Aggressive Gastric Adenocarcinoma after Eradication of Helicobacter Pylori: A Rare Case Report

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Abstract

Background

Gastric adenocarcinoma is closely linked to Helicobacter pylori infection. While early gastric cancer has a slow doubling time (577–3,462 days), late-stage disease progresses more rapidly (69–305 days).

Case Presentation

A 39-year-old non-smoker female presented with loss of appetite, vomiting, dysphagia, generalized weakness, and significant weight loss. Initial endoscopy was normal, but biopsy revealed H. pylori-associated gastritis, treated with triple therapy. Symptoms persisted for six months post-eradication. Imaging later showed a hard, nodular mass in the gastric corpus and body, confirmed as invasive diffuse adenocarcinoma. She underwent palliative total gastrectomy with Roux-en-Y esophagojejunostomy.

Conclusion

This case highlights a rare occurrence of rapidly progressive gastric carcinoma developing shortly after H. pylori eradication, emphasizing the need for close follow-up in symptomatic patients.

Keywords: *Advanced gastric cancer, Case report, Roux-en-Y esophagojejunostomy, Signet ring cell carcinoma.*

Introduction

Gastric adenocarcinomas (GA) are malignant tumors of the gastric mucosal glands. GA is the fifth most common carcinoma and ranks fourth in global cancer-related mortality[1]. While environmental and dietary factors contribute to its risk, Helicobacter pylori infection is the major cause, associated with 75% of gastric cancers worldwide[2]. On average, GA takes seven years to progress from mucosa to submucosa and an additional 2–3 years to become advanced cancer[3]. Although GA progression after H. pylori eradication has been reported, it remains rare and poorly understood.

We report a case of unusually rapid GA progression within 6 months of H. pylori eradication.

Case Report

A 39-year-old non-smoker female with no known comorbidities presented to the outpatient department with a six-month history of loss of appetite and vomiting, generalized body weakness for four months, and dysphagia for three months. Vomiting occurred immediately after intake of solid food and was neither blood-stained nor bile-stained. These symptoms were associated with significant unintentional weight loss

(from 58 kg to 52 kg). She also reported progressive dysphagia with a sensation of a foreign body in the throat. Additionally, she experienced melena three to four times during the past week.

There was no significant family history of similar symptoms. Six months earlier, she had visited another clinic where an upper gastrointestinal (GI) endoscopy revealed no abnormalities (Figure 1). However, biopsy from that visit showed Helicobacter pylori-induced chronic active gastritis. She was treated with a triple therapy regimen, but her symptoms persisted despite eradication.

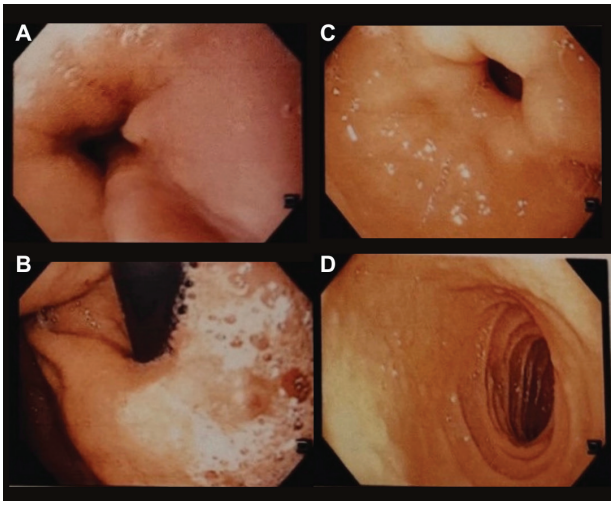

Figure 1. *Upper Gastrointestinal endoscopy showing (A) Gastroesophageal junction (B) Fundus (C) Antrum and (D) First part of duodenum with normal impressions of the GI tract six months ago.*

On her current presentation, the general examination revealed only pallor. Abdominal examination showed a palpable epigastric mass measuring 2 × 2 cm. Laboratory findings were within normal limits, except for microcytic anemia with a hemoglobin level of 8.0 g/dL. Helicobacter pylori testing from biopsy was negative. However, upper GI endoscopy revealed a hard, nodular mass over the corpus and body of the stomach, highly suggestive of malignancy (Figure 2). Biopsy and histopathology confirmed invasive diffuse adenocarcinoma, characterized by ill-defined glands, signet ring cells, and pleomorphic hyperchromatic nuclei (Figure 3). These findings were in stark contrast to the gastroduodenoscopy and biopsy results obtained six months earlier.

