Lesson of the Week

Failure of chemoprophylaxis to prevent meningococcal disease

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The incidence of meningococcal disease among household contacts of patients with the disease is considerably greater than that among the general population.¹ Chemoprophylaxis is generally recommended to reduce this risk² (D M Jones, J D Abbott, unpublished report 1981, No 12, Communicable Disease Surveillance Centre) but may fail because it is given too late, because the infecting organism is resistant or inaccessible to the agent chosen, or because the drug is not given to the entire household.¹ We describe an outbreak of meningococcal disease lasting 12 weeks in an extended family in which selective prophylaxis failed to prevent two secondary cases.

Case reports

CASE 1

A 6 month old infant was admitted to hospital with a three day history of lethargy and vomiting. She was pale, unresponsive, and feverish. She had stiffness of the neck, Kernig's sign, and a tense fontanelle, but there were no petechiae. Meningitis was confirmed by lumbar puncture and treatment with penicillin and chloramphenicol started. *Neisseria meningitidis* group B was subsequently isolated from blood and cerebrospinal fluid, and chloramphenicol was stopped. She made an uneventful recovery and was discharged 10 days after admission. *N meningitidis* was not isolated from further blood cultures and nose and throat swabs collected before her discharge.

The patient's two sisters, aged 2 and 4, received prophylaxis with rifampicin 5 mg/kg twice daily for five days starting on the day after her admission. Throat swabs were collected from them and 19 other household contacts (playmates and members of the extended family). *N meningitidis* group A but not group B was isolated from the baby's uncle.

CASE 2

The 4 year old sister of the baby in case 1 was admitted one week later (12 days after completing her course of rifampicin) with a one day history of vomiting, fever, and abdominal pain. On admission she was confused but without meningism. There was tenderness on movement of both elbow joints and a macular rash on her arms. Her symptoms remitted spontaneously, and she was discharged after 48 hours. On the next day, however, *N meningitidis* group B was isolated from a blood culture and nose swab that had been collected on admission. These isolates were later shown to be of the same serotype as those in case 1—namely, Frasc type 8/11. Both isolates were susceptible to penicillin, rifampicin, tetracycline, and sulphonamides (minimal inhibitory concentration of sulphadiazine <1 mg/l). She was readmitted for treatment with penicillin, after which further blood cultures gave negative results.

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The increased risk of meningococcal disease among close contacts of infected patients may persist for many months

As in case 1, when *N meningitidis* had been isolated rifampicin was given to her siblings and on this occasion also to her parents. Swabs were then taken from them and the other household contacts. *N meningitidis* group B, Frasc type 8/11, was isolated from an aunt.

CASE 3

Eight weeks later a 16 month old cousin of the patients in cases 1 and 2 (the daughter of the aunt from whom *N meningitidis* group B had been isolated) was admitted with a brief history of vomiting, fever, and lethargy. She had a rapidly developing purpuric rash and opisthotonus and responded only to painful stimuli. Two hours later she had convulsions, which were generalised at first but later focal, centred on the right side of the face. She was treated with plasma infusions, large doses of hydrocortisone, benzylpenicillin, chloramphenicol, phenobarbitone, and later diazepam by continuous infusion. Blood taken for culture was contaminated with two strains of *Staphylococcus epidermidis*; there was no growth in the cerebrospinal fluid, but group B meningococcal antigen was shown by latex agglutination. During her recovery left hemiparesis became evident and necrotic skin overlying the larger purpuras required replacement by grafts. Three months later she had minor residual left hemiparesis but no evidence of intellectual deficit or personality change.

Rifampicin was given to her 3 year old brother, her parents, and other household contacts (32 on this occasion); all except a 6 month old infant received minocycline. Dosage schedules were as used by Munford *et al.*³ These subjects were swabbed two days after completion of their prophylaxis; none yielded *N meningitidis*.

Discussion

This outbreak highlights several pitfalls in the control of non-epidemic meningococcal disease. Firstly, prophylaxis was restricted initially to the siblings of the index case on the grounds that secondary cases are rare among adults. The risk of disease is indeed much greater in children, but the prevalence of carriage may be higher among adult contacts, who may subsequently infect or reinfect those previously given prophylaxis.¹ Prophylaxis with rifampicin is currently recommended for household and other intimate contacts, room mates in dormitories, and preschool children in day nurseries.² Deviations from the optimum chemoprophylaxis have been noted in the United States, where the Meningococcal Disease Surveillance Group judged that in only a minority of 326 cases studied was appropriate prophylaxis given to household members.¹ The patients affected in our outbreak, however, were members of an extended family with many young children, who frequently slept in each other's houses. Thus prophylaxis should perhaps have been offered to all members of

the extended family whether or not they were direct contacts of the patients.

The second point that we learnt from this outbreak was that transmission may occur over a fairly long period. An aunt of one of the patients (case 2) was identified as a carrier of the outbreak strain. We experienced difficulty in identifying the isolate in our laboratory, attempts to type the strain being inconclusive. Confirmation of identity and type was therefore sought from the Meningococcal Reference Laboratory, Public Health Laboratory, Manchester, but it was not until 14 days after collection of the swab and 19 days after presentation of case 2 that the type was confirmed. The general practitioner was informed and advised that the risk of further cases at this stage was small. This advice may have been influenced by the observations that meningococcal disease tends to occur shortly after first acquisition of the organism, if at all⁴; that the spread of meningococci and meningococcal disease during epidemics is rapid⁵; and that most secondary cases in families occur within two weeks.⁶ No further action was taken, and seven weeks later this carrier's daughter was admitted to hospital with meningococcal septicaemia. A similar outbreak of group B meningococcal disease has been described in which the fourth case, a 4 year old girl, had been exposed to meningococcal carriers for at least 13 months but had yielded negative cultures on four separate occasions.⁷ A later study showed very slow rates of transmission of meningococci between intimate contacts in families without overt disease.⁸ Thus it may be dangerous to base assumptions about the interepidemic spread of meningococci in families on experience gained during epidemics.

