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Meningococcal septicaemia and meningitis: a rising tide

Meningococcal septicaemia ranges clinically from a fulminating lethal illness to a low grade infection. The course seems to be determined by host resistance rather than by the strain of the organism, and unfortunately not enough is known about resistance or about the factors that dictate the transition from carriage to invasion. What is clear, however, is that early recognition and treatment are vital—and any delay may be catastrophic.

Neisseria meningitidis has been classified into several groups with group B responsible for most of the infections in Britain and Europe over the past 15 years.[1,2] There has, however, been a progressive increase in group C infections in recent years.[1] Less than 10% of infections are caused by groups A, Y, and W135.[3] Most cases occur in children aged under 5, but there has been a striking increase in the incidence of infection among teenagers and young adults.[4] The number of cases of meningitis notified in England and Wales in 1987 rose sharply to 1090, approaching the peak of 1974[3]; the number notified in the first four weeks of January 1988 was 214, compared with 142 and 70 in the corresponding periods for 1987 and 1986 (personal communication, Communicable Disease Surveillance Centre).

Clinical infection with the meningococcus occurs after a pre-existing carrier state, when the organisms reside in the nasopharynx. There is no predictable relation between the carrier rate in the community and the incidence of infection.[1,5,6] Bacteraemia is probably the primary event in all forms of infection,[5] though it has been suggested that the meninges might be infected by direct spread through the cribriform plate.[6] Subjects with deficiency of the terminal components of complement are definitely more predisposed to meningococcal infection.[7,8]

The clinical features may relate to the septicaemia and endotoxaemia, to metastatic infection, and to immunological effects. Fulminating septicaemia may present with startling suddenness, causing aggressive behaviour, rapid deterioration in consciousness, fever, shock, cardiac and renal failure, and disseminated intravascular coagulation. The patient may be dead within 48 hours. The most important tell tale sign is the rash; diagnosis has to be empirical and treatment urgent. In contrast, chronic meningococcaemia causes an illness that may last weeks or even months[5] with a rash (recurring every 48 to 72 hours[5]), joint pains, malaise, and fever. Bad prognostic factors include extremes of age, peripheral purpuric lesions, shock, absence of meningitis, absence of leucocytosis, hyperpyrexia, disseminated intravascular coagulation, and cardiac or renal failure.[9-11]

The severity of the systemic features such as shock, purpura, disseminated intravascular coagulation, and organ failure may relate to complement activation,[12] release of tumour necrosis factor,[13] and deficiency of certain important plasma proteins[14]—and possibly to the meningococcus's ability to impair B lymphocyte function.[15] The rash may be petechial, macular, or purpuric; the petechial rash is generally associated with meningitis and a better outcome than the purpuric rash, which is associated with fulminant septicaemia.[11] The lesions result from a local Schwartzman reaction, circulating immune complexes, or disseminated intravascular coagulation.[16] The rash may be confused with a viral exanthem[5] or a drug induced vasculitis.[17] Fulminating purpura may be associated with bleeding into the adrenals and refractory shock.

Metastatic infection, most often in the leptomeninges, is the commonest presentation. So pre-eminent is this that meningitis is often thought to be essential for the diagnosis, but the meninges may not be affected in every case. They may, however, be affected without the typical signs of meningeal irritation—namely, neck stiffness and a positive Kernig's sign. Lumbar puncture clinches the diagnosis in most cases but in overwhelming infections the expected excess of neutrophils and protein in the cerebrospinal fluid may be absent, and there may be no neutrophilia in peripheral blood. Rarely the appearance of the cerebrospinal fluid may mimic those in aseptic or tuberculous meningitis.[18,19] An urgent Gram stain of the fluid is therefore essential, though the organisms may not be seen if antibiotics have been given. Other unusual forms of metastatic infection include polyarthritis, pericarditis, pneumonitis, and infection of the genitourinary system.[20-24] Immunological effects usually occur later in the course of the illness, often during recovery, when chemotherapy may have been completed. The effects are probably due to circulating immune complexes and may produce a rash, polyarthropathy, fever, and pericarditis.[25]

