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Neurological Causes of Diaphragmatic Paralysis in 11 Alpacas (*Vicugna pacos*)

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Background: Diaphragmatic paralysis is a relatively uncommon medical condition in animals not reported in alpacas.

Objectives: Describe the signalment, physical examination, diagnostic testing, clinical, and histopathologic findings related to diaphragmatic paralysis in alpacas.

Animals: Eleven alpacas with spontaneous diaphragmatic paralysis.

Methods: A retrospective study examined medical records from a 10-year period and identified 11 alpacas with confirmed diaphragmatic paralysis admitted to Washington State University and Colorado State University Veterinary Teaching Hospitals between September 2003 and October 2009.

Results: The 11 alpacas ranged in age from 2 to 12 months. Fluoroscopic imaging confirmed the presence of bilateral diaphragmatic paralysis in the 7 alpacas that were imaged. Arterial blood gas analyses showed hypercapnea, hypoxemia, and low oxygen saturation. Seven alpacas died or were euthanized between 2 and 60 days after onset of respiratory signs. Histopathologic examination of tissues found phrenic nerve degeneration in the 6 alpacas that were necropsied and additional long nerves examined demonstrated degeneration in 2 of these animals. Two animals had spinal cord lesions and 2 had diaphragm muscle abnormalities. No etiologic agent was identified in the alpacas.

Conclusions and Clinical Importance: The etiology for diaphragmatic paralysis in these alpacas is unknown. A variety of medical treatments did not appear to alter the outcome.

Key words: Alpaca; Diaphragmatic paralysis; Degeneration; Fluoroscopy; Phrenic nerve.

Diaphragmatic paralysis has been described in a few animals, including a pony, a dog, a llama, and 3 cats.[1–5] The condition can be hemilateral or bilateral, with bilateral showing more severe signs of respiratory distress. Diaphragmatic paralysis in animals and humans has been attributed to central or peripheral nervous system trauma or compression, degeneration of the phrenic nerves that innervate the diaphragm muscle, or myopathies.[2,4–6] Unilateral diaphragmatic paralysis can be asymptomatic; however, animals or humans with bilateral diaphragmatic paralysis typically show signs of respiratory distress and demonstrate a paradoxical abdominal respiratory pattern in which the abdominal musculature contracts inward on inspiration.[1–3,6] Blood work can be unremarkable if an inflammatory condition is not present, but arterial blood gas samples might demonstrate the presence of alveolar hypoventilation and ventilation-perfusion mismatch with a respiratory acidosis, hypercapnea, hypoxemia, and metabolic compensation.[1,3,5,6] These findings are not specific to the diaphragmatic paralysis condition. Diagnosis of diaphragmatic paralysis can be challenging to confirm. Diagnostic imaging used in animals and humans includes radiography, ultrasonography, and fluoroscopy.[3,4,6] Other diagnostic tools such as respiratory inductive plethysmography and pneumotachography, and electromyogram testing, have been utilized to confirm a diagnosis but are not commonly available to many veterinary practitioners.[1,2,6] Prognosis is dependent on the inciting cause of the diaphragmatic paralysis and whether the condition is hemilateral or bilateral.

This study reports on the signalment, clinical signs, clinicopathologic findings, diagnostic techniques, treatment outcome, and necropsy results in 11 juvenile alpacas with diaphragmatic paralysis. A single etiology for these cases was not determined because a variety of lesions were observed.

Abbreviations:

pCO_2	partial pressure of carbon dioxide
pO_2	partial pressure of oxygen

Materials and Methods

Case Selection

Medical records of alpacas with a clinical diagnosis of respiratory disease between January 2000 and December 2009 were examined at the Washington State University Veterinary Teaching Hospital and Colorado State University Veterinary Teaching Hospital. Cases were excluded if a diagnosis of diaphragmatic paralysis was not confirmed through diagnostic imaging or histopathologic identification of phrenic nerve degeneration. Information collected from the medical records included signalment, history and duration of clinical signs, physical examination findings, diagnostic tests performed, treatments provided, and necropsy results or final outcome.

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Results

Signalment and History

Records identified 11 alpacas that presented between September 2003 and October 2009 and were diagnosed with diaphragmatic paralysis. There were 6 females and 5 intact males between 2 and 12 months of age (median 4.3 months old). The alpacas were born in Washington (n = 3), Idaho (n = 4), Colorado (n = 3), or Arizona (n = 1). One farm had multiple animals affected (n = 4) and 2 farms (Arizona and Colorado) had crias with similar signs that died before the presenting animal. All alpacas presented for signs of respiratory distress or labored breathing. Four alpacas had signs of ataxia before presentation. The duration of clinical signs before presentation ranged from 1 to 32 days.

