Diagnosis and surgical treatment of an intracranial cyst in an alpaca cria

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Case Description—A 7-day-old female alpaca was examined because of an acute onset of diffuse central neurologic deficits.

Clinical Findings—Diagnostic imaging with CT and MRI identified an intracranial cyst occupying approximately one-third to one-half of the dorsal portion of the cranial cavity, markedly displacing the cerebral hemispheres bilaterally.

Treatment and Outcome—Initial surgical management via trephination and needle drainage was only transiently effective at resolving the neurologic signs. Craniotomy and drainage and removal of the cyst lining resulted in a sustained improvement in neurologic status, and the cria remained clinically normal and well grown at follow-up 5 months after surgery.

Clinical Relevance—This report represented the first description of the successful treatment of an intracranial cyst in a New World camelid. (*J Am Vet Med Assoc* 2012;240:1501–1506)

A 7-day-old female Alpaca cria was examined at the University of Wisconsin Veterinary Medical Teaching Hospital because of anorexia and ataxia of 1 day's duration. It was accompanied by its dam and a clinically normal twin cria. Parturition had been assisted, but both crias appeared clinically normal in the first 5 days after parturition. The affected cria developed anorexia and hypermetric ataxia and paresis the day prior to examination. The ataxia and paresis had been rapidly progressive and was grade 4 of 5 (modified Mayhew neurologic scale[1]) on examination. The cria had no affinity for the dam and was unable to locate the udder but did have a suckle reflex. On physical examination, the rectal temperature was 38.3°C (101.0°F), heart rate was 200 beats/min, and respiratory rate was 60 breaths/min. A 1-cm reducible umbilical hernia was present. The cria was small (5.4 kg [11.88 lb]) for 7 days of age but was in good body condition. The small size was attributed to being a twin. On neurologic examination, the cria was obtunded, had a head tilt to the right, and had a compulsive, mildly hypermetric, ataxic gait. The results of postural reaction testing and the finding that the cria had a tendency to lean to the right were suggestive of a right-sided anatomic localization. The lesion was localized to the right cerebrum, brainstem, and cerebellum. The compulsive behavior was consistent with a lesion in the cerebrum or thalamus, whereas the ataxia, head tilt, and hypermetria were consistent with a lesion in the vestibular system and cerebellum. The obtundation may have been the result of a lesion in the brainstem or cerebrum. The results of a CBC and serum biochemical panel were within reference limits. An IV polyurethane catheter was placed in a jugular vein,[a] and a lumbar CSF sample was collected. There was mild blood contamination of the CSF sample, but no cytologic abnormalities were noted and the total protein concentration was within reference limits. An infectious etiology was considered unlikely because of the results of CBC and CSF analysis and serum protein concentration, which suggested adequacy of passive immunoglobulin transfer. Given the age of the cria, hypoxic ischemic encephalopathy was also considered unlikely. An indwelling nasoesophageal tube was placed to facilitate feeding. Treatment was initiated with vitamin E (30 U, PO, q 24 h), thiamine (10 mg/kg [4.5 mg/lb], IV, q 24 h), flunixin meglumine (0.5 mg/kg [0.23 mg/lb], IV, q 12 h), ceftiofur sodium (5 mg/kg [2.3 mg/lb], IV, q 12 h), and polyionic fluids (50 mL/kg/d [22.7 mL/lb/d]). The cria was fed 10% of its body weight in alpaca milk replacer/d over 12 feedings via the nasoesophageal feeding tube. Over the following 12 hours, the patient's neurologic status notably worsened. The cria became stuporous, with persistent vertical nystagmus, a right-sided head tilt, and anisocoria with mydriasis of the left and miosis of the right pupil. A pupillary light response was present but sluggish in the mydriatic left pupil. A commercial hypertonic saline (7.2% NaCl) solution[b] (4 mL/kg [1.82 mg/lb], IV) was administered as a bolus for treatment of possible increased intracranial pressure, and the cria was anesthetized for diagnostic imaging. Premedication was performed with midazolam sodium (2 mg, IV) and butorphanol tartrate (1 mg, IV) followed by induction of anesthesia with propofol (120 mg, IV), endotracheal intubation, and maintenance of anesthesia via isoflurane in oxygen. Computed tomography of the skull and cra-

