Incidental detection of tubular esophageal duplication in gastric cardia malignancy

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Congenital esophageal duplications represent about 15% of digestive tract duplications. We report a 38-year-old man who presented with longstanding heartburn and recent dysphagia. Endoscopy showed communicating tubular duplication of the lower esophagus with ulceroproliferative growth at the gastric cardia extending into the lower esophagus. Histology of resected specimen showed poorly differentiated adenocarcinoma of gastric cardia without evidence of Barrett’s esophagus. [Indian J Gastroenterol 2004;23:192]

Key words: Barrett’s metaplasia, esophagus, gastroesophageal reflux

Fig: Endoscopy showing tubular duplication at lower esophagus

Duplication of the esophagus is the second most common (around 10%-15%) duplication of the gastrointestinal tract, after the ileum. They are of two types, cystic and tubular, the latter being less common. They are thought to develop in the third week of embryonic development because of failure of vacuoles to coalesce properly, preventing recanalization of the esophageal lumen. Most esophageal duplications are located in the lower third. A 38-year-old man presented with heartburn and retrosternal pain for 3 years. He had mild dysphagia to solids and liquids since 3 months. He also reported weight loss of 7 Kg in 3 months. There was no history of vomiting or GI bleed. He had no significant family history and was a non-smoker and non-alcoholic. Physical examination was unremarkable.

Routine hemotlogical and biochemical investigations were normal. Upper GI endoscopy showed two esophageal lumina starting from 24 cm (Fig). The endoscope could be passed into the stomach through both lumina but there was ulcerated and edematous mucosa in one of the duplicated segments. Rapid urease test for Helicobacter pylori was negative. Barium esophagogram showed two esophageal lumina in the lower esophagus, both emptying into the stomach. Biopsy of the edematous mucosa near the gastroesophageal junction showed poorly differentiated adenocarcinoma of gastric cardia without evidence of Barrett’s esophagus. CT scan of thorax and abdomen did not reveal local or distant spread.

The patient underwent radical esophagectomy. Operative specimen showed communicating tubular duplication of the gastroesophageal junction and there was an ulceroproliferative growth in the cardia. Histology of the specimen showed poorly differentiated adenocarcinoma of gastric cardia extending to one of the duplicated lumina. There was no evidence of lymph node spread or intestinal metaplasia in the lower esophagus.

He developed odynophagia 6 months later. Endoscopy showed a large ulcer near the anastomosis; biopsy showed no malignancy. He responded to proton pump inhibitor.

Tubular duplications may communicate at both ends or may be closed at one end. Our patient had longstanding heartburn probably due to gastroesophageal reﬂux. His gastroesophageal junction had two openings and a common distal esophageal wall, which led to poorly functioning anti-reflux mechanism.

Malignancy involving cystic duplication in gastric cardia malignancy in partial duplication of esophagus have been reported earlier. However, in our patient there was no evidence of esophageal malignancy or intestinal metaplasia. Thus duplication of esophagus was most likely an incidental finding in our patient.

References

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