Hydropneumopericardium and oesophagitis: a non-fatal case

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ABSTRACT Hydropneumopericardium is a very rare and usually fatal complication of peptic oesophageal ulceration. The patient reported here survived and the report resembles one previously made about a child. In both patients failure to show the fistula radiologically or on endoscopy suggests that rapid spontaneous healing had occurred, and that this was responsible for survival.

Pneumopericardium can arise from gas-producing organisms within the pericardium, or from fistulae communicating with the bronchus, oesophagus, stomach, or the exterior (Meyer, 1948). Communication with the alimentary tract is most commonly a consequence of benign or malignant ulceration of the oesophagus or stomach (Gossage *et al*, 1976).

Stephenson *et al* (1958) reported a case of hydropneumopericardium due to a benign oesophagopericardial fistula, and reviewed 12 other published cases. Seven were traumatic (five of which were due to a foreign body), two were due to a benign ulcer, one to a diverticulum, one to a tuberculous abscess, and in the other two the cause was uncertain. None of the patients survived.

However, Dons *et al* (1964) reported a child with a hydropneumopericardium and oesophagitis, who survived after pericardial drainage.

Case report

A 52-year-old mentally defective man had been investigated 18 months previously for dysphagia. Radiology, oesophagoscopy, and histology at that time had confirmed the presence of a benign peptic stricture, which had been dilated. His present admission was precipitated by an acute attack of breathlessness and collapse, during which he first appeared "grey" and clammy and then vomited, the whole episode lasting for two and a half hours. Five days before admission he had fallen, and this had resulted in low backache. For the preceding three weeks, he had suffered a productive cough, with dyspnoea on exertion.

When first seen in the casualty department, however, he was no longer shocked and only slightly dyspnoeic at rest. Examination showed a pyrexia of 37.5°C, a heart rate of 110/min, and a blood pressure of 110/60 mmHg, but no other cardiovascular abnormalities were detected. There was thought to be a small left pleural effusion. A chest radiograph (fig 1) appeared to confirm this. Over the next two days he became afebrile but

Fig 1 Admission PA chest radiograph, showing small left pleural effusion and a hydronpneumopericardium. Arrow indicates pericardium.

Fig 2 Chest radiograph taken three days after admission. Hydropneumopericardium is now more obvious.

hypotensive (BP 85/60 mmHg), with a persisting tachycardia. Dysphagia for solids and occasional vomiting were now the only symptoms.

Three days after admission a further chest radiograph showed a large hydropneumopericardium (fig 2), features of which were now realised to have been present on the original film (fig 1). A barium swallow and meal showed a hiatus hernia with severe gastro-oesophageal reflux and an apparently benign lower oesophageal stricture, but no fistula was seen.

The next day, needle aspirations of the pericardial and pleural fluids were performed. The aspirates were both slightly cloudy and pale yellow in colour, with occasional pus cells, but no growth of organisms was obtained.

The patient remained well but two days later he started taking ampicillin for a pneumococcal chest infection.

Three days later, a thoracotomy was performed, since the hydropneumopericardium remained unchanged radiologically.

Operative findings

There was a bloodstained slightly turbid pleural effusion. The pericardium was opened behind the phrenic nerve and there was a similar pericardial effusion. Both layers of the pericardium were thickened and the pericardial space was obliterated

by posterior and inferior adhesions. No attempt was made to mobilise the heart. The pericardium was drained into the left pleural space and an intercostal drain introduced.

Postoperative progress

The patient remained well, and two weeks after operation the hydropneumopericardium had disappeared. An oesophagoscopy was performed before discharge, and this showed intense oesophagitis with white sloughing mucosa from a level of 25 cm down to the gastro-oesophageal junction. A short peptic stricture was observed 30 cm from the incisor teeth, but it was not dilated on this occasion. No areas of chronic ulceration or oesophageal tears were seen.

The patient was discharged on medical treatment for oesophagitis.

Blood tests during his hospital stay had shown a mild iron deficiency anaemia, and a blood urea of 8.5 mmol/l but normal plasma electrolytes, liver function test results, proteins, and serum thyroxine concentrations. No tubercle bacilli were seen in the aspirates, or subsequently cultured; viral antibody titres and the test results for antinuclear factor were negative. A Rose-Waaler test result was weakly positive.

The patient had remained well and was putting on weight when reviewed two months after dis-

charge from hospital, but was still reluctant to take solids. A chest radiograph was normal. Since then, the stricture has required repeated dilatation.

Discussion

Although no oesophagopericardial fistula was apparent, this was probably the cause of the patient's hydropneumopericardium. No gas-forming organisms were cultured from the pericardial fluid, and there was no suggestion of disease of the stomach or bronchi, which might have resulted in a fistula. Peptic oesophageal ulceration can penetrate locally, and may affect the aorta, bronchus, pleura, and lung, as well as the pericardium. Two of the 13 cases of non-neoplastic oesophagopericardial fistulae reported by Stephenson *et al* (1958) were due to peptic ulceration. All 13 cases were fatal, however, in contrast to the present case.

Failure to show the fistula radiologically, or a mucosal tear on endoscopy, together with the rapid recovery, suggests that the fistula was small and healed spontaneously. Similarly, in the only previously reported survivor after an oesophagopericardial fistula, a 6-year-old child (Dons *et al*, 1964), no fistula was detected radiologically, and no oesophageal mucosal perforation was seen,

even though at operation a fistula was shown leading from the pericardium into the oesophageal wall. Thus survival in both these cases was related to rapid spontaneous healing of the presumed fistula.

I thank Professor R H Girdwood and Mr P R Walbaum for permission to report on this patient, who was under their care, and for their helpful comments.

References

- Dons, N, Eriksen, K R, Ryssing, E, and Therkelsen, F (1964). Pyopneumopericardium with oesophagopericardial fistula. *Acta Chirurgica Scandinavica*, **128**, 766-770.
- Gossage, A A R, Robertson, P W, and Stephenson, S F (1976). Spontaneous pneumopericardium. *Thorax*, **31**, 460-465.
- Meyer, H W (1948). Pneumopyopericardium. *Journal of Thoracic Surgery*, **17**, 62-71.
- Stephenson, S E, Maness, G, and Scott, H W (1958). Esophagopericardial fistula of benign origin. *Journal of Thoracic Surgery*, **36**, 208-213.

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