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Case Report Rapport de cas

Bacterial meningitis after dental extraction in a 17-year-old horse

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Abstract — Dental extractions in horses may result in bacteremia, which can lead to systemic complications. Bacterial meningitis following oral cheek tooth extractions in a 17-year-old Thoroughbred gelding is described in this report. The bacterial meningitis was confirmed by histopathology. The gelding was presented for evaluation of intermittent fever, loose feces, and mild colic signs which started 5 days after cheek tooth extraction. This case illustrates a rare complication associated with oral tooth extraction in a horse and highlights the unusual presenting features of meningitis.

Key clinical message:
Bacterial meningitis secondary to oral cheek tooth extraction should be considered as differential diagnosis; particularly in cases with the development of pyrexia a few days after the procedure.

Résumé — Méningite bactérienne après extraction dentaire chez un cheval de 17 ans. Les extractions dentaires chez les chevaux peuvent entraîner une bactériémie, ce qui peut amener des complications systémiques. Un cas de méningite bactérienne à la suite d'extractions buccales de dents jugales chez un hongre pur-sang de 17 ans est décrite dans ce rapport. La méningite bactérienne a été confirmée par histopathologie. Le hongre a été présenté pour évaluation d'une fièvre intermittente, de selles molles et de signes de coliques légers qui ont commencé 5 jours après l'extraction de la dent jugale. Ce cas illustre une complication rare associée à l'extraction dentaire orale chez un cheval et met en évidence des caractéristiques inhabituelles de la méningite.

Message clinique clé :
La méningite bactérienne secondaire à l'extraction buccale des dents jugales doit être considérée comme un diagnostic différentiel, en particulier dans les cas de développement d'une pyrexie quelques jours après l'intervention.

(Traduit par Dr Serge Messier)

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Extraction of equine cheek teeth can result in complications, although recent improvements in minimally invasive oral extraction techniques have led to a lower incidence of complications (1–3). Recognized post-operative complications secondary to dental extractions in horses include nonhealing alveoli due to dental fragments or alveolar bone sequestrum formation; which can result in oroantral or oronasal fistula formation, orofacial sinus tract formation, or regional osteomyelitis (4). Dental extractions and periodontal procedures can result in bacteremia in horses, humans, and dogs (5–10). Case reports of systemic complications as a result of dental extractions in horses have documented abscessation of masseter muscle as well as extensive thrombophlebitis of the jugular, facial, and rostral cervical veins (11), facial cellulitis progressing to an orbital abscess (12), and bacterial endocarditis (13,14).

The findings of a horse, which developed bacterial meningitis without sinusitis following oral cheek tooth extraction, and later confirmed by histopathology are described in this report.

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Case description

A 17-year-old Thoroughbred gelding in good body condition [492 kg body weight (BW), body condition score 5/9] was presented to the William R. Pritchard Veterinary Medical Teaching Hospital (VMTH), University of California Davis, for evaluation of intermittent pyrexia of unknown origin and mild colic signs of 5-days duration. The gelding had a prior history of endodontic disease. The 109 and 110, and 210 teeth (Modified Triadan numbering system) had all been extracted between 2 to 6 y before presentation for the treatment of complicated crown-root fractures.

Ten days before presentation to the VMTH, the 111 and 211 teeth were extracted for treatment of severe periodontitis diagnosed by identification of increased periodontal probing

depths of 10 to 15 mm and mobility index of 3 (15). The extraction of the 111 tooth was complicated by excessive hemorrhage and external replacement resorption (ankylosis) of part of the mesial tooth root. The extraction of the 211 tooth had no apparent complications. The gelding received 1 dose of gentamicin sulphate (Gentamicin; Vetone, Boise, Idaho, USA), 6.6 mg/kg BW, IV, once, prior to extraction, and was prescribed trimethoprim-sulfamethoxazole [Bactrim DS (sulfatrim); Aurobindo, East Windsor, New Jersey, USA], 30 mg/kg BW, PO, q12h for 10 d, after surgery. Five days after the extractions, the gelding was reported to have intermittent fevers. On the day of referral to the VMTH, the gelding had started to show mild colic signs, his rectal temperature was 38.9°C and he passed soft feces. Administration of flunixin-meglumine (Banamine Inj; Merck, Madison, New Jersey, USA), 1.1 mg/kg BW, IV, once, did not improve his condition and he was referred to the VMTH for further diagnostic procedures and treatment.

