

Case Report

Carcinoma Stomach- Atypical Presentation.

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Abstract

Introduction: Most patients with gastric cancer are symptomatic. Weight loss and persistent abdominal pain are the most common symptoms at initial diagnosis. Approximately 25 percent of patients with gastric cancer have a history of gastric ulcer. Grossly thickened stomach has many differential diagnosis, including both benign and malignant and in latter adenocarcinomas are most common.

Case report: We present a young female of forty two years, not a known case of any chronic illness, who presented with early satiety, decreased oral intake, vomiting and weight loss for last eight months. All the symptoms were gradually progressive and were not relieved by symptomatic therapy by private practitioner. Her ultrasonogram revealed thickened stomach with suspicious lesion in liver which was confirmed on CT scan abdomen. Her upper gastro-intestinal endoscopy revealed grossly thickened stomach with gastric outlet obstruction and plenty of food residue even after 24 hours fasting. Her first endoscopic biopsy revealed lymphocytic gastritis but in view of strong suspicion, repeat endoscopic biopsy was done which revealed adenocarcinoma. The PET CT revealed malignancy limited mainly to stomach. The surgical oncologist consultation was taken and patient is being worked up for surgery followed by chemotherapy.

Conclusion: Proper treatment depends on right diagnosis but sometimes atypical presentations or findings of investigations can hinder timely diagnosis which can be countered by repeat testing, as was done in our case by performing repeat endoscopic biopsy of stomach.

Keywords: Endoscopy, Vomiting, Adenocarcinoma, Thickened Stomach, Weight loss

INTRODUCTION

Gastric cancer is the sixth most common cancer and the fourth most common cause of cancer-related death in the world [1]. Gastric cancer was once the second most common cancer in the world. In most developed countries, however, rates of stomach cancer have declined dramatically over the past half century due to widespread use of refrigeration, which led to increased consumption of fresh fruits and vegetables; decreased intake of salt, which had been used as a food preservative; and decreased contamination of food by carcinogenic compounds arising from the decay of unrefrigerated meat products. Salt and salted foods may damage the gastric mucosa, leading to inflammation and an associated increase in DNA synthesis and cell proliferation. Other factors likely contributing to the decline in stomach cancer rates include lower rates of chronic *Helicobacter pylori* infection, thanks to improved sanitation and use of antibiotics, and increased screening in some countries [2]. Gastric cancer remains difficult to cure, primarily because most patients present with advanced disease and even who present in the

most favorable condition and who undergo curative surgical resection often experience recurrent disease. However, advances in adjuvant therapy are resulting in improved survival [3]. Around 40% of cancers develop in the lower part, 40% in the middle part, and 15% in the upper part; 10% involve more than one part of the organ. The intestinal type of non-cardia gastric cancer is generally thought to arise from *Helicobacter pylori* infection, which initiates a sequence that progresses from chronic non-atrophic gastritis to atrophic gastritis, then intestinal metaplasia, and finally dysplasia. This progression is known as Correa's cascade. Gastric cancer may often be multifactorial, involving both inherited predisposition and environmental factors [4]. Environmental factors implicated in the development of gastric cancer include diet, *Helicobacter pylori* infection, Previous gastric surgery, Pernicious anaemia, Adenomatous polyps, Chronic atrophic gastritis, Radiation exposure and smoking. Worldwide, however, gastric cancer rates are about twice as high in men as in women [2]. The median age at gastric cancer diagnosis is 68 years; fewer than 2% of cases occur in persons younger than 35 years [5]. The gastric cancers that occur in younger patients may

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represent a more aggressive variant or may suggest a genetic predisposition to development of the disease. Early disease usually has no associated symptoms; however, as disease advances, then patient may develop indigestion, nausea or vomiting, dysphagia, postprandial fullness, loss of appetite, melena, hematemesis, weight loss. Late complications include pathologic peritoneal and pleural effusion, obstruction at gastric outlet, gastroesophageal junction, or small bowel, jaundice and cachexia [6].

CASE REPORT

We present a young female of forty two years, not a known case of any chronic illness, who presented with early satiety, decreased oral intake, vomiting and weight loss for last eight months. All the symptoms were gradually progressive and were not relieved by symptomatic therapy by private practitioner. In the beginning she started having early satiety for which she consulted some private doctor who treated him on line of gastritis with proton pump inhibitors and pro-kinetics but for no relief. Later on, her oral intake decreased and she started having vomiting in evening or night time and she lost ten kg of weight over eight months. The complete hemogram revealed normocytic normochromic anemia with hemoglobin of 10 mg%, total proteins were slightly low with 6 gm but rest all parameters including serum electrolytes, liver & renal function tests, blood sugar, thyroid profile,

