Achalasia complicated by oesophageal stricture

A case report

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Summary

A case of achalasia in which the Heller operation failed to provide symptomatic relief and was complicated by the development of an oesophageal stricture is presented. The reasons for the occurrence of oesophageal strictures after performance of the Heller operation are described and the management of this infrequent complication is discussed.


There is almost universal agreement that the surgical procedure of choice in the management of achalasia is the (modified) Heller operation. In this operation an extramucosal myotomy is performed on the narrow inferior segment of the achalasic oesophagus; the narrow segment can then relax sufficiently to allow food to pass into the stomach. The Heller operation has in fact proved so satisfactory in maintaining sustained relief from dysphagia that some authorities now recommend the procedure to almost every patient with achalasia in preference to another surgical procedure may prove necessary. In this article the management of a patient with achalasia complicated by oesophageal stricture is discussed.

Case report

The patient, a 28-year-old man, was admitted to Baragwanath Hospital, Johannesburg, in February 1981, having a 5-year history of dysphagia, regurgitation, substernal discomfort and weight loss. In 1978 a diagnosis of achalasia had been made at another hospital on the basis of clinical and radiological findings, and a Heller operation had been performed. No details of the postoperative course are known but after the operation the patient could only swallow liquids. By October 1980 he suffered total dysphagia. A nasogastric tube had been inserted and the patient was fed a liquid diet through this tube for the next 5 months.

On admission (with the nasogastric tube still in situ) the patient complained of a chronic cough in addition to total dysphagia. There was clinical and radiological evidence of aspiration pneumonitis and the chest radiograph (Fig. 1A) also revealed a large air-fluid level in the posterior mediastinum. Barium swallow examination (Fig. 1B) showed a grossly dilated oesophagus which was totally obstructed at the distal thoracic end. However, when contrast medium was introduced through the nasogastric tube no radiographic abnormalities of the stomach or duodenum were evident.

Oesophagoscopy was carried out and the oesophageal mucosa was carefully inspected and appeared healthy, but there was a tight fibrotic stricture at the lower end. Oesophageal manometry failed to reveal any evidence of peristaltic activity within the oesophagus. Although the manometer could not be introduced through the stricture to measure the resting pressure of the lower segment and the mecholyl test was not performed, a provisional

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The diagnosis of achalasia of the oesophagus complicated the presence of a stricture and aspiration pneumonitis was made.

Attempts to forcibly dilate the tight distal stricture were considered contraindicated by the increased risk of perforation in this case and by the likelihood that even if dilatation was possible it would prove to be of temporary benefit only. Intensive chest physiotherapy with bronchodilators and antibiotics was necessary in view of the aspiration pneumonitis.

On the day before operation the nasogastric tube was removed, considerable force being required to dislodge it from the strictured area, and oesophageal lavage was performed.

The operation

The abdomen was opened through the previous midline incision. The intra-abdominal oesophagus and cardio-oesophageal junction were densely adherent to the undersurface of the diaphragm and the left lobe of the liver, suggesting that the Heller operation may have been complicated by oesophageal perforation and leakage. The stricture in the distal oesophagus measured 2 cm in length and extended into the cardio-oesophageal junction, the external diameter of which had now been reduced to less than 1 cm. It was felt that in view of the length of the stricture, the severe scarring and stenosis at the cardio-oesophageal junction any attempt to repeat the Heller operation or to perform a cardioplastic procedure was contraindicated.

The choice of operation now lay between oesophagogastrectomy, oesophageal resection with intestinal interposition or oesophageal bypass. Oesophageal bypass using the whole stomach was selected on the grounds that it was the simplest and safest procedure, avoiding the necessity for thoracotomy and excision of the dilated oesophagus. The stricture was then excised for histological examination. The lower end of the oesophagus and the cardio-oesophageal junction were closed.

The greater curvature of the stomach was mobilized by ligation and division of the gastrocolic ligament outside the vascular arcades, preserving the right gastro-epiploic vessels. The vasa brevia, left gastro-epiploic and left gastric vessels were ligated and divided. The lesser omentum was divided with preservation of the right gastric vessels. The duodenum and head of the pancreas were mobilized by an extended Kocher manoeuvre and a pyloromyotomy was performed. The mobilized stomach was now laid aside and a retrosternal tunnel fashioned by blunt dissection. The left side of the neck was then opened using a J-shaped incision (Fig. 2). The cervical oesophagus was mobilized and divided, the distal end being closed, thereby completely excluding the achalasic oesophagus (Fig. 3, left). The proximal end of the cervical oesophagus was brought out for anastomosis to the gastric fundus which had been drawn up to the neck by the retrosternal route. A 1-layer oesophagofundic anastomosis was performed, using interrupted non-absorbable sutures.

Histological examination of the strictured area revealed fibrosis and chronic oesophagitis with no evidence of malignancy. An absence of ganglia in the resected specimen...
supported the clinical diagnosis of achalasia. The postoperative course was complicated by right basal pneumonia, which responded to treatment with chest physiotherapy and antibiotics, and by transient anastomotic leakage. However, the neck anastomosis had completely healed by the 3rd week after operation and the patient was able to resume an oral diet.

Results

In July 1981 the patient was submitted to a repeat barium swallow and fluoroscopic screening examination and to oesophagogastrotomy, biopsy specimens of the oesophageal and gastric mucosa being taken. At that time the patient admitted to occasional difficulty in swallowing certain solids such as hard meat and dry bread. However, the barium swallow (Fig. 3, right) failed to reveal any evidence of anastomotic stricture and the retrosternal stomach was seen to empty rapidly. Endoscopic examination showed a widely patent oesophagofundic anastomosis, an absence of bile in the stomach and no macroscopic evidence of oesophagitis or gastritis. Histological examination revealed no abnormalities of the oesophageal and gastric mucosa. Computed tomography revealed that the excluded oesophagus had now become a mucocele (Fig. 4).

