Sclerotherapy for bleeding oesophageal varices — a fatal complication

A case report

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Summary

Although injection sclerotherapy for the control of bleeding varices is considered a relatively safe procedure, major complications can occur. A case is presented in which delayed perforation of the oesophagus due to ulceration led to a fatal outcome. The technique used and complications of the procedure are briefly discussed.

Injection sclerotherapy for the treatment of bleeding from oesophageal varices and the prevention of further haemorrhage has gained widespread acceptance. Several reports have indicated the efficacy and relative safety of the procedure. Minor side-effects such as transient postoperative retrosternal discomfort, fever and tachycardia have been described and are well recognized. Serious problems are infrequent and procedure-related deaths are rare. We would like to report our experience with a patient who died 3 weeks after injection sclerotherapy owing to transmural ulceration and perforation of the lower oesophagus.

Case report

A 62-year-old Black man was admitted to King Edward VIII Hospital, Durban, on 8 February 1983 following repeated attacks of haematemesis and melena. He was known to be a heavy drinker and had previously been hospitalized elsewhere for similar episodes. On admission he appeared jaundiced and pale and had a flapping tremor of the hands, marked ascites and lower limb oedema. A clinical diagnosis of liver cirrhosis with portal hypertension and bleeding from oesophageal varices was made and the appropriate treatment was instituted. A barium meal examination had already demonstrated oesophageal varices, and endoscopy confirmed the presence of large varices in the lower half of the oesophagus which were not bleeding at the time. No other abnormalities in the upper gastro-intestinal tract were identified as possible sources of haemorrhage. The serum albumin level was 23 g/l, the globulin level 47 g/l, the bilirubin level 52 mmol/l and the prothrombin index 66%.

The varices were not bleeding when the patient underwent injection sclerotherapy on 16 February. This was done under general anaesthesia with the flexible Olympus GIF-K endoscope after the oesophagus had been intubated with the fenestrated Williams variceal tube. Three columns of varices were injected near the oesophagogastric junction with 5 ml, 5 ml and 3 ml of ethanolamine oleate respectively. The injections were made into the lumen of the varices and the tube was rotated immediately after injection to obtain obliteration of the vessel lumen. No undue problems were encountered. The day after the procedure the patient was well and did not complain of discomfort. Once he had been started on a liquid diet he was discharged and booked to return in 2 weeks' time for a repeat procedure. Six days after the sclerotherapy he was reported to be asymptomatic with a clear chest on auscultation.

On 1 March he was readmitted and was now found to be dyspnoeic, tachycardic and febrile and slightly confused. The ascites had improved on medication, but the epigastrium was tender and he had evidence of a large left-sided pleural effusion. This was drained by tube thoracostomy and 4½ litres of yellow-brown pus was evacuated. Culture subsequently yielded a Klebsiella species. Soon after the drainage procedure, his condition deteriorated abruptly and he went into septicemic shock and had an irreversible cardiorespiratory arrest.

At autopsy three ulcers were found in the lower oesophagus, one of which was large and had penetrated the full thickness of the oesophageal wall and perforated into the left hemithorax. There was fresh blood in the stomach due to recent bleeding from the ulcers. Some varices were still present (Fig. 1). Microscopy showed mucosal destruction and transmural ulceration with fibrosis of the submucosa and muscularis and thrombosis in multiple dilated venules. There was also evidence of purulent meningitis, and multiple micro-abscesses were noted in both kidneys (Figs 2 and 3). The liver was markedly cirrhotic, micronodular and siderotic. No Mallory hyaline bodies were observed. Swabs taken from the purulent meninges again yielded a Klebsiella species.

Discussion

Since Crafoord and Frenckner introduced the idea of injection sclerotherapy in 1939 the procedure has been popularized by Macbeth, Johnston and Rodgers and Terblanche et al., and it is now being used in many centres in the world as an effective and relatively non-invasive means of controlling and preventing haemorrhage from oesophageal varices. The peri-operative mortality rate ranges between 5% and 20%, depending on the technique used and the patient's condition. In patients with advanced liver disease major operations are associated with a prohibitive mortality rate, and in such situations sclerotherapy offers distinct advantages either as temporary or permanent treatment. Recurrence of bleeding is the major drawback, however, and even after multiple injections exsanguination is not unusual.

Sclerotherapy can be performed via either the rigid oesophagoscope or the flexible endoscope. The advantage of the former is the oesophageal distension obtained with interruption of the flow in the varices and good access with a wide-bore suction
Fig. 1. Large ulcer in the oesophagus with perforation near oesophagogastric junction. Some varices are still present. There is fresh blood in the stomach.

