Proteus mirabilis meningitis and cerebral abscess in the newborn period

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SUMMARY Three cases of Proteus mirabilis meningitis in neonates are reported, in 2 of which abscess formation was proved neuroradiologically. All neonates with P. mirabilis meningitis warrant a CAT scan, as does any newborn infant with meningitis who has a continuing pleocytosis after adequate treatment with antibiotics.

P. mirabilis is not commonly implicated as a causative organism in neonatal meningitis; in 1976 Hoffman et al.3 reported 2 cases of cerebral abscess caused by this organism. More recently Darby et al.4 reported an infant with a chronic brain abscess secondary to P. mirabilis meningitis. During a 6-month period we had 3 further cases, each from a different unit in this region; this suggests that this organism is becoming a common cause of meningitis with cerebral abscess formation.

Case reports

Case 1. This boy was born in a consultant maternity unit by spontaneous vertex delivery at 38 weeks' gestation weighing 3.12 kg, there being no antenatal or perinatal complications. She was transferred to a local GP unit at age 24 hours where she developed, 12 hours later, a high fever and became irritable and shocked. She was immediately transferred to the neonatal intensive care unit where she was found to be jaundiced, tachypnoeic, and to have a smelly umbilicus.

At 2 days CSF was not obtained for technical reasons, the only positive findings being P. mirabilis grown from the umbilical swab and from blood culture. A diagnosis of septicaemia secondary to omphalitis was made and gentamicin and penicillin were started, the organism being subsequently found to be sensitive to gentamicin. A repeat lumbar puncture was done because of convulsions, CSF showing a pleocytosis (polymorphs $28 \times 10^6/l$, lymphocytes $2 \times 10^6/l$, glucose 1.6 mmol/l, and protein 0.4 g/l), but culture gave no growth. In view of this further finding, chloramphenicol was added to the drug regimen. Her condition gradually improved and she was allowed home aged 27 days having received 21 days' parenteral antibiotic treatment. At discharge she was normal neurologically with a clear CSF.

Nine days later, aged 36 days, she was readmitted to this hospital after suddenly becoming irritable; on examination she was convulsing spasmodically. CSF showed $48.0 \times 10^6/l$ polymorphs, no glucose, and a heavy growth of P. mirabilis. She was initially treated with gentamicin parenterally and intrathecally, and chloramphenicol parenterally; the latter was changed to co-trimoxazole when culture sensitivities were obtained. The day after admission a burr hole was performed with the intention of inserting a CSF reservoir, but at operation pus was aspirated, culture also growing P. mirabilis. CAT scan (Fig. 1) showed bilateral frontal abscess formation. Despite repeated surgical drainage and adequate treatment with antibiotics she died 19 days after admission. Necropsy confirmed the clinical and radiological findings.

Case 2. This boy was born in hospital at 38 weeks' gestation weighing 2.95 kg, there being no antenatal

![Fig. 1 Case 1. CAT scan showing the abscess cavity as a large area of reduced absorption in the left frontal region (arrowed); there is moderate ventricular enlargement.](http://adc.bmj.com/)

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Problems. Delivery was by breech and there were no neonatal problems, discharge being after 48 hours. He was admitted to hospital at age 5 weeks with a 3-day history of excessive crying and convulsions.

He was pyrexic, unresponsive, and had staring ‘sun-setting’ eyes. The anterior fontanelle was tense, head circumference being 39·5 cm with separated sutures; there was also increased extensor tone. Investigations were all negative, lumbar puncture being withheld because of raised intracranial pressure. A few days later he was transferred to the neurosurgical department in Derby because of a rapidly increasing head circumference. Subdural aspiration yielded 200 ml pus, culture growing *P. mirabilis.* CAT scan (Fig. 2) showed a large frontal abscess. (Subsequent scans showed hydrocephalus with possible loculation in the lateral ventricles.) Despite treatment by aspiration, adequate parenteral and local antibiotics (gentamicin, ampicillin, and co-trimoxazole), and CSF shunting, the head circumference continued to increase and it was felt that further surgery would be of little value. When last seen at age 10½ months, he was retarded and functioned at a 2-month level. His head circumference was stable but future prognosis is poor; the latest CAT scan shows minimal cerebral mantle.