Discussion

Early failure of the Heller operation to relieve the symptoms of achalasia usually means that myotomy was incomplete and re-operation is indicated; the development of an oesophageal stricture is, however, a very unusual complication of this procedure. In a review of 2118 patients who underwent the Heller operation, Postlethwaite found only 19 cases complicated by stricture formation, an incidence of less than 1%.

An oesophageal stricture can arise in three ways:

1. Unsuspected perforation of the oesophageal mucosa at the time of the Heller operation. Leakage from the oesophagus may then be followed by peri-oesophageal inflammation and scarring. This was the most likely cause of the stricture in this patient and this view was supported by the finding of dense peri-oesophageal scar tissue at laparotomy. Undoubtedly the continuous presence of a foreign body, the plastic nasogastric tube, aggravated the tendency to stricturing in this case.

2. Replacement of the myotomy area by constricting fibrous tissue in the absence of oesophagitis, possibly due to organization of a peri-oesophageal haematoma. This has been reported occasionally and should be suspected when the patient suffers recurrent dysphagia years after the original operation.
3. Stricture associated with reflux oesophagitis, reflux occurring in 3-6% of patients who have undergone the Heller operation. Here the onset of dysphagia is most likely to be preceded by heartburn and occasionally by other complications of reflux oesophagitis, such as haemorrhage.

There is an increased risk of cancer of the oesophagus in long-standing achalasia, an incidence of 3 - 7% having been reported. This cause of dysphagia must be excluded by careful endoscopy before management of the oesophageal stricture is undertaken. Forcible dilatations of the stricture may then be tried. Where dilatation is contraindicated or proves unsuccessful, surgical treatment will be necessary. The surgical procedure chosen will depend on the nature and cause of the stricture.

When the stricture is due to fibrous healing of the myotomy a repeat Heller operation is indicated. The older methods of cardioplasty, where the lower segment and cardio-oesophageal junction is simply incised longitudinally and sutured transversely in a manner analogous to the Heneck-Mikuticz pyloroplasty, lead to severe reflux oesophagitis and have now been abandoned. Oesophagogastrostomy, in which the gastric fundus is anastomosed to the dilated oesophagus above the stricture, is likewise an invitation to free reflux and therefore contraindicated.

Any repair carried out to widen the cardio-oesophageal junction should incorporate an antireflux procedure, although it would seem illogical to add a potential mechanical obstruction to the lower oesophagus when the body of the oesophagus is essentially aperistaltic. However, widening of the strictured area with a fundic patch and combining this with a Nissen fundoplication appears to be an appropriate procedure as good results have been reported for this operation in uncomplicated achalasia. It could therefore be applied to the management of strictures of the cardio-oesophageal junction.

However, when severe scarring and stenosis of the distal oesophagus and cardio-oesophageal junction prevent the safe performance of a local repair, as in the case reported, a more radical approach will be necessary. Oesophageal resection with colon or jejunal interposition and also oesophagogastrectomy with excision of the redundant oesophagus have been used in cases of advanced achalasia. These techniques can be applied to the problem of achalasia complicated by oesophageal stricture, but there are certain disadvantages. Oesophagogastrectomy involves the performance of a thoracotomy which is not without risk in patients who have suffered severe pulmonary damage from chronic aspiration. If anastomotic leakage occurs it is likely to be complicated by fatal empyema. The risk of leakage is even greater in the intestinal interposition procedures in which bowel, which is of a smaller calibre, is anastomosed to the grossly dilated oesophagus and three anastomoses are required.

Bypass of the obstructed oesophagus using the whole stomach is not a new procedure, successful management of benign oesophageal strictures by means of this technique first being reported by Kirschners in 1920. The stomach has an excellent blood supply and will reach easily into the neck, where an oesophagofundic anastomosis can be performed without difficulty. The retrosternal tunnel is simple to fashion and avoids the necessity for thoracotomy. Should anastomotic leakage occur, as happened transiently in this patient, it will be superficial into the neck and not likely to cause death.

No ill-effect has resulted from complete exclusion of the thoracic oesophagus in this case, a similar observation having been reported in patients with malignant obstruction of the oesophagus undergoing gastric bypass with oesophageal exclusion. As expected from previous clinical and experimental evidence, the excluded thoracic oesophagus has become an asymptomatic mucocele in the posterior mediastinum (Fig. 4).

Treatment of achalasia, either by dilatations or the Heller operation, does not appear to protect the patient against the development of carcinoma. In the average case, yearly check-ups with endoscopy are advised. In the present patient the risk of malignancy is probably less now that the oesophagus is no longer subject to retention oesophagitis. Nevertheless it is planned to re-examine the excluded oesophagus by annual computed tomography; detection of a space-occupying lesion would necessitate thoracotomy and excision of the excluded oesophagus.

The retrosternal stomach has been seen on fluoroscopic screening to empty rapidly through the pyloromotomy, and it can be assumed that the acute angle now existing between the pyloro-antral region and the duodenum (Fig. 3, right) has limited the occurrence of biliary reflux. It would therefore appear that retrosternal gastric bypass with exclusion of the thoracic oesophagus has been an appropriate procedure in this patient with achalasia and a severe distal oesophageal stricture.

**Conclusion**

Oesophageal strictures are infrequent complications of the Heller operation for achalasia. If they are not suitable for or resistant to dilatation, the usual surgical management of these strictures will be either a repeat Heller operation or local cardioplastic repair combined with an antireflux procedure. Where more radical surgery is required, oesophageal bypass using the whole stomach provides an attractive alternative to oesophagogastrectomy or resection of the stricture with intestinal interposition.

**REFERENCES**