On presentation the horse was bright, alert, and responsive. No cranial nerve deficits were noticed on presentation. Heart rate and respiratory rate were elevated [68 beats/min (bpm) and 20 breaths/min (brpm), respectively] with no cardiac murmurs, arrhythmias, or increased bronchovesicular sounds noted on auscultation. There was no nasal discharge. Borborygmi were mildly decreased in all quadrants. Rectal temperature was 37.7°C. A complete blood (cell) count (CBC) revealed a mild leukocytosis [11 600 cells/μL; reference range (RR): 5000 to 11 600 cells/μL), a neutrophilia (9752 cells/μL; RR: 2600 to 6800 cells/μL)], and a hyperfibrinogenemia (5 g/L; RR: 1 to 4 g/L). No other significant clinical pathology abnormalities were identified. Abdominal ultrasound revealed hypomotile small intestine. A nasogastric tube was passed, and no net reflux was obtained. No abnormalities were palpated on rectal examination. Abdominocentesis yielded grossly normal peritoneal fluid. Abdominal radiographs showed no clinically significant abnormalities.

A possible colonic impaction was suspected, and medical treatment was instituted. An intravenous catheter was placed in the left jugular vein (Mila Extended Use MILACATH; MILA International, Florence, Kentucky, USA), 14 Ga × 13 cm. The horse received intravenous polyionic fluids (Vetivex 5L; Dechra, Overland Park, Kansas, USA), 10 L, IV fluid bolus, followed by a constant rate infusion, 3 mL/kg BW per hour. He also received enteral fluids which consisted of 5 L of water and $MgSO_4$ (Swan Epsom Salt; Smyrna, Tennessee, USA), 1 g/kg BW *via* nasogastric tube once. Feed was withheld for the first 24 h of hospitalization. The oral antimicrobial treatments were changed due to concerns for antimicrobial associated colitis. Gentamicin sulphate (Vetone), 6.6 mg/kg BW, IV, q24h was administered. He was also started on omeprazole (GastroGard; Merial, Duluth, Georgia, USA), 4 mg/kg BW, PO, q24 h.

Initially the horse responded to medical management. He remained bright and alert and was passing normal manure with no pyrexia recorded in the initial 12 h of hospitalization. Refeeding was initiated and was well-tolerated.

Twenty-four hours after admission, the horse became dull and pyrexic (38.8°C), and was treated with flunixin-meglumine (Banamine Inj; Merck), 0.55 mg/kg BW, IV, once. Forty-eight hours after presentation the horse was noted to be straining and was observed to be dribbling urine intermittently. Interspersed with these episodes the horse was noted to urinate in a normal manner. Urinalysis did not reveal significant abnormalities. The horse continued to be pyrexic each day 38.8 to 39.7°C but responded to flunixin-meglumine (Banamine Inj; Merck), 0.55 mg/kg BW, IV, q12h. The horse maintained a good appetite and was passing normal manure. A nasal swab for equine herpesvirus-1 was negative on polymerase chain reaction (PCR). The intravenous catheter site was examined every 6 h and there were no significant abnormalities.

Given the history of previous oral dental extractions, a dental examination was performed under standing sedation with detomidine (Dormosedan Inj; Zoetis, Kalamazoo, Michigan, USA), 5 mg *via* IV catheter 4 d after presentation (14 d after the initial extraction). The sedation level was adequate for performance of an oral examination and dental radiographs, and no ataxia was noticed. There were no significant extraoral findings. Teeth with a known history of previous extraction were noted to be missing (109 and 110, 210 and 211). A small retained fragment of the 111 palatal root was identified within the previous extraction site, with the remainder of the tooth missing. The alveoli of the previously extracted 111 and 211 teeth otherwise had healthy granulation beds which had filled in approximately 80% of the alveolus. The retained fragment of the palatal root of 111 was removed with gingival elevators and determined to be 1.6 cm in length and 1 cm at its widest diameter. No local anesthesia was used for the fragment removal. Sinus and dental radiographs were taken following removal of the retained tooth fragment, and other than the previously identified missing teeth, no clinically significant findings were identified.

