Stricture Associated with “Inlet Patch”

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We report the case of a 75 year old gentleman who presented with a five year history of intermittent dysphagia and symptoms of “acid reflux”. He was found to have a stricture in the upper esophagus on endoscopy. Biopsy of the stricture revealed the presence of heterotopic gastric mucosa consistent with an inlet patch. After treatment with a proton pump inhibitor daily for 3 months, his symptoms of dysphagia improved. He did not require dilation. The reported incidence of inlet patches, their presentation, possible complications and need for surveillance in selected cases is reviewed in this case report.

INTRODUCTION

A n inlet patch is a velvety, orange-red or salmon-colored patch of tissue that represents a remnant of embryonic heterotrophic gastric mucosa. It is usually located in the upper third of the esophagus but heterotrophic gastric mucosa can occur at any site from the esophagus to rectum. Its size varies from 0.2 to 5 cm. Inlet patches are usually circumferential and can be flat, depressed or may have raised margins. They are most commonly located on the lateral and posterior surface of the esophagus.

The incidence of inlet patch has been reported to be anywhere from <0.1% to 10% in adults. The incidence even approaches up to 20% microscopically in pediatric autopsy reports as documented by Rector et al.

This congenital anomaly of the esophagus is usually asymptomatic and incidentally found on upper endoscopy. In certain instances however, it can result in serious complications including ulceration, bleeding and perforation. In some cases it is also the cause of chronic cough and vocal cord dysfunction. Rare cases have been reported of adenocarcinomas and esophageal web and stricture formations associated with inlet patches.

CASE STUDY

R.V. was a 75 year old Caucasian gentleman who presented with a 5 year history of intermittent dysphagia and “acid reflux” symptoms. He reported dysphagia to both solids and liquids. His reflux symptoms included burning and postprandial epigastric pain. He denied history of nausea, vomiting, weight loss or melena. His symptoms were partially relieved by over-the-counter antacids. His past medical history was significant for hypertension, hyperlipidemia and carpal tunnel syndrome. He was undergoing radiation therapy for prostate cancer and had a remote history of tonsillectomy and adenoidectomy. He used alcohol on social occasions and denied cigarette and illicit drug use. His medications included beta-blockers, angiotensin-converting enzyme inhibitors and bisacodyl PRN for constipation.
Esophagastroduodenoscopy (EGD) demonstrated a mucosal ring/stricture in the upper esophagus associated with an orange-red mucosal patch with erosion (see Figures 1 and 2). These findings were located approximately 22 cm from the superior incisors. Biopsy specimens from the stricture and surrounding mucosa were obtained; histopathology was consistent with inlet patch.

The stricture (see Figures 1 and 2) appeared edematous and friable but allowed easy passage of the endoscope. A patch of salmon-colored mucosa was also visible adjacent to the stricture.

No dilation was considered necessary at the time of endoscopy. The patient was started on a trial of medical therapy with esomeprazole 40 mg daily. He demonstrated dramatic improvement of reflux and dysphagia symptoms over the course of three months. Follow-up EGD that was performed after twelve months demonstrated inlet patch with healing erosion (see Figure 4).

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DISCUSSION

Inlet patches are rare entities that are usually asymptomatic and benign. The presence of columnar-lined mucosa in the proximal esophagus is usually considered to be congenital. In the embryo, differentiation of the esophagus starts as stratified columnar epithelium which is bidirectionally replaced by stratified squamous epithelium in the mid-esophagus in a cephalo-caudal direction. Incomplete development of the squamous mucosa could isolate an area of columnar mucosa in the proximal esophagus.

According to a study conducted by Tang et al., inlet patches with parietal cells are a more common histologic type compared to others, it is the presence of these acid-producing cells in the esophagus that lead to ulceration. The healing process of ulceration can lead to fibrosis which subsequently develops into strictures or mucosal ring formations. To the best of our knowledge, there have been only four cases reported in the literature of stricture formation with inlet patches.

Continuous swallowing of alkaline saliva has a neutralizing effect on the acidic pH produced by the heterotopic gastric mucosa of the inlet patch, thus lessening the severity of symptoms as in the case of our patient. If symptoms and stricture formation are mild, patients can respond to conservative medical therapy. Depending on their severity, these ulcers and strictures can lead to symptoms ranging from mild heartburn to severe dysphagia. For dysphagia that persists despite medication, esophageal dilatation is necessary.

We recommend consideration of inlet patch in the differential of esophageal strictures of unclear etiology. We also recommend that any incidentally found inlet patch on endoscopy if thought to be causing the symptoms should be biopsied to identify the presence or absence of parietal cells because the presence of parietal cells are more likely to cause complications such as ulceration, bleeding, perforation and stricture formation. Some inlet patches are associated with development of adenocarcinoma of esophagus necessitating regular follow-up and evaluation.

Because malignant transformation of heterotrophic gastric mucosa (HGM) to adenocarcinoma is an exceedingly rare event, prior researchers have suggested regular endoscopic surveillance of HGM carriers is not indicated. However, other studies have found the presence of gastroesophageal reflux disease to be a common feature between adenocarcinomas of the upper esophagus arising from HGM and those arising from Barrett’s mucosa. Therefore, we recommend screening for “symptomatic HGM” at the same interval as that recommended for Barrett’s esophagus by the latest American College of Gastroenterology guidelines. After documenting two EGDs with biopsy negative for dysplasia, patients should undergo surveillance EGD with biopsy every three years.

References

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