Brain Abscess Due to *Arcanobacterium haemolyticum* after Dental Extraction

To the Editor—It has been suggested that 5%–20% of brain abscesses are presumably associated with oral infections or dental procedures [1–4], in which organisms belonging to the oropharyngeal flora, such as *Arcanobacterium haemolyticum*, are involved [5, 6]. This organism has been documented in cases of pharyngitis and wound infections [7], but rarely in systemic infections [7–9] and even less in brain abscesses [10]. We describe the case of a patient who developed a brain abscess due to *A. haemolyticum* infection after undergoing dental extraction procedure.

An 18-year-old man without a remarkable medical history, except for repeated periodontal manipulations, was admitted to the hospital with headache, vomiting, aphasia, weakness in his left extremities, behavior and mood alterations, and fever. Three months before admission, he had been treated for periodontitis and dental caries in a primary dental clinic. He underwent extraction of multiple teeth. He had been well until 15 days before hospital admission, when intense headache and vomiting developed. Seven days before hospitalization, weakness in his left extremities, behavior and mood alterations, and fever. Three months before admission, he had been treated for periodontitis and dental caries in a primary dental clinic. He underwent extraction of multiple teeth. He had been well until 15 days before hospital admission, when intense headache and vomiting developed. Seven days before hospitalization, weakness in his left extremities became worse, and he was unable to stand or walk. A brain CT scan revealed a left-sided hypodense fronto-parietal lesion with a small zone of clear hemolysis on 5% sheep blood agar. On the basis of the Gram stained smear of the abscess sample showed pleomorphic gram-positive coryneform bacteria. After 48 h at 37°C, aerobic culture grew min-

The isolate was identified as *A. haemolyticum* [11]. The isolate was found to be susceptible to penicillin, ceftriaxone, gentamicin, clindamycin, doxicicline, and vancomycin, but resistant to trimethoprim-sulfamethoxazole and ciprofloxacin by disk diffusion method. From the seventh day post-surgery, the patient received penicillin G (24 μU intravenously, daily for 21 days). Four weeks later, the patient was successfully discharged from the hospital with no subsequent complications.

*A. haemolyticum* is a catalase-negative, gram-positive, or gram-variable rod whose morphology is dependent on the growth media and conditions [11]. This species (formerly *Corynebacterium haemolyticum*) is an infrequent cause of pharyngitis in children and young adults. It is occasionally isolated from wound infections and abscesses and is found in patients with meningitis, pneumonia, pyothorax, and septicemia [7–13]. To our knowledge, after a review of the indexed literature, we consider this to be the second reported case in which *A. haemolyticum* is documented as the etiological agent of a brain abscess (previously, 1 case in a child was reported) [10] and the first in an adult patient. Although *A. haemolyticum* is susceptible (thus far, universally) to penicillin by in vitro MIC testing, treatment failure despite adequate doses of phenoxymethylpenicillin has been documented [11, 13–15]. Most studies have found that *A. haemolyticum* is susceptible to all antimicrobials tested, except trimethoprim-sulfamethoxazole [11, 13–16]. In the current case, the isolate was also resistant to ciprofloxacin.

This case illustrates the aggressive and serious nature of systemic odontogenic infections in which resistant strains could be producing severe neurological complications. The careful identification of *Arcanobacterium* species and its corresponding antimicrobial susceptibility test is important, so a complete understanding of the role of these organisms in disease and proper management can be realized [17]. As was seen in our case, *A. haemolyticum* can be resistant to additional drugs other than trimethoprim-sulfamethoxazole (ciprofloxacin, in this report) and can cause severe life-threatening diseases.

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.
References


2. Schlamser SE, Backman K, Norrby SR. Invasive staphylococci isolates in bone biopsy samples, compared with swab samples, and percutaneous bone biopsy specimens, for patients with diabetic foot osteomyelitis, Senneville et al. [1] found coagulase-negative staphylococci much more frequently in bone specimens than in swab samples (25.6% vs. 4.6%; P < .001). As outlined in the accompanying editorial, this finding was rather unexpected, because coagulase-negative staphylococci are microorganisms with little suspected virulence [2]. If confirmed, these data may have an impact on the choice of antimicrobial regimen used in these patients, because coagulase-negative staphylococci are usually considered to be contaminants in such conditions.

According to the authors, "the finding of a higher proportion of coagulase-negative staphylococci isolates in bone biopsy samples, compared with swab samples, was independent of the findings of their microbiological laboratory, which identified all of the organisms cultured from both bone and swab samples (including bacteria from the skin flora) in accordance with the protocol they established in 1996 in their diabetic foot clinic" [1, p. 61]. However, in the article they refer to [3], in which Senneville and colleagues discussed similar patients with the same procedures, although they observed similar discrepancies (in 31 patients with both swab and bone biopsy specimen cultures, coagulase-negative staphylococci were never cultured from swabs, while they were found in 8 bone biopsies; P < .01), Senneville and colleagues’ interpretation of this finding was much different: “this was likely to be related to the non-report of coagulase-negative staphylococci from superficial samples by our laboratory” [3, p. 929]. Could the authors clarify what made them change their interpretation between the 2 studies?

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

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References


Reply to Tattevin et al.

To the Editor—As noted by Tattevin et al. [1], in the 17 patients (not “31 patients,” as they wrote) with 20 episodes of diabetic foot osteomyelitis reported in 2001 by us an our colleagues [2], coagu-