The liver showed focal necrosis with intrasinusoidal fibrin, and the spleen showed recent infarction and associated fibrin. The kidneys contained fibrin deposits in the capillary loops of some glomeruli but no hypertensive vascular changes (fig 2). The adrenals showed thickened cortices composed of a mixture of

extremely hypertrophied compact and clear cells. Large islands of clear cells and focal necrosis in association with intrasinusoidal fibrin were present in some areas (fig. 3). The vertebrae showed a cellular marrow and evidence of microfracture.

Comment

The haematological parameters in association with scattered microthrombi at necropsy are strong evidence for the occurrence of disseminated intravascular coagulation in this case.

Release of tissue thromboplastin from necrotic tumour has been implicated in the induction of a hypercoagulable state (Amundsen *et al.*, 1963) but the small size of the tumour and absence of invasion strongly militates against this being a significant mechanism in this patient.

We think that the cushingoid state played a pivotal part in engendering disseminated intravascular coagulation in this case and are unaware of any previous case reports describing this association. The danger of such a state being engendered and increasing the morbidity and mortality of Cushing's syndrome seems to be great in view of the tendency to high levels of clotting factors, depressed reticuloendothelial system, increased susceptibility to infection, and in some cases associated tumours.

We are indebted to Professor D. S. Munro for permission to report this case.

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FIG. 3—Adrenal cortex showing cellular hypertrophy and intrasinusoidal fibrin. (Phosphotungstic acid and haematoxylin. $\times 220$).

Meningococcal Pericarditis without Meningitis

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Epidemics of meningococcal infection have become uncommon with improvements in standards of hygiene and reduction of overcrowding in susceptible groups. Isolated cases continue to be seen but are very rare without meningitis. We have been able to find only one previous report of meningococcal pericarditis occurring in the absence of meningitis (Orgain and Poston, 1939).

Case Report

A 17-year-old Welsh girl was admitted with sharp central chest pain, worse on inspiration, which had come on suddenly two hours previously. Past medical history and symptomatic inquiry produced no explanation.

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On examination she was pale, distressed, and short of breath but not cyanosed. Her temperature was 38.5°C, pulse rate 120/min and regular, blood pressure 140/85 mm Hg, and the jugular venous pressure was not raised. No abnormality was found on auscultation of the heart. The haemoglobin was 9.6 g/100 ml, E.S.R. 5 mm in one hour (Westergren), white blood count 15,700/mm³ (80% neutrophils). A chest radiograph showed nothing abnormal. An E.C.G. showed minimal S-T rise in leads I, aVL, V5, and V6. Later that day she developed neck stiffness, but lumbar puncture produced normal, sterile cerebrospinal fluid.

Fourteen hours after admission her blood pressure dropped to 70/40 mm Hg, jugular venous pressure was raised 5 cm, pulse rate was 140/min, and a pericardial rub was heard at the left lower sternal edge. She was still in considerable pain. An E.C.G. showed further S-T rise now involving leads I, II, aVL, aVF, and V1 to V6 (fig. 1). With the raising of the foot of the bed, the

FIG. 1—E.C.G. lead I on the first day, after operation, and six months later.

blood pressure rose to 100/50 mm Hg. She was treated with aspirin until two days later, when three blood cultures taken on admission grew *Neisseria meningitidis*, and treatment with penicillin, to which it was sensitive, was started. After treatment for seven days, the jugular venous pressure remained raised and there was pulsus paradoxus. A pleural rub and bronchial breath sounds were noted over the left lower lobe. A clinical diagnosis of pericardial effusion and left lower lobe consolidation was supported by a chest radiograph showing an enlarged cardiac shadow and collapse consolidation of the left mid and lower zones (fig. 2). The effusion was tapped on five successive days, with short-lived improvement each time. The pericardial fluid was sterile but contained a heavy deposit of polymorphs.

FIG. 2—Chest radiographs on the eighth day, (A) before and (B) after pericardial aspiration.

Two weeks after admission a left thoracotomy was carried out at which blood clots and soft yellow fibrinous material were evacu-

ated from the pericardial cavity and stripped off the ventricles. Postoperatively, an E.C.G. showed generalized T wave inversion. She required digoxin and diuretics for heart failure but improved rapidly; the temperature settled and penicillin was stopped a week after operation. She was discharged home a month after admission and was symptom-free after two months. Ten months after discharge she had a baby, at which time an E.C.G. showed nothing abnormal.

Comment

It is postulated that meningococcal meningitis arises as a result of pharyngeal infection causing a bacteraemia with meningeal seeding. Any organ is susceptible, but the most common extrameningeal sites are the lungs, joints, eyes, serous membranes, testicles, and heart (Lukash, 1963). The incidence of cardiac complications has never been established. Lowe and Diamond (1948) found the incidence of pericarditis in various series of meningococcal meningitis to vary from 0 out of 654 to 12 out of 280, while for myocarditis it ranged from 0 out of 280 to 58 out of 144. This variation may be explained by rather vague clinical criteria for the diagnosis of myocarditis, but Gore and Saphir (1947) reported 256 cases of meningococcal infection studied post mortem, out of which 111 had microscopical evidence of myocarditis.

The present patient undoubtedly had pericarditis in the absence of meningitis. Whether there was additional myocarditis is less certain, but the development of heart failure after pericardectomy suggests the possibility. The accompanying T wave changes on the E.C.G. may have been due to myocarditis or to anoxia of the peripheral myocardium from pressure of the effusion (Urschel *et al.*, 1945).

Another unusual feature was the time of onset of pericarditis in relation to the illness. Her first symptom was pain, presumably due to pericarditis. In a series of 12 patients reported by Herrick (1918), pericarditis occurred one to three weeks after the onset and Lukash (1963) concluded that "pericarditis is a feature of the later period of the acute stages of the disease."

Orgain and Poston (1939) described the only other reported case of meningococcal pericarditis without meningitis. The patient was a 32-year-old clergyman who presented with central chest pain of two weeks' duration. Blood cultures were initially sterile, though pericardial fluid culture produced the organism. There was no evidence of meningitis. He was treated with sulphonamide and antimeningococcal serum and made a complete recovery.

We would like to thank Dr. P. A. Thorn for allowing us to report this case.

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