viral screen, urine, electrocardiogram and chest x-ray were essentially normal. At this point of time ultrasonogram (USG) abdomen was done which revealed thickened stomach with multiple suspicious lesions in liver. Thus, for conforming ultrasonogram findings CECT scan abdomen was done which confirmed the findings of USG abdomen. Her first upper gastro-intestinal endoscopy revealed grossly thickened stomach with gastric outlet obstruction and plenty of food residue even after 24 hours fasting. The histopathological gastric biopsy revealed lymphocytic gastritis but in view of strong suspicion, repeat endoscopic biopsy was done which this time revealed adenocarcinoma. Now, for deciding about operability of the lesion, PET CT was done which showed mildly metabolically active heterogeneously contrast enhancing asymmetrical soft tissue density mural thickening showing differential hyperenhancement of thickened mucosal folds, involving distal body, antrum and pylorus of stomach with proximal dilatation of stomach and no significant perilesional extraluminal fatty stranding as described- likely primary malignant disease. Faintly metabolically active, few subcentemetric soft tissue density deposits in right infamesocolic region close to duodenum loop on right side- lymph node? Metastasis? No definite scan evidence of any other abnormal hyper metabolic disease focus elsewhere in body was noted. The surgical oncologist consultation was taken and patient is being worked up for surgery followed by chemotherapy.

Figure 1. CECT Scan Abdomen Showing Grossly Thickened Stomach (Arrow) & Liver Metastasis (Circles).

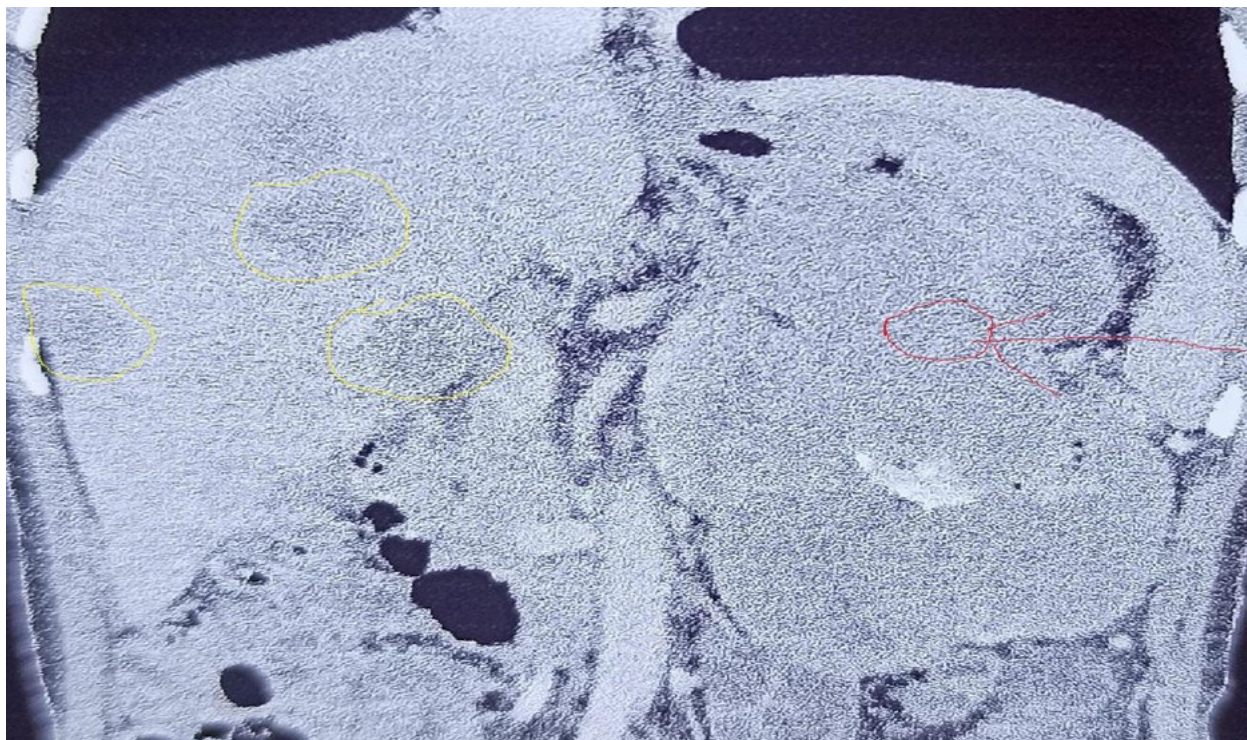
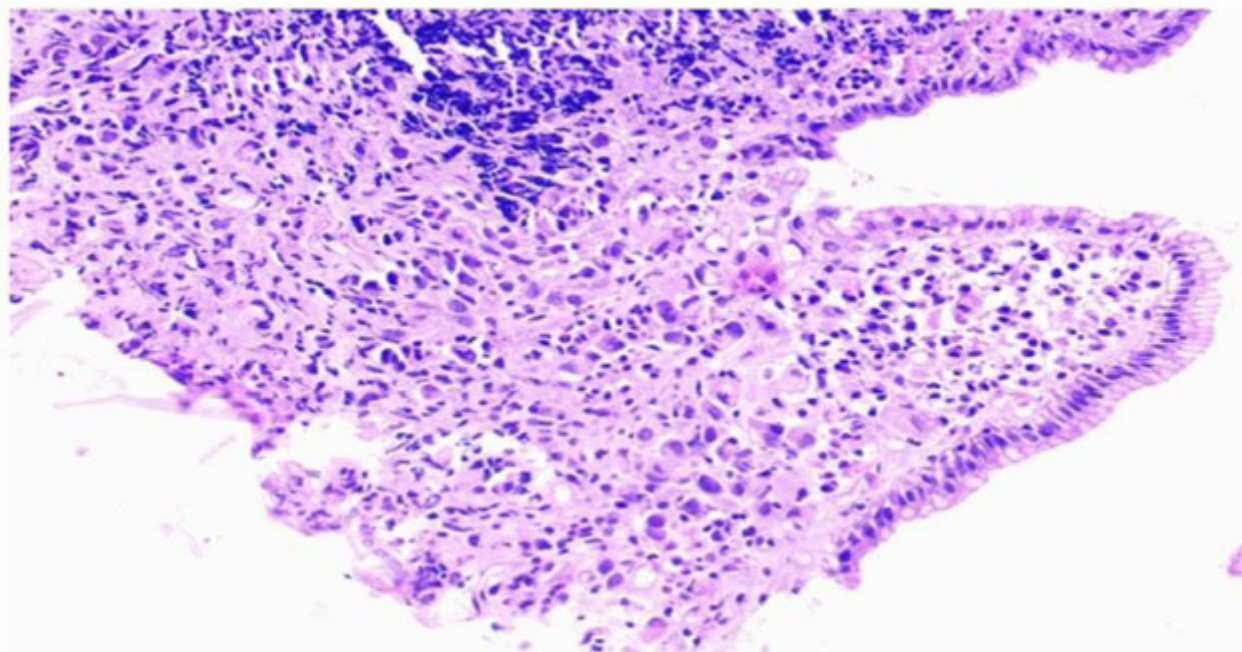


