

An unusual cause of massive pleural effusion in pregnancy

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ABSTRACT A 40 year old woman at 30 weeks of her eighth pregnancy presented with acute onset of dyspnoea and a large left pleural effusion after the onset of premature labour. A barium enema showed diaphragmatic rupture with intestinal contents in the thorax. Repair was accomplished through simultaneous left subcostal and thoracic incisions.

When rupture of the diaphragm occurs it usually results from major trauma to the abdomen, as in automobile accidents or crush injuries. Any condition associated with increased intra-abdominal pressure, however, may result in diaphragmatic rupture. We present the case of a woman 30 weeks pregnant with twins, who suffered rupture of her diaphragm with subsequent development of a pleural effusion while in premature labour.

Case report

A 40 year old woman (gravida 8, para 7) after 30 weeks of a twin pregnancy presented with nausea, vomiting, and abdominal pain of 12 hours' duration, which she did not think were caused by uterine contractions. Her pregnancy had been remarkable only for three weeks of nausea, vomiting, and diarrhoea, which had resolved spontaneously three weeks earlier. She had no history of abdominal trauma and her previous pregnancies had been uncomplicated.

She saw her private physician, who noted nothing abnormal on physical examination and found her to be in early labour. The premature labour was treated with intravenous magnesium sulphate and she was transferred to our institution for further monitoring. At the time of transfer she was no longer in active labour, and physical examination again showed nothing remarkable. Her blood pressure was 94/52 mm Hg, heart rate 120 beats/min, respiratory rate 18/min, and temperature 37°C. Ultrasound examination of the uterus showed two fetuses of a size appropriate for gestational age; there was no evidence of fetal distress.

Twelve hours after admission the patient developed dyspnoea on minimal exertion. Examination showed absent breath sounds in the lower left lung field and dullness to percussion in the same area. Abdominal examination showed no audible bowel sounds. With the patient breathing room air arterial oxygen tension was 7.5 kPa, arterial carbon dioxide tension 4.0 kPa, and pH 7.46.

A chest radiograph (fig 1) showed a large left pleural

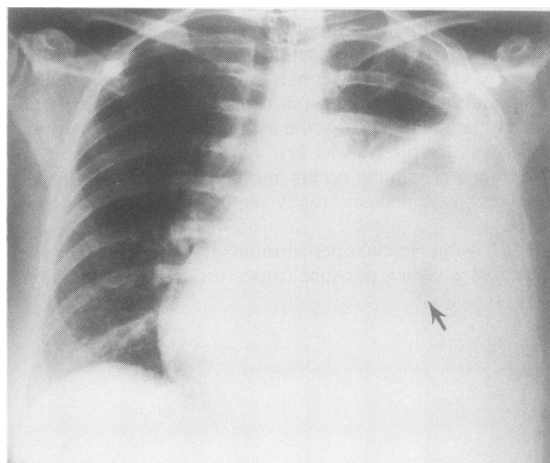


Fig 1 Chest radiograph showing a left pleural effusion, tracheal deviation to the right, and an extrapulmonary lucency in the left lung field (arrow).

effusion with a radiolucent area near the superior border suggesting extrapulmonary gas. Thoracocentesis with ultrasound guidance resulted in the removal of 1 litre of serosanguinous fluid; biochemical investigation indicated a transudate with pH 7.5, amylase 20 U/l, lactate dehydrogenase 82 IU/l, protein 10.0 g/l, and glucose 103 mg/100 ml (5.7 mmol/l) (coincident serum values were lactate dehydrogenase 141 IU/l, protein 50.5 g/l, glucose 110 mg/100 ml (6.1 mmol/l)). No organisms were seen in the Gram stained pleural fluid.

A ventilation-perfusion scan was non-diagnostic and a pulmonary angiogram showed normal vessels compressed in the left upper thorax. Thirty six hours after admission a repeat ultrasound examination of the area of dullness showed air filled structures above the diaphragm. A dilute barium enema (fig 2) showed a focal narrowing in the transverse colon with bowel above the left hemidiaphragm. Betamethasone was given to stimulate fetal lung maturation and intravenous magnesium sulphate was continued to control labour.

