Obstruction of the Esophagus by Diseased Ectopic Gastric Mucosa

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In the past decade, advances in anesthesiology, antibiotics, pre- and post-operative care and surgical technique have greatly increased the scope of thoracic surgery. Among those profiting by these advances, is the individual suffering from esophageal obstruction.

Recently reported benign lesions producing esophageal obstruction include acalasia, chemical obstruction, congenital obstruction, webs, congenital strictures, perforation (spontaneous and traumatic) and fistulae. It is our purpose to present two cases of esophageal obstruction due to diseased ectopic gastric mucosa and to emphasize the desirability of carefully evaluating the cause of obstructive lesions in order to remove the possibility of overlooking a non-malignant lesion. Inasmuch as diagnosis of such lesions today is often based upon direct visualization by esophagoscopy and by barium studies, it is possible to believe that some so-called malignancies of the esophagus are in truth quite benign and amenable to surgical correction. It has been the experience of many thoracic surgeons and pathologists that repeated attempts at biopsy of such lesions have failed to prove the presence of a cancer. In such instances, the clinical diagnosis of carcinoma, although unsupported, often prevails and is accompanied by unjustified pessimism. On the basis of the following two cases it is believed that a more detailed study of possible benign lesions of the esophagus is mandatory.

Ectopic gastric mucosa is not an infrequent finding. It has been reported in the upper third of the esophagus, the lower third of the esophagus, within gastric cysts of the mediastinum, chronically inflamed gall bladders, cysts of the pancreas, Meckel's diverticula, chronic tuberculous ulcers of the colon and in teratomas. These islands of gastric mucosa, no matter where they may occur, are subject to all the diseases and malformations to which gastric mucosa is heir. These include acute and chronic inflammations, ulcerations, polypoid hyperplasias, and hypertrophies. When symptoms arise from such ectopic foci, they usually are due to ulceration of the focus with subsequent hemorrhage and/or pain. In each of the cases presented here, however, the primary symptom was esophageal obstruction which later proved to be caused by ectopic foci of gastric mucosa which had become diseased.

Case 1: A 45 year old white female entered the hospital because of vomiting and dysphagia which had its onset six months prior to admission and was

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ac companied by a sensation of food lodging in the lower esophagus. This condition became progressively worse until two weeks prior to admission, at which time two attempts at esophageal dilatation were made. These afforded some relief but on the day prior to admission she became much worse, was nauseated, vomited and could retain only fluids.

History and physical examination were essentially negative. She showed no evidence of weight loss. Routine laboratory studies were normal. Esophagograms showed a sharply outlined obstruction at the junction of the middle and lower thirds of the esophagus (Figure 1). On November 1, 1951 esophagoscopy was performed and an obstructing lesion was seen 30 cm. from the gum margin. A biopsy was obtained and bougies were passed in an attempt to dilate the obstruction. The pathologic diagnosis of the biopsy specimen was esophagitis, ulcerative, severe. The inflammatory reaction was so severe that the tissue pattern was destroyed and much of the specimen consisted merely of fibrino-purulent exudate. No specific epithelium was identifiable.

The pathologist in his report made a supplementary comment that from the

![FIGURE 1 (Case 1): Esophagograms demonstrating an esophageal obstruction at the junction of the middle and lower thirds of the esophagus.](image)
microscopic appearance the lesion might possibly be ectopic nests of gastric mucosa. On November 6, 1951 the endoscopic examination was repeated, and a second biopsy was obtained (Figure 2). This was reported as, “1. Gastric mucosa, ectopic, polypoid, of the esophagus. 2. Esophagitis, chronic, ulcerative, severe. 3. Esophagitis acute, focal, severe.”

With each of the endoscopies, attempts had been made to dilate the esophagus, but these had not been successful. An exploratory thoracotomy was advised.

On December 7, 1951 with the patient in the conventional left lateral position, the thorax was opened. After the rib spreader had been placed, the pulmonary ligament was dissected up to the level of the inferior pulmonary vein. The mediastinal pleura was then opened and the esophagus mobilized. The wall of the lower 8 cm. of the esophagus was found to be markedly thickened and adherent to the surrounding structures. After freeing up the involved area, the esophagus was incised in a longitudinal direction. The lumen was completely obstructed. A biopsy of the entire thickness of the wall was taken, and submitted for frozen section. The report on the biopsy was as follows, “Esophagitis chronic and acute, ulcerative, severe. Hyperplasia polypoid, adenomatous, of ectopic gastric mucosa within the esophagus.” The diaphragm was then opened, and the lower segment of the esophagus was resected. Examination disclosed characteristic gastric mucosa (Figure 3). An anastomosis between the fundus and the esophagus was carried out without difficulty. Further details of the operation technique have been omitted because they have become standardized. Her post-operative course was uneventful.

Case 2: A 54 year old white female experienced difficulty in swallowing for six months. This had its onset one month following cholecystectomy. The dysphagia consisted of an uncomfortable low substernal sensation on swallowing solid food. This was relieved by liquids. These symptoms persisted and progressed to the point where only liquids could be taken. There had been a 30 pound weight loss, during this period. Routine laboratory studies were essentially negative. Esophagograms revealed an obstructing lesion of the esophagus in the lower third (Figure 4).

