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Original Research Paper

General Surgery

A RARE CASE OF METASTATIC GASTRIC LINITIS PLASTICA PRESENTING AS AN ACUTE ABDOMEN IN A YOUNG FEMALE

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ABSTRACT Linitis plastica is an uncommon entity of stomach which is strongly associated with genetic predisposition. On histopathological examination, there are few or scanty cellular infiltration of hypertrophic submucosal layer of stomach in the background of abundant filamentous strands and scar like tissue. Owing to the presence of these strands, it is referred to as linitis (due to resemblance to linen). This case report highlights the management of acute complication of gastric malignancy in a 31 years old female. The patient was referred for evaluation of suspected abdominal tuberculosis based on imaging and a plethora of symptoms. On second day of admission patient presented with features of hollow viscus perforation. She was treated with Modified Graham's omental patch closure and feeding jejunostomy. She was discharged on the fifth postoperative day and referred for palliative chemoradiation. This case report highlights the management of a perforated metastatic linitis plastica of stomach presenting as an acute abdomen.

KEYWORDS : Linitis plastica, Surgery, Gastric cancer, Metastases

INTRODUCTION

Gastric adenocarcinomas can be classified into two varieties-intestinal and diffuse types. While reduced smoking trends and effective Anti-H. pylori eradication therapies have decreased the incidence of intestinal type of adenocarcinomas, diffuse gastric cancer cancers tend to rise in incidence [1]. Gastric carcinomas can present with plethora of symptoms and complications. Diffuse gastric cancers are associated with poor outcome and reduced five year survival rates compared to intestinal type of gastric cancers.

Case Report

A 31 years old female who is a homemaker from Broadway, Chennai was referred from a private hospital for evaluation of suspected abdominal tuberculosis based on contrast imaging of abdomen. She presented with complaints of early satiety, breathlessness on exertion and upper abdominal pain for the past 1 month. Patient was apparently normal 1 month ago after which she developed early satiety after food intake-specific to solids, insidious in onset and gradual in progression. Complaints of dull aching upper abdominal pain- intermittent in nature and aggravated after food intake. History of recent change in abdominal pain- dull-aching, continuous type of pain and radiating to the back. History of easy fatiguability MMRC Grade- II. No history of hematemesis or malena. No history of abdominal distension, obstipation or vomiting. No history of dysphagia or regurgitation. No history of high colored urine, clay colored stools or generalized itching. No history of bone pain. No known medical comorbidities. No past history of any surgeries. History of loss of 5 kilograms of weight over the past 1month. History of loss of appetite for the past 1 month. No history of any substance abuse. Patient attained menarche at the age of 13 years. Regular menstrual cycles with normal flow- 3/30. Patient has one daughter, P1L1. Breast fed child her child for 1.5 years duration. No history of malignancy related deaths among family members.

On examination, patient was thin built and undernourished. Pallor was present. There were no signs of dehydration. Vitals were within normal limits. On examination of abdomen, there was mild tenderness in epigastric region. No palpable mass or positive shifting dullness. Per-rectal examination was normal.

Course in the Hospital,

On second day of admission while awaiting for imaging reports, patient developed sudden epigastric pain. Vitals-Tachycardia. No hypotension or desaturation. Patient also developed guarding and rigidity confined to upper abdomen. Blood investigation revealed anaemia (Hemoglobin -9 gm/dl). Packed red cell transfusion was started. Emergency X-ray erect abdomen was performed. Air under diaphragm/ crescent sign was present on Xray. Patient was immediately given fluid resuscitation with dextrose normal saline solution. Ryles tube and Foley's catheter was inserted and abdominal girth charting was done. She was prepared for exploratory laparotomy after emergency contrast enhanced scan of abdomen. CECT abdomen-intravenous contrast revealed extraluminal air pockets. An irregular circumferential wall thickening noted in the fundus, body and cardiac region of stomach. Evidence of multiple enlarged lymph nodes noted in the para-aortic, retrocaval and mesenteric region largest measuring 1.2 x 1.4 cm. Evidence of heterodense lesion of size 3.9 x 2.8cm noted in left adrenal -possibility of metastasis. Minimal free fluid in abdomen. Impression: Features suggestive of malignant growth of fundus, cardiac region and body of stomach with hollow viscus perforation. Mild ascites

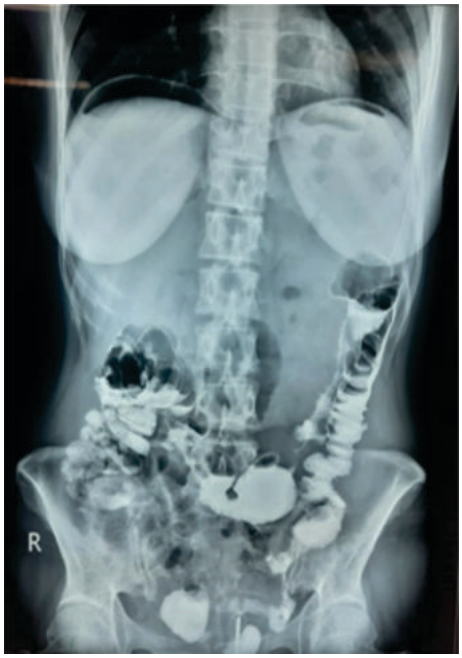

Fig 1: Xray erect abdomen showing crescent sign suggestive of hollow viscus perforation.