A blood culture is essential before starting treatment, though it may take up to a week for the organism to grow,

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and the culture result may be negative in some cases. When antibiotics have been given before culturing blood or cerebrospinal fluid, diagnosis may still be confirmed by countercurrent immunoelectrophoresis or latex agglutination (though their value is doubtful in group B infections).[6 26] In fulminant disease treatment must be started immediately without waiting for results of investigations (or a consultant opinion). A recent DHSS circular (3 February 1988) has suggested that doctors should give penicillin to patients with suspected meningococcal infection before transferring them to hospital; this may be particularly sensible in circumstances where there may be a delay. The antibiotic cover should be broad spectrum and the popular regimen is intravenous benzylpenicillin 3-4 MU (60 000—70 000 units/kg in children) four hourly and chloramphenicol 1 g (20-25 mg/kg in children) six hourly. The chloramphenicol may be withdrawn once meningococcal infection has been confirmed. The recommended duration of treatment varies from one to two weeks, though shorter courses have been successful.[27-29] An alternative effective drug is ceftriaxone.[30]

Overwhelming sepsis should ideally be treated in an intensive therapy unit with close haemodynamic, biochemical, and haematological monitoring. The patient may need assisted ventilation, circulatory support, or renal dialysis. The possibility of acute haemorrhagic adrenal failure must be kept in mind and hydrocortisone given if there is any suspicion that the patient might have it. The endotoxaemia may be severe enough to kill despite these measures. It might be treated by plasmapheresis, leucapheresis, and exchange transfusion,[31 32] but further studies are needed before these measures are recommended.

Though penicillin is almost invariably effective in systemic infection it may not be adequate in clearing the carrier state,[33] and it is generally (though not universally) agreed that after the penicillin the patient should be given rifampicin 600 mg or 10 mg/kg body weight (5 mg/kg for children under 1 year) twice daily for two days.[4] Chemoprophylaxis is indicated only for close family contacts,[33] especially children and young adults. Sulphonamides, which have been widely used in the past, are not reliable agents for prophylaxis because of the high incidence of resistant strains; they may be used when a strain associated with a specific outbreak is known to be sensitive. Rifampicin is therefore the prophylactic drug of choice (at the dosage mentioned), though rare failures have been reported. Vaccination is not generally recommended except in epidemics, and vaccines are available only against groups A, C, Y, and W135, but not group B.[4 34] Mortality in this condition remains depressingly high—about 10% in meningitis, rising to as much as 50% in septicaemia without localising signs.[4 34]

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After the horror

Post-traumatic stress disorder is formal terminology for a common condition, the psychological problems that follow exposure to events such as the Bradford football stadium fire, the Zeebrugge ferry disaster, the Vietnam war, or some less well publicised and more private horror such as assault or rape. These psychological problems may be severe, prolonged, and disabling, and they include intrusive memories, flashbacks, anxiety, numbing of feelings, and irritability. The account of a survivor of the Bradford soccer stadium fire illustrates phenomena associated with the condition.

It's hard for me to talk about it. I go hot and cold and I can't stop me hands trembling. I didn't used to be able to talk about it at all . . . I thought I was beginning to get over it but then I went to the memorial service and that brought it all back . . . I didn't feel I could go on, I wanted to talk to somebody because I was feeling very disturbed. . . . One of my main things was I felt very lonely and couldn't talk to anyone properly and I was feeling very scared and vulnerable all the time and having bad dreams. And I did have very bad guilt feelings. Everyone in the stand had been rushing about, it was every man for himself and I felt very bad about that. I was all the time thinking why was it me that had got out, had I pushed others aside and just saved myself? . . . I've only been back to the ground once since, so far . . . I forced myself to go . . . I did it as a test. I came through it and so I know now I'm getting stronger in myself.[1]

The past decade has seen a blossoming of interest in such experiences, but they have been recognised for well over a century. Attempts to find meaning in the folly that was the Vietnam war and growing acceptance of the Vietnam veteran as a victim rather than a perpetrator have added new

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