therapy, and possibly, exposure to stressful situations during hospitalization. Early diagnosis and treatment determined a favourable prognosis.

Introduction

The stomach is a highly vascularized organ within the gastrointestinal tract, making necrosis at this level uncommon. Acute necrotizing gastritis can occur as a stage of phlegmonous gastritis, where microorganisms cause necrosis and gangrene in the stomach wall, instead of an isolated intramural abscess with a clear etiology[1,2]. Suspecting gastric wall necrosis may be possible when air is detected in ultrasound or radiological studies of the stomach. The presence of air accumulation can be associated with various clinical situations, each with different prognoses depending on factors such as age and comorbidities[3,4].

Necrotizing gastritis is a condition associated with high morbidity and mortality rates, especially in infants. It is often related to sepsis, asphyxia, malformations, and cardiac surgery. Although less common than necrotizing enterocolitis (NEC), it shares several clinical features and treatment options. Typical symptoms include abdominal distention, melenic stools, hematemesis, or coffee-ground vomiting. Abdominal distention is commonly observed in preterm infants, while hemorrhage and vomiting are more frequent in term infants[2,5].

Gastric infarction and necrosis represent acute abdominal emergencies that require rapid intervention, usually involving surgery. There is limited data on patients with hematologic malignancies who develop necrotizing gastritis. Some case reports have described patients with acute leukemia who developed acute phlegmonous necrotizing gastritis during the neutropenic phase of induction chemotherapy and were successfully treated. Therefore, it is important to consider the diagnosis of this condition in immunocompromised patients[6,7].

The objective of this study is to present a clinical case of necrotizing gastritis, a rare condition that should be considered in immunocompromised patients with associated risk factors.

Clinical Case

A 7-year-old male patient diagnosed with T-cell precursor acute lymphoblastic leukemia was completing a cycle of induction chemotherapy with the PETHEMA 2013 protocol (L-asparaginase, Vincristine, and Daunorubicin + continuous prednisone for over a month). During his hospital stay, he developed a 12-day clinical presentation of epigastric abdominal pain and occasional vomiting. Physical examination revealed severe tenderness upon palpation of the epigastric region, with visible peristalsis but no history of obstruction. Laboratory tests showed moderate neutropenia and an increasing level of C-reactive protein (CRP).

Initially, acute pancreatitis was suspected, but it was ruled out as pancreatic enzymes and an abdominal CT scan were normal. A possible acid peptic disease due to steroids was then considered, and the decision was made to continue medical treatment with proton pump inhibitors and prokinetics while assessing tolerance to the oral route.

However, the following day, the patient experienced intensified abdominal pain at night, accompanied by multiple episodes of vomiting, hematemesis, hydroelectrolytic imbalance (severe hypokalemia), hyperglycemia, severe neutropenia, and signs of shock. As a result, the patient was transferred to the pediatric intensive care unit (PICU). An emergency esophagogastroduodenoscopy (EGD) was performed, which revealed acute hemorrhagic pangastritis consistent with necrotizing gastritis (Figure 1.a), and this diagnosis was confirmed by biopsy (figure 2).

Management included fasting, potassium replacement, fluid replacement, and continuous infusion of omeprazole and ondansetron. Laboratory tests and blood cultures yielded negative results with no bacterial growth. The patient's condition deteriorated to refractory shock despite catecholamine therapy, necessitating mechanical ventilation, antibiotic treatment, and multiple vasoactive support with norepinephrine, epinephrine, and vasopressin.