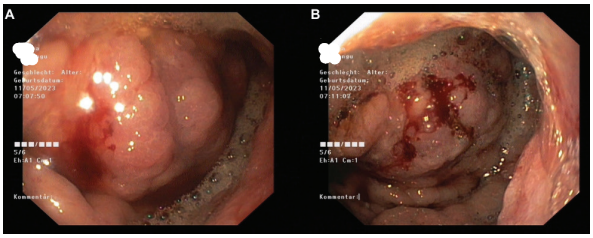

Figure 2. *Upper Gastrointestinal endoscopy showing hard nodular mass over (A) corpus and (B) body likely malignant.*

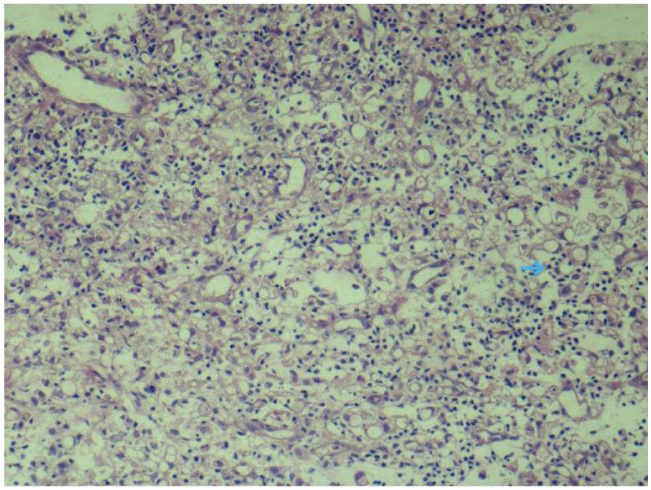

Figure 3. *Hematoxylin and eosin stain (20 X) showing invasive diffuse adenocarcinoma with ill defined glands, signet ring cells and pleomorphic hyperchromatic nuclei.*

An advanced endoscopic nasogastric tube was placed for enteral feeding purposes. Computed tomography (CT) scan found asymmetrical thickening of body and antrum of stomach with mass measuring 3.3 x 4.07 cm in lesser curvature at antrum. The mass was limited within gastric walls with ulceration and perigastric fats and locoregional lymphadenopathy with few homogeneously enhancing likely reactive left paraaortic lymph nodes from which metastasis can't be ruled out (Figure 4).

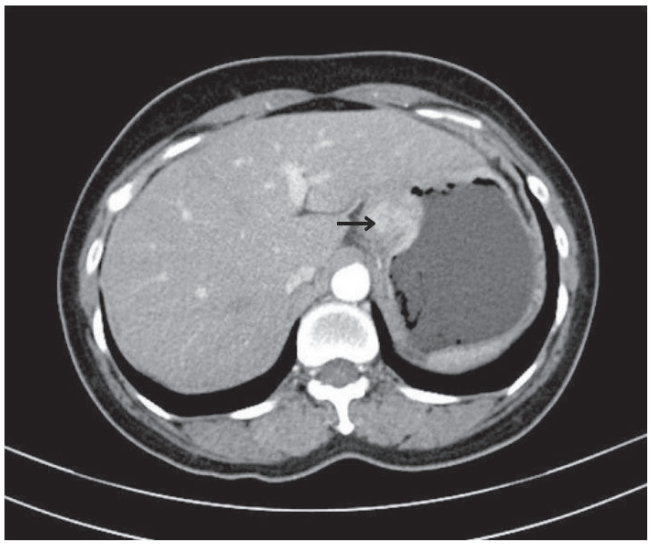

Figure 4. *Computed tomography (CT) scan showing asymmetrical thickening of body and antrum of stomach with locoregional lymphadenopathy.*

