TWO CASES OF BENIGN STRICTURE OF OESOPHAGUS AFTER GASTRECTOMY FOR PYLORIC STENOSIS

BY

DAVID W. HURST, M.D.

Surgical Registrar, Oldchurch Hospital, Romford

It is known that benign oesophageal stricture may occur as a complication of upper abdominal surgery, particularly the surgery of peptic ulcer. Benedict (1960) reviewed 1,938 oesophagoscopies and reported nine cases of benign inflammatory stenosis after total gastrectomy for carcinoma of the stomach and 12 after surgery for duodenal ulcer (subtotal gastrectomy in 10, repair of perforation in two). In a miscellaneous group it occurred once each after pyloroplasty, proximal partial gastrectomy, oesophagectomy for oesophagitis and stenosis, partial gastrectomy for gastritis, revision of posterior gastro-enterostomy and vagotomy, and several other related procedures. He also noted one case after subtotal gastrectomy for pyloric obstruction, and one after first-stage gastrectomy for pyloric obstruction.

On reviewing the literature, very few accounts of benign stricture of the oesophagus after uncomplicated gastrectomy can be found in which the cardiac sphincter mechanism is left reasonably intact. Benedict's two cases have been mentioned. McKeown (1958) described two cases, and listed a similar case reported by Straus in 1950. A few more cases have been reported, but have usually followed upon severe post-operative problems such as distal loop obstruction. Within a period of one month recently, two such cases were seen in a surgical unit where a large number of gastrectomies are performed annually; such a case has not been encountered before. Such an event deserves comment and an effort to consider the aetiology.

Case 1

A 40-year-old housewife first came for treatment on September 16, 1960, complaining of postprandial upper abdominal pain of five years' duration. It usually occurred about 30 minutes after eating and was relieved by alkali. For almost five years she had vomited a small amount at

least once daily. Recently she had begun to notice retrosternal burning pain after vomiting. A barium-meal examination on September 23 showed gross pyloric stenosis but no evidence of oesophageal abnormality (Fig. 1). Laparotomy on October 5 confirmed the x-ray findings, and the cause was a duodenal ulcer on the anterior wall. A standard three-quarters anterior Polya-Hofmeister gastrectomy was performed. The post-operative course was excellent and she went home on the tenth day.

When first seen at follow-up clinic she stated that, almost immediately after leaving hospital, food began to stick high up in the gullet. This was thought to be functional and she was reassured. Five days later she was

admitted as an emergency case, being unable to swallow even fluids. On November 22 a barium swallow revealed a simple stricture 5 in. (12.5 cm.) long and situated at the junction of the middle and lower third of the oesophagus (Fig. 2). Oesophagoscopy revealed much debris and inflammation of the upper oesophagus. Bougienage was unsuccessful, but two days later she swallowed a bougie herself (No. 8 English).

She has continued to pass bougies as an out-patient, gradually increasing the calibre up to No. 24 English. A histamine test meal on February 20, 1961, showed that she still had a very high total acidity and a normal free HCl. Recent x-ray examination showed some improvement in the stricture, and for the first time it was possible to demonstrate a hiatus hernia (Figs. 3 and 4).

Case 2

A 50-year-old man employed at an automobile factory had a history of postprandial pain over a period of 18 months. The pain was mid-epigastric, and occasionally radiated through to the back. At no time had he vomited, and there was no retrosternal pain. A barium meal on October 3, 1960, revealed a gross pyloric stenosis possibly due to a pre-pyloric ulcer (Fig. 5). Shortly after, a standard three-quarters anterior Polya-Hofmeister gastrectomy was

FIG. 1.—Case 1. Barium-meal radiograph, showing severe pyloric stenosis.

FIG. 2

FIG. 3

FIG. 4

FIG. 2.—Case 1. Barium swallow radiograph, showing a simple stricture of the oesophagus. FIG. 3.—Case 1. Barium swallow radiograph, showing the improvement after bougienage. FIG. 4.—Case 1. Barium-meal radiograph, revealing a hiatus hernia which may have been a sequel to the oesophageal stricture.

performed. His post-operative course was excellent, and he went home on the tenth day. A few days later he reported the rapid onset of severe dysphagia. A barium swallow revealed a benign stricture of the lower one-third of the oesophagus (Fig. 6). This was confirmed by oesophagoscopy, and bougienage was easily effected. He has continued self-bougienage, and very quickly gained 2 stone (12.7 kg.) in weight. A barium-meal examination in March, 1961 (Fig. 7), showed improvement in the stricture, but a hiatus hernia could not be demonstrated. Previous x-ray films had also failed to show this defect.

