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Clin Endosc 2024;57:697-699
https://doi.org/10.5946/ce.2024.121
pISSN: 2234-2400 • eISSN: 2234-2443

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CLINICAL ENDOSCOPY

A rare cause of progressive dysphagia

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Quiz

A 52-year-old man visited his primary care provider because of dysphagia that had persisted for three months. The symptoms was progressive and accompanied by epigastric pain. He lost 5 kg of weight over a 3-month period. The patient was diagnosed with hypertension and diabetes mellitus 2 or 3 years earlier but did not take his medication appropriately. He was a non-smoker, rarely drank alcohol, and had no significant family history of smoking. He had not undergone esophagogastroduodenoscopy (EGD) in the past ten years and did not report any abnormalities at that time. Initial EGD revealed multiple linear yellowish lesions in the distal esophagus that resembled subepithelial lesions. During examination of the distal esophagus, the esophagus did not expand well, and the endoscopist felt resistance during advancement of the endoscope. Therefore, the endoscopist withdrew the esophagogastroduodenoscope without examining the stomach and duodenum because of the risk of perforation.

The patient's vital signs were normal, and the physical examination yielded unremarkable findings. Laboratory tests revealed that the complete blood count, renal function test, and liver function test results were within normal ranges. A repeat EGD at our hospital revealed approximately 3 cm long, linear, slightly yellowish, protruding lesions between 37 cm from the upper incisor and the esophagogastric junction (Fig. 1).

What is the most likely diagnosis?

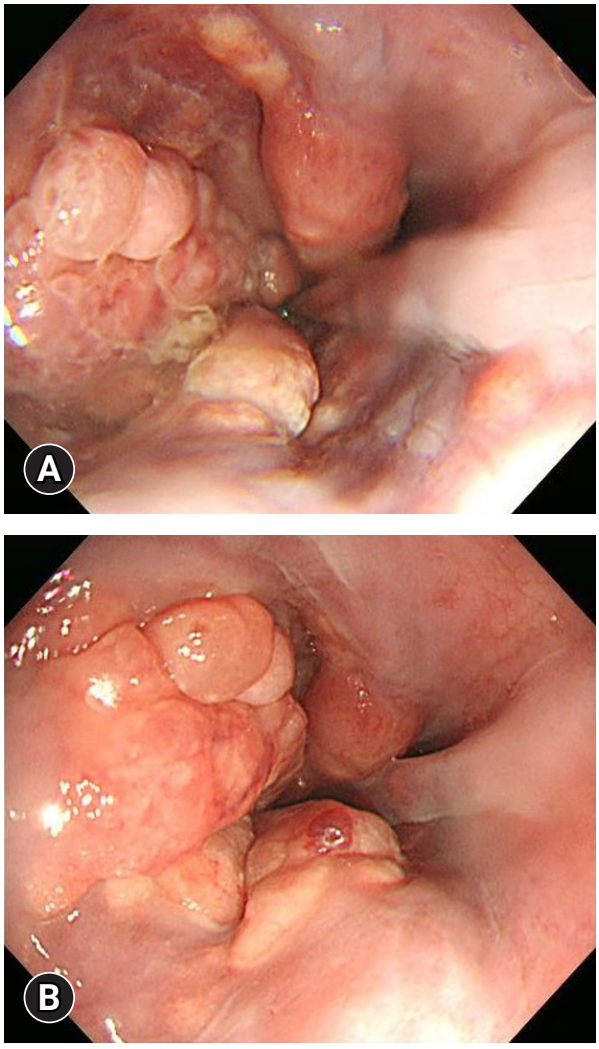

Fig. 1. Endoscopic findings of the esophageal lesions. (A) Not-expending esophagus. (B) Several linear, protruding lesions.

