

Oesophageal stricture: a complication of peptic ulcer

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Benign strictures of the oesophagus associated with hiatus hernia do not always follow the same pattern. Some patients have symptoms of reflux oesophagitis for years before slowly developing a stricture: others present with a sudden onset of dysphagia. The reason for this has not been explained, and the role of a true peptic ulcer as opposed to peptic oesophagitis in the aetiology of stricture has rarely been mentioned. It has been accepted that reflux oesophagitis produces benign strictures at or below the level in the gullet at which the mucous membrane changes from squamous to columnar.

Four cases I have seen in the last 18 months are of some interest since they indicate that strictures of the lower oesophagus may sometimes be caused by peptic ulcers. As none of the ulcers was resected the full histological pattern was not shown, but they occurred in patients with oesophageal reflux and behaved like peptic ulcers elsewhere. One of them was palpated at operation and felt like an ordinary gastric or duodenal ulcer, the surrounding oesophagus feeling quite normal. I have therefore referred to them as peptic ulcers in the lower oesophagus. The following is a summary of the case histories.

CASE REPORTS

CASE 1 A high-grade mentally defective man aged 67 was admitted to hospital on 28 January 1964 after a severe haematemesis. He had no previous history of heartburn or dysphagia. He was shocked and anaemic; the haemoglobin was 29%. A barium swallow and meal was reported as showing a moderate-sized hernia with coarse mucosal folds at the lower end of the oesophagus, but no stenosis was present. Oesophagoscopy on 2 February 1964 showed oesophagitis with considerable bleeding from the mucosa and no stenosis. It was thought that this was insufficient to account for the massive haemorrhage, and a laparotomy was carried out. No ulcer was present in the stomach or duodenum; this was confirmed by gastrotomy. A large hiatus hernia was present. While this was repaired, an ulcer on the right posterolateral wall of the oesophagus about 1.5 cm. in diameter was palpated. A vagotomy and pyloroplasty was performed as well as a repair of the hiatus

hernia. He made an uneventful recovery, and on 25 February 1964 a further oesophagoscopy was carried out. The oesophagitis was much improved and an ulcer with raised edges was seen. The edge of the ulcer was biopsied; it showed a moderately severe chronic inflammatory cell infiltration. There was no evidence of neoplasia. He was discharged, eating well, on 27 February 1964. He was next seen on 5 May 1964 complaining of vomiting and an inability to swallow for two to three weeks. A barium swallow showed a tight stricture near the lower end of the oesophagus with foreign bodies retained in the oesophagus above it. He was admitted dehydrated and ill. An oesophagoscopy was carried out. The foreign bodies, a halfpenny and five buttons, were removed. At 40 cm. there was a tight stricture which was dilated with difficulty.

CASE 2 A man aged 64 was seen on 8 June 1964 with dysphagia, having been referred by a colleague. He had had epigastric pain half an hour after meals for about one year. He had a brisk haematemesis on 25 December 1963, which was treated with bed-rest, diet, and oral iron. A barium meal on 12 March 1964 had shown a small hiatus hernia with gastro-oesophageal reflux. No stricture was noted at the time, but on later review it appears that there may have been minimal narrowing present above the hernia. An oesophagoscopy on 7 July 1964 showed oesophagitis and a stricture with granulation tissue. A biopsy was taken of this tissue and a further barium swallow was arranged. The biopsy showed chronic inflammation only. The barium swallow showed that there was a tight stricture with an ulcer crater in the stricture. The duodenal cap was also deformed.

CASE 3 A woman aged 72 was seen on 20 July 1964, having been referred by a colleague on account of dysphagia. She had been admitted to hospital on 20 February 1964, having vomited a cupful of bright red blood for the first time in her life that morning. She had had epigastric pain after meals for five years and occasional heartburn, but had had none for the past three months. She was not shocked. The haemoglobin was 69%. A barium meal on 29 February 1964 showed a moderate-sized hiatus hernia with free reflux and no stenosis. She made a good recovery on a gastric diet with alkalis, and her haemoglobin level rose steadily. She remained well until May 1964, when she complained of dysphagia for the first time. She

had no pain or heartburn. A barium swallow and meal on 5 May 1964 showed a lower oesophageal stricture with a probable ulcer crater. Reflux could not be produced. An oesophagoscopy was carried out; this showed a tight organic stricture at 39 cm. which could with difficulty be dilated to size 19 bougie; there was little oesophagitis above it.

