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Spontaneous Gastric Perforation: A Rare And Fatal Presentation Of Primary Gastric Lymphoma – Case Report

Taha Kabbaj, Badr Kabila, El Weld Moustapha, Kaid Mohamed, Dato Habib, Noufou Moctar, Othman Alaoui, Omar El Aaoufir, Leila Jroundi, Zakaria El Mouatassim

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I. Introduction

Primary gastric lymphoma (PGL) is a rare clinical entity accounting for the majority of extra-nodal non-Hodgkin lymphoma (EN-NHL). The most common histological subtype is the primary gastric diffuse large B-cell lymphoma (PG-DLBCL) accounting for 40 %–70 % of all primary gastric non-Hodgkin's lymphomas[1,2].

The clinical manifestations of PG-DLBCL are nonspecific and easily misdiagnosed due to its unspecific symptoms of the digestive tract[3].

The PGL is more likely to occur in patients older than 50 years, but patients in the second decade of life could be also affected. Males are 2 to 3 times more likely to develop PGL than females.[4,5,6]

The treatment of PG-DLBCL includes surgery, radiotherapy and chemical immunotherapy. Under proper treatment, PG-DLBCL has a good prognosis, with a 5-year overall survival rate of nearly 90%[7]. In some instances, it might present as spontaneous perforation of the stomach or perforation related to chemotherapy. At this point, surgical intervention is warranted in most cases[9].

II. Observation

A 50-year-old female patient, with no significant past medical history, presented to the emergency department for chronic epigastric pain evolving over two months, recently exacerbated over the past week. She also reported anorexia and progressive weight loss. On clinical examination, the patient was febrile at 38°C, pale, and exhibited marked tenderness in the epigastric region without signs of guarding or palpable masses. The abdomen was soft, and her vital signs were initially stable.

Initial laboratory investigations revealed moderate anemia (hemoglobin at 10.5g/dL), a mild elevation of CRP at 35 mg/L, and no leukocytosis.

An abdominal CT scan was performed, revealing irregular thickening of the gastric wall suggestive of a tumor.**(Figure1)** A diagnostic esophagogastroduodenoscopy confirmed the irregular thickening of the gastric wall, and gastric biopsies were obtained for histopathological analysis.

Due to family-related issues, the patient refused hospitalization and was discharged on symptomatic treatment with omeprazole while awaiting the pathology results.

The patient did not return as scheduled and was lost to follow-up for approximately two months. She eventually presented again to the emergency department with intense abdominal pain, severe deterioration of her general condition, and clinical signs of peritonitis.

On examination, the patient appeared pale, with prolonged capillary refill time (>3 seconds), hypotension(90/60mmHg), tachycardia(120bpm), tachypnea(26 breaths/min), oxygen saturation at 95%, and a fever of 38.8°C.Abdominal examination revealed a distended, immobile abdomen with generalized guarding, a positive Blumberg sign, and diffuse tympanism.

Biological investigations showed significant leukocytosis at 19,500/mm³ with neutrophil predominance, a CRP level elevated to 197 mg/L, and normocytic anemia with hemoglobin at 9.5 g/dL.

Meanwhile, the histopathological analysis confirmed the diagnosis of gastric lymphoma, showing complete effacement of the normal tissue architecture, replaced by a diffuse infiltrate of large atypical lymphoid cells. These cells exhibited pleomorphic nuclei, prominent nucleoli, and vesicular chromatin, features consistent with malignant proliferation **(Figure 2)**.

Immunohistochemical staining confirmed the B-cell lymphoid origin of the tumor cells with positive CD20 expression **(Figure 3A)**. The immunohistochemical profile also showed negativity for CD10, suggesting

an activated (non-germinal center) subtype **(Figure 3B**). The Ki-67 proliferation index was elevated, exceeding 70%, indicating a high proliferative activity **(Figure 4A**). Additionally, tumor cells were negative for epithelial markers CK AE1/AE3, excluding an epithelial origin and confirming the lymphoid nature of the proliferation **(Figure 4B).** Altogether, these findings were consistent with a diagnosis of diffuse large B-cell lymphoma.
Given the clinical deterioration and histopathological findings, secondary gastric perforation was suspected.

