She was admitted to this hospital in April 1975, when she complained of persistent dyspnoea, malaise, and chest pain. She was thin, dyspnoeic at rest, but not cyanosed or anaemic. There were widespread crepitations throughout both lung fields and some expiratory bronchospasm. Investigations were arranged because of a history of diarrhoea and the results were as follows: haemoglobin 13.6 g/dl, white cell count $11 \times 10^9/l$ ($11\,000/mm^3$), total protein 65 g/l, albumin 42 g/l, and globulin 23 g/l. The result of the latex test for rheumatoid arthritis was negative. Serum carotene was $1.0 \mu\text{mol/l}$ ($55 \mu\text{g}/100 \text{ ml}$), serum vitamin B₁₂ 875 ng/l (normal range 150-900 ng/l), and serum folate 0.8 $\mu\text{g/l}$. Within five hours 16% of a 25-g dose of xylose was excreted. There was no antinuclear factor. Calcium concentration was 2 mmol/l (8 mg/100 ml). Faecal fats were normal. Chest x-ray examination showed diffuse fibrotic changes throughout both lung fields—overall appearances that would be consistent with farmer's lung. Jejunal biopsy findings were reported as follows: "Stunting of mucosal villi with an increase in the chronic inflammatory cell content in the lamina propria. The appearances are those of partial villous atrophy."

Discussion

Both of these patients presented initially with respiratory symptoms and gluten enteropathy was only subsequently detected. In retrospect, however, symptoms had been present for some time. Non-specific symptoms are the presenting factors in many adults with coeliac disease and in any population there are probably many people with undiagnosed coeliac disease. Recently Berrill *et al*¹ have suggested that there is a strong case for routine jejunal biopsy in patients with bird fancier's lung. The same may apply to farmer's lung.

In most of the recent reports^{1,2,17} avian antigens have been detected in the patients with pulmonary disease associated with coeliac disease but the role of these antigens is not clear. The recent report of Berrill *et al* (incorporating two cases originally described in 1970¹⁸) showed that five out of 16 patients with bird fancier's lung had total or subtotal villous atrophy and had positive avian precipitins with a history of contact with birds.¹ In another report, however,³ avian antigens were not implicated in any of the three cases of diffuse interstitial lung disease found among a population of 24 patients with coeliac disease. In a case of coeliac disease associated with extrinsic allergic alveolitis reported by Scadding² precipitins to pigeon antigens were

present in the blood, and bronchial challenge with pigeon serum produced a febrile reaction after four hours.

Morris *et al*¹⁷ found precipitins against various avian antigens in coeliac patients, although there was no history of close contact with birds. Close contact is not necessary, however, as evidenced by cases of pigeon fancier's lung reported in the wives of two pigeon fanciers who were only indirectly exposed to avian antigens.¹⁹ A patient of my own who is a poultry farmer had coeliac disease first diagnosed when he was referred because of positive avian precipitins without pulmonary disease. Tests for allergic alveolitis were done by his general practitioner because the patient had complained of general ill-health and weight loss. This patient responded well to a gluten-free diet and the avian precipitins became negative.

Further screening of patients with coeliac disease and diffuse lung disease associated with immunological abnormalities including intrinsic allergic alveolitis is necessary to tell how often these conditions co-exist.

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SHORT REPORTS

Inversion of cervix uteri at caesarean section

Partial inversion of the fundus uteri is sometimes observed during caesarean section due to forcible or unwise traction on the cord while attempting to deliver the placenta, but inversion or "invagination" of a partly effaced and dilated cervix into the lower uterine segment at the time of caesarean section is probably never seen by most obstetricians. The extreme rarity of this condition is probably why it has not been reported.

Case report

A 29-year-old primigravida, whose expected date of delivery was 16 June 1975, was admitted to the university obstetric unit on 14 June for her delivery. Since on obstetric examination there was some doubt about the presentation, an x-ray film was ordered which showed a "breech" with extended legs and arms. As the pelvis was adequate and the baby's weight assessed at 2500-3000 g, an assisted breech delivery was contemplated. She went into spontaneous labour on 24 June and an internal examination showed a non-effaced cervix about 6 cm dilated. During labour the fetal

heart rate suddenly became rapid (varying from 160 to 180/min), and since even at this stage (nearly 12 hours after the onset of labour) the cervix was only partly effaced and 6-8 cm dilated a caesarean section was decided on. A routine lower segment caesarean section was performed and the fetus delivered without much difficulty except for some resistance encountered while delivering the head. When the suturing of the uterus was about to start we noticed a pouch-like protrusion, which partly occupied the lower segment. The upper end of the pouch, which extended to almost the level of the uterine incision, was open with a somewhat wavy margin while below it communicated with the vagina (see fig). As the identity of this "pouch" appeared uncertain, and it was lying freely mobile within the lower segment, its open upper margin was partly sutured to the decidua of the lower segment with interrupted catgut. This was wrong as at the end of the operation when the vagina was swabbed we noticed that there was hardly a cervix to be felt, although it had been only partly effaced just before the caesarean section. It was only then that the possibility of invagination (inversion) of the cervix was suspected, which was what had actually happened.

