Case report

Broncho-oesophageal fistula: a late complication of endoscopic variceal sclerotherapy

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SUMMARY A case is reported of delayed broncho-oesophageal fistula presenting several weeks after fiberoptic injection sclerotherapy for oesophageal varices in a patient with chronic active hepatitis who eventually died from bronchopneumonia. Such serious complications of injection sclerotherapy should be kept in mind with the increasing popularity of this method of early treatment of bleeding oesophageal varices.

In recent years there has been increasing interest in the use of endoscopic injection sclerotherapy for the treatment of bleeding oesophageal varices.¹⁻¹⁰ Rigid^{1-6 9 10} and fiberoptic^{3 7 8 10} endoscopes have been used in this technique and a number of modifications have been introduced to enable compression of varices to be achieved during the injection procedure.^{7 8 10-12} The complications arising from sclerotherapy range from substernal pain,^{1 2 7-9} oesophageal bleeding,^{4 5 8 9} ulceration and sloughing of the oesophageal mucosa,^{2 5 8 9} and early oesophageal perforation^{2 5 8 9} with peri-oesophageal leakage,^{1 2 5} thoracic empyema,^{1 4} and pleural effusion,⁷ to delayed oesophageal necrosis occurring five to 14 days later^{1 4 13} and late oesophageal stenosis.^{1 2 5 8 14}

We have used injection sclerotherapy as the treatment of choice of bleeding oesophageal varices in our hospital for the last year and wish to report an unusual complication of broncho-oesophageal fistula formation presenting several weeks after sclerotherapy treatment without any intervening episode to suggest oesophageal perforation. This does not appear to have been reported previously.

Case report

A 53 year old man with HBsAg-negative and autoantibody-negative chronic active hepatitis with

cirrhosis on prednisolone for six months was admitted with melaena, hepatosplenomegaly, and ascites. Endoscopy demonstrated bleeding oesophageal varices and a percutaneous splenoportogram demonstrated a patent portal vein with large oesophageal varices. After resuscitation, endoscopic variceal sclerotherapy was performed under general anaesthesia using an Olympus GIF-K forward-oblique endoscope and a prototype Fujinon over-tube using a technique similar to that of Williams and Dawson.¹² A total of 24 ml 5% ethanolamine oleate was injected into four rows of varices at the gastro-oesophageal junction. No further bleeding occurred from this time and substernal pain after the procedure disappeared within 24 hours. Radiograph of the chest at this time was normal, and he was discharged four days later free of symptoms and ascites on prednisolone and spironolactone. At two weeks' review he was well but at one month he complained of mild dysphagia and odynophagia for solids with coughing when he lay down. Radiograph of the chest was normal. Three weeks later he was readmitted with worsening productive cough of yellow sputum but unchanged dysphagia. On examination he had lost weight, there was minimal ascites, unchanged hepatosplenomegaly, no signs of hepatic encephalopathy, but signs of lung consolidation in the right lower zone. A clinical diagnosis of pneumonia and late oesophageal stricture after sclerotherapy was made and radiograph of the chest showed bilateral bronchopneumonia, worse on the right.

At endoscopy, varices were not evident but in the lower third of the oesophagus a large circular defect

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replaced the right lateral wall immediately above the gastro-oesophageal junction communicating with a deep cavity lined by granulation tissue in the base of which was seen a small bronchus. Biopsy of the cavity edge revealed inflammation only. The gastro-oesophageal junction was slightly narrowed but admitted the Olympus GIF P2 endoscope (diameter 8 mm) without difficulty. The remainder of the upper gastrointestinal tract was normal. A diagnosis was made of broncho-oesophageal fistula due to delayed necrosis of the oesophagus after injection sclerotherapy. A barium study confirmed the presence of a cavity outside the lower oesophagus on the right (Fig. 1) communicating with a right lower lobe bronchus which filled only when the patient lay down and this reproduced his cough.

Despite supportive measures bronchopneumonia worsened, septicaemia developed, and he died nine weeks after sclerotherapy. Necropsy showed evidence of widespread bronchopneumonia with a 3 cm cavity in the right lower lobe contiguous with a 1.5 cm defect in the right lateral wall of the oesophagus just above the gastro-oesophageal junction (Fig. 2). It contained an open bronchus in its base. No varices were visible, there was 700 ml of ascitic fluid, the liver weighed 900 g and had the appearance of macronodular cirrhosis. Histopathology demonstrated obliterated submucosal oesophageal veins but patent peri-oesophageal veins.

Fig. 1 Barium study demonstrating large cavity to the right of the lower oesophagus (fluid level) with narrowing of the oesophagus immediately above it.

Fig. 2 Necropsy view of opened oesophagus with a probe demonstrating communication between oesophagus and cavity in right lung viewed posteriorly.

Discussion

The mechanism of delayed oesophageal necrosis in our patient after injection sclerotherapy was presumed to have been the result of inadvertent submucosal extravasation of sclerosant. This has been shown to produce coagulation necrosis in dogs¹⁵ and in man,^{3 10 13} although perivascular injection is recommended as a technique in some centres^{10 13 14} to induce submucosal fibrosis and obliteration of varices. Intravascularly injected sclerosant rapidly disperses in a cephalad direction if no compression technique is used^{3 10 16} and accidental perivascular leakage may occur. By adding radiographic contrast medium to the sclerosant solution and performing injection under fluoroscopy, we have found that the sclerosant solution remains within the varix injected while the varix is both positioned in the over-tube window and after rotation of the over-tube to achieve compression. The quantity of sclerosant we used in our patient was comparable with that used by others.^{1 2 8 9 12 14} It is, therefore, difficult to understand how extensive extravasation of sclerosant could have occurred in our patient. It is possible that the concomitant administration of corticosteroids played some part in minimising the local reaction around oesophageal necrosis and allowed the slow development of a peri-oesophageal cavity within the right lung and subsequent erosion into a bronchus.

All currently reported experience with injection sclerotherapy shows it to be a highly effective method of arresting acute haemorrhage from oesophageal varices in about 90% of cases.^{1 2 6 7 9 13} Major complications of sclerotherapy have occurred in 2–44%^{1 2 7 9 13} of injection treatments, although many are primarily consequences of rigid oesophagoscopy such as oesophageal tears^{2 5 8 9} and leakage.^{1 2 5} Our case report represents the only major complication of our experience so far with 26 patients, giving an overall mean complication rate from the literature of about 12%. The number of recent reports would suggest that endoscopic sclerotherapy is becoming more popular as the preferred treatment for the management of patients with bleeding oesophageal varices, and the possibility of a major complication such as that described here should be considered in the event of new oesophageal or chest symptoms developing after treatment.

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