Hence, patient was taken for emergency laparotomy. Intraoperative findings- 0.5 x 0.5cm perforation about 5 cm below esophago-gastric junction near lesser curvature. Stomach was contracted and firm in consistency. Stomach was posteriorly adherent with the pancreas. About 200ml of ascitic fluid was aspirated and sent for cytology and culture sensitivity. Numerous omental deposits of size approximately 2x2cm were noted over greater omentum. Multiple serosal nodules of size approximately 1 x1cm were noted over transverse colon. Biopsy was taken from ulcer edge for histopathological confirmation of malignancy. Intraoperative features suggestive of malignant gastric perforation in a suspected case of metastatic carcinoma of stomach. Modified Graham's omental patch closure with Witzel feeding jejunostomy was performed.

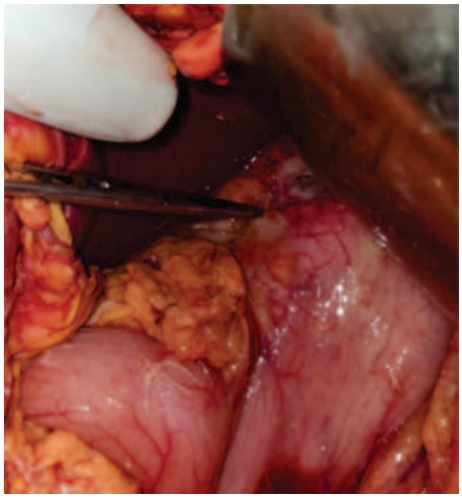

Fig 2: Intraoperative picture showing gastric perforation near OJ junction

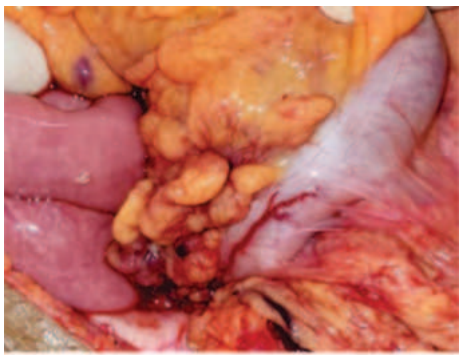

Fig 3: Intraoperative picture of numerous serosal nodules over transverse colon and omental deposits

Post Operative Course:
She was extubated after procedure and shifted to intensive care unit for monitoring. She was started on feeding jejunostomy feeds on the 3rd post operative day. Histopathology report revealed signet ring adenocarcinoma of stomach. She was started on palliative chemoradiation therapy and was discharged on the fifth postoperative day.

DISCUSSION

Linitis plastica was first described by Dr.Arthur Scout as a specific type of malignancy of stomach in the year 1953. On histopathological examination, there are few or scanty cellular infiltration of hypertrophic submucosal layer of stomach in the background of abundant filamentous strands and scar like tissue. Owing to the presence of these strands, it is referred to as linitis due to resemblance to linen. They are loosely classified as Borrmann type- IV cancer based on endoscopic examination. The characteristics of these class of tumors are extensive desmoplasia, predilection for younger females, increased propensity for vascular and lymphatic dissemination and low incidence of liver metastasis. Linitis plastica is also characterized by dyscohesive signet ring cells with propensity for submucosal spread. Histologically in line of Japanese classification these tumors are microscopically classified as scirrhous, medullary and intermediate types. The scirrhous type has a strong prediction for submucosal invasion and endoscopically appear as engorged gastric rugosities. The scirrhous nature of tumor is attributed to chemical interaction between neoplastic cells and CAF (cancer associated fibroblasts) via paracrine factors like TGF-beta, FGF7 and HGF [2].

Molecular Pathogenesis of Linitis Plastica

The three important pathways linked with the pathogenesis of linitis plastica are Hippo, KEGG and PI3KT-AKT pathways. The activation of ANGPTL4 oncogene by HIF-alpha (Hyopoxia inducible factor) secreted by tumor cells in response to hypoxia upregulates the genetic pathways that are responsible for tumor growth and lymphatic dissemination. CDH-1 is the most commonly involved mutation in linitis plastica. Methylation induced silencing of CDH-1 gene results in the deficiency of essential cell adhesion molecular E-cadherin. Micro RNA 143 expression via TGF-beta/ SMAD pathway is responsible for desmoplastic reaction and its increase is strongly correlated with peritoneal metastasis and invasion. The practical application of molecular pathogenesis lies in the development of novel therapeutic agents like tranilast (TGF-beta/ SMAD pathway inhibitor), bemarituzumab (monoclonal antibody against FGFR26) and linsitinib (CD44-IGF1R inhibitor) [3].

