if this child presents without any signs of respiratory distress. Children already in respiratory distress should have optimal medical treatment with forced intravenous fluids, corticosteroids in high doses, and bronchodilators. Depending on the clinical course and blood gas analysis, mechanical ventilation may be required. Very high airway resistance must be overcome, and respiratory adjustment should be done as in severe bronchial asthma. Hypoxaemia can be avoided by increasing FrO2. Respiratory acidosis may be corrected by infusion of THAM (not sodium bicarbonate), especially when high Paco2 interferes with vital functions.

Summary

Two cases of powder aspiration are reported. A 74-month-old girl showed a classical course with an asymptomatic period of 3–4 hours, then severe respiratory distress developed. Acute respiratory insufficiency made tracheal intubation and mechanical ventilation necessary for 10 days. Complications included insufficient alveolar ventilation, atelectasis, pneumothorax, and superinfection. But the baby recovered with some residual radiological changes in the lungs. A 13-month-old boy was treated immediately after massive powder aspiration by tracheal intubation and bronchial wash-out. The postoperative course was uneventful and no respiratory distress developed.

Powder aspiration leads to severe bronchiolar obstruction with a delay of several hours and has a high mortality rate. The best results in treatment are obtained by immediate intubation and bronchial wash-out, even in the absence of respiratory symptoms. Artificial ventilation may be necessary with the special problem of overcoming very high airway resistance. Corticosteroids and bronchodilators may be helpful.

References


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Pyogenic meningitis in chronic gastroenteritis and marasmus

We report a series of cases of infants presenting with a severe illness with gastroenteritic symptoms in whom the initial cerebrospinal fluid (CSF) was normal but pyogenic meningitis developed during the course of the illness. Clinical presentation was atypical and the development of meningitis was unexpected and unpredictable. This unusual combination prompted this paper.

Case reports

Case 1. A male aged 3 months was readmitted 10 days after a previous episode of chronic gastroenteritis, with a history of diarrhoea for 2 days. He was 5% dehydrated, opisthotonic, and after rehydration was below the 3rd centile for weight. Lumbar puncture yielded sterile CSF (Table). Enteropathic E. coli O127/B8 was cultured from the stool.

Opisthotonus persisted; 2 weeks after admission the diarrhoea was severe, he was pyrexial, a lumbar puncture again yielded sterile CSF (Table). A week later the diarrhoea was blood-stained, pyrexia and opisthotonus persisted, a third lumbar puncture yielded sterile CSF (Table). X-rays of chest and abdomen were normal. Improvement occurred until 6 weeks after admission, another pyrexial episode prompted a fourth lumbar puncture which now yielded purulent CSF (Table). After treatment for meningitis total nerve deafness was diagnosed.

Case 2. A male aged 8 months was admitted with a history of diarrhoea for the previous month. He was
### Short reports

