Subdural abscess secondary to covert dental sepsis

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Summary
The bacterial flora of a subdural abscess in a 17-year-old male, with radiological evidence of unilateral infection of the maxillary and frontal sinuses, was typical of that encountered in the dental sulcus. Extensive examination revealed no primary focus of infection other than apical infection in the 2 upper first molar teeth, which were extracted. Treatment with ampicillin, gentamicin and metronidazole rapidly controlled the subdural infection, and resolution, as evidenced by computerized tomography, was complete at 10 months.

Introduction
Intracranial sepsis is a well known complication of acute dental infection arising either spontaneously, or after dental surgery (Hollin, Hayashi and Gross, 1967). The relationship of chronic asymptomatic dental sepsis to intracranial infection is less firmly established. Ingham et al. (1978b) have described 2 patients with brain abscess in whom symptomless dental infection was considered to be the primary source of infection. The present authors report the clinical and bacteriological findings in a patient with subdural abscess in whom the primary source was covert dental sepsis.

Case report
A 17-year-old panel beater presented with a 2-week history of productive cough, sore throat, nausea and vomiting, followed within a day by frontal headache, gradually increasing in severity and exaggerated by straining and coughing. Three days before admission he noticed progressive weakness of the right leg, shivering and sweating. On examination, he was febrile and drowsy but not dysphasic. The right optic fundus was swollen and there was a dense, right homonymous hemianopia and pyramidal weakness of the right leg, with increased reflexes and an extensor plantar response.

The peripheral white cell count was 11.1 × 10⁹/l, the ESR 45 mm in the first hour and there was hyponastraemia. Computerized tomography (CT) revealed no abnormality other than widening of the interhemispheric fissure. Lumbar cerebrospinal fluid was at a pressure of 22 cm and contained 16 white cells/mm³, polymorphs and lymphocytes being in equal numbers. The CSF protein was 0.39 g/l, the sugar 4.2 mmol/l and it was sterile on culture. A further CT scan showed increased widening of the interhemispheric fissure with contrast enhancement of the edges, more on the left than the right, and partial ring enhancement in the left frontal pole, appearances consistent with a parafalcaline subdural empyema.

Midline frontal, left frontal, left temporal and left posterior burr holes were made and approximately 35 ml of thick pus aspirated. Five milligrams of chloramphenicol were instilled and a rubber drainage catheter inserted into the cavity. Two days later 5 ml of pus were aspirated from the drainage tube which was then removed.

Culture of the pus revealed the organisms listed in the table, and a blood culture taken on the day of operation yielded anaerobic streptococci, non-haemolytic streptococci, Streptococcus milleri (Lancefield Group F) and S. milleri (non-groupable).

Postoperatively, the patient received i.v. ampicillin (one g/6 hr), gentamicin (80 mg/8 hr) and metronidazole (500 mg/8 hr). Examination carried out to locate the source of infection revealed no abnormality in any system other than in the right maxillary
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and frontal air sinuses which, on X-ray, were opaque. Dental examination revealed 2 grossly carious upper first molar teeth, each had radiological evidence of apical infection associated with one or more of their roots. Removal of the infected teeth was carried out.

<table>
<thead>
<tr>
<th>Obligate anaerobes</th>
<th>Aerobes/facultative anaerobes</th>
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</thead>
<tbody>
<tr>
<td>Propionibacterium acnes</td>
<td>Streptococcus milleri</td>
</tr>
<tr>
<td>Fusobacterium polymorphum</td>
<td>(Lancefield Group F)</td>
</tr>
<tr>
<td>Veillonella alcalescens</td>
<td>S. milleri</td>
</tr>
<tr>
<td>Bacteroides melaninogenicus</td>
<td>(Lancefield Group C)</td>
</tr>
<tr>
<td>sub. sp. melaninogenicus</td>
<td>Anaerobic streptococci</td>
</tr>
<tr>
<td>Anaerobic diphtheroid</td>
<td>S. viridans</td>
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<td></td>
<td>Eikenella corrodens</td>
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<tr>
<td></td>
<td>Actinomyces odontolyticus</td>
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</table>

The patient made a steady recovery, complicated by focal epilepsy controlled by phenytoin. One month postoperatively he was clinically normal and remained so at review 18 months later. The CT scan appearance steadily improved with partial resolution of the empyema at 6 months and complete resolution at 10 months.

Bacteriology

Specimens of pus were processed within 30 min of aspiration. The material was inoculated on to MacConkey agar, and nutrient agar containing 5% horse blood (vol/vol), which were incubated aerobically at 37°C, and on to chocolate agar incubated in 5% carbon dioxide at 37°C. The pus was also inoculated on to duplicate plates of blood agar, and blood agar containing nalidixic acid at a final concentration of 50 mg/l as a selective medium (Ingham et al., 1978a), which were incubated anaerobically in an atmosphere of 90% hydrogen and 10% carbon dioxide in an anaerobic jar fitted with a cold catalyst. One set of plates was left for 5 days before examination.

Aerobic and facultatively anaerobic bacteria were identified by reference to Cowan and Steel (1974). The methods of Duerden et al. (1976) and Tharagonnet et al. (1977) were used to identify obligate anaerobes. Sensitivity to antibiotics was determined by the diffusion technique using paper discs containing metronidazole 5 μg and penicillin 1 unit, appropriate controls being included.

Discussion

The bacterial flora of the abscess was typical of that found in dental abscesses (Sabiston, Grigsby and Segerstrom, 1976) and did not resemble that reported in studies of frontal lobe abscesses stated to be of sinusitis origin, in which S. milleri was present in pure culture (de Louvois, Gortvai and Hurley, 1977). In the absence of any other infective focus, the organisms isolated from the blood culture almost certainly represented bacteraemia secondary to the intracranial infection.

The occurrence of maxillary sinusitis due to the extension of dental infection is well documented (Bauer, 1943; Maloney, 1968). This sinus opens via its ostium into the central part of the hiatus only a few millimetres posterior to the ostium of the frontal sinus (Gray, 1967). Dental infection may thus spread to these sinuses, causing the increased radiological opacity seen in this patient, hence progressing to intracranial sepsis.

Although organisms encountered in dental sepsis are usually sensitive to penicillin, this may not always be the case. Destruction of penicillin in the abscess cavity by penicillinase-producing strains of Bacteroides melaninogenicus (Pinkus, Veto and Braude, 1968), which may be present in dental infection (Sabiston et al., 1976), could reduce the efficacy of this agent against accompanying penicillin-sensitive bacteria. This effect has been demonstrated experimentally in mice (Hackman and Wilkins, 1976). Since there will necessarily be a delay before bacteriological results are available, initial treatment of intracranial infections must be based on the primary source of infection and the bacterial species likely to be encountered. The initial chemotherapy of subdural abscess should, in the authors' opinion, consist of a combination of antimicrobial agents with a broad spectrum of activity, such as metronidazole, gentamicin and penicillin. These should be continued until the primary source of infection has been identified and the sensitivity of the causative bacteria determined. The latter can usually be accomplished in a few days by a technique combining selective culture with direct sensitivity testing (Ingham et al., 1978a). Should this time chemotherapy may, if necessary, be modified and the primary source extirpated.

Should the nature of the bacterial flora indicate a possible dental focus as the primary source of infection it is essential that a dental opinion be obtained, as deep-seated dental sepsis may only be revealed by specialized techniques, such as intra-oral X-rays.

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References