Received: May 13, 2024 **Revised:** May 23, 2024 **Accepted:** May 24, 2024

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Answer

Endoscopic findings of an esophageal lesion were suspected of malignancy and thought to be an infiltration of gastric cancer rather than of esophageal origin. Some resistance was observed in the lower esophagus. We attempted to insert the endoscope into the stomach. Diffuse, easily friable hyperemic thickened mucosal changes with hypertrophic gastric folds were observed at the whole body of the stomach, with a relatively preserved antrum (Fig. 2). The endoscopic diagnosis was gastric linitis plastica (LP) with esophageal infiltration. Gastric and esophageal biopsies were performed to confirm gastric cancer. Histological examination revealed adenocarcinoma with less glandular structure and poor cohesion in the gastric lesion, which was diagnosed as adenocarcinoma of the poorly cohesive type (Fig. 3A). Additionally, signet ring cell infiltration was observed in the esophageal lesion, which was diagnosed as a signet ring cell carcinoma (Fig. 3B). Computed tomography (CT) and F-18 fluoro-D-glucose positron emission tomography-CT (F-18 FDG PET-CT) were performed to determine the extent of gastric cancer. Gastric cancer with distal esophageal invasion with metastatic lymph nodes in the perigastric, common hepatic, and abdominal para-aortic spaces and bone metastasis at T9 and L4 were found on F-18 FDG PET-CT (Fig. 4). Based on these examination results, the patient was diagnosed with advanced stage IV gastric cancer.

LP is a rare type of gastric cancer, found in 7% to 10% of all primary gastric cancer cases, characterized by thickening and

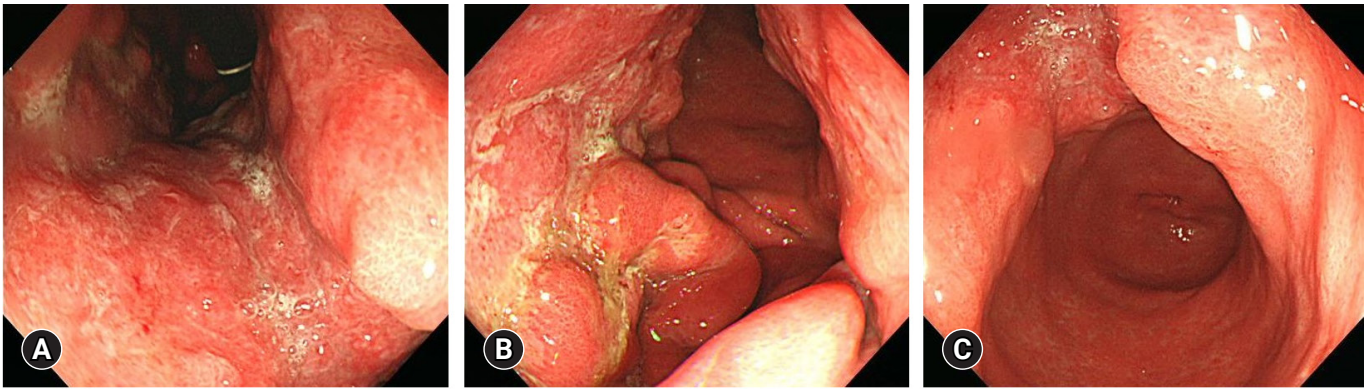

Fig. 2. Endoscopic findings of the stomach. (A) Diffuse wall thickening of the gastric body. (B) Hypertrophic gastric fold. (C) Relatively preserved gastric antrum.