#### Table  Sequential lumbar punctures in each case

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Hospital day</th>
<th>Indication</th>
<th>Cell count ((/mm^3))</th>
<th>Protein ((g/l))</th>
<th>Sugar (mg/100 \text{ ml} (\text{mmol/l}))</th>
<th>Bacteriology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>Opisthotonus</td>
<td>0</td>
<td>0-26</td>
<td>52 (2-9)</td>
<td>No growth</td>
</tr>
<tr>
<td>15</td>
<td></td>
<td>Pyrexia</td>
<td>0</td>
<td>0-30</td>
<td>60 (3-3)</td>
<td>No growth</td>
</tr>
<tr>
<td>24</td>
<td></td>
<td>Pyrexia, opisthotonus</td>
<td>1</td>
<td>0-28</td>
<td>70 (3-9)</td>
<td>No growth</td>
</tr>
<tr>
<td>41</td>
<td></td>
<td>Pyrexia</td>
<td>L 20 P 790</td>
<td>3-20</td>
<td>&lt;10 (&lt;0-6)</td>
<td>No organism</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>Empirical procedure in sick child</td>
<td>1</td>
<td>0-37</td>
<td>183 (10-2)</td>
<td>No growth</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>Right-sided convulsion</td>
<td>L 30 P 290</td>
<td>2-60</td>
<td>35 (2-0)</td>
<td><em>Diplococcus pneumoniae</em></td>
</tr>
<tr>
<td>3*</td>
<td>25</td>
<td>Pyrexia</td>
<td>L 70 P 3300</td>
<td>1-40</td>
<td>36 (2-1)</td>
<td><em>Diplococcus pneumoniae</em></td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>Empirical procedure in sick child</td>
<td>2</td>
<td>0-88</td>
<td>138 (7-7)</td>
<td>No growth</td>
</tr>
<tr>
<td>8</td>
<td></td>
<td>Pyrexia and spasticity</td>
<td>L 57 P 65</td>
<td>1-10</td>
<td>30 (1-7)</td>
<td><em>Diplococcus pneumoniae</em></td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>Empirical procedure in sick child</td>
<td>0</td>
<td>0-31</td>
<td>128 (7-1)</td>
<td>No growth</td>
</tr>
<tr>
<td>7</td>
<td></td>
<td>Bulging fontanelle</td>
<td>L 6 P 76</td>
<td>1-84</td>
<td>20 (1-1)</td>
<td><em>Salmonella adelaide</em></td>
</tr>
<tr>
<td>6</td>
<td>25</td>
<td>Stiff neck</td>
<td>0</td>
<td>0-34</td>
<td>83 (4-6)</td>
<td>No growth</td>
</tr>
<tr>
<td>30</td>
<td></td>
<td>Pyrexia</td>
<td>L 30 P 22 490</td>
<td>1-68</td>
<td>&lt;10 (&lt;0-6)</td>
<td><em>Salmonella untyped</em></td>
</tr>
</tbody>
</table>

*No preceding sterile CSF obtained. L = lymphocytes; P = polymorphonuclear cells.

10% dehydrated but no other clinical abnormalities were found. After rehydration his weight was still below the 3rd centile. Lumbar puncture yielded sterile CSF (Table). Diarrhoea continued, and on the third hospital day he had a right-sided fit but showed no new clinical findings. Lumbar puncture yielded purulent CSF (Table). Diarrhoea persisted for a further week. No organism was grown from the stool. He was left with severe central nervous system damage after treatment for his meningitis.

**Case 3.** A female aged 3 months was admitted with a history of 2 days of diarrhoea and vomiting. She was 10% dehydrated and after rehydration was below the 3rd centile for weight. Diarrhoea persisted, nonpathogenic *E. coli* was cultured from the stool. On the 25th hospital day the child was pyrexial; lumbar puncture yielded purulent CSF (Table). The meningitis responded to treatment but diarrhoea recurred intermittently until her discharge 2 months later when there was no detectable neurological deficit.

**Case 4.** A male aged 3 months was admitted with a history of haematemesis for the previous 4 days. He was 5% dehydrated, hypotonic, and had diarrhoea. His weight after rehydration was on the 3rd centile. Lumbar puncture yielded sterile CSF (Table). Haematemesis was not confirmed. Intravenous cloxacillin and kanamycin were started empirically. There was no electrolyte imbalance. On the fourth day the arms were spastic. A week after admission he still had diarrhoea and pyrexia and spasticity had involved the legs. Lumbar puncture yielded purulent CSF (Table). Penicillin replaced the other antibiotics. 10 days later, with spasticity persisting, he was taken home by his parents against medical advice.

**Case 5.** A female aged 2½ months was readmitted 9
days after a previous diarrhoeal illness. She was moribund, shocked, and 15% dehydrated, with a 5-
day history of diarrhoea. After rehydration her weight was below the 3rd centile. Lumbar puncture
yielded sterile CSF (Table). On the seventh day the diarrhoea was somewhat improved, but her font-
tanelle was bulging. Lumbar puncture yielded purulent CSF (Table). Later that day she died.
Post-mortem examination showed pus over the brain from which *Salmonella adelaide* was cultured.
This organism was not isolated previously from rectal swabs.