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**FIGURE 2**

*Figure 2 (Case 1):* Photomicrograph (x 100) taken of second esophageal biopsy, showing typical gastric mucosa with adjacent nest of squamous epithelium.

**FIGURE 3**

*Figure 3 (Case 1):* Photomicrograph (x 100) taken of resected segment of esophagus.
FIGURE 4 (Case 2): Esophagogram showing persistent filling defect and obstruction of the lower third of the esophagus.

FIGURE 5 (Case 2): Gross specimen. Resected portion of esophagus showing polypoid, hemorrhagic gastric mucosa.
FIGURE 6: Photomicrograph (×50) of esophageal biopsy. In this instance the entire tissue is partially solidified and disintegrating. Outlines of tubular structures can be seen, but the characteristic squamous epithelium is no longer present. No doubt, accounts for the erroneous diagnosis of chronic peptic ulceration in the absence of peptic ulcer disease. 

FIGURE 7: Photomicrograph (×50) showing transition from normal esophageal epithelium to ectopic pyloric gland type muccosa.
There was no evidence of congenitally short esophagus or of a hiatus hernia in either case.

On November 9, 1951 diagnostic esophagoscopy was carried out. There were accumulated secretions all the way down. After aspirating the secretions, a brief view was obtained of what appeared to be a tumor mass, but contact of this area with the suction tip produced severe bleeding. The bleeding continued and the lesion could not be revisualized. On November 13, 1951 esophagoscopy was repeated and this time the lesion was well visualized. It had the appearance of a very injected polyp (Figure 5). Biopsy was obtained which was reported as follows, "Hemangioma of the esophagus" (Figure 6). An exploratory thoracotomy was advised.

She was prepared in the usual fashion and on December 6, 1951 thoracotomy was performed as described in the previous case. After mobilizing the esophagus, an area similar in length to that of the previous case was encountered. Again the esophagus was opened longitudinally and complete obstruction was present (Figure 7). Biopsy and frozen section revealed "ectopic gastric mucosa." This was subsequently confirmed on the permanent section (Figure 8). Resection and anastomosis was carried out as in the previous case (Figure 5). The immediate post-operative course was uneventful. Eight weeks post-operatively she had difficulty swallowing solid foods. She was again esophagoscopyed. The site of the anastomosis appeared normal but somewhat narrowed. This was easily dilated. Subsequent dilatation was required on one other occasion.

Both cases are now doing well.

Discussion

In the first case, the lesion covered a large area showing peptic ulceration, cicatrization, and stenosis. In some places gastric mucosa was preserved but in other areas the ulceration had completely destroyed the usual mucosal structures. This process can be so extensive as to obliterate completely all semblance of the gastric mucosa. By virtue of the histopathologic processes, however, it is possible in many instances to consider ectopic gastric mucosa as an underlying factor in so-called widespread ulcerative, necrotizing esophagitis. Recently we have had a third case which was initially thought to be due to gastric mucosa. However, following resection of the stenosed area, it was found that the ulceration involved the entire surface and that it was no longer possible to identify the epithelial structures.

In the second case, the obstruction was due to polypoid hyperplasia of gastric mucosa. It is, by analogy, reasonable to suppose that within such islands of gastric mucosa, changes could occur similar to those within the stomach or Meckel's diverticula. The mechanical obstructing component of the disease in the esophagus, however, is far more important than in either of the lower locations.

Inasmuch as these lesions will mimic malignancy by virtue of their clinical signs of obstruction, weight loss, pain, bleeding, and x-ray patterns, we feel that the surgeon should possess conclusive proof of the essential malignancy of the lesion before he admits defeat. In the absence of positive histologic evidence of cancer, repeated attempts to establish a precise diagnosis should be made. If, after reasonable attempts, a diagnosis has not been established, the patient should be given the benefit of an exploratory thoracotomy.
SUMMARY

1) Two cases of esophageal obstruction due to diseased ectopic gastric mucosa are reported.

2) On the basis of these and similar cases, it is urged that the obstructive lesions of the esophagus should never be classified as malignant on a basis of clinical, roentgen, and endoscopic appearances alone. If definite histologic proof is not otherwise available, the patient should receive the benefit of surgical exploration.

RESUMEN

1) Se refieren dos casos de obstrucción del esófago debida a mucosa gástrica ectópica.

2) Sobre la base de estos y de casos similares es preciso que las lesiones obstructivas del esófago no sean clasificadas como malignas apoyándose en las apariencias clínica, radiológica y endoscópicas solamente. Si no se obtiene una prueba histológica concluyente el enfermo debe beneficiarse de una exploración quirúrgica.

RESUME

1) L'auteur rapporte deux cas d'obstruction oesophagienne dus à une ectopie pathologique de la muqueuse gastrique.

2) En se basant sur ces deux cas, et sur d'autres similaires, il est de première importance de savoir que le diagnostic de cancer ne doit pas être fait en présence d'obstruction oesophagienne, sur les seuls caractères clinique, radiologique ou endoscopique. Si par ailleurs, la preuve histologique ne peut être apportée d'une façon valable, il ne faut pas hésiter à faire bénéficier le malade de la chirurgie exploratrice.

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