An urgent abdominopelvic CT scan confirmed the diagnosis, revealing a gastric wall defect with perigastric pneumoperitoneum, associated with irregular circumferential wall thickening and large-volume peritoneal effusion**(Figure 5**).
Unfortunately, despite rapid intensive care management, the patient succumbed to the septic shock.

III. Discussion

Diffuse large B-cell lymphoma (DLBCL) is a subtype of aggressive non-Hodgkin lymphoma (NHL), accounting for 40%-50% of all NHLs. Unlike MALT lymphomas, often associated with Helicobacter pylori,DLBCL is more aggressive and prone to complications such as perforation.[8].

Perforation and peritonitis are known complications of GI lymphomas that can occur either at diagnosis or during the course of treatment.But it can also occur prior to treatment , and is typically due to deep tumor infiltration, ischemic necrosis from hypovascularization,or rarely, a response to recent .[9,10]

A comprehensive review by R. Vaidya et al. identified only 92 cas of perforation in 1062 patients diagnosed with intestinal lymphomas and only 16% present stomach perforation.which make it a very rare complication NHL [11]. Our case represents a spontaneous gastric perforation **prior to** any treatment, highlighting the rarity of such a presentation.

Clinically, spontaneous gastric perforation presents with signs of acute abdomen, including sudden severe epigastric pain, generalized guarding, fever, tachycardia, hypotension, and signs of septic shock.

However, symptoms may sometimes be masked by pre-existing chronic gastric pain, leading to delayed diagnosis.[12,13]

Histologically, MALT lymphomas show diffuse or nodular infiltration with lymphoepithelial lesions, while DLBCL features large atypical lymphoid cells with vesicular nuclei and prominent nucleoli. Immunohistochemically, both express CD20.

DLBCL subtypes vary in CD10 expression, and a high Ki-67 proliferation index (>50%) helps distinguish it from MALT lymphoma.[14,15]

Imaging, particularly abdominopelvic CT, plays a crucial role, confirming pneumoperitoneum, identifying tumor-related gastric wall thickening, perigastric fat infiltration, and associated lymphadenopathy, while excluding other causes of perforation.[16,17]

The treatment of primary gastric lymphoma has shifted away from surgery, toward chemotherapy regimens. In a time surgery is now limited to cases of perforation, hemorrhage, or obstruction due to the tumor

Management of spontaneous gastric perforation requires emergency surgery, typically exploratory laparotomy with gastric resection and peritoneal lavage.

Subsequent oncologic treatment includes R-CHOP chemotherapy for DLBCL and Helicobacter pylori eradication for MALT lymphomas if appropriate.[18,19]

Prognosis depends on the severity of peritonitis, timing of surgery, and lymphoma response to treatment. Postoperative mortality remains high, underscoring the importance of early diagnosis, aggressive management, and multidisciplinary follow-up to improve survival outcomes.[20,21].

IV. Conclusion

Spontaneous gastric perforation due to primary gastric lymphoma is a rare but life-threatening complication. While perforation is more commonly associated with chemotherapy, spontaneous cases prior to treatment are exceptional.Prompt imaging—particularly CT—is vital for early detection. Emergency surgical intervention remains the cornerstone of management, followed by systemic treatment. Given the high mortality risk, early diagnosis and timely multidisciplinary care are crucial for improving outcomes.