Diagnostic laparoscopy was performed to assess abdominal involvement and for surgical planning. It revealed omental deposits, but no parietal peritoneal deposits. Serosanguinous peritoneal fluid was collected and sent for cytological analysis. Intraoperatively, a hard mass was found at the lesser curvature of the stomach, extending proximally to the pylorus and 3 cm below the

gastroesophageal junction, involving the body of the stomach with serosal involvement. The posterior wall of the stomach with the mass was adherent to the superior border of the pancreas but was separable. No additional lesions were observed.

The patient underwent a palliative open total gastrectomy. The left gastric artery was ligated and divided, short gastric vessels were divided, and a Roux-en-Y esophagojejunostomy (end-to-side, antecolic) was performed. The patency of the anastomosis was confirmed intraoperatively. Pathological examination of the surgical specimen revealed a poorly differentiated, invasive diffuse adenocarcinoma. The patient remained stable postoperatively and during the one-month follow-up.

Discussion

The prevalence of gastric carcinoma among endoscopic patients in Nepal increased from 1.8% to 2.4% according to studies conducted in 2013 and 2021[4–5]. The antrum has been identified as the most common site, with chronic Helicobacter pylori infection being the principal cause of non-cardiac gastric cancer[6]. Epidemiological data suggest a male predominance, with a peak incidence in the 51–70 age group, accounting for more than 50% of cases, while patients below 40 years comprise only 6.2%[5].

Histopathologically, gastric cancers are classified into intestinal and diffuse types. The intestinal type is more commonly associated with chronic H. pylori infection, whereas the diffuse type is more prevalent in younger patients[7]. This case report presents a rare occurrence of diffuse-type adenocarcinoma in a young patient following H. pylori eradication.

In our case, the biopsy was negative for H. pylori following treatment with a triple regimen, and no abnormalities were seen on endoscopy. However, due to persistent symptoms, further investigations were performed after six months. CT imaging revealed a tumor involving the body and antrum, and histopathology confirmed invasive diffuse adenocarcinoma. A cohort study in Sweden reported a 0.2% prevalence of gastric carcinoma in patients post-H. pylori eradication[8]. Therefore, the development of the tumor in our patient, in the absence of family history and after eradication therapy, is extremely rare and of uncertain cause.

Time and tumor growth rate can help retrospectively estimate the timing of cancer onset and predict patient survival. However, growth rates vary significantly among individuals[9]. One study found that the risk of severe intestinal metaplasia and differentiated-type gastric cancer increases with age, suggesting it can take up to 10 years for cancer to develop through atrophic gastritis and intestinal metaplasia[3].

Several studies have also described the exponential growth nature of human cancers. Gastric cancer doubling times, assessed via X-ray imaging, ranged from 54 to 3,462 days. For early gastric cancer, doubling times are reported to be 577 to 3,462 days, while advanced gastric cancer ranges from 105 to 305 days[9]. In our case, the appearance of a large tumor within just 180 days after a normal endoscopy raises questions about the tumor's doubling rate and suggests unusually rapid progression.

Genetic testing and neoadjuvant chemotherapy were not performed in this case due to financial limitations, which could have contributed to improved diagnosis and prognosis. Further research is needed to explore the association between H. pylori and rapidly growing tumors in younger patients, along with more effective diagnostic approaches in such rare presentations.

CONCLUSION

Thus, unique growth of gastric adenocarcinoma after H. pylori eradication as presented in this case is a reminder of the ever evolving nature of cancer research and the need of clinicians to recommend long term surveillance for high risk patients and refine understanding for improvement of patient outcomes.

Consent

Written informed consent was obtained ensuring patient's anonymity.

Declaration of competing interest

There are no conflicts of interest.

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