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Short reports

Delayed rupture of the diaphragm presenting as gastric outlet obstruction

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Diaphragmatic hernia was first described in 1610 by Ambroise Paré, but it was not until 1886 that Riolfi reported the first successful repair of such an injury, caused by a stab wound. With the advent of the motor car and increasingly frequent road traffic accidents, traumatic herniation became more common and in 1925 Hedblom[1] found that, of a total of 378 cases managed operatively, 278 were traumatic in origin. In that series various techniques were used to close the diaphragmatic defect—direct suture, flap closure, or in some cases simply suturing abdominal viscera, usually stomach or colon, into the gap. In these early cases there was an operative mortality of 33·6%; as would be anticipated the mortality was increased considerably in the group complicated by strangulation.

More recent reviews show a similar pattern of aetiology, most diaphragmatic hernias being traumatic in origin, and the hernia being more commonly left sided, frequently containing the stomach.[2–4] Presentation is often delayed for many years, the symptoms varying from none whatsoever to those of life threatening cardiorespiratory compromise at an early stage.

The commonest presentation is of vague respiratory or abdominal complaints, on which may be superimposed obstruction of the gastrointestinal tract at any point from the oesophagus to the colon. In all series there appears a small group of uncertain aetiology; in a series of 112 cases from the Mayo clinic four patients gave no relevant history and in seven the diaphragm had been affected by an inflammatory process such as empyema or subphrenic abscess.[2] We describe here a case where a patient presented as an emergency with gastric outlet obstruction due to incarceration of the stomach in a large left sided diaphragmatic hernia some six months after successful decortication of a left sided empyema.

Case report

A 78 year old man with a 16 year history of peptic ulceration presented as an emergency in April 1983 with a perforated duodenal ulcer, which was treated by vagotomy and pyloroplasty. His postoperative progress was complicated by a left lower lobe pneumonia, which was treated with antibiotics. His temperature settled and he was allowed home, for review in the clinic. At the time of discharge he had a small persisting left sided pleural effusion. This effusion failed to resolve and in June he was readmitted for further investigation. The fluid from the effusion was sterile to culture, but the effusion persisted despite tube drainage and a pleural biopsy showed inflammatory tissue compatible with the wall of an empyema cavity. This was confirmed by computed tomography, which showed an empyema cavity with a wall about 1 cm thick.

In August 1983 he underwent decortication of this empyema cavity by an experienced thoracic surgeon and made an uneventful postoperative recovery. He was seen in the clinic in November and discharged from further follow up; at this stage his chest radiograph was satisfactory (fig 1). After a brief period of vague upper abdominal discomfort he presented again in March 1984 as an emergency with a two week history of nausea and vomiting, culminating in a 24 hour period when he had vomited everything he had taken by mouth. A nasogastric tube was passed and 1·5 litres of brown fluid aspirated. A chest radiograph on admission was interpreted as showing a raised left hemidiaphragm.

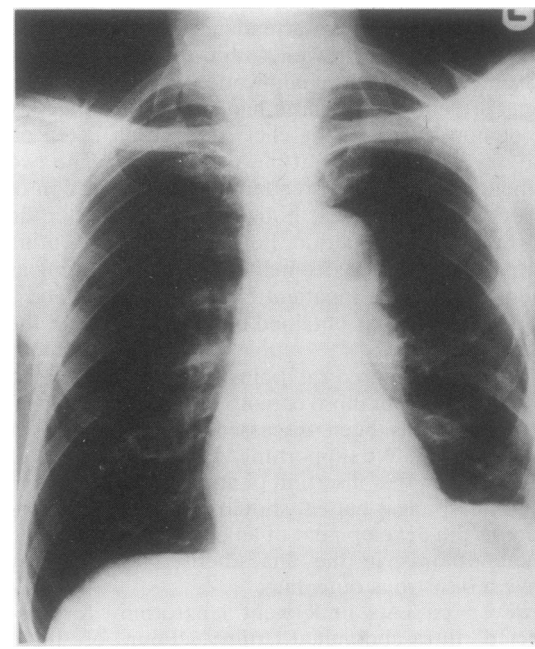

Fig 1 *Chest radiograph three months after the decortication showing a satisfactory postoperative appearance.*

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Attempts to recommence fluids provoked further vomiting and a barium meal examination was performed. This showed a large diaphragmatic hernia through which the stomach had prolapsed into the chest (figs 2 and 3). Parenteral nutrition was started and he subsequently underwent exploratory laparotomy. A large gastric volvulus through an 8 cm defect in the apex of the left hemidiaphragm was found. The stomach was returned to the abdomen and the spleen, which was adherent to the diaphragmatic defect, removed. The defect in the diaphragm was closed by direct suture with Ethiflex. Postoperative progress was uneventful and the patient was allowed home on the 10th postoperative day, eating normally. A follow up barium study confirmed the restoration of normal anatomy.