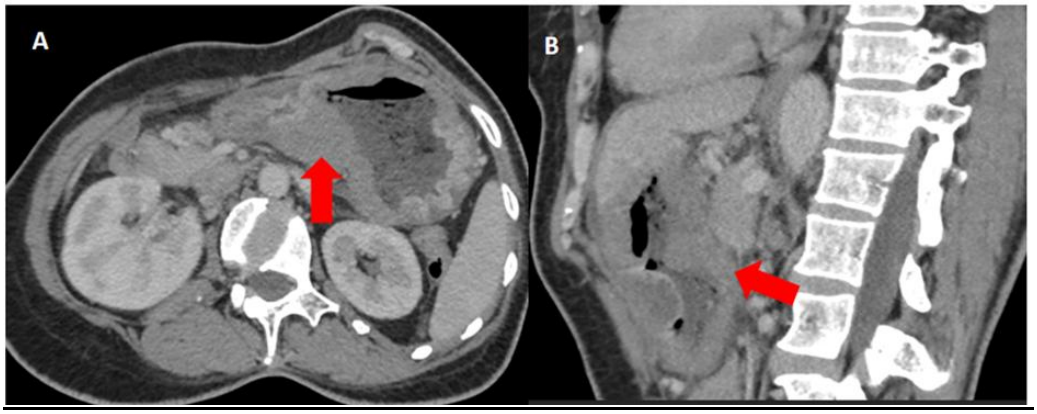

Figure1 : Contrast-enhanced abdominal CT scan (A: axial view, B: coronal reconstruction): showing irregular gastric wall thickening (red arrow).

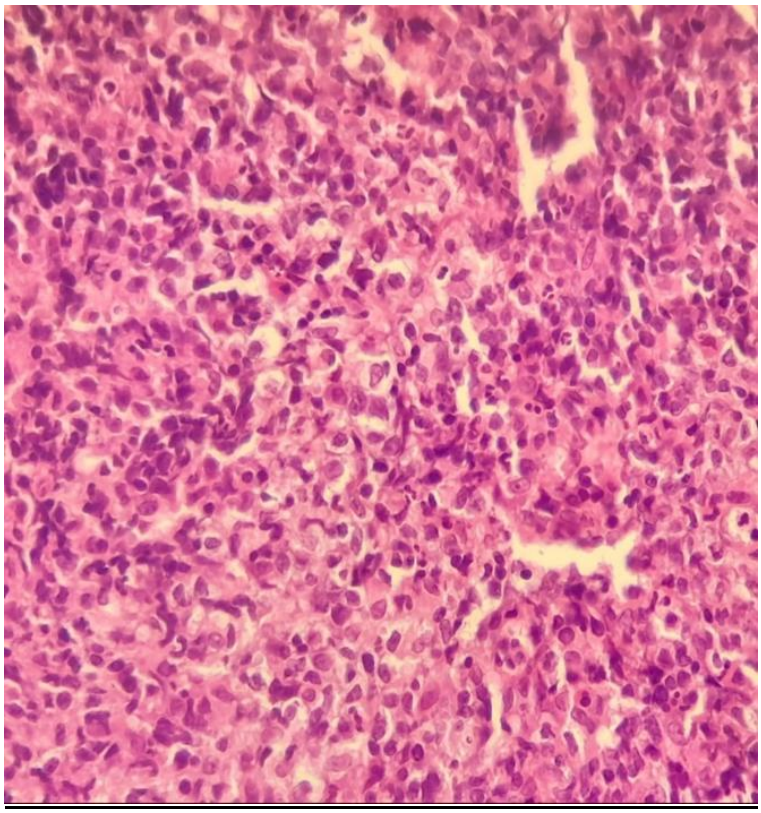

Figure 2: Histology (x400) : Effacement of the normal tissue architecture by a diffuse infiltrate of large atypical lymphoid cells, with pleomorphic nuclei, prominent nucleoli and vesicular chromatin

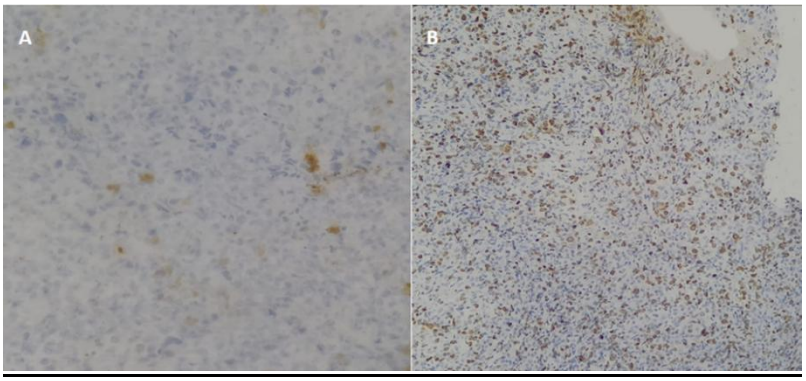

Figure 3 :A: Immunohistochemistry: CD20 staining (x400) : Diffuse membranous expression of neoplastic cells B: Immunohistochemistry: CD10 staining (x400) : Lack of immunostaining by neoplastic cells