Abbreviation

FLAIR Fluid-attenuated inversion recovery

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nial portion of the vertebral column showed a large fluid-attenuating non–contrast-enhancing lesion that displaced the cerebral hemispheres laterally, ventrally, and caudally (**Figure 1**). The lesion was located within the longitudinal cerebral fissure, extending from the rostral portion of the frontal bone rostrally along slightly over one-half the length of the fissure, with an estimated volume of 35 mL. No communication could be demonstrated between the lesion and the ventricular system. In addition, there were multiple, small, non–contrast-enhancing regions of hypoattenuation within the cranial cervical portion of the spinal cord. A diagnosis of an intracranial cyst with marked cerebral compression with secondary syringohydromelia or cervical spinal cord edema was made. Cerebellar herniation was not demonstrated on CT; however, this was suspected on the basis of the degree of mass effect and the rapid clinical deterioration of the patient.

An emergency trephination and drainage of the cyst was performed with the cria in sternal recumbency under continued general anesthesia and the head and neck secured with a cushion[c] and towels under the neck to prevent jugular vein compression. After standard sterile preparation and draping, a 3-cm incision was made centered over the vertex of the skull. The periosteum was elevated from the parietal bone with an osteotome. Hemorrhage was controlled with electrocautery. A hole was drilled with a handheld drill in the parietal bone on the right side 1 cm from the vertex. Hemorrhage was controlled with bone wax. The dura was penetrated with an 18-gauge needle, and 25 mL of cyst fluid was aspirated. The fluid protein concentration was 341.4 mg/dL, and there were no nucleated cells. The bone defect was sealed with a small piece of bone wax. The periosteum and subcutaneous tissues were closed with 3-0 polyglyconate synthetic absorbable suture[d] in a simple continuous pattern. The skin was closed routinely. Following decompression, the patient's neurologic deficits notably improved. The cria was able to nurse from the dam but remained grade 2 of 5 ataxic and paretic. The head tilt, anisocoria, and nystagmus had resolved. Intravenous fluid administration was discontinued, and esophageal tube feedings were reduced to 5% of body weight/d and then discontinued 2 days after surgery because the cria continued to nurse aggressively. Preoperative treatment with vitamin E, thiamine, flunixin meglumine, and ceftiofur were continued.

Three days following the emergency craniotomy, MRI was performed under propofol and isoflurane anaesthesia as before (**Figure 2**). A large T2-hyperintense, T1-isointense lesion was identified slightly to the right of midline, causing displacement of the cerebral hemispheres laterally, ventrally, and caudally and occupying the dorsal one-half to two-thirds of the dorsal