The 7 alpacas from Washington and Idaho received parenteral selenium and vitamin E at birth.[a] Eight alpacas received oral or parenteral vitamin A and D products at birth. *Clostridium perfringens* type C and D and *Clostridium tetani* vaccine was either given at birth if the dam was not vaccinated, or beginning at approximately 3 months of age.

None of the alpacas had a history of trauma or other major illness before clinical signs. The water source for all farms was well water. The farm with 4 affected animals evaluated their water source and it was found to have high calcium; no other abnormalities were noted. Alpacas <6 months old were still nursing but had access to grass pasture (Washington and Idaho) and grass or alfalfa hay (all locations). The yearling alpaca was provided grass hay and access to grass pasture. Trace vitamin and mineral supplements were provided through commercial alpaca pellets or free choice mineral salt mixtures. All alpacas were seen by referring veterinarians with no improvement observed after treatment with antibiotics, steroids, or nonsteroidal anti-inflammatory drugs, vitamin supplements, and supportive care.

Clinical Evaluation

Physical examinations revealed rectal temperature and heart rate within normal limits. All alpacas were tachypneic (48–72 breaths per minute; reference range 15–35 breaths per minute) with pronounced inspiratory effort including nostril flaring and paradoxical or reverse abdominal effort. Others respiratory signs observed included cyanosis (n = 2) and attempted open mouth breathing (n = 4). Lungs sounds were reported as "harsh" in 4 alpacas and rales or inspiratory stridor were ausculted in 2 other alpacas. All alpacas were ambulatory; however, 4 showed signs of ataxia. The alpacas weighed between 13.6 and 36 kg (mean 24.5 kg) and 4 were extremely thin with body condition scores of 1.5–3 out of 9.

Clinical Pathology Results

Blood was aseptically obtained from the jugular vein for CBC, serum chemistry, and vitamin and mineral panels (described below). CBC and serum chemistry panels were performed on all alpacas at admission and at multiple times for animals that were hospitalized. Arterial blood gas samples were obtained from the medial saphenous artery if the alpaca tolerated restraint without excessive respiratory distress.

On presentation, CBC abnormalities were mild and transient. Four alpacas demonstrated stress or physiologic leukograms with leukocytosis (median 28.7×10^3 cells/μL; range 21.2–34.0 $\times$ 10^3 cells/μL), mature neutrophilia (median 22.6 $\times$ 10^3 cells/μL; range 16.8–27.5 $\times$ 10^3 cells/μL, and monocytosis (median 1.7 $\times$ 10^3 cells/μL; range 1.3–2.3 $\times$ 10^3 cells /μL). Only 2 of the 4 alpacas demonstrated lymphopenia (median 0.5 $\times$ 10^3 cells/μL; range 0.4–0.6 $\times$ 10^3 cells/μL). One alpaca was leukopenic (5.9×10^3 cells/μL) with neutropenia (4.2×10^3 cells/μL). One alpaca had a lymphocytosis (7.9×10^3 cells/μL) and 1 alpaca had a mild decrease in hematocrit (24%). There were no other signs indicative of inflammatory or infectious processes as the fibrinogen and globulin levels were within normal limits for all 11 alpacas. All other CBC values were within normal reference values. Subsequent blood work showed values returned to within reference ranges.

Serum chemistry panels revealed transient increases in aspartate aminotransferase (median 487 U/L; range 305–810 U/L) activity in 5 alpacas and creatine kinase (median 762 U/L; range 103–1,799 U/L) activity in 8 alpacas. These increases were suspected to be caused by the respiratory effort, transport, and recumbency. Five alpacas were hyperglycemic (median 288 mg/dL; range 162–420 mg/dL) because of stress or administration of corticosteroids. One alpaca was hypoproteinemic with a total protein of 4.5 g/dL and hypoalbuminemia of 2.7 g/dL. Serum electrolytes were variable, but typically within reference ranges, and abnormalities reflected hydration status and anorexia.

Arterial blood gas samples were obtained at admittance from 7 alpacas. Six alpacas were hypercapneic (partial pressure of carbon dioxide [pCO_2] 50.6–79.9 mmHg; reference range 35–47 mmHg). All 7 were hypoxemic (partial pressure of oxygen [pO_2] 21.4–54.6 mmHg; reference range 62–96 mmHg) with decreased oxygen saturation (23.3–88.8%; reference range 99–100%). Four alpacas had respiratory acidosis (pH 7.202–7.34; reference range 7.35–7.45) and 2 had a compensatory metabolic alkalosis (bicarbonate 32.7–39.7 mEq/L; reference range 22–30 mEq/L). When the animals were administered intranasal oxygen supplementation, the oxygen saturation improved in 3 alpacas; however, the pCO_2, and pO_2, and respiratory patterns did not improve.

