failed to show any reason for her initial haematological problems but confirmed the presence of an infected haematoma in the thigh. There were no features of disseminated intravascular coagulation, but by this stage she had recovered clinically from this disorder.

Comment

This case illustrates the need to consider the diagnosis of disseminated intravascular coagulation in any patient with unexplained thrombocytopenia. There were no apparent precipitating factors or clinical features to suggest this disorder and, moreover, none were found at necropsy.

Diagnosis was dependent on the performance of routine laboratory investigations. A low fibrinogen concentration was particularly helpful, yet it was normal initially. That this should have been so emphasizes the need for serial assays of coagulation factors when a diagnosis of disseminated intravascular coagulation is being considered. Furthermore, a method measuring thrombin-clottable fibrinogen is to be preferred in these circumstances. The prothrombin time was slightly prolonged from the beginning, but did not alone raise the suspicion of disseminated intravascular coagulation. A long thrombin time was noteworthy. It is of interest that the partial thromboplastin time and the factor VIII level were normal. This re-emphasizes the observation that in disseminated intravascular coagulation a one-stage technique for measuring factor VIII levels may be misleading (Nossel *et al.*, 1969). Red cell fragmentation, absent at first, never was a major finding.

Confirmation of the diagnosis was strongly suggested by the response and sensitivity to heparin administration (see Chart). It is striking that a dose of 40 mg every six hours caused only a transient minor increase in fibrinogen and platelet levels, while a modest increase in dosage to 60 mg six-hourly was correlated with a significant rise in both (see Chart). A dilemma that is highlighted by this patient's illness is whether heparin should have been used at all and what indeed its role is in the treatment of disseminated intravascular coagulation.

The pathogenesis of the illness remains unknown. While idiopathic thrombocytopenic purpura is rare at this age—and hence not a diagnosis which should be accepted with equanimity

Platelet count, fibrinogen, and cryofibrinogen responses to steroid and heparin administration and withdrawal.

—the response to the administration and withdrawal of steroids seemed to support the diagnosis initially. It is probable, however, that the problem was one of disseminated intravascular coagulation *ab initio*. The steroid dependency is difficult to account for on this hypothesis and was certainly misleading. A fascinating alternative is that antigen-antibody reactions responsible for an initial idiopathic thrombocytopenic purpura-like illness precipitated subsequent intravascular coagulation.

This study was supported in part by grants from the National Institutes of Health (AM-00795, AM-05391, AM-05413, HE/AM-07652, AM-09115, and HE-12860-01).

References

- Nossel, H. L., Niemetz, J., Waxman, S. A. and Spector, S. L. (1969). *American Journal of Medicine*, **46**, 591.

Traumatic Diaphragmatic Transpericardial Hernia

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British Medical Journal, 1971, **2**, 629-630

Diaphragmatic tears due to blunt or penetrating wounds have been well documented since the original description by Ambroise Paré in the sixteenth century (quoted by Lindskog, 1961). We here report a case of traumatic rupture of the diaphragm and pericardium with herniation of abdominal viscera through the pericardial sac into the pleura.

Case Report

A 62-year-old writer was referred to the medical department for progressive anaemia. He had been admitted to hospital after a road accident 10 months previously with a fracture of the right acetabulum and blunt injury to the chest wall. The electrocardiogram showed ST elevations consistent with pericarditis, and in the case summary it was noted that "myocardial infarction cannot be excluded." He complained of weakness, effort dyspnoea, chest discomfort after meals, and intermittent constipation. He was pale, pulse 104, blood pressure 130/70. The chest was barrel-shaped and heart sounds were distant. Peristalsis could occasionally be discerned over the precordium. The abdomen appeared scaphoid in the prone position. The patient said that his abdomen had been protuberant before the accident and that he had noticed the sudden change and had even drawn attention to this fact.

Haemoglobin was 6.6 g/100 ml, haematocrit reading 26%. Occult blood was present in the stools. E.C.G. was normal. Chest x-ray film showed a large air bubble anterior to the heart shadow. On barium-enema examination part of the left colon was seen in the chest. Barium-meal examination showed volvulus of the stomach, with the fundus and pyloric region in the chest anterior to the cardiac shadow (Figs. 1 and 2).

When the lung was retracted at thoracotomy the heart was seen outside the pericardial sac, completely eviscerated. Most of the stomach and part of the large bowel together with the omentum were in the left pleural space, with a remnant of a torn mediastinal pericardium running across them (Fig. 3). With the abdominal viscera replaced a tear extending 15 cm could be seen through the base of the diaphragmatic pericardium and the diaphragm beneath it, running from the sternum posteriorly and to the left and through the muscular and tendinous layers of the dia-

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FIG. 1—X-ray film after barium enema. Remains of barium in left colon in left chest.

FIG. 2—Stomach in 180° volvulus almost wholly in chest anterior to heart.

phragm. The tear in the diaphragm was repaired in two layers with the remnants of the torn pericardium.

FIG. 3—Drawing at time of operation. Heart is outside pericardium, stomach, bowel, and omentum in the pleural cavity having gone through the mediastinal pericardium.

Postoperatively the abdomen took on its normal protuberant configuration. The patient has returned to full activity.

Comment

Crawshaw (1952) described the first case of traumatic hernia resulting from a tear of the diaphragm and pericardium. That the condition is rare can be seen from Robb's (1963) series of 410 cases of diaphragmatic hernia, of which 17 were traumatic; only one had a tear of the pericardium and diaphragm. All reported cases but one (Beddingfield, 1968) caused by a knife stab have been due to blunt trauma, usually as a result of a road accident.

In the so-called chronic stage cardiorespiratory embarrassment and/or dyspeptic symptoms predominate. In our patient symptoms were attributable to abdominal viscera in the chest with occult bleeding from the stomach in volvulus. The dramatic change in the configuration of the abdomen was an excellent clue to the migration of abdominal contents. This sign has not been described to our knowledge in cases of traumatic diaphragmatic tears. Absence of cardiac embarrassment is accounted for by the additional tear in the mediastinal pericardium, allowing for escape of bowel into the pleural space. In this respect our case differs from those previously reported where the pericardial sac also formed the pseudosac of the diaphragmatic hernia.

References

- Beddingfield, G. W. (1968). *Annals of Thoracic Surgery*, 6, 178.
- Crawshaw, G. R. (1952). *British Journal of Surgery*, 39, 364.
- Lindskog, G. E. (1961). *Journal of Thoracic and Cardiovascular Surgery*, 42, 1.
- Robb, D. (1963). *British Journal of Surgery*, 50, 664.