Two hours after the dental examination, the horse was sedated with detomidine (Dormosedan Inj; Zoetis), 2 mg *via* IV catheter, to facilitate a full abdominal ultrasonographic examination. Approximately 10 min later he became moderately ataxic. He was returned to his stall to recover from the sedation. Approximately 2 h later he was observed to be persistently circling to the right in his stall and he was ataxic (grade IV/V). Cranial nerve examination revealed an absent menace in the left eye and a delayed menace in the right eye and the horse was behaviorally blind. There was no evidence of other cranial nerve involvement. The ataxia of both thoracic and pelvic limbs, declining mentation, circling, and apparent blindness were most consistent with cortical or diffuse central neuroanatomical localization. In addition, the intermittent urine dribbling may have been associated with central neurological disease, has been reported in humans with eastern equine encephalitis (16). Because of safety concerns for personnel with the persistent walking, apparent blindness, and ataxia (grade IV/V), further neurological evaluation was not carried out. Treatment with intravenous fluids was continued and additionally dimethylsulfoxide (DMSO) (RIMSO 50; Valhoma, Oklahoma, USA), 1 g/kg BW, IV, once and flunixin-meglumine (Banamine Inj; Merck), 1.1 mg/kg BW, IV, once and minocycline (Minocycline hydrochloride capsules USP; Aurobindo Pharma, Hyderabad, India), 4 mg/kg BW, PO, q12h, were administered. The horse deteriorated and progressed to seizure. During an initial seizure

episode, a venous blood gas analysis demonstrated a lactate of 7.3 mmol/L, PCV 47%, and total protein 80 g/L. The seizure activity responded to midazolam (Midazolam Inj; Hospira, Lake Forest, Illinois, USA), 0.05 mg/kg BW, IV, once. Two and a half hours later the gelding experienced a second seizure, which again responded to intravenous midazolam (Midazolam Inj; Hospira), 0.05 mg/kg BW, IV, once. Additional treatment with phenobarbital (Luminal 60 mg tablets; Hikma, Eatonstown, New Jersey, USA), 10 mg/kg BW, PO once, was started; however, the horse experienced a third seizure, which was more violent than the first two. Clinical signs did not resolve after intravenous administration of midazolam (Midazolam Inj; Hospira), 0.1 mg/kg BW, IV once. Euthanasia was recommended and elected. The horse was anesthetized with ketamine (Ketalar Inj; Vetone, Boise, Idaho, USA), 2.2 mg/kg BW, IV and midazolam (Midazolam Inj; Hospira), 0.05 mg/kg BW, IV, once, and then pentobarbital (Euthasol inj; Virbac, Fort Worth, Texas, USA), 100 mg/kg BW, IV was administered.

Cerebrospinal fluid (CSF) was collected immediately after death from the atlanto-occipital space using aseptic technique. The cerebrospinal fluid appeared grossly abnormal (turbid and xanthochromic). Abnormal findings on cytology included a total protein concentration of 9.13 g/L (RR: 0.45 to 0.61 g/L) and an elevated total nucleated cell count of 7910 cells/mL (RR: 0 to 7 cells/mL) consisting of 93% neutrophils, 3% small mononuclear cells, and 4% large mononuclear cells. No organisms were seen on cytology, but the findings were consistent with marked neutrophilic inflammation (meningitis). Postmortem, but prior to histopathology results, the cerebrospinal fluid tested negative for West Nile virus with capture enzyme-linked immunosorbent assay (ELISA), *Listeria monocytogenes* with PCR, and *Neospora hughesi* on immunofluorescence assay (IFA). The CSF was positive for *Sarcocystis neurona* at 1:80 on IFA, and for Lyme disease on multiplex analysis (OSPF CSF:Serum ratio $>$ 4). The fluid was submitted for bacterial culture, but no growth was obtained.

