Diagnosis and surgical removal of brain abscesses in a juvenile alpaca

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**Case Description**—A 1-month-old female alpaca was examined because of progressive clinical signs consistent with an intracranial lesion.

**Clinical Findings**—Clinical signs included signs of depression, lethargy, tetraparesis, and neck weakness. Two large isointense intracranial masses could be seen on T1-weighted magnetic resonance images. On T2-weighted images, the masses contained concentric rings of hypointense and hyperintense material.

**Treatment and Outcome**—2 abscesses were removed via a craniotomy that incorporated removal of the sagittal crest and surrounding skull and transection of the sagittal sinus. The bony defect was replaced with polypropylene mesh. The alpaca recovered within 2 weeks and was fully integrated into the herd within 1 month after surgery.

**Clinical Relevance**—Findings indicated that surgical removal is a feasible means of successfully treating intracranial abscesses in juvenile alpacas. (J Am Vet Med Assoc 2007;231:1558–1561)

A 1-month-old female alpaca weighing 15 kg (33 lb) was brought to the Queen’s Veterinary School hospital with a history of dullness, swaying, and head nodding that had become progressively worse over the previous week. The dam had been brought to the United Kingdom from Australia during the last month of pregnancy to join a herd of approximately 200. For 3 days following parturition, the cria remained in sternal recumbency with its head raised but swaying from side to side; signs of depression were evident. Rectal temperature was 37.3°C (99.1°F), pulse rate was 100 beats/min, and respiration rate was 32 breaths/min. Results of abdominal palpation and auscultation of the heart and lungs were unremarkable. The cria was reluctant to support its weight and had deficits in foot placement and hopping reactions in all 4 limbs. Flexor reflexes were normal in all 4 limbs, and strong patellar reflexes were present. Pupillary light reflexes and menace responses were normal. There was no evidence of pain associated with spinal palpation or manipulation. The abnormal mentation and involvement of all 4 limbs combined with an absence of cranial nerve deficits suggested a diffuse forebrain lesion or possible metabolic disorder.

Diagnostic testing included a serum biochemical panel, CBC, urinalysis, and measurement of baseline serum bile acids concentration. No clinically important abnormalities except mild neutrophilia (12.9 × 10⁹ WBCs/L; reference range, 5.4 to 11.38 × 10⁹ WBCs/L) were identified. Cerebrospinal fluid taken via lumbar puncture contained a normal protein concentration (0.41 g/L); no nucleated cells were seen.

Ceftiofur (2 mg/kg [0.9 mg/lb], SC, q 24 h) was administered because of the possibility of an infectious cause for the neutrophilia and diffuse forebrain signs. Neurologic deterioration continued over the following 4 days, to the point that the cria became unable to stand or maintain sternal recumbency and ceased to suckle. The cria became increasingly unresponsive to the environment, lying predominantly in right lateral recumbency while grinding its teeth. When lifted, the limbs and neck were flaccid, but when lying in lateral recumbency, the cria would occasionally thrust its limbs out in rigid extension. Elevation of the head induced vertical nystagmus. The progressive neurologic signs increased suspicion of a space-occupying forebrain lesion associated with early cerebellar herniation at the foramen magnum. Differential diagnoses that were considered included hydrocephalus, neoplasia, parasitic cyst, and abscess.

