and Seeler and Shwiaki[4] suggest instead that repeated transfusions should be given to allow the distended sinusoids to undergo fibrosis. Nevertheless, in chronic hypersplenism the spleen does not become smaller with transfusion. Gradually increasing splenic size with thrombocytopenia, as observed in our patient, may indicate the need for urgent splenectomy. In such circumstances the risks of a pre-operative exchange transfusion, rather than serial transfusions with normal (HbAA) blood, might be justified. Regular checking of the platelet count at clinic visits, particularly in patients with an enlarging spleen, will facilitate the early diagnosis of hypersplenism.

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Arthritis after meningococcal meningitis

The cause of the lesions in meningococcal infections is not fully understood. Skin lesions may be due to the Schwartzmann reaction and "immunological factors,"[1] while circulating immune complexes have been shown in serious and fatal cases in West Africa and in a single case seen in Britain.[1] We have recently seen a further British case in which the clinical course and the laboratory results shed more light on the cause of the arthritis.

Case report

A schoolboy aged 13 complained of headache and pains in the legs. Later he vomited, developed a rash, and suffered progressive impairment of consciousness. About 24 hours afterwards he was admitted to hospital. On examination a petechial rash of the buttocks and legs was seen, he was feverish (38°), and blood pressure was 100/60 mm Hg. He was drowsy, the neck was hyperextended and stiff, and lumbar puncture showed a purulent fluid containing white cells $7{\cdot}9 \cdot 10^9$/l (7940/mm^3) (96% polymorphs), protein 492 g/l, and reduced sugar of 0·8 mmol/l (14·4 mg/100 ml). Gram-negative intracellular diplococci were seen, and the fluid and blood culture grew *Neisseria meningitidis* type C, sensitive to sulphadiazine. The patient was given 4-hourly intravenous sulphadiazine for 4 days and penicillin for 3 days. He improved the day after admission but arthritis of the wrists and knees became evident; his fever rose again and between days 4 and 9 was mostly over 38°, while 30 ml, 120 ml, and 60 ml of sterile joint fluid were removed from the knees on days 5, 10, and 12 respectively. Nevertheless, his general condition and the rash improved steadily and intravenous fluids were stopped on day 6 when the neck stiffness had almost disappeared. Since his meningitis and general condition improved while the arthritis and fever developed we thought an adverse immune response had occurred. He made a full recovery apart from a "dead" left ear.

Laboratory studies—Serum and joint fluid were examined for immune complexes by the method of Zubler *et al*[5] with the use of I^{125}-labelled complement (C1q) prepared here. Haemolytic complement (CH50) was measured by doubling dilutions of serum and joint fluids, and complement C3 and C4 by Mancini diffusion. Split products of C3 and C5 were measured by two-dimensional "rocket" electrophoresis. Cells from joint fluid specimens were washed, counted, applied to microscope slides in a cytocentrifuge, and fixed in 70% ethanol at −20°; antigen, antibody and βIC complement were detected by immunofluorescence. Serum collected on 5 February 1976 contained immune complexes, C3 and C5 split products, normal haemolytic complement, and slightly reduced C3 (see table). The joint-fluid CH50 titre, however, was less than 1/2 (albumin 23 g/l) and it contained immune complexes and little C3. The second fluid specimen contained more white cells (mainly polymorphs) and CH50 had returned to normal, but split products were still present. Cells from the exudate contained meningococcus type C antigen, immunoglobulin, and complement.

Comment

There was no clinical indication to aspirate the knee joint before chemotherapy, but as the patient had a positive blood culture, bacteria had probably reached the joints early in the disease. Later both the cells and the joint fluid contained immune complexes that were fixing complement, and we assume that these induced a complement-mediated sterile local inflammatory reaction. We believe that this reaction caused much of the patient's later general illness and fever, and most of his joint disease, and that it was developing while skin lesions were recovering. Similar processes may often occur in meningococcal disease, which may explain why some later components of the condition seem to be unaffected by chemotherapy.

We are grateful to Mrs Daphne Bird for carrying out "rocket" electrophoresis.

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Findings in serum and two specimens of joint fluids

Date	Specimen	White cells: Total (×10^9/l)	White cells: Polymorphs (%)	Complement activities (g/l): CH50	C3	C4	C3 split products	Immune complexes (%)*
5 Feb '76 ..	Joint fluid	9·6	90	<1/2	43	10	+	63
10 Feb '76 ..	Joint fluid	50	99	1/64			+	
5 Feb '76 ..	Serum			1/64	78	27	+	25

*Expressed as percentage radioactivity of specimen precipitated with trichloracetic acid. Several standards of human immunoglobulin are also included in the assay.
CH50 = haemolytic complement.