The brain was collected separately and submitted for rabies screening using fluorescent antibody testing before complete necropsy. Test results were negative. No significant oral lesions associated with the site of extraction were identified grossly. A 1-mm thick, soft, dark red to black plaque coated the spinal cord and filled the subdural space, extending from the C7 to C8 spinal nerves to the cauda equina. Epaxial muscles surrounding the T1 through T5 vertebrae were streaked with 1- to 2-mm long, linear, dark red to black regions that dissected through the muscle bellies. No other lesions pertinent to the clinical presentation were noted. Histopathology was performed on liver, kidney, spleen, lung, heart, lymph nodes, gastrointestinal tract, as well as the brain, spinal cord, cribriform plate, and alveolar bone of the 111 and 211 tooth extraction sites. The collected sections of alveolar bone were decalcified in 10% formic acid solution prior to evaluation and did not demonstrate significant histologic changes. The predominant histologic finding was a severe, pyogranulomatous, perivascular infiltrate that multifocally expanded the meninges surrounding the cerebrum, cerebellum, brainstem, and cervical spinal cord, and extended along the spinal nerves, as well as into the cribriform plate. A dense cluster of coccobacilli was identified within a meningeal vessel adjacent to the brainstem, and a Gram stain confirmed a mixed population of Gram-negative rods and cocci (Figure 1). Secondary lesions within the neural parenchyma included marked, diffuse gliosis, and perivascular, lymphoplasmacytic, and neutrophilic cuffing. Together, pyogranulomatous meningitis, and lymphoplasmacytic encephalitis with gliosis were indicative of chronic inflammation with continuing insult, supported by the bacterial embolus. Acute, subdural hemorrhage affecting the spinal cord from C7 to the cauda equina correlated with the gross findings, and was unassociated with significant inflammation; consequently, the hemorrhage was independently attributed to iatrogenic trauma.

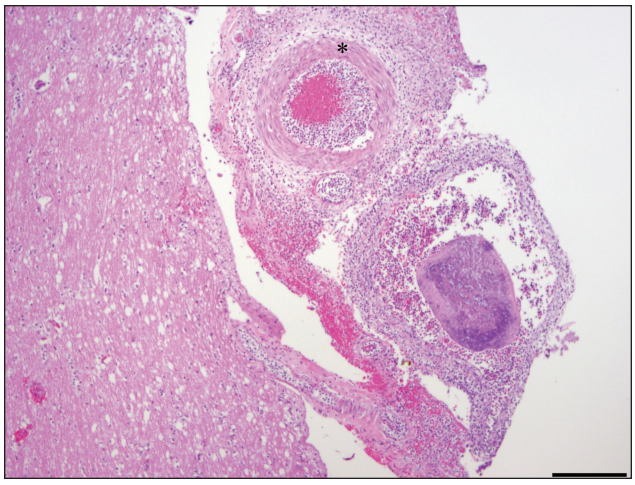

Figure 1. Large aggregate of mixed coccobacilli within a markedly inflamed meningeal vessel adjacent to the brainstem (asterisk). Hematoxylin and eosin (H&E). Scale bar = 200 μm.

Discussion

Complications following oral cheek tooth extraction in horses are reported to occur in 8 to 20% of cases and typically involve the alveolus and directly associated structures; such as, the maxillary or mandibular bone and the nasal cavity (1–3,17). Systemic infection following oral cheek tooth extraction has been reported rarely in horses (11–13,18). In humans, dental extractions are associated with a high risk for inducing transient bacteremia (19). Bacteria originating from the oral cavity can circulate in the bloodstream and cause infections such as endocarditis, meningitis, and brain or liver abscesses and can progress to sepsis in humans (20–22). A recent study reported transient bacteremia following cheek tooth extractions in horses (10). Bacteria can circulate in the bloodstream immediately after gingival disruption, but the bacteremia is usually cleared prior to the end of the dental extraction or periodontal procedure without resulting in sepsis, in both horses and humans (9,10).

The most commonly isolated bacteria from blood cultures obtained during cheek tooth extraction in horses were reported to be alpha-hemolytic streptococci and anaerobes such as *Fusobacterium* spp., and *Prevotella* spp., and facultative anaerobes such as *Actinomyces* spp. (10). In this case, Gram-negative rods and cocci were identified within a meningeal vessel. It would have been ideal to further characterize the bacterial genera,

as this would have potentially supported that the meningitis was a direct complication of the tooth extraction; but unfortunately, no bacteria were cultured. Oral bacterial flora shifts towards predominantly anaerobic and Gram-negative bacteria with periodontal disease. Specific bacteria associated with periodontal disease are *Prevotella* spp., *Porphyromonas* spp., and *Peptostreptococcus* spp. (23). Kern et al (10) showed that bacteria isolated from the swabs of samples of extracted teeth largely corresponded with those identified in blood cultures. Culturing the tooth root remnant perhaps was a missed opportunity in this case; however, it would have been difficult to justify before receiving the histopathology results, given the ambiguity of the clinical signs.