Discussion

Both of these patients suffered from an equally severe pyloric stenosis. They were relatively young and fit, with no coincidental diseases. A four-day period of gastric lavage was carried out in each case, using Ryle's tubes. Identical operations were performed and the immediate post-operative course was excellent in each case, with removal of the tubes on the second day. In Case 1 a hiatus hernia could only be shown post-operatively, and in Case 2 this defect of the cardia has not been demonstrated despite careful search.

A review of the literature brings out the following factors important in the aetiology: incompetence of the cardia; pyloric obstruction and hypersecretion; suction tubes; and infection and thrombophlebitis from adjacent lesions.

Incompetence of the Cardia

Allison (1948), in discussing peptic ulcer of the oesophagus, focused attention on the importance of the mechanism of the cardia in protecting the oesophagus from reflux. Besides describing the many factors that contribute to a competent cardia, he enumerated ways in which incompetence might develop: insertion of a rigid tube—for example, a Souttar tube through the cardia—surgical trauma, paralysis or lack of tone of the muscles as seen in very debilitated or flabby obese subjects, and, perhaps most important of all, the presence of a sliding mediastinal hernia of the cardia. These views are supported by many authors, and it is accepted that a sliding hiatus hernia is one of the commonest predisposing factors in oesophagitis. In Case 1 no hiatus hernia could be demonstrated until after the stricture was well established, while in Case 2 no hiatus hernia had ever been shown despite repeated investigations. It is possible that the defect in Case 1

may have been a sequel to the oesophageal stricture. It has also been shown (Rudstrom, 1954) that with hypersecretion in the stomach a regurgitation could develop, with subsequent changes in the oesophagus, even when the cardia was competent. He reported six cases of oesophageal stricture associated with duodenal ulcer, none of which had an associated hiatus hernia, and in support of his observations quoted Wangenstein and Leven's (1949) series of six similar cases.

Pyloric Obstruction and Hypersecretion

In both cases pyloric stenosis was severe, but only in Case 1 was vomiting an important feature. Larson, Layne, and Howard (1942) observed the passive function of the oesophagus in vomiting, but cited the mechanical factors of rapid dilatation and excessive movement of the oesophagus leading to small fissures in the mucosa as being sufficient to cause ulceration. Pyloric stenosis is often encountered in reports of oesophageal stricture after upper abdominal surgery, and the obstructive vomiting must play an important part in the damage to the lower oesophagus. Rudstrom (1954) listed the ulcer disease, the hypersecretion, and the pyloric stenosis as the primary factors involved.

In Case 1 a post-operative histamine test meal showed a very high total acidity and a normal free HCl, so that pre-operatively there must have been marked hypersecretion. In Case 2 the post-operative histamine test meal showed a very low total acidity and no free HCl. It is possible that in the first case damage to the oesophagus due to the daily vomiting had been established before operation, and the early onset of a stricture was only coincident with or perhaps accelerated by pre- and post-operative suction tubes, and by any minor derangement of the cardia resulting from the gastrectomy. In Case 2 it is interesting to note that, besides the complete absence of vomiting throughout the history, the patient was almost achlorhydric as a result of his gastrectomy. Wangenstein and Leven (1949) felt that gastrectomy was the logical treatment for oesophageal stricture as it eliminated hypersecretion. Vomiting was not a factor in Case 2, and an adequate gastrectomy did nothing to prevent a serious stricture.

Suction Tubes

Vinson (1941) stated that the use of tubes might contribute to oesophageal reflux by rendering the cardia

FIG. 5

FIG. 6

FIG. 7

FIG. 5.—Case 2. Barium-meal radiograph, showing severe pyloric stenosis. FIG. 6.—Case 2. Barium swallow radiograph, showing a simple stricture of the oesophagus which occurred post-operatively as in Case 1. FIG. 7.—Case 2. Barium swallow radiograph, showing considerable improvement in the stricture after bougienage.

partially incompetent. Many other authors have reviewed the importance of suction tubes in causing damage to the oesophagus, and all seem to agree that it plays a minor part which may become more significant if oesophageal injury has already occurred. McKeown (1958) in reporting two similar cases made the interesting observation that after gastrectomy in each the Ryle tubes were left in only 72 hours, and strictures developed. He felt that they were the result of regurgitation of alkaline fluid through the Polya type stoma and hence into the oesophagus, and sought to prevent this by converting both to a Billroth type of anastomosis. After the second operation the tubes were left in for five days but the strictures disappeared.