Case 3. This boy was born in hospital at 35 weeks’ gestation by vertex delivery, birthweight 2·5 kg, Apgar score 8, and was admitted to the neonatal unit. There had been no antenatal care and at artificial rupture of membranes, done to augment labour, there was offensive liquor. He developed mild hyaline membrane disease requiring umbilical catheterisation and continuous positive airways pressure. On day 4 he had a series of short generalised convulsions with apnoea requiring intubation. At this stage CSF showed 11·0 × 10⁶/l polymorphs and a growth of *P. mirabilis.* IV chloramphenicol and intrathecal gentamicin and hydrocortisone were given. His condition deteriorated and he required assisted ventilation for a short time. Gradually he improved, but at one month CSF still showed a pleocytosis, despite prolonged intrathecal therapy. He was transferred for neuroradiological investigations after a gamma scan had shown increased uptake in the frontal area. An air ventriculogram showed hydrocephalus without any evidence of a mass lesion. His condition gradually deteriorated despite treatment and he died aged 6 weeks; permission for necropsy was refused.

Discussion

Cases 1 and 2 had abscess formation proved both radiologically and surgically. In our third patient (Case 3) evidence was not forthcoming, but as he had an increased uptake on gamma scan in the frontal area and a continuing pleocytosis, he may have had a frontal abscess or an area of indolent cerebritis. We assume that in all patients the source of septicemia and subsequent seeding of the *P. mirabilis* organisms, either directly or by septic thrombosis in the frontal areas, was via the umbilicus. Only Case 1 had a scalp electrode during labour. Although abscess formation in pyogenic meningitis is always worrying, it seems striking that *P. mirabilis* meningitis was complicated by frontal abscess in 2, and probably in all 3, babies described in this report.

Neonatal meningitis still continues to be a major problem and carries a high mortality and morbidity. The incidence varies, the rates in Goldacre’s series⁴ and the Collaborative Perinatal Research Study in the USA⁵ being 0·26 and 0·46/1000 live births respectively. In Goldacre’s series⁴ the incidence of *P. mirabilis* was only 2 in 726 cases of neonatal meningitis.

We feel that any neonate with meningitis who has a continuing pleocytosis despite adequate treatment with antibiotics should have neuroradiological investigation in the form of a CAT scan. In the light of our experience and that of other authors⁸ it would seem worthwhile obtaining early CAT scans in all neonates who have *P. mirabilis* meningitis, where an abscess seems to be a common complication and may also be silent.

Fig. 2 Case 2. CAT scan after injections of meglumine, diatrizoate, and sodium diatrizoate (Urograin 290). The walls of both lateral ventricles show uptake of contrast producing a halo effect indicating severe ventriculitis. The frontal abscess cavity is arrowed and communicates with the right lateral ventricle.
Metoclopramide poisoning in children

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SUMMARY 15 children with metoclopramide (Maxolon) poisoning are reported. One of the 5 children accidentally poisoned developed slight extrapyramidal signs. All 10 children who experienced extrapyramidal side effects while being treated with metoclopramide had received a dose greater than that recommended by the manufacturer of 0.5 mg/kg per day. Dystonic reactions are likely to occur if the recommended dose is exceeded, but individual susceptibility to metoclopramide and the cumulative effect of repeated doses of the drug may also be important.

Metoclopramide (Maxolon), a chlorbenzamide derivative, is an antiemetic drug which has been available in the UK since 1967. It is regarded as a safe drug with few side effects and has been recommended for habitual vomiting, drug-induced vomiting, and as an adjunct to x-ray examination of the upper alimentary tract. However, alarming extrapyramidal side effects have been reported in children receiving the recommended dose, and in children who have taken the drug in excess. We have looked at a number of children who required admission to this hospital after accidental poisoning by this drug or as a result of its extrapyramidal side effects.

Patients and method

Between January 1967 and December 1978, 15 children were admitted to this hospital with metoclopramide poisoning. Most of the cases occurred during the last 5 years (Figure). The clinical details are summarised in Table 1.

Table 1 Clinical information on 15 children with metoclopramide poisoning

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Sex</th>
<th>Dose ingested (mg)</th>
<th>Duration from administration to symptoms (hours)</th>
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<tr>
<td></td>
<td>2 6/12</td>
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<td>75</td>
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<tr>
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<td>3 6/12</td>
<td>15-3 F</td>
<td>75</td>
<td>4-9</td>
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<td>2 1/12</td>
<td>12</td>
<td>100</td>
<td>8-3</td>
<td>-</td>
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<tr>
<td></td>
<td>1 5/12</td>
<td>9-6 M</td>
<td>60</td>
<td>6-3</td>
<td>-</td>
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<tr>
<td>Therapeutic poisoning</td>
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<td></td>
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<td>24</td>
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<td>40</td>
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<td>30</td>
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<td>10</td>
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<tr>
<td></td>
<td>9 6/12</td>
<td>30 M</td>
<td>70</td>
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M L Smith and D Mellor

Arch Dis Child 1980 55: 308-310
doi: 10.1136/adc.55.4.308

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