Toxicology, Vitamin, and Mineral Results

Serum (n = 6), whole blood (n = 3), and liver (n = 4) trace and macro mineral levels were measured in various animals with most values within normal limits. Minerals measured included arsenic, barium, cadmium, cobalt, chromium, copper, iron, manganese, molybdenum, lead, sulfur, zinc, and selenium. Selenium (serum, blood, or liver) was within reference ranges in 8 of 9 alpacas except for an alpaca located in Colorado (0.07 ppm; reference range 0.12–0.20 ppm). Arsenic (liver, n = 3) and lead (liver, n = 3; blood, n = 1) concentrations were below

detectable limits. Serum vitamin E concentrations were obtained on 9 alpacas. Levels were low in 3 alpacas located in Colorado (22–135 μg/dL; reference range 150–600 μg/dL).

Immunodiagnostic Testing

Various serologic, culture, immunohistochemistry, electron microscopy, and PCR diagnostic tests were performed on different alpacas, with more extensive testing performed on earlier cases. Serum, bronchoalveolar lavage fluid, cerebrospinal fluid, feces, and tissues samples were tested in select alpacas. Alpacas were negative for bluetongue virus (n = 1), border disease virus (n = 1), bovine viral diarrhea virus (n = 1), caprine arthritis encephalitis virus (n = 1), equine herpes virus-1 (n = 3), fungal infections (n = 1), llama adenovirus (n = 2), *Neospora* (n = 1), and *Mycoplasma* (n = 1). Three of 5 alpacas had mild increases in West Nile virus antibody titers; however, only 1 had a history of vaccination. Two alpacas were tested for myasthenia gravis by administering edrophonium[b] or measuring acetylcholine receptor antibody concentrations and both were negative. One alpaca was negative for exposure to organophosphates. One alpaca was tested for coronavirus and was positive on fecal samples; however, phrenic nerves were negative on immunohistochemistry using both bovine and feline assays.

Diagnostic Imaging

Lateral and dorsoventral thoracic radiographs were obtained on 9 alpacas within 1 day of arrival at the hospital. Three alpacas had radiographic signs consistent with pulmonary atelectasis, 5 alpacas exhibited findings suggestive of interstitial pneumonia, and 1 alpaca was noted to have cranial displacement of the diaphragm.

Thoracic fluoroscopy was performed on 8 alpacas and demonstrated findings consistent with bilateral diaphragmatic paralysis. Two healthy juvenile alpacas were also evaluated to determine normal diaphragm motion. A diagnosis of diaphragmatic paralysis was made based on minimal to absent craniocaudal movement (< one-half of a vertebral body) of the diaphragmatic crura during inspiration and expiration. The reports also commented on significant inspiratory intercostal expansion and cranial displacement of the diaphragm regardless of respiratory phase in 2 alpacas.

Thoracic ultrasound was performed on 1 alpaca and showed minimal excursion of the diaphragm and minimal comet tails over the pleura. This same alpaca had neutropenia but no other signs supportive of an infectious or inflammatory process.

Other Diagnostic Testing

Follow-up neurologic examinations were performed on the 4 alpacas that appeared to be ataxic on presentation. Hind limb gait or placement abnormalities were present in the 4 alpacas and 3 also had abnormalities in the front limbs. Peripheral lesions were suspected in 3 alpacas. Cervical lesions were suspected in 1 alpaca, but the owner declined additional imaging diagnostics. Cranial nerves were normal in 3 of the 4 alpacas, and the 4th alpaca had decreased menace and pupillary light responses and a mild head tremor. No muscle atrophy was appreciated on palpation of the alpacas.

Electromyogram testing of 1 alpaca showed normal activity in the intercostal and caudal cervical muscles but little to no electrical activity in the diaphragm, indicating abnormal muscle activity. Caudal cervical area and quadriceps muscles biopsies results[c] from the same animal found that the caudal cervical muscles appeared normal; however, the quadriceps muscle sample showed small groups of atrophic fibers indicative of early or mild neuropathy. This alpaca did not show other signs of a peripheral neuropathy and was not ataxic.

Other diagnostic testing included echocardiography (n = 4) and bronchoalveolar lavage and upper airway endoscopy (n = 1) with no abnormalities noted.

Treatment

Upon arrival at the veterinary teaching hospital, 8 alpacas demonstrating severe respiratory difficulty were provided supplemental oxygen by mask or nasal oxygen cannula. Treatments instituted included antibiotics (n = 10), nonsteroidal anti-inflammatories (n = 5), thiamine hydrochloride (n = 3), oral vitamin E (n = 5), selenium and vitamin E (n = 2), vitamin A and D (n = 2), furosemide (n = 2), gastroprotectants (n = 3), albuterol (n = 1), fenbendazole (n = 2), ivermectin (n = 1), partial parenteral nutrition (n = 1), and transfaunation with bovine rumen contents (n = 1). None of the alpacas appeared to improve with any medical therapy while hospitalized.