Case 6. A female aged 5 months was admitted with pneumonia; her weight was below the 3rd centile.
She recovered from the pneumonia, but at 3 weeks had severe diarrhoea. Nonpathogenic *E. coli* was
cultured from the stool. After 4 days of diarrhoea she had bilateral otitis media and a stiff neck.
Lumbar puncture yielded sterile CSF (Table). A week later culture of the continuing diarrhoea yield-
ed enteropathic *E. coli* O126/B16 and an organism of the *Salmonella* group. Chloromycetin and gentami-
cin were started intravenously. The following day, because of continuing pyrexia, a further lumbar
puncture was performed which yielded purulent CSF (Table). The child died 6 days later.

Discussion

Meningitis was totally unexpected in these patients. Lumbar puncture was done for specific neurological
findings in 2 cases, one with focal seizures (Case 2) and one with a bulging fontanelle (Case 5). The other
4 infants underwent the procedure as part of the investigation of unexplained fever, a situation
reminiscent of the clinical setting of neonatal meningitis.

Episodic pyrexia is frequently seen in our patients with chronic gastroenteritis and investigation often
shows no cause. The fact that in this series 2 children died, 3 other children had neurological deficits
ranging from gross impairment (Case 2) to deafness (Case 1), and only one child recovered adequately,
suggests avoidable delay in diagnosis on our part. However, even retrospectively we could find no
clues as to when meningitis started in these patients, in whom the onset of the disease was insidious and
unremarkable.

In all except Case 3, a sterile CSF preceded the finding of purulent CSF. Case 3 had been in hospital
for 25 days before purulent CSF was found. It is reasonable to assume that these cases all developed
meningitis while in hospital, a rare event in clinical practice. Previous authors have reported cases in
which initial CSF cell counts and biochemistry may be normal and the existence of bacterial meningitis
only becomes evident upon positive bacterial culture from the CSF in the following 24 hours (Moore and
Ross, 1973). Sometimes initial lumbar puncture may yield completely normal CSF which is sterile on
culture, but a positive blood culture is followed by the finding of purulent CSF at some stage in the
next 48 hours (Wegeforth and Latham, 1919; Fisher et al., 1975). The shortest time interval
between the lumbar punctures in our series was 3 days (Case 2). In the other cases the interval ranged
from 5 to 17 days. The initial lumbar puncture in Case 2 may have localized a bacteraemia to the
meninges, but the same mechanism seems extremely unlikely in the other 5 cases.

Pneumococcal infections account for more than 50% of severe infections occurring in children who
have undergone splenectomy. The infections occur within 18 months of the splenectomy and are
commonest in children under 2 (Ellis and Smith, 1966). The same authors point out that the spleen
increases its mass by a greater amount than any other organ during the first year of life. Our patients
were all severely malnourished and it could be postulated that the combined effects of splenic
growth retardation and thymolymphatic atrophy, which has been reported in severe protein-calorie
malnutrition (Smythe et al., 1971), plus aberrations in splenic blood supply consequent on recurrent
dehydration caused by persistent diarrhoea, led to a relative asplenia with heightened susceptibility to
pneumococcal infections. The 2 cases of salmonella meningitis can be more easily explained on the basis
of a persisting source of infection in the gut and the known tendency for the organism to spread via the
blood.

This series of cases illustrates the dangers of meningitis stealing silently into the clinical picture of
children with long-standing diarrhoea. The difficulty of detecting the onset of meningitis in these
cases is great. It seems that the only safeguard against missing the diagnosis is to be constantly
alert to the possibility and to include a lumbar puncture as part of the evaluation of any unexplained
fever or other change in clinical state.

Summary

Pyogenic meningitis occurred in 6 infants in the course of chronic gastroenteritis, in 5 of whom a
normal CSF had been recorded earlier in the illness. Clinical signs of meningitis were often absent. The
problems of diagnosis and management and of possible predisposition to pneumococcal meningitis
in children suffering from chronic gastroenteritis are discussed.
Short reports

References


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Neonatal hypocalcaemia after intrauterine exposure to anticonvulsant drugs. *Birgitte Friis and H. Sardemann.*

Pyogenic meningitis in chronic gastroenteritis and marasmus.

G P Wyatt and J C Marcus

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