The cria was anesthetized with a combination of diazepam, ketamine, and sevofoamine, and magnetic resonance imaging of the head was performed with a 0.2T permanent magnet. Examination of sagittal and transverse T1-weighted images (repetition time, 800 milliseconds; echo time, 20 milliseconds) revealed a dome-shaped calvarium with caudoventral displacement of both cerebral hemispheres and compression of the lateral ventricles. The falx cerebri was deviated to the right, and there was apparent smoothing of the gyri in the left hemisphere with poor differentiation of white matter from gray matter, suggestive of a large, spheroid isointense mass in the left cerebral hemi-
sphere (Figure 1). On transverse images, the mesencephalic aqueduct was displaced toward the right, and on sagittal images, the cerebellum was severely compressed rostrocaudally, with an indentation caudally, suggestive of a high pressure within the caudal fossa secondary to subtentorial herniation of the cerebral hemispheres. On T2-weighted images (repetition time, 3,000 milliseconds; echo time, 80 milliseconds), 2 masses could be seen. One was located rostro-dorsally on the right and was compressing the olfactory lobe. The other was larger and was overlying the left cerebral hemisphere. The bulk of the larger mass was heterogenous with an isointense centre surrounded by a large area of hypointensity. Surrounding the bulk of the mass was a hyperintense ring interior to a hypointense ring separating the mass from the normal-appearing brain. Most of the smaller mass was hypointense with a surrounding high-signal-intensity ring. Examination of fluid attenuated inversion recovery images did not reveal any differences in signal intensities of the lesions, but effectively highlighted the severe compression of the lateral ventricles. Examination of gadolinium-enhanced T1-weighted images revealed contrast-enhanced rings around the 2 isointense masses. The imaging diagnosis was intracranial abscess formation.

Regular monitoring revealed a decrease in the cria's heart rate to 80 beats/min 5 hours after anesthetic recovery and deterioration of its neurologic status to a complete lack of awareness of the surrounding environment. These findings, in combination with the poor response to antimicrobial treatment and the dorsal location of the 2 masses, allowing straightforward surgical access, prompted immediate craniotomy.

The cria was again anesthetized with a combination of diazepam, propofol, and sevoflurane and positioned in sternal recumbency with its head elevated. A midline skin incision exposed the underlying calvarium, which was extremely thin with complete deficits in some places, making it impossible to create a bone flap for later replacement. Thus, Rongeur forceps were used to remove the bone and expose the larger of the 2 masses seen on magnetic resonance images. A 20-gauge spinal needle was inserted into the mass, and a small plug of thick, white caseous material was removed from within the bore of the needle, confirming that the mass was an abscess. However, it was not possible to remove the contents by aspiration. Therefore, the craniotomy site was expanded with the aim of total excision of the abscess and surrounding capsule. The sagittal crest was removed and the sagittal sinus transected. Bleeding from the sagittal sinus remained a persistent problem until sufficient access was gained to apply bipolar diathermy directly to the vessel; hemostasis, in the meantime, was achieved by use of surgical cellulose. Incision of the dura exposed the surface of the abscess. The capsule of the abscess was dissected away from normal brain tissue to permit complete excision. During this procedure, the capsule tore at the site of the previous needle puncture, revealing thick, creamy caseous material. Some of this material was removed to ease manipulation of the capsule and reduce the potential for contamination. Samples were submitted for bacteriologic culture and susceptibility testing, and treatment with amoxicillin-clavulanate (20 mg/kg [9 mg/lb], IV, q 6 h) and metronidazole (15 mg/kg [6.8 mg/lb], IV, q 8 h) was begun. The craniotomy site was extended further rostrally on the right side to expose the second abscess, which was excised in its entirety with the capsule. The extensive incision of the dura mater that was required for removal of the 2 abscesses involved incising through the falx cerebri so that the cerebral hemispheres were no longer suspended from the arch of the cranial vault and sank ventrally. After irrigation of the cranial cavity, the calvarial deficit was covered with polypropylene mesh sutured to the remaining bone with 2-0 polypropylene. The subcutaneous tissue

![Figure 1](image_url)
and skin were closed routinely, and a stent bandage was applied to the skin incision. Because of low blood pressure caused by blood loss from the sagittal sinus, 2 boluses (14 mL and 6 mL) of succinylated gelatin colloid4 were administered during surgery and a constant-rate infusion (2 mL/kg/h) was administered for 90 minutes. After surgery, a transfusion of 260 mL of whole blood from the dam was given.