The dose of rifampicin (5 mg/kg twice daily) given to juvenile contacts of two patients (cases 1 and 2) may have been suboptimal. Doses of rifampicin recommended for prophylaxis against meningococcal disease are as follows: adults and children aged over 12, 600 mg twice daily for two days; children aged 1-12 years,

10 mg/kg twice daily for two days; and infants aged 3-11 months, 5 mg/kg twice daily for two days.² Sulphadiazine may be preferable in outbreaks in which the infecting strain is susceptible, but it cannot be relied on for "blind" prophylaxis because of widespread resistance to sulphonamides. (D M Jones, J D Abbott, unpublished report 1981, No 12, Communicable Disease Surveillance Centre).

Thus this outbreak serves as a reminder that although most secondary cases of meningococcal disease occur within two weeks after the index case, some may present months later. Prophylaxis that does not include all members of an extended family may fail to prevent secondary cases, and even optimal prophylaxis may fail.⁷

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Is the BCG vaccination of the elderly considered useful in the control of tuberculosis in areas where the disease is prevalent?

BCG vaccination will provide important protection against the development of naturally acquired tuberculosis for about 15 years. There is evidence that with age tuberculin sensitivity as determined by Mantoux testing gradually diminishes unless there is reinfection. There is certainly evidence that the general level of immunity to tuberculosis diminishes with advancing age: the incidence of tuberculosis has fallen much more in the young than in the elderly. Clear evidence in the form of a controlled study is not available to show how effective BCG may be in the elderly so it is difficult to advocate BCG vaccination of the elderly. Even so, until there is more precise evidence that vaccination would cause an adverse reaction its use in elderly people would seem to be reasonable.—G W POOLE, consultant respiratory physician, London.

Anonymous. Tuberculosis in old age. *Tubercle* 1983;64:69-71.

Grange JM, Gibson Julia, Osborne TW, Collins CH, Yates MD. What is BCG? *Tubercle* 1983;64:125-39.

Anonymous. Is BCG vaccination effective? *Tubercle* 1981;62:219-21.

A woman in her mid 30s had hypertension, pre-eclampsia, and a caesarean section at 38 weeks in a recent pregnancy. She had a lumbar chordal epidural anaesthetic and in the immediate puerperal period developed a Bell's palsy. She now wants another baby. What advice should be given?

The cause of idiopathic facial paralysis (Bell's palsy) is uncertain, though viral neuropathy or entrapment neuropathy are possibilities. There is an association between Bell's palsy and pregnancy: among pregnant women the incidence is 45/100 000 births compared with 17/100 000 a year among non-pregnant women of the same age group.¹ In one series of 42 cases associated with pregnancy, 31 occurred in the third trimester, six in the first two trimesters, and five in the first two weeks post partum.¹ There is no obvious relation with parity. In the general population there is an association between Bell's palsy and hypertension, but no association has been found with pre-eclampsia.¹ Although transient trigeminal palsy has been described after epidural analgesia,² Bell's palsy has not been linked with otherwise uncomplicated epidural analgesia. The prognosis in pregnancy is no worse than among non-pregnant women,¹ and recovery is complete in about 80% of

cases. Prednisolone treatment is still controversial and may be inappropriate in certain cases,³ but if it is to be used treatment should be started promptly.⁴ So far as I know recurrent Bell's palsy in consecutive pregnancies has not been described (though it is difficult to know how many patients have embarked on another pregnancy). Although this woman cannot be completely reassured, there seems to be no reason to warn her against having another baby.—JAMES OWEN DRIFE, senior lecturer in obstetrics and gynaecology, Leicester.

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In many years of consultant practice I have observed that if the "maintenance medication" of psychotic patients stabilised on drugs is gradually tapered off their psychotic illness has invariably relapsed. This emphasises, perhaps, the need for continued medication, but when this relapse of psychosis was treated with the same neuroleptic drug or drugs as before, with gradual increase to maximum recommended dosage when necessary, there seemed to be an uncharacteristic lack of response. In many such patients a change to a different type of neuroleptic—for example, phenothiazine to thiothixene type—seemed to be effective in alleviating psychotic symptoms. Is this a recognised phenomenon and if so what is the explanation?

It is undoubtedly true that dosages of neuroleptic drugs necessary to treat acute psychotic episodes are much higher, sometimes many times, than dosages adequate for maintenance. Patients in an acute episode can tolerate much higher dosages of a neuroleptic drug without developing side effects than when they are in remission. Perhaps the patients referred to required even higher doses than the "maximum recommended" to bring their acute psychosis under control. There is certainly evidence, however, that patients who fail to respond to one neuroleptic drug may show considerable improvement when another is substituted.¹—J P LEFF, honorary consultant psychiatrist, London.

1 Leff JP, Tress KH. The response of untreated chronic schizophrenics to haloperidol and haloperidol decanoate. *International Clinical Psychopharmacology* suppl 1 (in press).