Case Report Rapport de cas

Mycotic encephalitis, sinus osteomyelitis, and guttural pouch mycosis in a 3-year-old Arabian colt

Barbara Hunter, Patrick N. Nation

Abstract — Mycotic encephalitis caused severe ataxia and other neurologic deficits in a horse. The finding of a single, large focus of cerebral malacia, with histopathologic evidence of fungal elements, suggested infection was a result of direct transfer from the frontal sinuses, rather than hematogenous spread from the guttural pouch.

Résumé — Encéphalite mycotique, ostéomyélite des sinus et mycosie de la poche gutturale chez un poulain arabe âgé de 3 ans. Une encéphalite mycotique a causé une ataxie grave et d’autres déficits neurologiques chez un cheval. La découverte d’un grand foyer unique de malacie cérébrale avec une preuve histopathologique d’éléments fongiques a suggéré que l’infection était un transfert direct des sinus frontaux, plutôt qu’une propagation hématogène provenant de la poche gutturale.

A 3-year-old, male Arabian horse was presented to Edmonton Equine Veterinary Services in late spring with a history of acute, profuse nasal bleeding and severe ataxia of unknown duration. The horse had been turned out to pasture the previous fall with 7 other horses. The herd was being fed timothy hay from round bales, and heated water from an automatic water dispenser. One other horse in the herd displayed mild ataxia 2 wk after the presentation of this patient. The remaining 5 horses appeared normal.

Case description

Physical examination revealed normal temperature, pulse, and respiratory rate. The frontal bones had several hard, domed protrusions that were smooth, circular and measured between 8 mm and 2 cm in diameter, and were more prominent on the left than the right. An abbreviated neurologic assessment showed grade 4/5 neurologic deficits (1). The horse was severely ataxic on all 4 limbs and he preferred to lean his right side against a wall. Menace responses were bilaterally absent, but direct pupil-lary light reflexes (PLR) were present in both eyes. Tongue tone was reduced and moderate dysphagia was present. Despite his neurologic deficits, the horse was alert. He tended to be hyper-reactive to auditory and tactile stimuli.

A brief endoscopic examination of the upper airway revealed right laryngeal hemiplasia. The guttural pouches were not entered despite the history of nasal hemorrhage, as the level of ataxia displayed by the horse made a thorough endoscopic examination of the upper airway hazardous to both personnel and equipment. Radiographs of the frontal and maxillary sinuses showed a non-union fracture of the left frontal bone with bony callus proliferating around the fracture site. Descending from the callus toward the molar tooth roots was a peculiar, radiopaque structure with multiple foci of lysis.

The left frontal sinus was trephined in order to surgically sample the mass seen on radiographs. The mass was firm and easily delineated from the surrounding structures. Biopsies were submitted for histopathology, and material collected was sent for bacterial and fungal cultures.

Blood drawn for a complete blood (cell) count (CBC) on initial examination showed a significant monocytosis at $9.6 \times 10^9/L$ (normal: 0.0 to $0.46 \times 10^9$ cells/L). The packed cell volume was low at 0.26 L/L (normal: 0.32 to 0.52 L/L), but the remainder of the hemogram was normal. Biochemical analysis of the serum showed a hyperglycemia (9.1 mmol/L; normal: 2.6 to 6.1 mmol/L), hyperbilirubinemia (52 mmol/L; normal: 9 to 49 mmol/L), and an elevated creatine kinase (808 IU/L; normal: 30 to 357 IU/L). These findings were consistent with stress, recent hemorrhage, and muscle trauma.

A severe deficiency in serum iron (3 mmol/L; normal: 17 to 37 mmol/L) combined with the monocytosis was consistent with a significant chronic inflammatory process. A repeat CBC 48 h following the initial examination and treatment showed a progression in the inflammatory response with a slight increase in the monocytosis (10.1 $\times 10^9/L$) and a hyperfibrinogenemia (6.0 g/L; normal: 2.0 to 4.0 g/L).

Treatment was initiated with oral erythromycin (Apo-Erythro-S; Apotex Inc; Toronto, Ontario), 25 mg/kg body weight (BW),
q12h, rifampin (Sanofi Aventis, Laval, Quebec), 5 mg/kg BW, q12h, and intravenous flunixin meglumine (Flunazine; Vétoquinol; Lavaltrie, Quebec), 1.1 mg/kg BW, q24h, while awaiting biopsy results. Clinical signs worsened despite treatment, so the horse was euthanized with an overdose of intravenous sodium pentobarbital (Euthanyl Forte; Bimeda-MTC; Cambridge, Ontario), 128 mg/kg BW. The head was submitted for necropsy.

