Gastric Lymphoma: Severe Gastric Outlet Obstruction Warranting Stent Placement

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ABSTRACT

Primary gastric lymphoma is an uncommon entity accounting for <5% of primary gastric neoplasms and 10-15% of all non-Hodgkin lymphomas. It may present with nonspecific symptoms or features of gastric outlet obstruction (GOO). GOO can develop prior to or during treatment due to disease or post-treatment scarring. The obstruction can be both intrinsic or extrinsic. Endoscopic balloon dilatation and surgical gastrojejunostomy have been tried with variable success. Gastroduodenal stenting with self-expanding metal stents (SEMS) has been used lately with excellent results but mainly in the palliative management of gastric carcinoma with GOO or in benign GOO. We present a case of gastric diffuse large B cell lymphoma on RCHOP chemotherapy who developed severe GOO leading to profound metabolic alkalosis and electrolyte imbalances, ultimately warranting an enteral stent.

Key Words: *Primary gastric lymphoma, Gastric outlet obstruction, Diffuse large B cell lymphoma, Enteral stent.*

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INTRODUCTION

Primary gastric lymphoma (PGL) is the most common type of extra-nodal lymphoma, representing 30-40% of all extra-nodal non-Hodgkin lymphoma cases.[1,2] Diffuse large B cell lymphoma (DLBCL) is the predominant subtype of PGL.[3] It typically presents with non-specific symptoms, *e.g.*, epigastric pain, anorexia, or weight loss. Rarely, the presentation may be complicated by a perforation or obstruction. Gastric outlet obstruction (GOO), intrinsic or extrinsic, can develop prior to or during treatment due to disease or post-treatment scarring and fibrosis. Endoscopic balloon dilatation and surgical gastrojejunostomy have been tried with variable success. Gastroduodenal stenting with self-expanding metal stents (SEMS) has been used lately with excellent success rates of 80-90% but mainly in the palliative management of gastric carcinoma with GOO or in benign GOO.[4] Usually, the obstruction by PGL resolves with initial chemotherapy sessions but rarely, it can be severe at presentation or may worsen due to post-chemotherapy scarring demanding an enteral stent for resolution of symptoms.

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CASE REPORT

A 35-year male presented in the medical oncology clinic with a history of abdominal pain, significant weight loss, and generalized weakness for four months. There was neither history of gastritis or other co-morbidities, nor any family history of cancer. He had undergone gastroscopy and biopsy (distal body and antrum) which showed DLBCL. His performance status was good and his vitals were within the normal range. Physical examination revealed a well-nourished male with mild epigastric tenderness, no palpable lump, no organomegaly, and normal bowel sounds. Laboratory investigations showed hemoglobin (Hb) of 8.4 g/dL, white blood cell (WBC) count, 11000/uL, serum potassium (K), 4.19 mmol/L (Normal: 3.3-5.1), serum sodium (Na), 136 mmol/L (Normal: 136-145), and serum bicarbonate (HCO_3), 27 mmol/L (Normal: 22-29). Hepatitis B surface antigen was positive. Positron emission tomography/computed tomography (PET/CT) scan showed the hypermetabolic gastric body and antral tumor without any locoregional flurorodeoxyglucose (FDG)-avid lymph nodes. Bone marrow aspiration and trephine biopsy showed no evidence of involvement by DLBCL. The disease was staged as IE gastric DLBCL. It was planned to give R-CHOP chemotherapy (rituximab, cyclophosphamide, doxorubicin, vincristine, prednisolone) for 6 cycles with interim PET after 3 cycles and radiotherapy after completion of chemotherapy. He was started on tablet entecavir by the gastroenterology team for positive hepatitis B. He did quite well until he received his second cycle of chemotherapy, after which he developed persistent non-projectile vomiting particularly a few hours post-meals. He also developed constipation but was