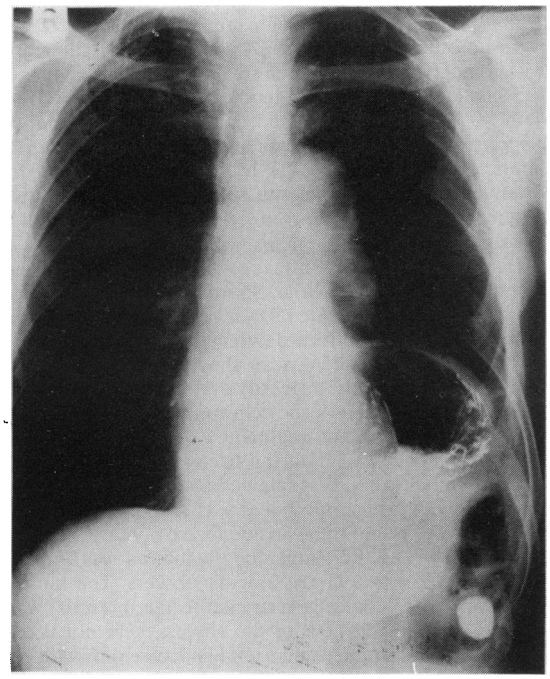

Fig 2 *Barium swallow showing the stomach herniating through the apex of the left diaphragm.*

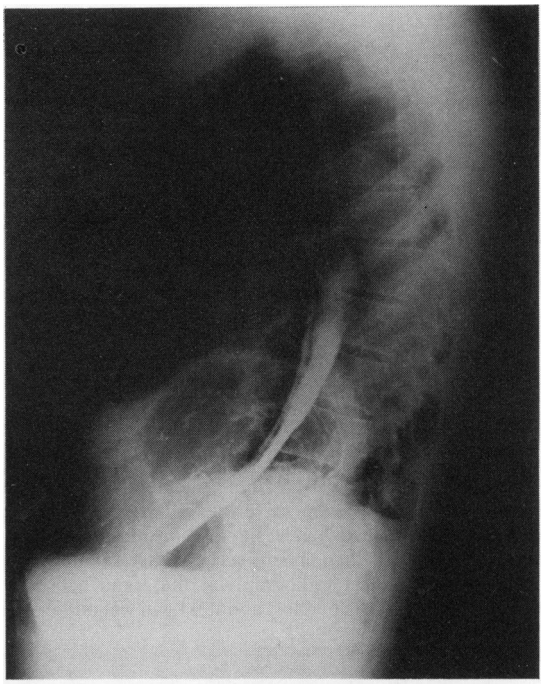

Fig 3 *A lateral film obtained during the barium study.*

Discussion

In retrospect the chest radiograph of March 1984 clearly shows a diaphragmatic hernia; most diaphragmatic hernias can be diagnosed with plain radiographic examination alone—although, as in this case, contrast studies may provide useful additional anatomical information. In cases where there is doubt about the diagnosis artificial pneumoperitoneum may be used to outline the diaphragm but this procedure is not without hazard.[5]

The aetiology of this patient's hernia is presumably related to weakening of the left hemidiaphragm by the infective process of the empyema and/or the subsequent decortication. The diaphragm appeared normal on the follow up radiographs; it is postulated that it subsequently ruptured spontaneously or as the result of some minor forgotten trauma, so that the hernia became apparent only when the stomach passed up through the defect and became obstructed. Although trauma remains the most common cause of diaphragmatic hernia an appreciable proportion will arise in other ways. Presentation is commonly delayed[2] and even in cases due to trauma the causal event may have been forgotten by the patient. A high index of suspicion on the part of the clinician will ensure early diagnosis and appropriate treatment.

We thank Miss A Jones for typing the manuscript.

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