Figure 2. Histopathology Section Showing Adenocarcinoma Stomach

DISCUSSION

The differential diagnosis of a thickened stomach wall includes a wide range of both benign and malignant conditions. Benign causes include inflammatory conditions like gastritis, peptic ulcer disease, crohn's disease, and menetrier's disease, eosinophilic & granulomatous gastritis, tuberculosis, amyloidosis, zollinger ellison syndrome while malignant causes include gastric adenocarcinoma and lymphoma. Other less common causes include granulomatous gastritis, amyloidosis, and metastasis to the stomach. Gastric adenocarcinoma is a common cause of malignant thickening, which can be focal or diffuse (e.g., Borrmann type 4, also known as linitis plastica). Lymphoma can present as a focal, irregular mass or as a segmental/diffuse, symmetric thickening, sometimes with homogeneous enhancement. Gastrointestinal stromal tumours (GISTs) are less common but can cause a mass-like thickening. Metastatic Cancer from other primary sites can involve the stomach secondarily, causing wall thickening. In our case, there were many unexpected things like first of being a female, normally carcinoma stomach is predominantly seen in males. Second was age factor, normally carcinoma stomach is seen in old age but our patient was forty four-year-old. There were no risk factors, she was non-smoker, no family history or gastric symptoms or gastric surgery in past. There were no radiation exposure and she was non-obese. The first hint of malignancy of stomach came from ultrasonogram abdomen and CECT scan abdomen. The endoscopic finding of grossly thickened stomach with significant food residue, despite being prolonged fasting, pointed towards malignancy. The

first endoscopic stomach biopsy revealed just lymphocytic gastritis but in view of clinical presentations and radiological/endoscopic findings, a wise decision of repeat biopsy proved right. Hence, final diagnosis of adenocarcinoma stomach was made and PET CT scan also confirmed that disease was localized to stomach only. Normally, gastric adenocarcinomas do not present with such gross thickened mucosa except in linitis plastica.

CONCLUSION

Proper treatment depends on right diagnosis but sometimes atypical presentations or findings of investigations can hinder timely diagnosis which can be countered by repeat testing, as was done in our case by performing repeat endoscopic biopsy of stomach.

Conflict of Interest: Authors declare that there was no conflict of interest and no financial support was taken for publication of this case report and same was published after due consent from the patient.

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