CASE 4 A man aged 64 was seen on 31 December 1964, having had a haematemesis earlier in the day. He admitted to indigestion and heartburn for many years, and complained that food had been sticking at the lower end of the sternum for three months. On examination he was not shocked nor anaemic. He had evidently had only a small haematemesis. No further bleeding occurred. A barium swallow on 13 January 1965 was reported on as follows: 'There is a large hiatus hernia with an oesophageal peptic ulcer and a peptic stricture. No other abnormality is detected in the oesophagus, stomach or duodenal cap or loop.' On 18 January 1965 he complained that he could not swallow solids at all. He was advised to enter hospital for oesophagoscopy, but he delayed this for personal reasons until 18 March 1965. At that time he stated that his heartburn was unchanged in severity but his swallowing was much improved and he could then swallow solids. Oesophagoscopy on 19 March 1965 showed mild oesophagitis and a healing ulcer posteriorly just below the limit of the squamous epithelium. There was mild stenosis. Gum elastic bougies up to size 17 passed easily. The hernia was repaired and a vagotomy and pyloroplasty was carried out. On 31 March 1965 he said he was eating better than he had done for years.

DISCUSSION

The first three cases have the following points in common: an absence of previous symptoms of dysphagia; a sudden haemorrhage, presumably from a peptic ulcer, shown later in each case; a hiatus hernia without any stricture on a barium examination at the time of the haemorrhage but the development within a few months of a stricture of sufficient severity to make the swallowing of solids difficult or impossible.

Until recently I had thought that a true peptic ulcer at the lower end of the oesophagus was rare, but perhaps it is just a question of not seeing them easily at oesophagoscopy when there is concomitant oesophagitis. I have had a number of patients referred to me in the last two years who have been admitted to hospital with haematemesis. Barium examination has shown only a hiatus hernia with no ulcer in the oesophagus, stomach or duodenum. I have carried out an oesophagoscopy, usually a few weeks later, and have seen only a mild oesophagitis. An acute gastric erosion has usually been postulated as the cause of the

haemorrhage. This view was held for case 1 until the ulcer was palpated at laparotomy. Perhaps some of the others in fact bled from oesophageal ulcers which were not detected on barium examination, as is sometimes the case with gastric ulcers.

Oesophageal ulcers frequently seem to straddle the junction between the squamous and columnar epithelium as stomal ulcers do with the gastric and jejunal mucosa. When ulcers occur at this level in the oesophagus, there seems to be no reason why they should not follow the same course as peptic ulcers elsewhere and have the same complications. Acute massive haemorrhage occurs at this site just as in other sites. Stenosis is a common sequel to pyloric ulceration and also follows a gastric ulcer at times. It was a sequel to an oesophageal ulcer in the first three cases described. In the first two cases, if haemorrhage had not occurred, the onset of dysphagia would have been the first symptom drawing attention to the oesophagus when the stenosis developed. With a duodenal ulcer, a pyloric stenosis can occur as a result of congestion and oedema, which then resolves as the ulcer heals. This sequence of events was closely imitated in the oesophagus in case 4, although resolution of the stenosis was not complete at the time of surgery. Gastric ulcers sometimes undergo malignant change. Adenocarcinoma occurs in association with hiatus hernia, and it seems possible that this could be due to malignant change in a peptic ulcer. Peptic ulcers in the oesophagus perforate at times just as they do elsewhere.

These four cases have suggested that peptic ulcers of the oesophagus may play a greater part in the pathology of the lower oesophagus than has previously been considered. They indicate that an oesophageal stricture can develop rapidly following a peptic ulcer and that this may be the aetiology of those strictures which present with dysphagia without appreciable symptoms of previous oesophagitis.

SUMMARY

Four cases of haematemesis are presented with peptic ulcers just above a hiatus hernia. Oesophageal stenosis was a sequel in each case. It is postulated that a rapid onset of dysphagia is sometimes due to a peptic ulcer of the oesophagus. It is also indicated that peptic ulcers of the oesophagus behave like other peptic ulcers and may be liable to all the complications that occur in other sites. They may also be more common than is generally realized.