swelling of both ankle joints the next day. He complained of limping. At 16:00 hours on 15 September he developed sudden pain in the left side of the scrotum, which rapidly increased in severity. The purpuric rash now involved both lower extremities, the perineum, the suprapubic region, and both buttocks. The patient was the second of three boys in the family. He was born at full term, delivered normally, and the birth weight was 3.5 kg. There was no family history of any bleeding diathesis nor was there a history of bleeding or a similar episode of anaphylactoid purpura in this child.

The child was in severe pain from the obviously inflamed swelling on the left side of the scrotum. He was afebrile. A typical urticarial and purpuric rash with the distribution described together with arthralgia of both ankles suggested a diagnosis of Henoch-Schönlein syndrome. There were many purpuric spots on the scrotum particularly on the left side, which was swollen and looked inflamed. Both testes were palpable. The left testis was drawn-up and tense, felt enlarged, and was very tender. The right testis was normal. A diagnosis of Henoch-Schönlein syndrome with vasculitis of the left testis was made. In view of the clinical findings torsion of the left testis was suspected, and we decided to explore the scrotum.

At operation all the layers of the left testis were haemorrhagic and oedematous. It was considerably enlarged, blue, and congested due to the obvious torsion. The cyst of Morgagni was congested and haemorrhagic. Vasculitis was also present. The testis was untwisted and its colour returned to normal in a few minutes. It was then fixed in the usual manner. The right testis was not explored.

The patient was well postoperatively; the oedema in the scrotum disappeared and there was no increase in the extent of the purpuric rash or in the arthralgia. He remained well until 22 September 1973 when he had bloody diarrhoea with melaena and vomiting. The urine contained a few red blood cells but there was no other evidence of glomerulonephritis. He complained of severe pain in the abdomen. He responded to treatment and his symptoms quickly improved. On 26 September 1973 the haemoglobin was 12 g/100 ml; W.B.C., 11,600/mm³; platelets 315,000/mm³. He was symptomless and the urine and stools were negative for blood. When he was discharged on 7 October 1973 he had no purpuric rash or arthralgia, and the scrotum was normal.

Comment

This patient was admitted to hospital with the classical

features of the Henoch-Schönlein syndrome, the rash and arthralgia appearing before the testicular involvement. There was no evidence of glomerulonephritis though the severity of the gastrointestinal lesion was shown by the bloody diarrhoea. Testicular involvement in the Henoch-Schönlein syndrome is uncommon despite an incidence of 38% in 20 cases reported by Sahn and Swartz (1972). Apart from these and the cases reported by Fitzsimmons, we know of only five other reported cases (Noussias, Blandy and Ward-McQuaid, 1969). None of the published cases of testicular involvement was complicated by testicular torsion, though some were surgically explored (Noussias, Blandy and Ward-McQuaid, 1969). Vasculitis and bleeding into the gastrointestinal tract is a common complication of the Henoch-Schönlein syndrome and presents with abdominal pain and sometimes bloody diarrhoea. Vasculitis in the gastrointestinal tract is rarely the site for an intussusception (Gairdner, 1948) and this can make diagnosis difficult as in this case, where vasculitis in the testis was complicated by testicular torsion. When testicular involvement occurs in the Henoch-Schönlein syndrome vasculitis is undoubtedly the cause of the pain and swelling in the testis. In the present case oedema and haemorrhagic vasculitis were seen in the testis at operation. Vasculitis in the testis predisposes to testicular torsion just as vasculitis in the gastrointestinal tract predisposes to intussusception. Sahn and Schwartz (1972) thought surgical exploration unnecessary when testicular involvement complicates the Henoch-Schönlein syndrome. In view of the testicular torsion in this patient we recommend that it should be considered as a differential diagnosis and that surgical exploration should be carried out when there is testicular involvement.

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Unusual Presentation of Tuberculosis

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Unusual presentations of tuberculosis in the middle-aged and elderly are becoming more common, and yet in many cases the diagnosis is made only at necropsy. Often leucopenia is present, especially when the spleen is involved (Evans *et al.*, 1952; Fountain, 1954; Medd and Hayhoe, 1955; Cooper, 1959; Glasser *et al.*, 1970; Hansson *et al.*, 1972). The clinical condi-

tion is frequently diagnosed as either a malignant blood disease or a "connective-tissue disorder" and the patient treated with corticosteroids or cytostatic agents. In the present case of disseminated tuberculosis the clinical picture of arthralgia and fever and the presence of the L.E.-cell phenomenon and leucopenia led to a presumptive diagnosis of systemic lupus erythematosus (S.L.E.).

Case Report

A 53-year-old man with fever of unknown origin and arthralgia was referred to us in May 1972. He had previously had excellent health, apart from a non-specific chorioretinitis diagnosed in 1965, and was seen regularly by his doctor. In January 1972 he suddenly developed arthralgia. In addition he had a slight, non-irritating rash on his chest, which lasted two weeks, and experienced temperatures of up to 39°C in the evenings. Antipyretics had no effect but the fever caused him little discomfort. During the six months before referral he had lost 5 kg in weight.

Physical examination showed nothing abnormal; in particular there was no enlargement of the lymph nodes, liver, or spleen and the skin was normal. Pulse rate 88/min; blood pressure 130/70 mm Hg; E.S.R. 99 mm in the first hour; packed cell volume 33%; W.B.C. 2,900/mm³ (50% non-segmented neutrophils, 33.5% segmented neutrophils, 1.5% monocytes, 15% lymphocytes).