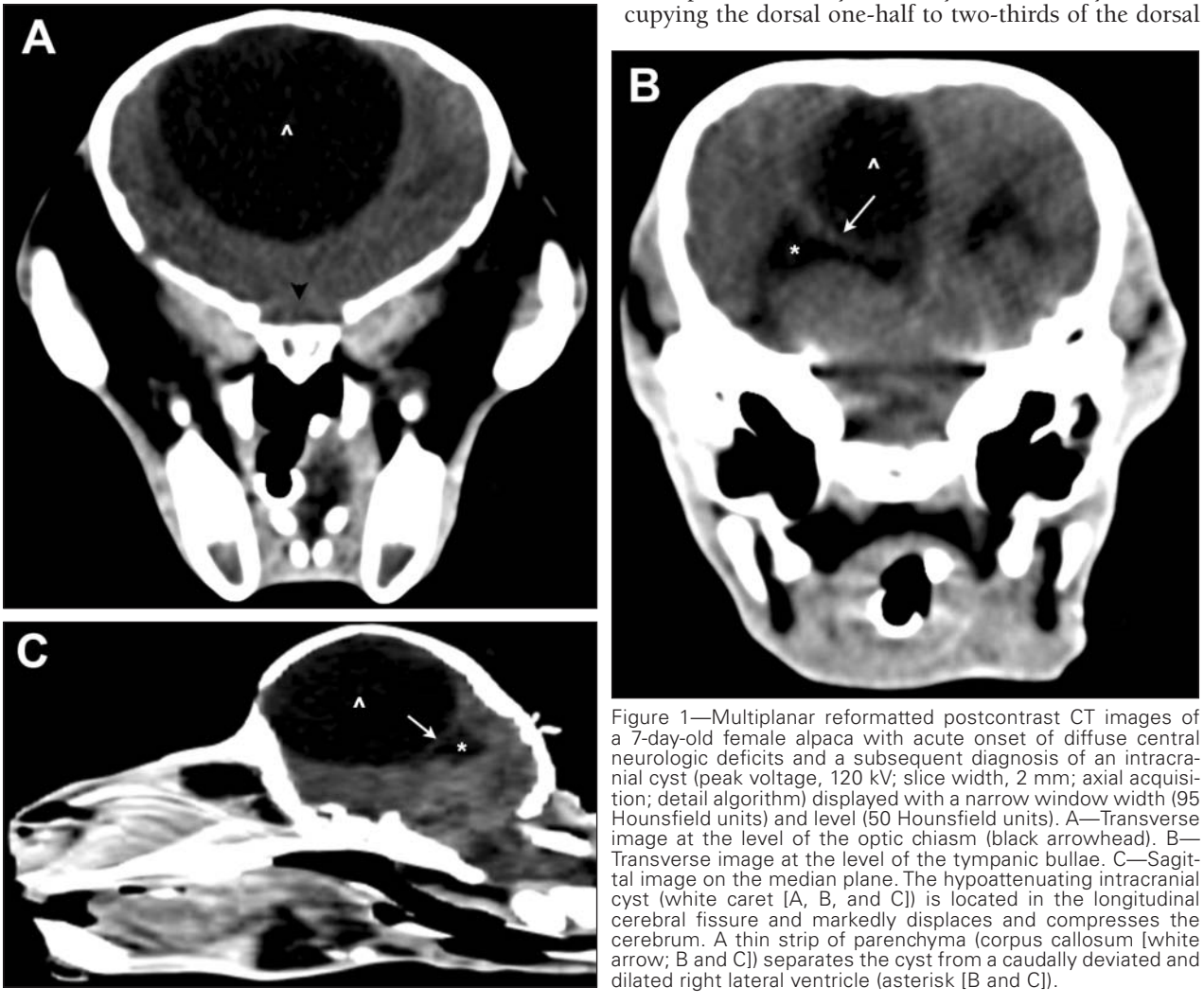

Figure 1—Multiplanar reformatted postcontrast CT images of a 7-day-old female alpaca with acute onset of diffuse central neurologic deficits and a subsequent diagnosis of an intracranial cyst (peak voltage, 120 kV; slice width, 2 mm; axial acquisition; detail algorithm) displayed with a narrow window width (95 Hounsfield units) and level (50 Hounsfield units). A—Transverse image at the level of the optic chiasm (black arrowhead). B—Transverse image at the level of the tympanic bullae. C—Sagittal image on the median plane. The hypoattenuating intracranial cyst (white caret [A, B, and C]) is located in the longitudinal cerebral fissure and markedly displaces and compresses the cerebrum. A thin strip of parenchyma (corpus callosum [white arrow; B and C]) separates the cyst from a caudally deviated and dilated right lateral ventricle (asterisk [B and C]).

