stances, when the alcoholic is also depressed and has suicidal tendencies, it would seem wise to avoid the drug altogether.

I should like to thank HM Coroner for Bedfordshire for permission to publish details of these cases; the scientific officers at the Home Office Forensic Science Laboratory, Aldermaston, Reading, who performed the analyses; and Dr J C Valentine, consultant pathologist, who performed the post-mortem examination in cases 1 and 5.

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Department of Histopathology, Bedford General Hospital, Bedford MK42 9DJ
JOAN M HORDER, MRCPATH, DMJ(PATH), senior registrar

Meningitis due to chloramphenicol-resistant Haemophilus influenzae type b

Two strains of *Haemophilus influenzae* resistant to chloramphenicol have been reported from the USA,[1] and a resistant non-typable strain has been isolated in Holland.[2] We report here a case of meningitis due to chloramphenicol-resistant *Haemophilus influenzae* type b that occurred in Oxfordshire in July 1977.

Case report

A 19-month-old girl presented after two days' illness with drowsiness, irritability, stiff neck, and vomiting. Cerebrospinal fluid (CSF) showed $8{\cdot}5 \times 10^9$/l white cells, mainly polymorphs, and numerous pleomorphic Gram-negative rods on microscopy. The peripheral blood showed a neutrophil leucocytosis of $17{\cdot}8 \times 10^9$/l. Both CSF and blood grew *Haemophilus influenzae* type b.

Immediate treatment was begun with chloramphenicol 150 mg intravenously every 4 hours (90 mg/kg/24 h). After 72 hours the level of consciousness, tachycardia, and fever had not improved and a further CSF specimen showed $2{\cdot}0 \times 10^9$ white cells/l, with organisms still present on microscopy. This CSF specimen and a further blood culture also grew *Haemophilus influenzae.*

The original sensitivity plate was then reappraised and the zone of inhibition around the 25-μg chloramphenicol disc was shown to be slightly smaller than that given by a control strain. A test for penicillinase production by the organism gave a negative result. Chloramphenicol was therefore stopped and treatment changed to ampicillin 10 mg intrathecally once, and 400 mg intravenously every 4 hours (250 mg/kg/24 h).

Steady clinical improvement followed. The CSF was sterile after three days and pyrexia and tachycardia resolved after 10 days. Oral amoxycillin was substituted after seven days in doses up to 2 g/24 h and stopped after 14 days, since when the child has remained well.

The minimum inhibitory concentration of chloramphenicol for this organism was 8 mg/l (control strain 0·5 mg/l) and that of tetracycline 32 mg/l (control strain 1·0 mg/l), using the agar plate incorporation method.

A strain of *Haemophilus influenzae* type b resistant to chloramphenicol and tetracycline was isolated from the throat of the 4-year-old brother, but non-typable strains from the mother, father, and babysitter were sensitive to both antibiotics.

The patient had been previously well, apart from otitis media four months earlier, which had been treated with penicillin. The rest of her family had also been well and had used neither chloramphenicol nor tetracycline in the previous five years.

Comment

The only other strain of chloramphenicol-resistant *Haemophilus influenzae* type b so far reported caused meningitis in a 9-month-old infant from Philadelphia; it was also tetracycline resistant. This resistance was probably plasmid-mediated, as in the non-typable strain isolated in Holland.[2] This has grave implications for future treatment. Ampicillin has been considered to be a safe and effective antibiotic for treating infections caused by *Haemophilus influenzae* but increasing numbers of treatment failures have been reported; these are explained by inadequate doses, relatively poor penetration of meninges, or bacterial resistance due to the production of β-lactamase.[3] Intravenous chloramphenicol penetrates readily into the CSF and has previously been considered to be active against all strains of *Haemophilus influenzae.* The possibility of resistance to either or both these antibiotics emphasises the importance of performing sensitivity tests whenever invasive strains are isolated and of reculturing CSF and blood when early clinical response is unsatisfactory. A less potent chloramphenicol disc—for example, 5 μg—might facilitate recognition of resistant strains. As advocated by the American Academy of Pediatrics,[4] combination of chloramphenicol and ampicillin as initial treatment for *Haemophilus influenzae* meningitis may be considered. Antibiotic antagonism is, however, a theoretical objection and combination therapy may actually increase the risk of long-term complications.[5]

We are grateful to Dr J D Baum for permission to report details of this case.

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Departments of Paediatrics and Pathology, Churchill Hospital, Oxford
ANN-LOUISE KINMONTH, MB, senior house physician
C N STORRS, MRCP, DCH, paediatric senior registrar
R G MITCHELL, DM FRCP, consultant microbiologist

Psittacosis masquerading as rheumatic fever

It is now generally accepted that the diagnosis of rheumatic fever is based on the modified Jones's criteria,[1] in which evidence of a preceding streptococcal infection is paramount. We record a case in which an initial clinical diagnosis of rheumatic fever was made but in which subsequent serological evidence indicated infection by *Chlamydia psittaci* rather than streptococci.

Case report

A 39-year-old salesman was admitted with a two-week history of general malaise, sweating, and shivering associated with a sore throat and an unproductive cough. Three days before admission he had developed acute pain and swelling and redness of the wrist, metacarpophalangeal, and proximal interphalangeal joints. This developed into a flitting arthritis, which affected the elbow, knee, ankle, shoulder, and temporomandibular joints. Two days before admission he had developed a red, patchy rash proximally on all limbs, which subsequently spread on to the trunk. Four days before admission his GP had prescribed oxytetracycline and soluble aspirin. He had no relevant medical history apart from longstanding, recurrent low back pain.

On admission the abnormal findings were that he was clammy, febrile (38·5°), and peripherally cyanosed, with signs of acute arthritis affecting carpal and metacarpophalangeal joints. He had a rash with the typical appearance of erythema marginatum affecting his trunk and proximal aspects of limbs. His pulse was 108/min and regular; the respiratory rate was 20/min; and there was a soft systolic ejection murmur over the aortic area. An electrocardiogram showed sinus tachycardia with right bundle-branch block. A chest *x*-ray film showed minor patchy shadowing at the right lung base. The results of routine biochemical tests were all normal, but he had a mild normochromic normocytic anaemia of 12·6 g/dl, with a WBC 13×10^9/l (neutrophilia) and ESR 116 mm in first hour (Westergren). Treatment with penicillin V 500 mg six-hourly and soluble aspirin 600 mg four-hourly with bed