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Results of liver function tests were normal with the exception of alkaline phosphatase (126 units/l.). Serum iron was 30 $\mu\text{g}/100\text{ ml}$. Blood urea nitrogen and serum creatinine were within normal limits. A few typical L.E. cells were found and, in addition, free homogenized purplish nuclear material was present in the preparation. Antinuclear Ig-activity was revealed by immunofluorescence up to a serum dilution of 1:20. No antibodies were detected to natural or denatured DNA. Serum complement level was normal. Latex fixation test gave a negative result. Chest x-ray film was clear, with no suspicion of parenchymal foci. There was no response to a tuberculin test with 10 U purified derivative. Cultures of blood and urine were negative.

Diagnosis of S.L.E., obscure infection, and paraneoplastic syndrome were considered. Gastrointestinal radiography, skeletal survey, liver scanning, cholecystography, pyelography, and coeliac axis arteriography all failed to show a neoplasm and no evidence for an occult infection could be found. Thus the presumptive diagnosis was S.L.E., though the biological evidence was meagre.

The patient was given 60 mg prednisone daily and within a few days the fever abated and he was discharged for observation at home. Three days later, however, the fever returned. On the 13th day he was readmitted because of massive melaena. He had a high temperature and was jaundiced and covered with a thick petechial rash, mostly on the legs. His general condition had deteriorated markedly and he had lost 9 kg. His weight was then 55 kg (six months earlier it had been 70 kg). Physical examination still showed no enlargement of lymph nodes, liver, or spleen. The presence of intra-abdominal fluid was suspected. Auscultation and a chest film were inconclusive.

Laboratory data were as follows. Packed cell volume 23%; W.B.C. 550/mm³ (46% non-segmented neutrophils, 39% segmented neutrophils, 1% eosinophils, 2% myelocytes, 2% monocytes, 10% lymphocytes); platelets 40,000/mm³. Serum electrophoresis showed an acute-phase pattern with an increase of α 2-globulin (19%) but was otherwise within normal limits. Standard cultures of blood, urine, and bone marrow were negative. Bone marrow showed rich cellularity with a myeloid: erythroid ratio of 4:1; normal erythropoiesis; a myeloid series with a shift to the left and increased granulation; an increase of reticulum cells, which appeared singly or in clusters of 3 to 5 cells, often oval in shape; 3% plasma cells; and 15% lymphocytes. L.E. phenomenon and nuclear immunofluorescence were absent. Serum complement level was normal.

In view of these findings the diagnosis of S.L.E. could no longer be entertained. The bleeding was probably secondary to the development of an ulcer, especially in connexion with the diminution of the number of platelets.

An infectious aetiology was then considered in order to explain the marked inflammatory picture. With the reticulum cell reaction of the bone marrow, in conjunction with the peripheral leucopenia and the aggravation produced by cortisone treatment, emphasis was given to tuberculosis as the presumptive diagnosis. The patient was placed on broad-spectrum antibiotics and anti-tuberculosis agents (cephalosporin 2 g, isoniazid 600 mg, and ethambutol 1,200 mg daily). This led to a dramatic improvement. Five days after starting this therapy his temperature became normal. His appetite improved and he gained weight. After 10 days cephalosporin was removed from the regimen. Within two months he had gained 6 kg in weight. At the time of writing he was perfectly well.

During this patient's second admission cultures were made on Löwenstein's medium. Six weeks later, when he had already recovered under antituberculosis treatment, cultures of bone marrow and sputum were reported as positive for *Mycobacterium tuberculosis* (cultures of urine and cerebrospinal fluid remained negative). Laparoscopy, performed after therapy had been started, showed a few granulomatous lesions on the peritoneum. The fibrinous material obtained was examined microscopically after special staining; in one preparation a few acid-fast bacilli were detected.

Comment

In many cases atypical forms of tuberculosis are diagnosed only at necropsy, especially when they occur as haematological disorders (Hansson *et al.*, 1972). Proudfoot *et al.* (1969) showed that nearly half of the cases of disseminated tuberculosis in adults are "cryptic," presenting with a normal x-ray picture, neutropenia, pancytopenia, or a leukaemoid reaction. In most of these cases a therapeutic trial with antituberculosis drugs may be diagnostic and life-saving, as in our case. Furthermore, the diagnosis of tuberculosis was established in this patient by the results of the cultures and the finding of acid-fast bacilli in the peritoneal aspirate. Since specific treatment reversed the entire illness, including the wasting syndrome and arthralgia, S.L.E. was excluded. The early picture presented by the patient thus has to be explained as being part of the tuberculous infection.

L.E. cells are rarely found in chronic tuberculosis (Hollander, 1966). In 1953 we saw a similar case of "smouldering" tuberculosis of the reticuloendothelial system with arthralgia, positive L.E. cell phenomenon, and rash. In leprosy, another mycobacterial infection, L.E. cells have also been observed in a few cases with the demonstration of antinuclear factor activity in the serum (Bonomo *et al.*, 1965). The mycobacterium may produce in a few patients a pathological autoimmune profile, possibly in connexion with the known adjuvant effect of mycobacteria.

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