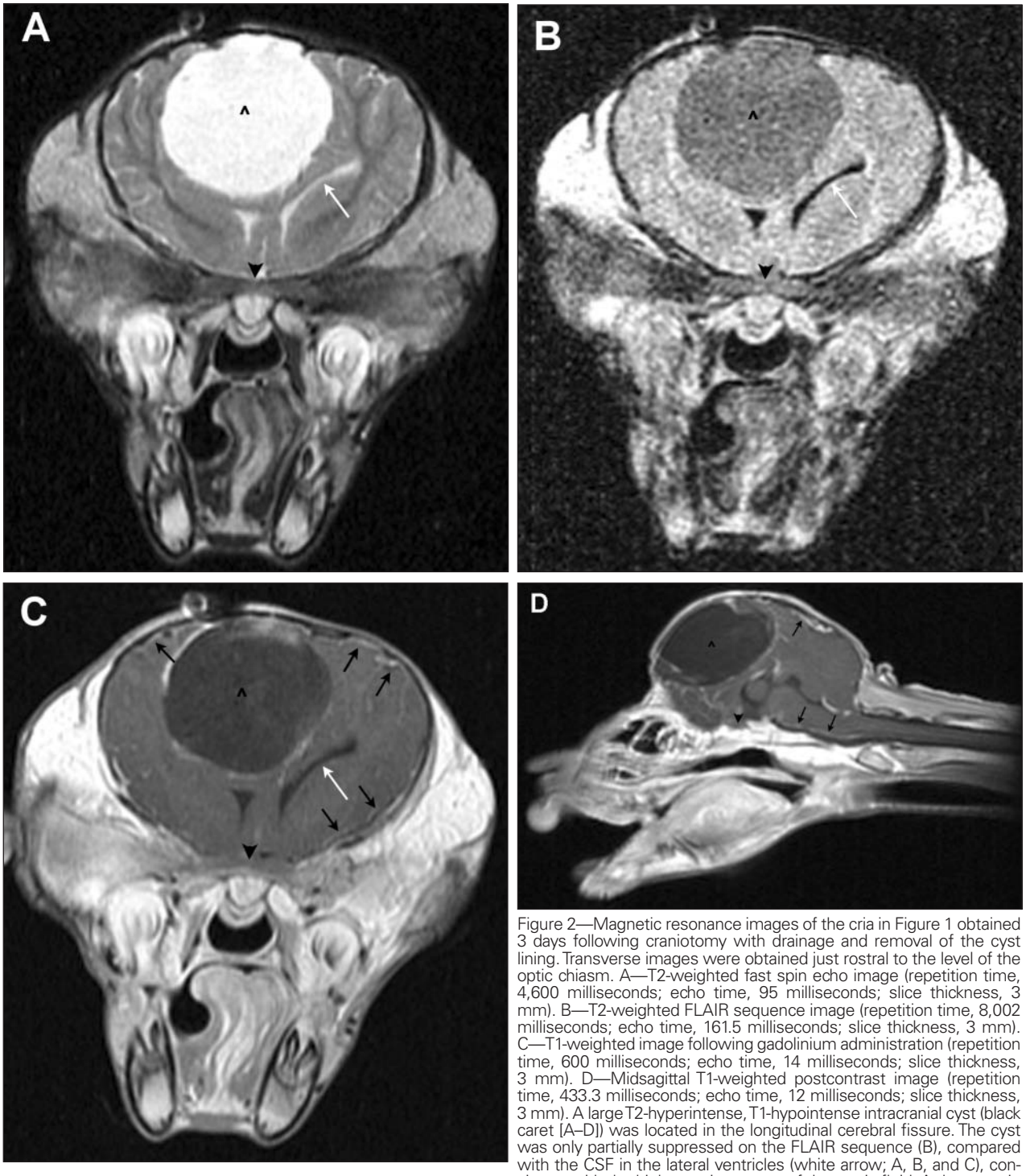

Figure 2—Magnetic resonance images of the cria in Figure 1 obtained 3 days following craniotomy with drainage and removal of the cyst lining. Transverse images were obtained just rostral to the level of the optic chiasm. A—T2-weighted fast spin echo image (repetition time, 4,600 milliseconds; echo time, 95 milliseconds; slice thickness, 3 mm). B—T2-weighted FLAIR sequence image (repetition time, 8,002 milliseconds; echo time, 161.5 milliseconds; slice thickness, 3 mm). C—T1-weighted image following gadolinium administration (repetition time, 600 milliseconds; echo time, 14 milliseconds; slice thickness, 3 mm). D—Midsagittal T1-weighted postcontrast image (repetition time, 433.3 milliseconds; echo time, 12 milliseconds; slice thickness, 3 mm). A large T2-hyperintense, T1-hypointense intracranial cyst (black caret [A–D]) was located in the longitudinal cerebral fissure. The cyst was only partially suppressed on the FLAIR sequence (B), compared with the CSF in the lateral ventricles (white arrow; A, B, and C), consistent with the high protein content of the cystic fluid. A decrease in the size of the intracranial cyst is evident, compared with CT images obtained at the same level (A and C). Meningeal enhancement was present on the postcontrast images (black arrows) and likely represents sterile meningitis from previous craniotomy.