Fig. 2. Purulent meningitis caused by Klebsiella septicaemia.

cannula, but major complications have been encountered. Instrumental perforation occurred in 3 out of 117 cases reported by Johnston and Rodgers and in 2 out of 22 in Terblanche et al.’s earlier report. The rigid oesophagoscope might also induce bleeding on introduction and will not enable the endoscopist to deal with gastric varices if this is required. For these reasons the flexible fibre-optic endoscope is now used in most centres. Several devices have been designed to help distend the varices before injection, interrupt blood flow and compress the vessels afterwards so that the sclerosing agent can be of maximum benefit. The fenestrated Williams tube we use allows the varix to protrude into the lumen of the tube and makes accurate injection possible. Although we prefer to use general anaesthesia, which will allow the use of an oesophageal over-tube, there are several advocates of local anaesthesia and mild sedation. We are using ethanolamine oleate and aim for injection within the lumen of the varix. Acute thrombosis and damage to the vascular endothelium will result in variceal obliteration. Without doubt some of the sclerosant will be misdirected and end up in the perivascular space, where it will initiate an inflammatory response. This is not necessarily detrimental, since the subsequent fibroplasia will also help to occlude the varices. In a large series of 640 patients in Germany a 1% solution of laureth 9 (1 ml at a time up to a total of 50 ml) was injected into the oesophageal wall adjacent to varices. Major complications occurred in 64 patients (10%), with ulceration resulting in incomplete wall necrosis in 13 cases and complete wall necrosis with mediastinitis and pyothorax in 11. Sivak has reported the American experience in an overview of 1305 procedures in 610 patients. The sclerosant most often used was 5% sodium morrhuate, injected intravascularly via a flexible endoscope. A total of 118 complications were encountered (19% per patient, 9% per procedure). The incidence of oesophageal ulceration was 4% per procedure. We have documented a similar incidence.

Oesophageal ulceration starts within a few hours as a response to the sclerosing agent, and develops fully in 7 - 10 days. Oedema can lead to narrowing of the lower oesophagus, but the inflammation normally resolves within a few weeks. The development of a fibrotic stricture requiring dilatation is rare and has not occurred in any of our patients. This is the first case of severe
transmural ulceration and perforation of the oesophagus we have seen. The clinical and histological evidence suggests that the leak occurred during the second week after sclerotherapy, by which time most patients will have been discharged from hospital. Minor side-effects of sclerotherapy are seen regularly but usually disappear within 48 hours. These include retrosternal discomfort, dysphagia and odynophagia, fever, tachycardia and arrhythmias, changes in the prothrombin index and partial thromboplastin time, and small pleural effusions. They are presumably related to the sclerosant entering the general circulation and to local effects. 7-16

Sclerotherapy does therefore seem to be associated with certain problems, but if we are prepared to accept these, as well as the high incidence of recurrent bleeding and the need for continuous follow-up and repeat procedures, it may still be the treatment of choice when major surgical procedures pose a high risk because of severely compromised liver function.

REFERENCES


Primary cardiac hydatid disease

A case report

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Summary

A young Coloured man whose only symptom was that of minimal dyspnoea on exertion, on examination had signs of infundibular stenosis which were confirmed by cardiac catheterization. Additional features were left anterior hemiblock and cardiomegaly as delineated radiologically. Cross-sectional echocardiography revealed a very large cystic mass located within the interventricular septum which encroached upon the right ventricular outflow tract. This cystic mass was further delineated by cardiac cine angiography. It is postulated that this mass was a primary cardiac echinococcal cyst and was directly responsible for the complications of left anterior hemiblock and the infundibular obstruction. The patient declined surgery and a definitive pathological diagnosis could therefore not be made. If this is a hydatid cyst then it is the second case reported in the literature diagnosed by two-dimensional echocardiography and in which left anterior hemiblock has been recorded. The clinical features, complications and surgical correction are briefly outlined.

Case presentation

The patient was a 27-year-old Coloured man from South West Africa/Namibia whose only complaint, mild dyspnoea on exertion, attributed to being overweight and unfit. In June 1982 he went to his general practitioner with influenza, at which time a cardiac murmur was first detected. He worked for a building construction concern and had not been in a sheep-rearing area.

The physician who examined him in SWA could find no evidence of cardiac failure. Further findings reported were a blood pressure of 125/85 mmHg, a 'significant systolic murmur' heard maximally just below the pulmonary area and a widely split second heart sound with reduced intensity of the pulmonary component. A 'right ventricular lift' was noted as well as 'pulsation in the left second and third interspace'. Chest radiography documented a 'rather globular heart' as well as 'prominence in the area of the right atrium and right ventricle'.

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