Four days after admission the patient underwent surgical repair of her diaphragmatic hernia via simultaneous left thoracic and subcostal incisions. An 8 cm defect was noted in the central tendon of the left hemidiaphragm, through which part of the stomach, small intestine, omentum, and transverse colon had herniated. These were reduced and a 2 cm section of gangrenous small bowel, which had been incarcerated in the defect, was resected. Both fetuses remained

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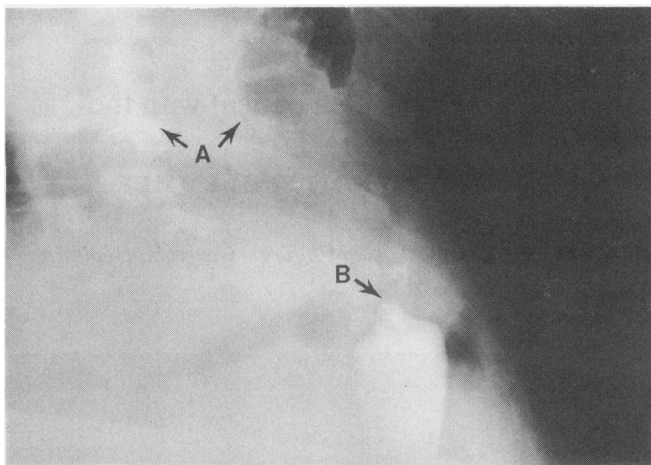


Fig 2 Barium enema showing contrast filled viscus in the left hemithorax (A) and narrowing of the colon (B) as it herniates through the left hemidiaphragm.

stable throughout the procedure and the patient did well postoperatively.

The patient went into labour three days after operation and was delivered by caesarean section under epidural anaesthesia. Twin 1, a girl weighing 1930 g, developed respiratory distress and died at 5 days. Twin 2, a 1630 g boy, did well and was discharged home with the mother.

Discussion

Rupture of the diaphragm without a history of trauma is a rare and potentially lethal complication of pregnancy.¹⁻³ The presentation and treatment are similar to those of rupture due to trauma. In women diaphragmatic rupture from any cause is relatively rare. Among 190 000 hospital admissions reviewed by Diddle and Tidrick, only 11 women had non-hiatal diaphragmatic hernia.³ They did not specify whether these were associated with trauma or pregnancy. In reviewing reports published since 1966 we could find no cases of non-traumatic rupture of the diaphragm in pregnancy.

Symptoms associated with herniation of abdominal contents across the diaphragm may be due either to intestinal obstruction, leading to nausea, vomiting, and abdominal distension, or to cardiopulmonary embarrassment with palpitations, dyspnoea, cough, or chest pain. The variety of symptoms makes the diagnosis of diaphragmatic rupture difficult.

A large pleural effusion is an unusual manifestation of diaphragmatic rupture, and when it occurs is usually exudative.⁴ We suggest that the rapid onset, chemical characteristics, and volume of fluid in this case may be explained by the fluid dynamics of the small intestine. The normal small bowel is capable of exchanging large volumes of fluid between the bowel lumen and the vascular space. In 30 minutes, 5 litres of fluid is transported from the blood to the

lumen and 5.5 litres moves from the lumen into the vascular space. This net movement of fluid into the blood is reversed in cases of intestinal obstruction, and fluid is lost into the lumen and bowel wall. The transudate from the serosal surface then collects as free fluid, the volume of which depends on the extent to which the intestine is affected.⁵

Once the possibility of diaphragmatic rupture is considered, the diagnosis may be confirmed by chest radiography, by passage of a nasogastric tube, or, as in our case, by barium contrast studies. Chest radiographic criteria for diaphragmatic hernia include an arch like shadow suggesting a raised diaphragm, extrapulmonary lucencies or densities above the diaphragm, shift of the heart or mediastinum away from the defect, and plate like atelectasis adjacent to the raised hemidiaphragm.⁶

We present this case as illustrating a potentially fatal cause of pleural effusion in pregnancy. Although rare the diagnosis, once considered, is easily confirmed and definitive treatment is available.

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