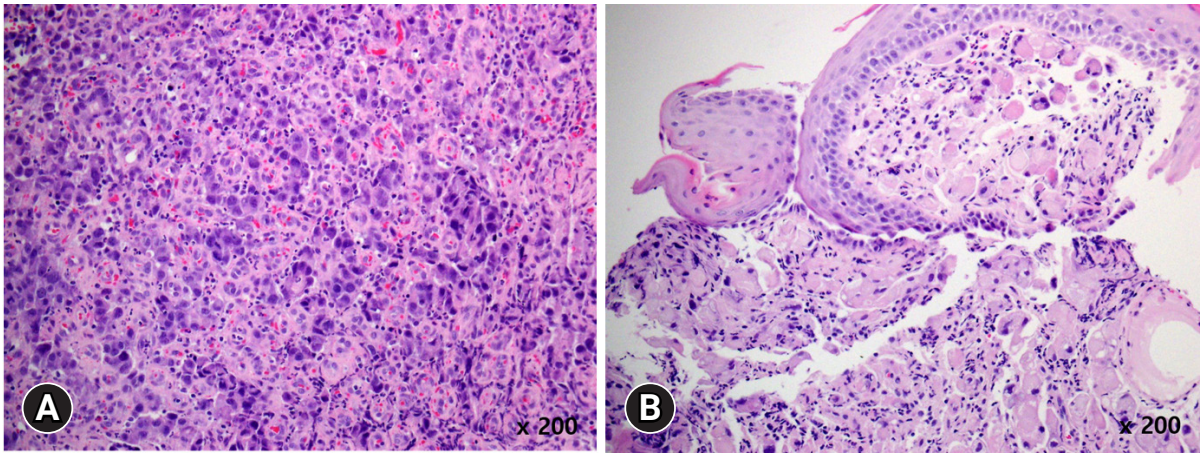

Fig. 3. Histologic findings. (A) Stomach: adenocarcinoma with less glandular structure and poor cohesion that represents poorly cohesive type adenocarcinoma (hematoxylin & eosin stain, ×200). (B) Esophagus: signet ring cell infiltration that represents signet ring cell carcinoma (hematoxylin & eosin stain, ×200).

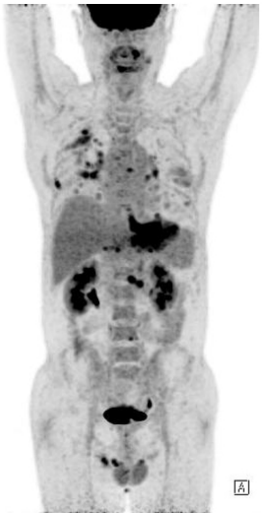

Fig. 4. Coronal view of F-18 fluorodeoxyglucose positron emission tomography-computed tomography. Diffuse hypermetabolic wall thickening is shown in the stomach with infiltration into the esophagogastric junction. Multiple hypermetabolic lymph nodes are shown in the perigastric, common hepatic, and abdominal para-aortic spaces. Additionally, focal lytic lesions are shown in the T9 and L4 vertebral bodies with hypermetabolism.

stiffness of the gastric wall due to diffuse tumor infiltration.[1] Some physicians use LP interchangeably with either Borrmann type 4 advanced gastric cancer or scirrhous adenocarcinoma. However, gastric LP differs from Borrmann type 4 advanced gastric cancer or scirrhous adenocarcinoma in terms of the primary site of involvement, gross findings, and clinical course.[2]

LP usually occurs in the upper body of the stomach rather than the gastric antrum, as in this patient, with the diffuse infiltration of tumor cells in the gastric submucosa and muscularis propria layers.[2,3] Therefore, the gastric wall is prominently rigid and thickened, with extensive hypertrophic gastric folds.[4] "Leather bottle" was a term used to describe the gross appearance of the LP, which had a limited expansion of gastric volume even when inflated.[5] Esophageal involvement in LP also occurs along the submucosal layer; however, it rarely presents as a prominent submucosal infiltrate, as in this patient.[6]

Patients with LP have a poor prognosis.[7] A recent National Cancer Database study reported that the mean overall survival for patients undergoing surgery with chemotherapy and/or radiation, surgery alone, chemotherapy and/or radiotherapy alone, and no treatment was 28.4, 17.1, 12.3, and 8.1 months, respectively.[8] The patient, in this case, received palliative chemotherapy. However, the disease progressed, and the patient died eight months after diagnosis.

Conflicts of Interest

Sung Eun Kim is currently serving as a KSGE Publication Committee member; however, she was not involved in peer reviewer selection, evaluation, or the decision process in this study. The other authors have no potential conflicts of interest.

Funding

None.

Author Contributions

Conceptualization: SEK; Data curation: JJ, SEK; Investigation: SEK, SJO; Visualization: SEK, SJO; Writing–original draft: JJ, SEK; Writing–review & editing: all authors.

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