Gross postmortem examination was performed on the head and neck to the level of C4. The 3 swellings, originally noted on physical examination of the frontal bone, were so hard that they had to be sectioned with a band saw. Upon sectioning, foci of lysis and necrosis of the internal bone structure were evident. Internally in the right frontal sinus there was a round, nodular, fibrous proliferative mass growing from the internal surface of the right frontal bone ventrally into the right frontal sinus. This measured 1.5 cm in diameter at its base, and protruded approximately 1.5 cm into the sinus cavity. The mass was covered by a layer of inflammatory debris on which white fungal colonies were growing freely and were readily visible to the unaided eye (Figure 1).

The left guttural pouch was grossly normal. The mucous membranes of the right guttural pouch were covered with a mixture of fibrin, blood, and necrotic debris. The mucosa over and the connective tissue around the stylohyoid bone were remarkably thickened, in places to a depth of 1.5 cm, with proliferating fibrous tissue, an inflammatory infiltrate, necrotic debris and colonies of fungi (Figure 2).

In the brain there was a 6 cm × 1 cm area of yellow discoloration and softening of the lateral right cerebral cortex, with general loss of grey matter. Even following formalin fixation, which normally significantly hardens brain tissue, the affected area was palpably softened. The neuropil in the anterior aspect of the lesion was disorganized and lacked a clear difference between grey and white matter.

Histopathologic examination of the wall of the right guttural pouch over the stylohyoid bone revealed multiple layers of fibrous connective tissue of different densities. The whole mass was infiltrated by a mixed population of mononuclear inflammatory cells. Towards the surface there were blood, necrotic debris, macrophages, giant cells, and clusters of neutrophils, as well as tangled masses of fungal mycelia and sporangia (Figure 3). The fungi were typical of Aspergillus species, and the genus of the fungus was later verified by fungal culture results. Species was not identified. Sections of the nodular proliferation of the right frontal bone revealed a similar reaction to that over the stylohyoid bone with a layered mass of fibrous tissue covered by a smaller amount of inflammatory debris than in the guttural pouch, and distinct colonies of Aspergillus spp. growing on the surface. There was some proliferation of the underlying bone, but most of the mass was fibrous tissue. Sections of the brain from the most softened area revealed diffuse, mycotic encephalitis, with destruction of the neuropil and replacement with a disorganized mass of microglia, macrophages, giant cells, and fungal mycelia. There were large empty spaces in the cortex that were traversed by blood vessels.

**Discussion**

Mycotic encephalitis is a rare cause of neurologic disease in the horse, and previously reported cases have implicated hematogenous spread from an infected guttural pouch as the mode of infection. As in previous reports, this case did have unilateral guttural pouch mycosis. Mycotic infection of the guttural pouch can have catastrophic sequelae, as infection often damages the major vessels and nerves that traverse the pouch. The most catastrophic of these sequelae include uncontrollable epistaxis, and aspiration pneumonia secondary to pharyngeal paralysis (2,3).

Guttural pouch mycosis is one of the more common diseases to affect the guttural pouch, with Aspergillus spp. being the most common causative agent (4,5). The fold of mucosa over the internal carotid artery (ICA) is a site of specific predilection for mycotic infection, possibly because of the higher local temperature, or the rhythmic pulsing of the artery (2). Cases in which mycotic infection has avoided the ICA and instead invaded the maxillary artery have also been reported, although this is far less
other vessels. and the lack of invasion of the right internal carotid artery or localization of infection to the right lateral lobe of the brain, from the guttural pouch seems unlikely in this case given the nasal cavity via the cribriform plate. Hematogenous spread of infection to the brain most likely occurred from the frontal sinus bones, it is likely that infection of the frontal sinus has been reported in humans (13), but not in equids. Mycotic encephalitis is a rare sequela of guttural pouch mycosis and previous reports have indicated a hematogenous spread. In this case, the mycotic encephalitis seems to have been a sequel to a fungal sinusitis that was seeded at the same time as the unilateral guttural pouch mycosis.

This case is particularly interesting due to the combination and severity of pathology. Mycotic encephalitis is a rare sequela to guttural pouch mycosis and previous reports have indicated a hematogenous spread. In this case, the mycotic encephalitis appears normal on gross pathology. Given the presence of a lesion of the maxillary artery in two horses. Can Vet J 1984;25:393–242.


References


