

A rare complication of oesophageal sclerotherapy

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Endoscopic sclerotherapy is a standard procedure for the treatment of oesophageal varices. It is generally regarded as a safe procedure with low morbidity and mortality. However, there are a number of rare and fatal complications associated with this procedure. We report a case of pericardial effusion developing after oesophageal sclerotherapy. The patient developed cardiac tamponade requiring pericardiocentesis. This complication is uncommon, but its true incidence may be underestimated. When pericardial effusion is diagnosed early, this potentially fatal complication can be treated without serious sequelae.

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Introduction

Endoscopic sclerotherapy was first reported in 1939 for the treatment of bleeding oesophageal varices. In recent years, it has become a standard procedure for controlling variceal bleeding, and its efficacy has been proven in a number of controlled trials.¹ This procedure, although generally regarded as safe, is associated with a number of rare and sometimes serious complications. This article describes a rare complication of endoscopic sclerotherapy—pericardial effusion and cardiac tamponade.

Case report

A 47-year-old woman suffering from cirrhosis of the liver associated with hepatitis B presented with sudden haematemesis. Upper gastrointestinal endoscopy revealed five columns of grade 3 to 4 varices. Endoscopic sclerotherapy was performed with a standard end-view fiberoptic endoscope. Five per cent ethanolamine oleate was used as the sclerosing agent and was given intravariceally with free hand technique.

The first and second sclerotherapy sessions were performed with an interval of two weeks. Eth-

anolamine oleate, 15 ml, was injected intravariceally at the first session. The patient did not develop any complications afterwards. Sclerotherapy was repeated two weeks later, and ethanolamine oleate, 15 ml, was again injected intravariceally. After 24 hours, the patient experienced chest pain associated with shortness of breath and orthopnoea. A physical examination showed that she was in respiratory distress with elevated jugular venous pressure. Her blood pressure was 110/80 mmHg, and her chest was clear on auscultation. Heart sounds were normal and no pericardial rub was audible. An electrocardiogram (ECG) showed normal axis with T wave inversion over V2 to V3. A chest X-ray taken at this juncture revealed enlarged cardiac shadow when compared with old films (Fig 1). An urgent echocardiogram showed a large pericardial effusion with impaired diastolic filling (Fig 2).

Due to her clinical deterioration and confirmation of a large pericardial effusion, pericardiocentesis was performed; 700 ml of blood-stained fluid was tapped and a pericardial drain was left in situ. A further 500 ml of fluid was drained the following day. Culture of the fluid for bacteria and *Mycobacterium* spp. were negative. Pericardial biopsy showed no evidence of malignancy or inflammation.

Follow up echocardiograms performed two weeks and one month later showed no re-accumulation of fluid. Her oesophageal varices were eradicated with endoscopic variceal ligation.

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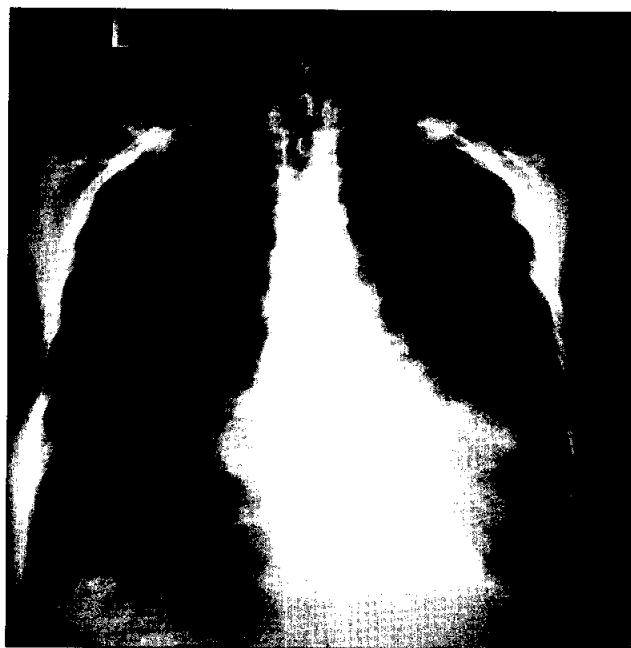