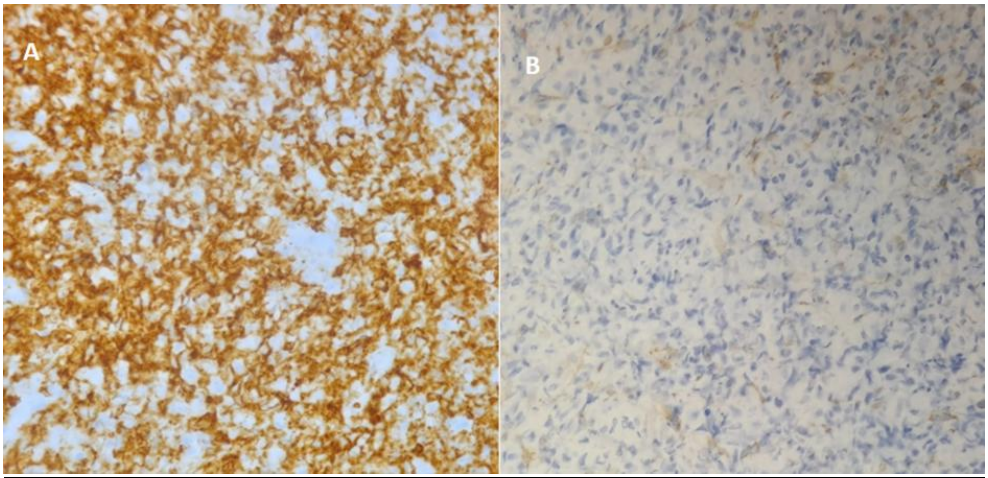

Figure 4 : A: Immunohistochemistry: CK AE1,AE3 staining (x400) : Lack of immunostaining by neoplastic cells B:Immunohistochemistry: Ki-67 staining (x200) : Nuclear expression by 70% of neoplastic cells

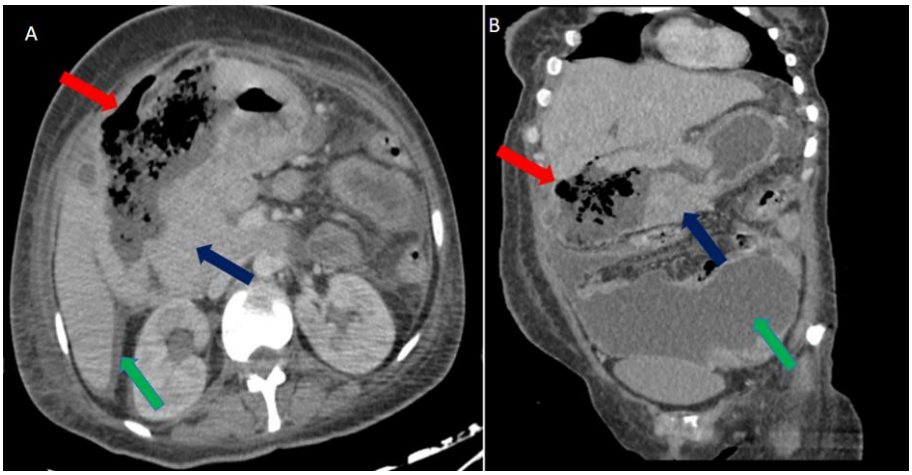

Figure 5: Contrast-enhanced abdominal CT scan (A: axial view, B: coronal reconstruction): Shows a gastric wall defect with perigastric pneumoperitoneum (red arrow), associated with circumferential wall thickening (blue arrow) and Large-volume ascites(green arrow)

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