Peripheral blood was taken for a full blood count and the measurements were within normal limits. Her current weight was 5 kg. Her anthropometric increased and the plantar response was bilaterally extensor. The child was not alert, the tone of all the limbs was otherwise normal. On central nervous system examination, the child had neck retraction and uprolling of eyes for the previous 2 days. The child was born at term by normal vaginal delivery in the same hospital with a birth weight of 2.8 kg. She was on exclusive breast feeding.

On physical examination, her pulse rate was 130 min⁻¹, respiratory rate was 50 min⁻¹ and temperature was 39.3 °C. On head to toe examination, the anterior fontanelle was full and pulsatile (size 1.5 × 1.5 cm). Physical examination was otherwise normal. On central nervous system examination, the child was not alert, the tone of all the limbs was increased and the plantar response was bilaterally extensor (upgoing). Her current weight was 5 kg. Her anthropometric measurements were within normal limits.

Peripheral blood was taken for a full blood count and the report showed a total leukocyte count of 12 000 cells mm⁻³ with 65% polymorphonuclear leukocytes, 31% lymphocytes, 2% monocytes and 1% eosinophils. The general blood picture showed mild to moderate hypochromia with microcytosis. Hepatic and renal profiles were within normal limits. Chest X-ray (postero-anterior view) was normal. Urine culture was sterile. Cytological analysis of cerebrospinal fluid (CSF) showed a white cell count of 1000 cells μl⁻¹, 70% neutrophils, 2% monocytes and 1% eosinophils. The general picture was normal. CSF biochemistry revealed that proteins were raised (200 mg dl⁻¹) (normal CSF proteins: 20–45 mg dl⁻¹) and glucose was reduced (35 mg dl⁻¹) (normal CSF glucose level: >50 mg dl⁻¹ or 75% of serum glucose). Her plasma glucose was 75 mg dl⁻¹ (normal plasma glucose: 50–120 mg dl⁻¹). On direct microscopy, Gram staining of the CSF showed Gram-positive cocci in clusters. CSF culture showed growth of large greyish white, non-haemolytic colonies which were adherent to the blood agar surface. On Gram staining, Gram-positive cocci in clusters were again observed. A capsule was noticed on capsular staining. The organism was weakly catalase-positive, and coagulase- and oxidase-negative. It hydrolysed aesculin and liquefied gelatin, fermented glucose, trehalose, mannose and sucrose and did not ferment mannitol, rhamnose, raffinose, arabinose or xylose. It did not grow in 6.5% NaCl. On the basis of these characteristics, the organism was identified as Stomatococcus mucilaginosus. The blood culture also grew Gram-positive cocci which were subsequently identified as Stomatococcus mucilaginosus. Antimicrobial susceptibility testing was performed using NCCLS (2006) guidelines by the Kirby–Bauer disc diffusion method on Mueller–Hinton agar plates. The strain was multidrug-resistant. It was susceptible to only vancomycin (disc potency 30 μg) and cefotaxime (30 μg). It was resistant to gentamicin (10 μg), co-trimoxazole (trimethoprim/sulfamethoxazole 1.25/23.75 μg), erythromycin (15 μg), tetracycline (30 μg), ciprofloxacin (5 μg), amikacin (30 μg), ampicillin (10 μg), cefazolin (30 μg) and oxacillin (1 μg).

Empiric antibiotic treatment was started immediately with cefotaxime and amikacin prior to obtaining culture and an antibiotic susceptibility report. After obtaining the antibiotic susceptibility report, further treatment continued with cefotaxime alone. However, as the child continued to be febrile, it was decided to add vancomycin. The child recovered well subsequently with no neurological sequelae and was discharged after 12 days.
**Discussion**

Although *Stomatococcus mucilaginosus* is a normal commensal of the human oral and upper respiratory tract flora (Gordon, 1967), it is increasingly being implicated in serious diseases such as endocarditis (Prag et al., 1985), septicaemia and catheter-related sepsis (Poirier & Graudreau, 1989). The most common risk factors are indwelling venous catheter, cancer, cardiac valve disease, intravenous drug abuse and severe neutropenia (Prag et al., 1985; Ascher et al., 1991b; Relman et al., 1987; Weinblatt et al., 1990). Infections due to *Stomatococcus mucilaginosus* are probably under-reported because it can be misidentified as *Staphylococcus aureus*, *Micrococcus* or *Streptococcus* sp. because of the presence or absence of catalase activity and coagulase negativity (Ascher et al., 1991a). The problem is compounded when a laboratory is using commercial identification kits such as API Staph-Indent or Microscan systems which have not incorporated the organism into their databases (Ascher et al., 1991a). We identified our isolate based on its strong adherence to the agar surface, Gram-positive nature, weak catalase positivity, coagulase negativity, positive aesculin and gelatin hydrolysis and fermentation of glucose, sucrose, maltose, mannose and trehalose (Gordon, 1967; Ascher et al., 1991a; Bergan et al., 1970; Bergan & Kocur, 1982).

Since *Stomatococcus mucilaginosus* is part of the normal flora of the mouth, the most likely portal of entry appears to be the oral cavity following violation of the mucous membrane barrier (Ascher et al., 1991a). We presume that breakdown of the oral mucous membrane subsequent to an underlying oral infection could lead to haematogenous seeding of the CSF. This is the first reported case of meningitis in a healthy 2-month-old child. Prior studies have reported meningitis in premature and immunocompromised children (Ascher et al., 1991a).

The other unusual feature in this case was the absence of neutropenia; the haematological profile showed neutrophilia when meningitis was diagnosed. *Stomatococcus mucilaginosus* meningitis has been reported predominantly in neutropenic patients, patients with malignancies or patients who were otherwise immunocompromised.

Tracing the drug-resistance profile of *Stomatococcus mucilaginosus* over the last 20 years, penicillin was considered the drug of choice in the 1980s with a study in 1989 first reporting penicillin resistance (Pinsky et al., 1989). Thereafter, incremental drug resistance was observed over the years and vancomycin is now emerging as the effective empiric treatment in invasive *Stomatococcus mucilaginosus* infections (Poirier & Graudreau, 1989; Pinsky et al., 1989). In our study too, *Stomatococcus mucilaginosus* was multidrug-resistant and susceptible only to vancomycin and cefotaxime. However, the patient responded well only to vancomycin. We believe that cefotaxime may not have been effective despite good *in vitro* activity because of the presence of oxacillin resistance. In fact, it appears that oxacillin resistance should be interpreted as resistance to all cephalosporins, as is the case with meticillin-resistant *Staphylococcus aureus* (MRSA), despite *in vitro* activity. It should be understood that cross-resistance with meticillin extends to all β-lactam antibiotics, including cephalosporins. The reason for *in vivo* failure of cefotaxime could be poor penetration of the drug in the bacteria or due to its increased efflux or a change may occur in the penicillin-binding protein, as is seen in MRSA (PPB2a, which is a low binding protein). However, this issue should be explored further.

We believe that Gram-positive cocci in clusters having weak/ negative catalase activity and producing sticky colonies that adhere to the agar surface should be suspected of being *Stomatococcus mucilaginosus* and appropriate tests should be performed to confirm the identity of the bacteria.

Given the initial difficulty often encountered in differentiating *Stomatococcus mucilaginosus* from coagulase-negative staphylococci and occasionally from group D *Streptococcus*, vancomycin may be a reasonable component in an empiric antibiotic regimen (Weinblatt et al., 1990; Pinsky et al., 1989). Vancomycin can be begun empirically in such cases, as no report of vancomycin resistance has been reported yet, to be replaced with other drugs once an antibiotic susceptibility report is available.

**References**