Forty-eight hours after surgery, the cria was pyrexic (40.0°C [104°F]). A CBC and serum biochemical testing revealed neutrophilia (28.6 × 10³ neutrophils/L; reference range, 3.4 to 11.38 neutrophils/L) and hyperfibrinogenemia (6 g/L; reference range, 1 to 4 g/L) consistent with inflammation. Bacteriologic culture of samples obtained during surgery yielded Fusiformes spp susceptible to amoxicillin and metronidazole. Therefore, antimicrobial treatment was continued as before for an additional 7 days. Flunixin (1 mg/kg [0.45 mg/lb], IV, q 8 h) was administered for 5 days for pain relief.

The cria’s neurologic status gradually improved over the following 10 days. Immediately after surgery, the animal was responsive and could maintain sternal recumbency with its head raised. However, it was unable to stand or suckle and was fed via a nasogastric tube. Five days after surgery, the cria could walk with sling support, but struggled to stand unaided. One week after surgery, the cria started to suck milk from a bottle and could walk unaided with hobbles on its pelvic limbs. Ultrasonography of the liver, spleen, kidneys, bladder, umbilicus, and heart did not reveal any abnormalities or any source of infection. Ultrasonography through the cranial deficit revealed fluid accumulation dorsal to the brain; the gyri appeared regular, and the ventricles were in a normal position, suggesting that the brain had partially returned to its normal morphology.

Twelve days after surgery, the cria was able to stand, walk, suckle from the dam, and graze grass unaided and was discharged from the hospital. One month after surgery, the cria was fully integrated into the herd and was behaving and feeding normally.

Discussion

Brain abscesses occur commonly in newborn farm animals.1–3 Seeding of bacteria in the brain is thought to occur by extension from a primary infectious disease process involving the head (eg, cranial sinuses, nasal passages, middle ear, or head wound), via hematogenous spread from other organs, or as a result of systemic infection.2,4 However, it is not always possible to identify the infectious cause.2,3 In the cria described in the present report, no focus of infection was identified, but possible causes of the brain abscesses included hematogenous spread from the dam in utero and neonatal septicemia secondary to failure of passive transfer of immunity.5

Magnetic resonance images for the case described in the present report were strongly suggestive of a diagnosis of brain abscess.1,5 Typically, mature abscesses have an isointense to hypointense core on T1-weighted images and an isointense to hyperintense core with a hypointense capsule on T2-weighted images. Abscesses containing semisolid material tend to be isointense,7 which was the case for the abscesses in this cria. For-
Selected abstract for JAVMA readers from the American Journal of Veterinary Research

Effect of show jumping training on the development of locomotory muscle in young horses
Nancy J. Rietbroek et al

**Objective**—To investigate whether training for show jumping that is commenced early after birth affects the characteristics of equine locomotory muscle.

**Animals**—19 Dutch Warmblood horses.

**Procedures**—Horses were assigned to a trained or not trained (control) group. After weaning, training (free jumping [2 d/wk] that was alternated with a 20-minute period of exercise in a mechanical rotating walker [3 d/wk]) was started and continued until horses were 3 years old. Fiber type composition (determined from myosin heavy chain [MyHC] content), fiber area, diffusion index (area supplied by 1 capillary), citrate synthase activity, and Na\(^+\),K\(^+\)-ATPase content were assessed in gluteus medius muscle specimens collected at 0.5, 1, 2, and 3 years.

**Results**—Developmental changes included an increase in MyHC fiber type IIa and a decrease in type IIad; increases in fiber area, diffusion index, and citrate synthase activity, and a decrease in Na\(^+\),K\(^+\)-ATPase content. The MyHC fiber type I and type IId were detected in high and low proportions, respectively. Training increased Na\(^+\),K\(^+\)-ATPase content, but did not affect other variables.

**Conclusions and Clinical Relevance**—In horses, show jumping training at an early age resulted in increased Na\(^+\),K\(^+\)-ATPase content of the deep portions of the gluteus medius muscle. The lack of training effects on the other muscle characteristics can partly be explained by the fact that an appropriate (aerobic) fiber type composition was already established at training commencement. These data also suggested that the developmental changes in equine muscle represent sufficient adaptation to meet the demands of this specific training. (Am J Vet Res 2007;68:1232–1238)