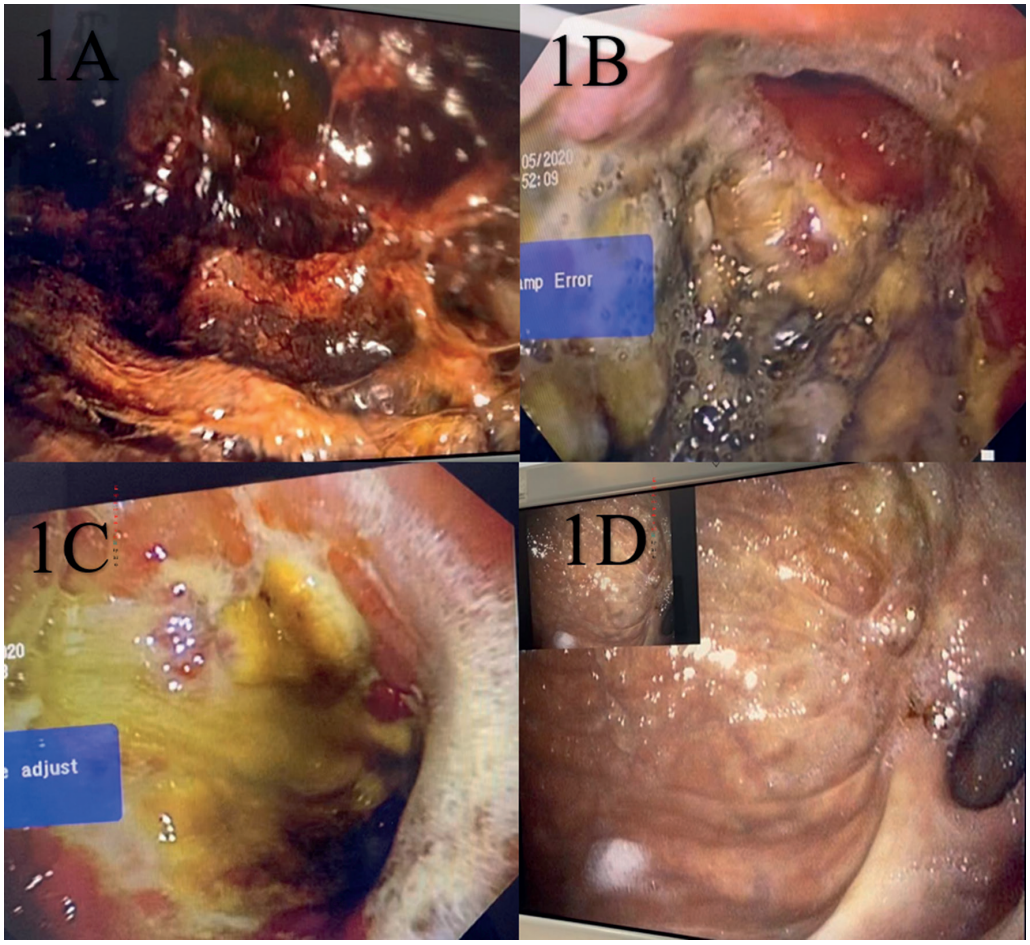

Figure 1. Endoscopic evolution: A. Day 0: Extensive necrotic involvement of the entire gastric mucosa, with loss of gastric folds, areas of recent hemorrhage, and fibrin septa. B. Day 15: Gastric necrosis confined to the greater curvature of the gastric body, with improvement of necrosis in the antrum and gastric fundus. C. Day 30: Necrotic area involving 10% of the greater curvature of the gastric body before the corpoantral junction. D. 6 months: Resolution of necrosis with patchy erythema and pale areas, and formation of a 1 cm diameter pseudodiverticulum before reaching the corpoantral region.

After overcoming the critical condition, parenteral nutrition was initiated on the second day due to prolonged bowel rest. On the sixth day, a follow-up EGD showed persistent necrotizing gastritis. Proton pump inhibitors and sucralfate were administered. After 30 days of enteral rest, another EGD was performed, revealing a significant improvement in extensive necrosis of the gastric body, fundus, and antrum, with only 20% of the greater curvature of the gastric body still affected by necrosis (figure 1.b). Enteral nutrition (polymeric diet) was considered based on tolerance, progressing to a soft diet without irritants or lactose.

The patient started the oral route and showed adequate in-hospital tolerance for 72 hours, leading to hospital discharge with double doses of proton pump inhibitors and sucralfate. The patient was advised to remain vigilant for any alarming signs. During the follow-up visit 2 months after discharge, the patient remained hemodynamically stable. The monitoring EGD showed complete improvement of necrotizing gastritis, with remaining areas of fibrosis in the antral-body region (figure 1.c).

Chemotherapy treatment was postponed for a month and a half after the resolution of the critical condition associated with necrotizing gastritis. The same high-risk PETHEMA protocol was resumed, but with reduced doses (50% of the cytostatic) during the intensification stage. In the subsequent stage, the protocol continued with the usual doses. End-of-treatment analyses were conducted, and the patient has been in complete remission without complications associated with the underlying pathology for two years to date.

Discussion

Necrotizing gastritis is a serious and rare complication, likely resulting from the rich vascularization supplied by branches of the celiac trunk and the superior mesenteric artery. It is challenging to diagnose both clinically and histopathologically. In this type of gastritis, although the four main vessels supplying the stomach remain patent, necrosis primarily occurs due to transmural infection. This condition can affect both immunocompetent and immunosuppressed individuals, including patients with leukemia, as in our case[7,8].

Acute gastric ischemia has been associated with various conditions such as disseminated intravascular coagulation, severe mesenteric ischemia, adrenal insufficiency, vasopressin infusion, endoscopic dissection, and sclerotherapy[9]. Relevant risk factors contributing to the pathogenesis include cardiovascular disease, hemodynamic instability, alcohol intake, malnutrition, diabetes, hypoxemia, trauma, and immunosuppression[1].

In immunocompromised patients like our patient with T-cell precursor acute lymphoblastic leukemia, who experienced severe neutropenia due to induction chemotherapy, the likelihood of developing this and other complications is increased[6]. Necrotizing gastritis is a serious and rare complication, with limited reports in the world literature, and it can lead to high mortality rates[2,6]. Although the association with a specific chemotherapy agent has not been clearly established, this complication has been observed in patients receiving certain cytotoxic agents in combination[8,12]. Given the patient's underlying diagnosis of severe neutropenia at the time of presentation, it is suspected that the damage mechanism was multifactorial[9,10].