Fig 1. Chest X-ray showing features of pericardial effusion

Discussion

Although endoscopic variceal sclerotherapy has now been well recognised for the treatment of bleeding oesophageal varices, it is not without complications. As many as 25% to 50% of patients have chest pain or discomfort after sclerotherapy, and low-grade fever occurs in approximately 10% to 15% of patients. Oesophageal ulcers are very common, occurring in about 40% to 50% of cases.² More serious complications include bleeding, pleural effusion, and oesophageal perforation. There have been isolated reports of bizarre complications such as broncho-oesophageal fistula,³ respiratory distress syndrome, and brain abscess.⁴

This case illustrates another rare complication of sclerotherapy—pericardial effusion and cardiac tamponade. The pericardial effusion was probably directly related to the sclerotherapy because symptoms developed within 24 hours of the procedure and no other causes could be found. On reviewing the literature, only seven cases have been reported.⁵⁻⁸ The exact pathogenesis is uncertain. The oesophagus is a relatively thin-walled organ, and the lower third is just posterior to the fibrous pericardium. Direct injection of the sclerosant into the pericardial cavity is theoretically possible, especially when using a longer injecting needle with the needle directed anteriorly. Another possibility is that injection of sclerosant into the oesophageal wall

produces an intense inflammatory reaction involving the adjacent tissues. Radiological evidence demonstrates that mediastinal and pleural inflammation are frequent after endoscopic sclerotherapy, although most of these changes are mild and transient.⁹ This probably explains why chest pain is so common after sclerotherapy.

Chest pain is a common presentation. Six of seven of the previously reported cases complained of chest pain, while the remaining one had a fever and a pericardial rub. Our case eventually required peri-cardiocentesis because there was cardiac tamponade present. Three of the previously reported cases also required pericardiocentesis, all of them yielding blood-stained fluid. The remaining cases resolved spontaneously. Two patients eventually needed pericardiectomy for constrictive pericarditis. In one patient, symptoms suggestive of constrictive pericarditis presented six months after sclerotherapy.⁵

The injecting method for our patient was the intravariceal approach, which would seem less likely to give rise to a severe inflammatory response than the less popular paravariceal approach. This was also the method used for most of the reported cases. There is no satisfactory explanation for this. One hypothesis is that there is extravasation of sclerosants after an intended intravariceal injection. This complication is not restricted to a specific type of sclerosant. Sodium morrhuate was used in five cases while ethanolamine oleate was used for the other three, including our patient. Sclerosants are basically irritants, and probably all can cause severe inflammatory reactions.



Fig 2. 2 D Echocardiogram showing pericardial effusion

It is difficult to give definite recommendations to prevent this complication occurring as it is so rare, and no single factor has been incriminated as its cause. Smaller injection volumes, a shorter injecting needle (<4 mm), and tangential injection (especially when giving an anterior injection) may theoretically reduce this risk. Patients who develop significant chest pain and fever after sclerotherapy should be monitored for signs of pericarditis and cardiac tamponade. Considering that chest pain is such a common complaint after sclerotherapy, it is likely that this complication has been under-reported. Most cases subside spontaneously, but some will progress to cardiac tamponade or constrictive pericarditis. Since it is a readily treatable condition, its early diagnosis is of vital importance.

An alternative method for treating bleeding oesophageal varices is endoscopic variceal ligation, which has gradually gained popularity in recent years. It is comparable in efficacy to endoscopic sclerotherapy, but has fewer complications.¹⁰ Because it does not require the injection of sclerosants, complications related to the intense inflammatory reaction do not occur. In the future, it may become the treatment of choice for bleeding oesophageal varices.

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