passing flatus. Due to persistent vomiting, he could not come for the 3rd cycle of chemotherapy. Eventually, he crashed in the emergency with a 2-week delay from the due date of the 3rd cycle of chemotherapy. His vomiting could not settle in the past 4 weeks despite taking maximum antiemetics. He had not been able to pass stool since then but was passing flatus. He was hypotensive (blood pressure, 90/60 mmHg), tachycardiac (heart rate, 118/minute) and his oxygen saturation was 89% at room air. On examination, he was drowsy, emaciated, cachectic, dehydrated with sunken eyes, and had a doughy abdomen. Bloods tests were suggestive of severe metabolic alkalosis, hypokalemia and hyponatremia {Blood urea nitrogen, 44 mg/dL, creatinine, 0.57 mg/dL, serum albumin, 3.93 g/dL, K, 2.4 mmol/L, Na, 114 mmol/L, HCO_3, 48 mmol/Land raised troponin I 0.059 (Normal: <0.046)}. Computed tomography pulmonary angiogram (CTPA) did not show any evidence of pulmonary embolism or pulmonary pathology and the echocardiogram showed normal left ventricular ejection fraction. A restaging computed tomography (CT) scan was done which showed stable gastric antral tumor without locoregional lymphadenopathy; however, there was interval development of pyloric stenosis and gastric outlet obstruction (Figure 1a).

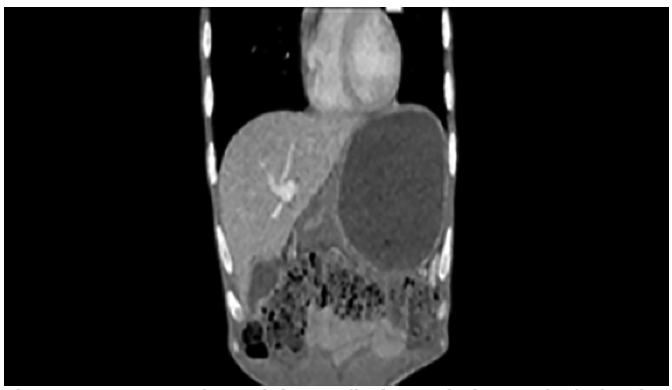

Figure 1a: CT scan chest abdomen (before pyloric stenting) showing gastric antral/pyloric tumor with interval pyloric stenosis leading to gastric outlet obstruction.

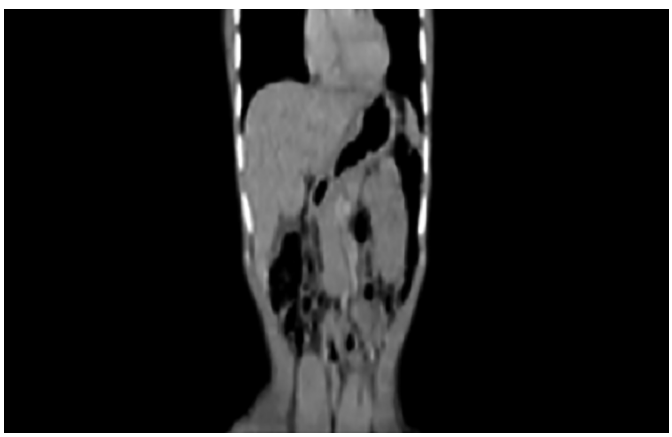

Figure 1b: PET/CT (after pyloric stenting) showing interval regression in hypermetabolic gastric body and pyloric thickening.

Further chemotherapy was held and oesophago-gastro-duodenoscopy (OGD) was performed which confirmed the CT findings of complete obstruction causing pyloric stenosis (Figure 2a). A naso-jejunostomy (NJ) tube was passed for feeding purpose and endoscopic biopsy was done, which showed moderate chronic focally active gastritis with focal intestinal metaplasia but no Helicobacter pylori, dysplasia or malignancy. After NJ feeding, metabolic alkalosis and electrolyte imbalances started to resolve {K, 3.65 mmol/L, Na, 135 mmol/L, and HCO_3, 26 mmol/L}. Unfortunately, the patient was still unable to eat and continued to have episodic vomiting leading to the displacement of his NJ tube after which vomiting worsened. He became very depressed due to his inability to eat and recurrent vomiting and even expressed his thoughts of leaving the hospital and wishing to go home to die. However, he agreed to have another OGD with or without pyloric stenting after a couple of sessions with a psychologist. He underwent endoscopic enteral pyloric stenting (Figure 2b), after which intractable vomiting finally resolved. He was also offered radiotherapy to the stomach. He then started to eat better and put on weight. An interim PET/CT scan was done later, which showed interval regression in the hypermetabolic gastric body and pyloric thickening (Figure 1b). He feels quite well now tolerating an oral diet and is receiving the remaining cycles of chemotherapy.