During severe shock, total gastric blood flow drops by nearly 90% and reaches nearly 0 in the body mucosa and fundus due to gastric vasoconstriction. Acute gastric ischemia may manifest with symptoms such as nausea, vomiting, upper gastrointestinal bleeding, abdominal distension, and symptoms related to underlying predisposing conditions[10], which could be related to the described case considering the presented signs and symptoms. Stomach distension often exacerbates gastric ischemia, as seen in events following the consumption of large meals or in individuals with bulimia[11]. At the tissue level, early gastric ischemic changes include capillary dilation, mucosal edema, vascular congestion, and superficial necrosis. These findings progress to coagulative necrosis of the mucosa, characterized by superficial erosions and necroinflammatory (fibrinopurulent) exudates. Persistent ischemia leads to full-thickness hemorrhagic necrosis and deep ulceration of the gastric wall. Reactive changes, such as mucin depletion, nuclear enlargement, hyperchromasia, and increased mitotic activity, occur as part of the pathophysiological changes in this condition[1,11].

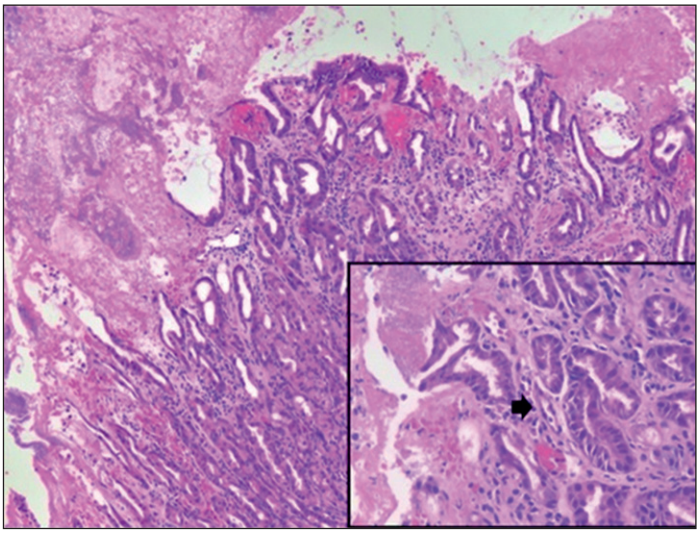

Figure 2. Histopathological description report of the patient. Microscopic ischemia shows gastric mucosa with extensive superficial ischemic necrosis and hemorrhage with abundant fibrinoid material, bacterial colonies, and reparative changes in superficial glands. The inset shows, at higher magnification, tortuous glands with cytoplasmic eosinophilia and nuclear hyperchromasia indicative of reparative changes, as well as foci of extravasated red blood cells, vascular congestion, and bacterial colonies (arrowhead). The diagnostic conclusion was severe acute necrohemorrhagic gastritis. Abundant cocci and bacillary bacterial colonies. No evidence of Helicobacter-like bacilli, fungi, or parasites.

The main diagnostic tests for necrotizing gastritis are upper gastrointestinal endoscopy with biopsy, as performed in the described case, and other specific imaging techniques for vascular studies, such as axial CT scan, CT angiography, and mesenteric angiography[12]. Endoscopic findings may vary depending on the etiology and duration of the condition and can include diffuse or irregular discoloration of the gastric mucosa (pale mucosa), loss of the mucosal vascular pattern, diffuse erosions, and ulcerations. Ischemic ulcers tend to appear near anastomotic sites[1,10].

Gastric necrosis typically presents as multiple ulcerations in the antral region, with a fibrinoid base. In severe acute cases, like the one described, areas of necrotic mucosa or submucosal emphysema may be observed. These areas are highly friable and bleed upon contact with the endoscope[1,10,13].

The role of histological study is usually limited, often revealing nonspecific gastritis. However, in our case, the diagnosis was confirmed through endoscopy and biopsies, ruling out other differential diagnoses such as neoplasms, drug-induced ulceration, and viral infection. Diagnostic endoscopy should always be considered in patients with suspected gastric ischemia, as it can confirm the diagnosis, assess the severity and

extent of gastric ischemia, and exclude other pathologies[13,14].

Treatment initially focuses on patient resuscitation with intravenous fluids and broad-spectrum parenteral antibiotics. Gastric decompression is achieved through a nasogastric tube, and enteral rest is recommended. Gastric necrosis and perforation generally carry a poor prognosis, with reported mortality rates ranging from 50% to 80%[6]. Fortunately, in the described patient, the clinical course was favorable, which could be attributed to the prompt management prompted by clinical suspicion[15].

Surgical management options for necrotizing gastritis include total or subtotal gastrectomy with gastrointestinal continuity reconstruction[1,13]. Surgical intervention is recommended in cases of gastric perforation, gastric volvulus, or severe gastric ischemia that do not respond to medical treatment[1]. However, in the presented case, surgical intervention was not required as the patient responded well to medical management on time.

Conclusions

Acute necrotizing gastritis is a rare condition that demands a high level of suspicion as an early diagnosis enables prompt and intensive management, significantly reducing mortality associated with this condition. The etiology of gastric necrosis is multifactorial, with a significant portion of cases being idiopathic. In the presented case, it is important to consider the following risk factors: immunocompromised state, prior administration of corticosteroids and cytotoxic drugs, as well as exposure to stressful situations during hospitalization.

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

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