portion of the cranial cavity. The cerebellum did not appear herniated into the foramen magnum. The mass only partially suppressed on FLAIR sequences, which was consistent with the proteinaceous nature of the cystic fluid removed at surgery. The intracranial cyst was smaller than identified by CT, with an estimated volume of 17 mL. The subjective increase in volume, compared with that calculated to be present following trephination, was believed to demonstrate the continued production of cystic fluid because the difference was greater than could be explained by innate errors in volume calculation. No communication between the ventricular system and the cyst could be identified. A 1.4 × 0.36-cm curvilinear T2-hypointense, T1-isointense, non–contrast-enhancing region that created a signal void on gradient echo sequences was located

along the caudodorsal margin of the cyst. Because no similar hyperattenuating region was present on the CT images, this lesion was determined to be an intracystic hematoma that had formed secondary to the emergency craniotomy. Contrast enhancement of the cyst lining as well as the meninges was present and likely represented sterile meningitis from the previous craniotomy. Additional MR findings included T2-hyperintense, T1-hypointense linear regions on either side of the central canal within the dorsal cervical portion of the spinal cord. This was consistent with spinal cord edema or syringomyelia as suspected on the CT scan. Furthermore, a 1.2 × 1.1 × 0.8-cm, T2-hyperintense, mildly T1-hypointense, well-defined, contrast-enhancing triangular region consistent with infarction was identified in the right occipital lobe immediately caudal to the intracranial cyst. A diagnosis of an intracranial cyst, possibly subarachnoid in location, was made.

The cria's neurologic status continued to improve, and by day 7 of hospitalization, 6 days following surgery, no neurologic deficits were appreciated. Flunixin meglumine and thiamine administration were discontinued on day 5 of hospitalization, but antimicrobial treatment was continued as before. However, 10 days after trephination and drainage, acute deterioration in the patient's status was noted. The cria had become grade 3 of 5 ataxic and paretic, obtunded, and unable to nurse. The cria was again anesthetized as previously and placed in sternal recumbency. A 3-cm incision was made lateral to the previous incision centered over the vertex of the skull. The bone wax was removed from the previously drilled hole. The dura was penetrated with an 18-gauge needle, and 30 mL of cyst fluid was aspirated. The fluid was blood tinged. The bone defect was again sealed with a small piece of bone wax. The periosteum, subcutaneous tissues, and skin were closed

