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Case Report

Meningitis Caused by *Streptococcus agalactiae* after Professional Tooth Cleaning: First Case

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Keywords
Meningitis · *Streptococcus agalactiae* · Group B streptococcus · Tooth cleaning · Case report

Abstract
We report the first case of meningitis caused by *Streptococcus agalactiae* (group B streptococcus; GBS) after professional tooth cleaning in a previously healthy patient. GBS is a common commensal of the human gastrointestinal and vaginal flora. Although occurrence in the oral flora is unusual, oral transmission and thus occurrence can be assumed.

Introduction

Streptococcus agalactiae or group B streptococcus (GBS) is a well-known and common pathogenic agent of neonatal sepsis and meningitis caused by the colonization of the maternal birth canal. The number of GBS infections in adults reported in recent years has increased. GBS infections are often seen in elderly patients or those with preexisting comorbidities (especially immunosuppressive diseases). Common GBS infections in adults are skin and soft tissue infections, osteomyelitis, or pneumonia [1].

Meningitis caused by GBS in adults is a very rare event. GBS meningitis is more often reported in patients with preexisting severe diseases or as a result of other primary infections (e.g., endocarditis) [1]. However, there are a few case reports of GBS meningitis in healthy patients without underlying diseases [2–4].

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Streptococcus agalactiae is a gram-positive, catalase-negative coccus and has beta-hemolytic properties. It is an apathogen inhabitant of the bowel and vaginal flora in colonized people. Transmission depends on sexual and eating habits [5]. Tooth brushing is known to cause transient bacteremia. Here we report the case of a previously healthy man with GBS meningitis in an immediate temporal context of professional tooth cleaning.

Case Report

A 56-year-old patient was admitted to a local hospital with headache, nausea/vomiting, and progressive impaired vigilance for some hours. In the morning of the same day, he underwent professional tooth cleaning. Under suspicion of meningitis, an empiric anti-infective treatment with ceftriaxone, ampicillin, acyclovir, and dexamethasone was initiated. The patient was taken to our hospital. He had reduced vigilance (sopor), a positive sign of meningism, and 38.5°C body temperature, but no focal neurological deficit. CSF diagnostics revealed granulocytic pleocytosis of 9,200 cells/μL (reference: <5 cells/μL) with increased levels of total protein (6,160 mg/L; reference: <450 mg/L) and lactate (11.1 mmol/L; reference: 1.7–2.6 mmol/L). Blood analysis showed an inflammatory constellation: leukocytes: 15,830/μL (reference: 3,900–9,800/μL), C-reactive protein: 149 mg/L (reference: <5 mg/L), procalcitonine: 5.34 ng/mL (reference: <0.05 ng/mL). The growth of *Streptococcus agalactiae* was found in venous obtained blood culture, and *Streptococcus agalactiae* DNA was detected in CSF by multiplex PCR. Contrast-enhanced cerebral computed tomography as well as CT of the paranasal sinus revealed no other primary focus of infection. Transesophageal echocardiography showed no signs of endocarditis, and X-ray of the chest was normal. Antibiotic treatment was reduced to ceftriaxone monotherapy after pathogen identification and was continued for 14 days. Cerebrospinal fluid pressure was significantly increased, with more than 50 cm water column in the first lumbar puncture. This was consistent with hydrocephalus malresorptivus and probably the cause of impaired vigilance. Several lumbar punctures for CSF removal were made. Under antibiotic treatment, the cell number of CSF was falling in the following examinations. The clinical signs were regressive with recent mild headache and mild cognitive impairment at discharge from the hospital.

Discussion/Conclusion

In our reported case, there are three uncommon aspects: (i) first report of bacterial meningitis after professional tooth cleaning; (ii) meningitis caused by streptococci in a previously healthy patient; (iii) *Streptococcus agalactiae* as a pathogen agent of meningitis after tooth cleaning.

This is the first case report of meningitis after professional tooth cleaning. The literature shows several cases of inflammatory cerebral diseases after dental treatments, but not after routine professional tooth cleaning. Fernando and Phipps [6] reported a case of meningitis after an uncomplicated tooth extraction. They detected *Streptococcus sanguis* as the pathogen. Three more cases of meningitis after tooth extraction or other dental treatments caused by *Streptococcus oralis* were also reported [7–9]. Yoshii et al. [10] described meningitis and subdural empyema arising from an infection after tooth extraction with detection of *Capnocytophaga* species in a previously healthy patient.

As mentioned before, there are some case reports of GBS meningitis in healthy patients. A predisposing disease does not seem to be a prerequisite for GBS infections. The flora of the mouth is especially diverse and contains over 700 bacterial species. Streptococci are main

inhabitants. Common representatives in the oral flora are *Streptococcus mitis* or *Streptococcus sanguis* [11]. Although *Streptococcus agalactiae* often colonizes the human gastrointestinal and vaginal mucosa, its presence in the oral flora is unusual. Since oral transmission by certain eating habits is described [5], at least a temporary occurrence of GBS in the oral flora can be assumed. In our patient, symptoms of bacterial meningitis with fever, headache, and meningism followed by disturbance of vigilance occurring within a few hours after professional tooth cleaning reflect an immediate bacteremia and hematogenous colonization of the meninges that was proven by positive blood culture and CSF PCR.

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Statement of Ethics

The authors have no ethical conflicts to disclose.

Disclosure Statement

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