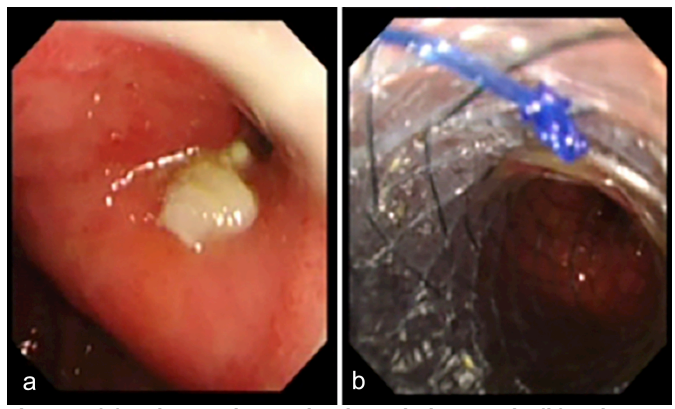

Figure 2: (a) Endoscopy image showing pyloric stenosis. (b) Endoscopy image showing pyloric stent in place.

DISCUSSION

PGL is defined as gastric lymphoma with or without the involvement of peri-gastric and abdominal lymph nodes. Adenocarcinoma and non-Hodgkin's lymphomas are the most common primary malignancies of the stomach. The predominant subtype of PGL is DLBCL; the other subtypes include mucosa-associated lymphoid tissue, follicular, mantle cell, and peripheral T cell lymphoma. DLBCL of the stomach is an aggressive lymphoma constituting 40-70% of all gastric lymphomas. It occurs more commonly in males, with a median age of occurrence of 50-60 years. Prognosis is determined by clinical factors like stage, serum lactate dehydrogenase levels, and performance status.[5,6] Primary gastric DLBCL typically presents with non-specific symptoms as seen in other common gastric conditions, *e.g.*, gastric adenocarcinoma, peptic ulcer disease, and non-ulcer dyspepsia. The most common presenting complaints include epigastric discomfort (84%), anorexia (46%), loss of weight (24%), nausea/vomiting (19%), and early satiety.[7] Very rarely, it may present with GOO. Kosch *et al.* reviewed 277

patients with PGL in a study and not a single patient presented with GOO.[8]

GOO due to gastric DLBCL generally resolves with the first few cycles of chemotherapy such as R-CHOP; however, rarely severe obstruction may still occur due to post-chemotherapy scarring or residual lymphoma. The consequences of severe GOO make it very difficult to deliver chemotherapy treatment leading to inferior outcomes. The completion rates for six cycles of R-CHOP and complete remission (CR) rates were significantly lower among those with GOO than those without.[4]

The GOO secondary to gastric DLBCL can be managed with gastroduodenal stenting or gastrojejunostomy (GJ). Gastric stents show better results than GJ as the former is less invasive and there is prompt relief of symptoms. Gastroduodenal stenting with SEMS has been preferred lately with excellent success rates of 80-90% but are mostly used in the palliative management of gastric carcinoma with GOO or in benign GOO. Although stents are not a long-term solution to issues of stent migration; they may, however, provide good temporary relief of symptoms in gastric DLBCL with GOO allowing completion of chemotherapy cycles and improving CR rates and outcomes.

To conclude, GOO due to primary gastric DLBCL is a rarity and is associated with inferior outcomes. This obstruction may need stent placement earlier rather than later. One must have a clinical suspicion and intervene early to avoid significant deterioration in performance status and life-threatening electrolyte imbalance making delivery of potentially curative chemotherapeutic treatment difficult.

PATIENT'S CONSENT:
The study was given exemption from the patient's written/informed consent by Institutional Ethical Review Board.

COMPETING INTEREST:
The authors declared no competing interests.

AUTHOR'S CONTRIBUTION:
JI: Data collection and report writing.
MZS: Literature review and discussion.
MS: Proof-reading of the final version.
SF: Concept of the manuscript and study design.
SWIB: Provided concept of the manuscript and supervised the study.

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