In view of this diagnosis it was decided to divide the sutures anchoring the invaginated cervix to the decidua and reduce the inversion. An attempt towards achieving this was made (nearly four hours after the caesarean section) from below (vaginally), under general anaesthesia, with the patient in the lithotomy position, and the manoeuvre proved successful—the anchoring sutures were identified and divided; the inverted cervix was then hooked with the finger and drawn down, in stages, into the vagina and the inversion corrected. The patient made an uneventful recovery and was discharged on 4 July.

Diagrammatic view of inversion of cervix uteri (as seen at time of the caesarean section).

Comment

The mechanism actually responsible for this complication, when it occurs in any patient, is perhaps very difficult to explain. Sometimes sheer unfamiliarity with very rare complications such as this may lead to perplexity in diagnosis as in this case.

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Subacute cardiac rupture after myocardial infarction

Although cardiac rupture after acute myocardial infarction is usually considered to cause immediate death, the condition of subacute rupture has recently been recognised.^{1 2} We describe the features of this condition and its treatment by surgery.

Case report

A man of 53 was admitted on 28 January 1974 with an anterior sub-endocardial infarct, which four days later extended to a transmural infarct. He was not given anticoagulants. A pericardial friction rub was heard on the sixth day. He was normotensive and his progress was uneventful until the 10th day, when a cardiac arrest occurred with profound nodal bradycardia. With resuscitation he regained consciousness after 30 minutes. His toes and fingers were cyanosed and cool, and there was gross distension of the jugular veins. There was a sinus tachycardia, an unrecordable blood pressure, weakly palpable femoral pulses without paradox, a gallop rhythm, and a soft pericardial friction rub. The electrocardiogram and chest x-ray films were essentially unchanged. Angiography 11 hours later showed a pericardial effusion and excluded pulmonary embolism (see fig). During attempted pericardial aspiration cardiac asystole developed, but with resuscitation his previous clinical state was restored.

Three hours later at left thoracotomy under partial cardiopulmonary bypass using the left femoral vessels, 200 ml of blood clot, some of it quite fresh, was removed from the pericardium where it had collected, especially over the posterior and right sides of the heart, compressing the right atrium and the great vessels. A large infarct of the whole of the anterior surface of the left ventricle extended to the interventricular groove. Dark plum-coloured blood was oozing from a flap-like fissure 1.5 cm long just below and anterior to the margin of the left atrial appendage, and also from multiple punctate sites. On the posterior surface of the infarct there was a lesion 0.5 cm long, which may have been produced surgically. As the infarct was

Right atrial cine angiogram demonstrating pericardial effusion.

too large to be excised, the two lesions were sutured with interrupted mattress sutures buttressed with teflon strips. On the second attempt the patient came off bypass with isoprenaline support. The pericardial cavity was infused with saline over the first 48 hours. Recovery was uncomplicated except for a left pleural effusion of 770 ml of straw-coloured fluid.

The patient has returned to full-time sedentary work and has mild dyspnoea (grade 2A). He is receiving digoxin, frusemide 40 mg, and Slow-K daily. He has signs of a large dyskinetic area over the previous infarct.

Comment

Cardiac rupture is usually considered to be sudden event with a complete tear through the ventricular wall. Pathological studies, however, often show a progressive recurrent leakage in a stepwise manner through the myocardium.^{3 4} Potentially, when the blood reaches the pericardial space it may compress the layers of the tract and stem further leakage. When the rupture into the pericardium occurs the patient will deteriorate suddenly and usually then proceed to a cardiac arrest with bradyarrhythmias and asystole. If the patient survives the initial rupture or is resuscitated from the arrest he then presents a characteristic syndrome. There is profound hypotension with severe right ventricular failure shown by gross jugular venous distension. There is, however, minimal evidence clinically or radiologically of left ventricular failure. Pulsus paradoxus may be absent.¹ Urgent angiography is needed to confirm the diagnosis and distinguish it from massive pulmonary embolism. Pericardial aspiration may be attempted but the definitive treatment is surgical⁵ where suture of the tears, rather than infarctectomy, may be sufficient.

We thank Dr John Walker for radiological help and advice.

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³ Levene, A, *British Heart Journal*, 1962, 22, 660.

⁴ Lautsch, E V, and Lanks, K W, *Archives of Pathology*, 1967, 84, 264.

⁵ *British Medical Journal*, 1972, 2, 310.

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