Primary sinus, nasal, submandibular, and periocular infections have been reported as causes of bacterial meningitis and brain abscessation (18,24,25). To the authors' knowledge this is the first report of bacterial meningitis after cheek tooth extraction without evidence of secondary sinusitis observable on radiographs. On initial presentation, meningitis was not a differential diagnosis for this case given the lack of neurological signs. Based on the chronicity identified in the histopathologic findings, the authors assume that the pyrexia was attributable to subclinical meningitis. Neurological signs did not progress until 4 d after initial presentation to the hospital (14 d after dental extraction). This is somewhat longer than reported in a case series of 5 horses that were presented with bacterial meningitis after sinus surgery. In these cases, clinical signs associated with the meningitis occurred 5 to 11 d after surgery (25). In retrospect, the intermittent urinary dribbling may have reflected neurological disease, which was noted 48 h after presentation. The reason for the delayed onset of clinical signs in this case is unknown but may have been due to the systemic antimicrobial (trimethoprim-sulfa combination) administration after tooth extraction.

Bacteria from paranasal sinus and odontogenic infections can gain access to the neurologic system by different routes including lymphatic vessels, blood vessels, or cranial nerves, as well as with osteitis and bony erosion or direct head trauma (24–27). In this case, healthy granulation tissue was present in the alveolus on oral examination and no bony abnormalities were noted on radiographic examination. In addition, no gross evidence on necropsy of local ascending spread of bacteria through the alveolar bone to the brain was apparent. However, the presence of a bacterial thrombus in a meningeal vessel suggests hematogenous spread of the infection.

The gelding was initially administered oral phenobarbital due to the cost of the intravenous formulation of the drug. It is possible that intravenous phenobarbital would have reached a steady state more quickly resulting in a better response (28). However, the mortality rate of horses with meningitis or meningoencephalomyelitis is 60 to 96% (29,30) and can reach 100% in horses with a history of bacterial meningitis secondary to an infectious disease of the head (18,24–26). In this case, the horse did not respond to initial therapy and continued to seizure, necessitating euthanasia.

No bacteria were seen on cytologic examination of the cerebrospinal fluid and bacterial culture did not reveal any significant growth of bacteria. This is not surprising, as a case series including horses with meningitis and meningoencephalitis only had a positive bacterial culture in 9% of antemortem cerebrospinal fluid samples (30). In addition, the horse was treated with antibiotics, which might have decreased the chance of identification of bacteria on cytology and culture. Findings on histopathology showed a population of mixed bacteria, which is a similar population to the bacterial growth of the cerebrospinal fluid in published case studies (24,30). Good penetration of the central nervous system can be achieved using macrolides, trimethoprim and sulfonamide, fluorinated quinolones, metronidazole, chloramphenicol, rifampin, and tetracyclines. The initial choice of an aminoglycoside was not appropriate (31,32); however, meningitis was not considered as a differential diagnosis at that time. The horse was switched to minocycline once neurologic signs were noticed, although the gelding was already in an advanced stage of the disease.

The positive CSF results for *Sarcocystis neurona* and *Borrelia burgdorferi* OSPF antibodies were likely due to altered blood brain barrier permeability and leakage of serum proteins considering the degree of inflammation within the CNS. Although it is impossible to completely rule these agents out, the histopathology was not consistent with either equine protozoal myeloencephalitis or Lyme disease.

This case illustrates an unusual complication associated with oral tooth extractions in a horse and highlights the unusual presenting features of meningitis. Although rare, clinicians should be aware of the potential risk of bacterial meningitis secondary to oral cheek tooth extraction, particularly with the development of pyrexia a few days after the procedure. Early diagnostic tests, such as a complete neurological examination and potentially CSF fluid collection, might be indicated if pyrexia persists and other potential causes of pyrexia are excluded. CVJ

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