The use of a Ryle tube was not thought to be an important factor here. It is conceivable that prolonged use of a tube could aggravate a pre-existing oesophagitis, but there was no proof of such disease, and use of the tube was very brief.

Infection and Thrombophlebitis from Adjacent Lesions

The relation between benign oesophageal stricture and coincident upper abdominal or thoracic lesions has never been clearly defined. Straus (1950) observed that most cases have in common the presence of a peptic ulcer, and the application of surgical trauma to the stomach or oesophagus. This trauma superimposed on chronic inflammatory disease of the upper abdomen might lead to retrograde thrombophlebitis in the lower oesophagus, and eventually to fibrosis and stricture. He felt that this could be explained anatomically in that the coronary vein drains blood from the stomach and receives tributaries from the lower oesophagus, and the lymphatics are closely allied. Allison (1948) observed that in reflux oesophagitis enlarged lymph nodes are always present around the lower end of the oesophagus, forming quite a large mass. He thought this might be a factor when the stricture is eccentric, especially in young children. Opposed to these theories is the fact that thrombosis cannot be shown microscopically, and also there may not be any coexistent inflammatory process. It is felt, therefore, that while these factors may be active in the pathogenesis, defects of the cardia and pyloric obstruction with vomiting are the most significant points in the aetiology.

Having considered several theories which have been brought forth to explain benign stricture of the oesophagus, it still remains that thousands of people have suffered from pyloric stenosis, but only a few have progressed to oesophageal stricture, with or without hiatus hernia and reflux. Larson *et al.* (1942) have suggested that regurgitation of gastric contents into the oesophagus is a normal process in a high percentage of cases. It appears, then, that other factors still unknown govern the response of the oesophagus to repeated exposure to acid gastric or alkaline jejunal contents.

Summary

Two cases are reported of benign stricture of the oesophagus following gastrectomy for pyloric stenosis. The rarity of this disease after uncomplicated gastrectomy is noted, and in reviewing the literature various aspects of the aetiology are assessed. Defects of the cardia and pyloric obstruction with hypersecretion are thought to be the most important factors, but other unknown forces may be involved.

I thank Mr. E. B. Whittingham for his advice and thoughtful criticism in the preparation of this case report.

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VESICAL HAEMORRHAGE AFTER MEGAVOLTAGE IRRADIATION

BY

D. P. B. TURNER, F.R.C.S.

Consultant Surgeon, Broadgreen, Mill Road, and Newsham
General Hospitals, Liverpool

Since the availability of megavoltage therapy in this region an increasing proportion of cases of vesical neoplasm from my surgical practice have been referred for this type of treatment. In general the type of case chosen for megavoltage irradiation has been the extensive papillary or ulcerating tumour regarded as too large for either transvesical diathermy excision with radon-seed implantation or segmental resection, and therefore the treatment has usually been an alternative to total cystectomy. The results have been, in the main, eminently satisfactory, and it is thought that this type of therapy given for selected cases of vesical neoplasm represents a definite advance on previous methods, both surgical and radiotherapeutic.

Among 17 cases treated, however, were four instances of persistent and serious vesical haemorrhage which occurred after the irradiation and which resulted in problems of the utmost difficulty in relation to their surgical management.

It is proposed here to describe these four cases, briefly to consider the cause, to discuss more fully the methods used to control the haemorrhage, and, finally, to formulate some conclusions concerning the management of this complication.

Case 1

A man aged 59 was first seen in hospital on December 14, 1957, because of painless haematuria. I.V.P. revealed a large papilliferous filling defect on the left lateral wall of the bladder, a small diverticulum, and normal pyelograms. Cystoscopy confirmed the presence of a large papillary tumour which looked benign. On January 10, 1958, a transvesical diathermy excision of this tumour, together with three other small seedling growths, was carried out. The tumours appeared to be non-invasive, and the histology was as follows: "The large papilloma measures 6 by 6 by 4.5 cm. There are three much smaller specimens. Histologically, they show considerable nuclear irregularity with numerous mitoses, but no evidence of invasion. The growths should probably be considered as carcinomata and they appear to have been completely removed."

In July, 1958, follow-up cystoscopy revealed the presence of multiple recurrences involving a large area of the bladder mucosa. Megavoltage therapy was given during the last two weeks of July. In November haematuria began; at first this was mild, but on January 7, 1959, the patient was readmitted with gross haematuria and a haemoglobin level of 37%. In spite of blood transfusions, "imferon,"