swelling of the legs. He was treated with rifampicin and dapsone from the outset, his reaction being controlled by prednisolone and thalidomide. Progress was uneventful, and nine weeks later leg oedema had virtually disappeared, superficial veins were normal, and he could breathe freely through his nose for the first time in several years.

Comment

This case is reported in detail because experience shows that in non-endemic areas, including Britain,[1] patients may present in extraordinary ways, often at special clinics, and with long delay before the diagnosis of leprosy is considered. The causes of brawny oedema of the legs in lepromatous leprosy almost certainly embrace factors concerned in non-specific panniculitis,[2] including cold, gravity, infection, stasis, and the unusual arrangement for the blood supply of subcutaneous fat. But additional factors may include (*a*) damage to autonomic nerves, causing increased capillary permeability[3]; (*b*) heavy parasitisation of endothelial lining cells by the bacillus; and (*c*) damage to blood vessel walls during immune-complex reactions.

Swelling of the hands and feet in leprosy may repay further study; the recent discovery[4] that after long periods of treatment for lepromatous leprosy the fingers may be the skin site with the highest bacterial load and the highest number of solid-staining bacilli originated in a clinical discussion on the significance of oedema in the fingers.

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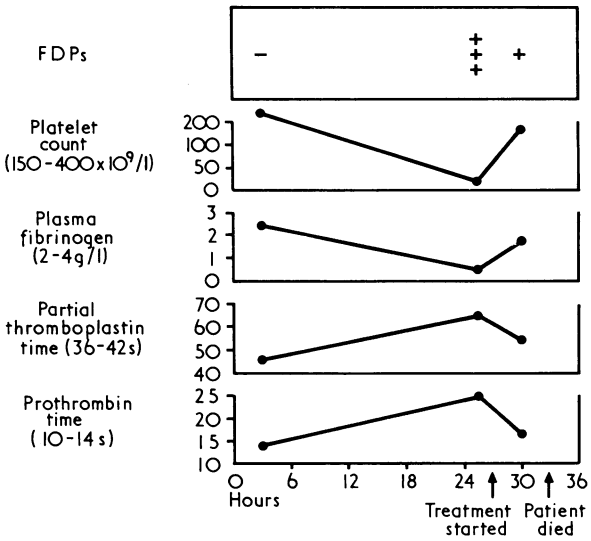
Dissecting aortic aneurysm and disseminated intravascular coagulation

Disseminated intravascular coagulation has numerous diverse causes.[1] Large clots, as in abruptio placentae and giant cavernous haemangioma, occasionally result in this syndrome. Severe disseminated intravascular coagulation is very rarely reported in acute aortic dissection.[2]

Case report

A 62-year-old woman with a history of hypertension was admitted to hospital as an emergency with acute pulmonary oedema. The previous day she had experienced mild retrosternal pain radiating to both arms. Examination showed acute heart failure with an early diastolic murmur maximal at the left sternal edge. All pulses were present and equal. The chest *x*-ray film showed widening of the aortic shadow in addition to pulmonary oedema. There was left ventricular hypertrophy and general T-wave flattening on the electrocardiograph. An arch aortogram showed a small false lumen in the media of the ascending aorta, without involvement of the major aortic branches. Surface scanning after intravenous ^{131}iodine-labelled fibrinogen showed a large clot in the false lumen. Conservative hypotensive therapy was instituted with methyldopa and guanethidine. Twenty-four hours later petechiae and bleeding from venesection and intravenous infusion sites were noted. The haemoglobin was 8·9 g/dl. Schistocytes and microspherocytes were seen on the blood film. The figure shows other coagulation parameters. The aspartate transaminase was 2400 IU/l (normal 10-30). Bleeding persisted and low-dose heparin (5000 units subcutaneously twice daily) treatment together with clotting factor and platelet supplements was begun. The bleeding diathesis improved over the next six hours (figure), but she then developed cardiac tamponade, collapsed, and died three hours later. Necropsy showed a 0·5-cm tear in the posterior wall of the ascending aorta and a 440-g clot containing ^{131}iodine-labelled fibrinogen in the false lumen. Aneurysmal rupture into the pericardium had occurred. The liver was intensely congested and microscopic examination showed considerable centrizonal necrosis. Fibrin clots were found in the microvasculature of the liver and kidneys.



Results of coagulation studies. Normal ranges are given in parentheses.

Discussion

Aortic aneurysm is an unusual cause of disseminated intravascular coagulation,[2] only two cases having been reported.[3 4] Abruptio placentae and giant cavernous haemangioma may also cause disseminated intravascular coagulation. The common denominator in all these conditions is the formation of a large extravascular blood clot, as seen in our patient.

The aetiology of disseminated intravascular coagulation may be classified into three processes[1]: (1) vascular endothelial cell damage, which activates Hageman factor and the intrinsic clotting system; (2) tissue injury, which activates extrinsic clotting; and (3) red-cell or platelet injury with the release of coagulant phospholipids. Probably all three processes were active in this patient. Large clots alone, as in abruptio placentae,[5] do not cause consumption coagulopathy: probably thromboplastic substances are released, initiating generalised intravascular coagulation as shown in this patient by the presence of fibrin clots in the microvasculature of both liver and kidneys. A further factor was probably liver injury, which, though rarely the sole cause of disseminated intravascular coagulation, is often a contributory aetiological factor.[1]

The definitive treatment for disseminated intravascular coagulation is removal of the cause: nevertheless, the administration of clotting factors, platelets, or heparin may be of temporary benefit.[1] Heparin therapy, although hazardous, was used with good results in the patient of Bieger *et al* as a prelude to surgery. In our patient low-dose heparin used together with clotting factors and platelets was beneficial and did not apparently contribute to her death.

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