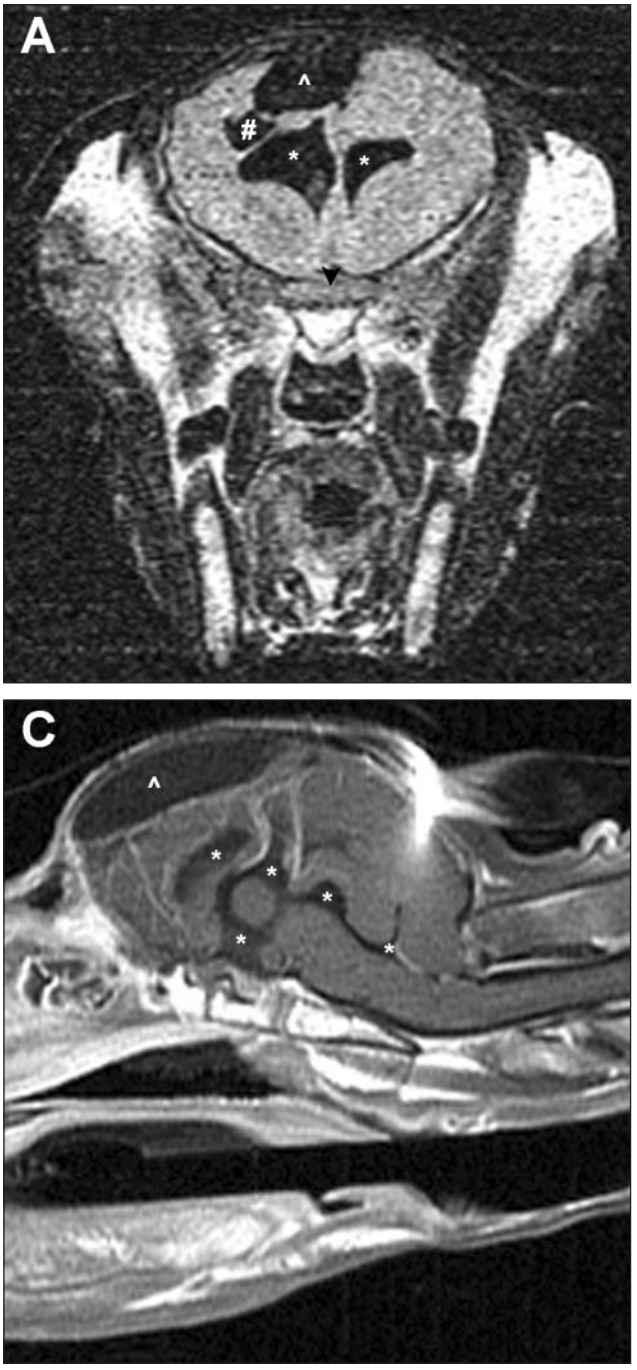

Figure 3—Magnetic resonance images of the cria in Figure 1 obtained 161 days following a third craniotomy (174 days after initial examination), with cyst drainage, debridement, and use of a temporalis muscle fascia graft to close the dural defect. Transverse images were obtained just rostral to the level of the optic chiasm. A—T2-weighted FLAIR sequence image (repetition time, 80,020 milliseconds; echo time, 153 milliseconds; slice thickness, 3 mm). B—T1-weighted image obtained after gadolinium administration (repetition time, 666.7 milliseconds; echo time, 19 milliseconds; slice thickness, 3 mm). Note the absence of meningeal enhancement. C—Midsagittal T1-weighted postcontrast image (repetition time, 516.7 milliseconds; echo time, 12 milliseconds; slice thickness, 3 mm). A moderately sized T2-hyperintense, T1-hypointense lesion (white caret [A, B, and C]) which suppresses on FLAIR sequences (A) was present in the location of the previous intracranial cyst. This region is most likely CSF that has filled in the void created by pressure necrosis of the cerebrum. An additional small region with similar MRI signal characteristics was present within the right cerebrum ventrolateral to the original cyst and corresponded to a parenchymal cistern identified at surgery (pound sign [A and B]). Mild compensatory dilation of the lateral ventricles secondary to cerebral atrophy is evident (asterisks [A, B, and C]).

as previously described. Again, the cria responded well to decompression, with notable improvement in neurologic deficits upon recovery from anesthesia.

On day 13 of hospitalization, 2 days following the second surgical decompression, the cria was again anesthetized for craniotomy and attempted removal of the cyst lining. The cria was anesthetized and prepared for surgery as previously described. The incision from the second surgery was reopened and extended rostrally to the level of the medial canthus of the eyes and caudally to the nuchal crest. Cutaneous hemorrhage was controlled by electrocautery. The periosteum was elevated from the parietal bone with an osteotome. An approximately 2 × 3-cm bone window was created with a variable-speed bone drill[e] within the parietal bone over the parietal lobe on the right side. An osteotome was used to remove the bone flap to expose the underlying dura mater. The dura mater was incised over the cyst. A high-frequency (18-mHz) ultrasound probe[f] placed over the cyst failed to identify a connection or thin membrane between the cyst and the lateral ventricles. The lining of the cyst was carefully removed. Hemorrhage was controlled with bipolar cautery. Because of moderate blood loss during surgery, a whole blood transfusion was administered. Thirty-six milliliters of the dam's blood was administered without complication. Following excision of the cyst lining, a small piece of temporalis muscle fascia was harvested to use as a graft to close the dural defect. The dura was closed with 4-0 polyglyconate synthetic absorbable sutures[d] in a simple interrupted pattern. Holes were drilled in the skull around the defect and also in the removed bone flap. The bone flap was secured to the skull with 3-0 nylon sutures[g] placed through the holes. The periosteum and subcutaneous tissues were closed in a simple continuous pattern with 3-0 polyglyconate synthetic absorbable suture.[d] The skin was closed with a subcuticular layer of 3-0 polyglyconate synthetic absorbable suture[d] and skin staples. Flunixin meglumine (0.5 mg/kg, IV, q 12 h) was given immediately prior to and for 48 hours following this surgery, and antimicrobial treatment was continued as before. The cria recovered uneventfully from anesthesia. Immediately following surgery, the ataxia and pareis persisted (grade 2/5) but mentation was normal and the cria interacted with the twin and the dam normally and was able to nurse effectively. Over the following 4 days, the ataxia and paresis improved to grade 1 of 5. The cria gained weight at an expected rate (500 g/d) during this time. Antimicrobial treatment was discontinued 3 days following the third surgery, and the cria was discharged 24 hours later.