Endoscopic assessment is the gold standard for evaluation of linitis plastica. The two mucosal patterns are giant waffle type and flat type. The giant waffle type structurally resembles Menetrier's disease, gastric lymphoma and metastasis while second type can be mistaken for atrophic gastritis. Other associated findings are loss of gastric distensibility and involvement of more than one thirds of total gastric mucosa. Negative gastric biopsy owing to scanty malignant cells in specimen can pose diagnostic challenges [2]. Endoscopic ultrasound guided submucosal biopsies are valuable as gastric mucosa is uninvolved in many cases of linitis plastica [3]. Gastric cancers may present with locoregional spread or distant metastasis at the time of presentation. Distant lymph nodes, liver and peritoneum are common sites of distant spread. The pattern of distant metastasis is governed by the type of gastric carcinoma. Diffuse gastric cancer predominantly spreads to peritoneum, bone, colorectal tissues and female reproductive tract while intestinal type of gastric cancer has a propensity for hepatic metastasis [4].

Gastric perforation can occur spontaneously in the setting of acid peptic disease(most common cause), malignant perforation, gastric volvulus, drug induced (Non-steroidal anti-inflammatory drugs) and ischemia of stomach. Gastric ulcers also occur in traumatic setting with instrumentation, surgery, endoscopy or erosion by foreign bodies/ battery. The survival rate following the repair of perforation strongly depends on the size (size more than 10mm is associated with high risk of peritonitis and mortality) and etiology of perforation. Pneumoperitoneum is characterized by air under diaphragm, Rigler's sign and foot ball sign in plain Xray which is not present in all patients. The sensitivity of contrast enhanced CT scan is much higher. CT scans can visualize extraluminal air pockets, features of enterocolitis, ascites and can even help to predict the possible site of perforation. Open or laparoscopic surgical management is the gold standard for treating gastroduodenal perforation. Thorough peritoneal lavage and Cellan-Jones/Modified Graham's pedicled omentoplasty is commonly used to address perforated gastric ulcer. Integrity of defect closure is assessed by ryles tube insufflation test (Tire test). Conservative management is

adapted only in cases of poor functional status and sealed perforation. Non-operative management is associated with the risk of intra-abdominal abscess and risk of missing out malignant growth in absence of intraoperative ulcer edge biopsy. Adjunctive treatments include post-operative course of Anti-H pylori regimen and proton pump inhibitor therapy to prevent recurrence [5]. About 10-16% off gastric perforation is attributed to malignancy etiology. However, they are commonly diagnosed post-operatively with histopathological report. There are significant controversies in the management of perforated gastric cancers. There are also reports of curative oncological resection (Radical gastrectomy with D2/D3 lymphadenectomies) carried at the time of emergency laparotomy for perforation. This one staged approach is strictly reserved for patient with presumed early stage, good functional status, low intensity of peritonitis and absence of comorbidities. Otherwise primary omentoplasty and delayed gastric resection can be planned for the patient after proper staging workup. The viability of peritoneally spilled gastric canrcinoma cells are questionable in the presence of inflammation [6].

The management of uncomplicated linitis plastica is brief described as follows. Diagnostic laparoscopy at the start of surgery identifies occult peritoneal and hepatic surface metastasis and avoids unnecessary radical curative resection [4]. While 41% of diffuse gastric cancers are unresectable at the time of presentation, remaining patients are managed with curative or palliative approaches. Total or partial gastrectomy with negative margins combined with D2 lymphadenectomy is the standard of care in curative surgery. Splenectomy might be required to clear hilar nodes [3]. Newer treatment modalities include PIPAC (Pressurized intraperitoneal aerosolized chemotherapy) and HIPEC (Hyperthermic intraperitoneal chemotherapy) for peritoneal dissemination [4]. However, linistic plastica is associated with poor survival and increased postoperative mortality in comparison with intestinal type of gastric cancer [3]. Perioperative chemotherapy is highly beneficial for all types of gastric cancer. There are numerous studies that show curative/palliative surgical resection with adjuvant therapy can prolong life expectancy in comparison with simple observation for locally advanced diffuse type of gastric cancers [1].

CONCLUSION

This case report outlines the management of a rare presentation of diffuse gastric cancer in a young female. This also highlights the importance of anticipating these rare possibities to aid in better patient care. The controversies and different surgical options for the management of perforated gastric carcinoma are briefly elucidated.

Conflict Of Interest: None

Informed Consent: Informed consent was obtained for publication of case report and clinical photographs of the patient.

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