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CASE REPORT

Ischaemic bowel within the thoracic cavity—An unusual cause of a pleural effusion

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Summary
Diaphragmatic defects are a rare complication following thoracic or upper gastrointestinal surgery. We present a case of a 78-year-old man who presented with ischaemic bowel that had herniated through such a diaphragmatic defect, 7 years after an oesophagogastrectomy for carcinoma. The patient was taken for an immediate laparatomy for resection of the infarcted bowel, and thereafter made an uneventful recovery. Patients found to have diaphragmatic defects should be considered for surgical repair to prevent this potentially life-threatening complication.

Case presentation

A 78-year-old man presented to the Accident and Emergency Department with a 24 h history of watery diarrhoea, vomiting and severe left-sided abdominal pain. He had become increasing dyspnoeic over the previous few hours, and described fevers and chills. He denied chest pain.

Prior to this acute illness he had been fit and well, with no active medical problems. He had undergone a curative oesophagogastrectomy 7 years earlier for a T2N0M0 oesophageal carcinoma. He had since been discharged from surgical follow-up. He took no medication and had no drug allergies.

On admission he was unwell, clammy and peripherally shut down. His pulse rate was 110 beats per minute, and his BP was 81/60 mmHg. His JVP was not elevated and he had a soft ejection systolic murmur. His respiratory rate was 16 breaths per minute, and his oxygen saturations were 95% on air. He had signs of a moderate right-sided pleural effusion with reduced breath sounds and dull percussion note. His

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abdomen was soft, but he was tender in the epigastric and right hypochondrial regions. Bowel sounds were absent, and digital rectal examination was normal.

Admission blood tests were as follows: haemoglobin 14.8 g/dl, white cell count 15,000 mm^{-3}, platelets 264 $\times$ 10^9 l^{-1}, sodium 138 mmol/l, potassium 4.1 mmol/l, urea 5.3 mmol/l, creatinine 111 μmol/l, alkaline phosphatase 204 U/l, alanine transferase 15 U/l, bilirubin 11 μmol/l, albumin 42 g/l, corrected calcium 2.27 mmol/l, C-reactive protein <5. An arterial blood sample taken on air showed a metabolic acidosis with respiratory compensation: pH 7.39, pCO_2 3.46 kPa, pO_2 12.5 kPa, HCO_3 15.3 mmol/l, BE-7.7. Electrocardiogram showed a sinus tachycardia with no signs of myocardial ischaemia. Chest radiograph showed a large left-sided pleural effusion (Figure 1). A diagnostic pleural tap revealed haemorrhagic fluid with pH 6.93 and protein of 45 g/l.

The patient was initially resuscitated with intravenous fluids and was treated with intravenous cefuroxime. Repeated arterial blood gas analysis on 40% inspired oxygen 2 h following admission showed a worsening metabolic acidosis: pH 7.13, pCO_2 3.4 kPa, pO_2 20.9 kPa, HCO_3 8.2 mmol/l, BE-19.2.

The rapidly worsening metabolic acidosis raised the possibility of bowel ischaemia. Furthermore, given the previous history of oesophagogastrectomy, the possibility of infarcted herniated bowel within the thoracic cavity was considered. An urgent abdominal and thoracic CT scan was therefore arranged. This revealed a left-sided diaphragmatic defect through which a long length of small intestine had herniated into the left pleural space. The small bowel was markedly dilated, fluid filed and showed abnormal circumferential thickening and enhancement. There was a large amount of fluid in the left pleural space between the small bowel loops, which together with the herniated bowel had led to compressive collapse of the left lung and mediastinal shift. The appearances were consistent with strangulated herniated bowel within the left hemithorax that had become ischaemic (Figure 2).

The patient was taken for an immediate laparotomy, at which most of the small bowel was found to be in the chest,

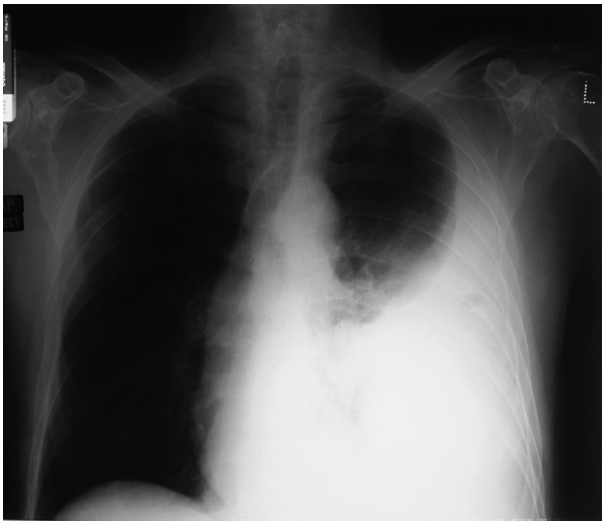

Figure 1 Chest radiograph on admission.

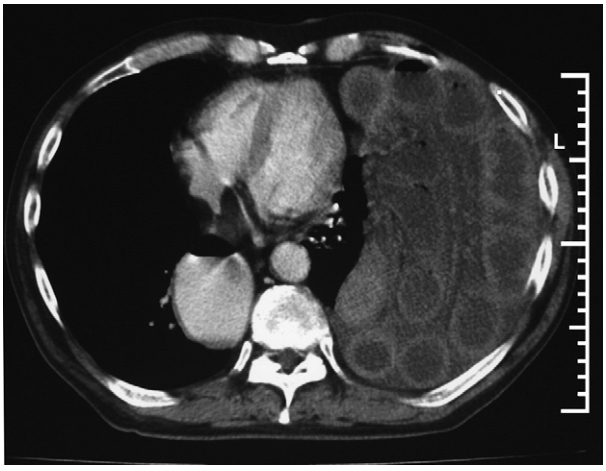

Figure 2 Computed tomographic scan of the thorax.

of which about half was infarcted. The gangrenous bowel was resected and the healthy bowel was returned to the abdomen. An end jejunostomy was formed, and the defect in the diaphragm was closed. Post-operatively the patient was transferred to the intensive care unit for continuing ventilation and treatment with vasopressors for septic shock. However, he made a rapid recovery, was extubated 24 h later, and was discharged from hospital following an uneventful recovery 10 days later. He remains well 6 months following discharge.

Discussion

Diaphragmatic hernia have been well described following blunt and penetrating trauma, and may occur in 3–7% of cases of thoracic or abdominal trauma.[1] Failure to diagnose the diaphragmatic defect at initial presentation can lead to presentation with strangulated bowel sometime many years following the original episode of trauma.[2] Diaphagmatic defects have also been described following coronary artery bypass grafting,[3] and oesophagectomy.[4]

Patients found to have diaphragmatic hernia of any aetiology should be considered for surgical correction of the defect to prevent the life-threatening complication of strangulated herniated bowel.

Patient consent

The patient has consented for this article to be published (signed consent form uploaded to site).

Competing interests

The authors have no competing interests.

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