The cria was examined 161 days following the final surgery. Results of the physical examination were normal, body weight and body condition score were appropriate, and no neurologic deficits were appreciated. The owners reported that the cria behaved normally in the herd. The cria was anesthetized and MRI was repeated (**Figure 3**). The intracranial cyst was reduced in size to 5.3 mL and was only mildly displacing the cerebral hemispheres laterally, caudally, and ventrally. The intracystic hematoma was no longer present. An additional small (0.4 mL) region with similar MRI signal characteristics was present within the right cerebrum ventrolateral to the original cyst and corresponded to a parenchymal cistern identified at surgery. No communication could be identified between these 2 lesions. Meningeal enhancement was no longer identified. There was mild compensatory dilation of the lateral ventricles secondary to cerebral atrophy. The right lateral ventricle was slightly larger than the left lateral ventricle. The parietal bone at the level of the craniotomy had reduced medullary signal intensity and was thicker than previously, indicating hyperostosis. The spinal cord edema had resolved. The previously described infarct in the right occipital lobe was smaller than previously described and was now T2-hyperintense, T1-hypointense to -isointense, and non–contrast-enhancing, consistent with a chronic infarct.

Discussion

To our knowledge, intracranial cysts have previously not been described in New World camelids. An intracranial teratoma and intracranial abscesses have been described, with successful surgical removal of the abscesses.[2,3] Arachnoid cysts develop as a result of a split in, or duplication of, the arachnoid membrane that surrounds the cyst cavity.[4–6] In human medicine, most arachnoid cysts remain stable throughout life, although some enlarge and others have been reported to spontaneously regress.[7] Mechanisms by which cysts enlarge are unknown, but 4 theories exist: a 1-way ball-valve mechanism causing entrapment of CSF, an osmotic gradient between cyst contents and CSF with subsequent fluid transport, active CSF secretion from cells in the cyst membrane, and a malformation of the arachnoid membrane or cerebral lobe agenesis.[8] In dogs and cats, intracranial subarachnoid cysts are thought to be congenital and appear to be more common in small-breed dogs. A cyst may be present without clinical signs, and clinical signs may worsen after hemorrhage into the cyst. The diagnosis may be achieved by CT, MRI, or ultrasonography.[9–14]

In human patients, the decision for surgical intervention is typically made on the basis of symptoms and their progression and the results of imaging studies. Surgical management is indicated for patients with cysts that cause symptoms because of neural compression or hydrocephalus.[15] The neurologic signs in this cria were presumably a result of increased intracranial pressure secondary to the mass effect of the very large cyst. Recurrence of these signs because of spontaneous refilling of the cyst following trephination prompted more specific surgical management. Neuroendoscopic exploration and fenestration of the cyst into a ventricle is the currently recommended technique in human neurosurgery. However, because of the lack of a small enough endoscope, craniotomy was elected in this patient. During surgery, no connection or thin membrane could be found between the cyst and the lateral ventricle. Because of a perceived higher risk of complications such as bleeding, adhesions, and occlusion of the opening from fenestration through what was assessed intraoperatively to be a relatively thick layer of brain parenchyma into the lateral ventricle, the decision was made to remove the cyst lining instead. Fenestration or

marsupialization into a ventricle should be considered in future cases.

The persistence of imaging abnormalities 6 months after surgical removal of the cyst is expected. Compression of the cerebral hemispheres likely induced pressure necrosis and, despite notable recovery of cerebral mass, complete resolution was unlikely to occur. The resolution of neurologic abnormalities highlights the tolerance of this species to some degree of cerebral parenchymal loss.

Intracranial cysts should be included as a differential diagnosis for central neurologic disease in neonatal New World camelids. The successful surgical management of an intracranial cyst with recovery of neurologic function to a level consistent with a normal quality of life in the patient in the present report should encourage treatment of future cases.

a. Mila International Inc, Erlanger, Ky.
b. Hypertonic saline (7.2% NaCl) solution, Phoenix Pharmaceutical, St Joseph, Mo.
c. Olympic Vac-Pac, Olympic Medical, Seattle, Wash.
d. Maxon, Syneture-Tyco Healthcare LP, Norwalk, Conn.
e. Surgairtome Two Drill, Zimmer Corp, Carpinteria, Calif.
f. Esaote MyLab70, Genova, Italy.
g. Monosof, Syneture-Tyco Healthcare LP, Norwalk, Conn.

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