Outcome

The onset of clinical signs to death or euthanasia (n = 6) was on average 26 days with a range of 2–68 days. Cause of death (n = 4) was respiratory arrest, suspected to be secondary to hypoxemia or muscle exhaustion. Euthanasia (n = 2) was performed because 1 alpaca's condition deteriorated and the other did not show clinical improvement after 3 weeks. Of the 5 alpacas that were discharged alive, 1 died 2 weeks later. None of the 4 surviving alpacas showed signs of ataxia or other gait abnormalities. Only 1 alpaca from Washington survived; she was a year old and had been showing signs for approximately a month before presentation. She was reexamined 6 weeks after discharge and her respiratory effort had significantly decreased, but an abdominal component remained, which increased when under stress. She is still alive. The other 3 surviving alpacas (1 female, 2 males) were from Colorado and were between 2.5 and 3 months old at discharge. Two had low vitamin E levels and supplementation was provided. Vitamin E levels on the 3rd animal were not evaluated but the alpaca was also supplemented. The respiratory signs appeared to resolve over time, and the 3 are still alive with no indications of respiratory abnormalities. None of the alpacas have experienced a relapse of clinical signs. The farm in Idaho had normal vitamin E levels in their herd

and placed all juvenile alpacas on oral vitamin E supplementation. This did not prevent occurrence of additional cases.

Postmortem Examination

Six alpacas received complete necropsy examinations. The alpaca that died after discharge did not have a necropsy performed. Gross necropsy findings were generally unremarkable and with most findings unrelated to the clinical signs. Emaciation (n = 3), pulmonary atelectasis (n = 2), bronchopneumonia (n = 1), thymic atrophy (n = 2), hypertrophic rectus abdominus muscles (n = 1), small ventricular septal defect (n = 1), and a maxillary abscess (n = 1) were reported. One alpaca had marked circumferential subarachnoid hemorrhage from C5 to T3 and more mild hemorrhage at T11–T13 and L4–L7 (suspected to be iatrogenic after spinal fluid collection attempt).

Histologic findings of various nerves, spinal cord segments, and diaphragm muscles were variable. Electron microscopy (n = 2 alpacas) and stains (hematoxylin and eosin stain, Luxol fast blue stain for myelin degeneration, and Bielchowsky stain for axonal degeneration) were used to further evaluate abnormalities. No signs of inflammation or infectious organisms were noted.

Distal axonal degeneration of 1 or both phrenic nerves was observed in all 6 alpacas (Fig 1). Four also showed myelin degeneration of the distal phrenic nerves. Myelin and axonal degeneration was observed in other major nerves including the radial, ulnar, sciatic, vagus, and femoral nerves in 2 alpacas. Degenerative changes to affected nerves were observed at the distal ends with variable degrees of progression proximally. No measurements were available. Examination of cervical and thoracic nerve roots did not show any abnormalities. Four alpacas showed only a peripheral neuropathy, while the other 2 showed axonal degeneration in the spinal cord at the cervical (C2–C5) (n = 1) and lumbar regions (L3–L5) (n = 1). The alpaca with cervical axonopathy also had similar signs in the mesencephalon region of the brain, which did not correlate with the origins of the phrenic nerve or respiratory centers of the brainstem. The cause of the lesion or the relationship with the diaphragmatic paralysis was not clear. The alpaca with the lumbar Wallerian degeneration and axonopathy (L3–L5) did not have histologic spinal cord lesions at the C5–T3 and T11–T13 areas of hemorrhage and no cause for the hemorrhage was identified. The clinical signs for this alpaca did not correlate with the lesions.

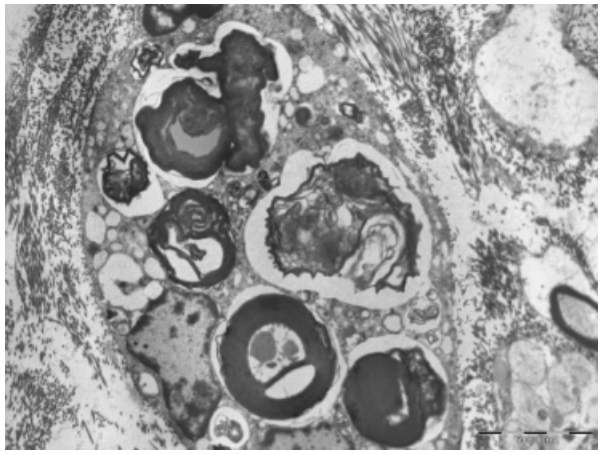

Fig 2. An ultrastructural image of the distal phrenic nerve of an alpaca with diaphragmatic paralysis shows several degenerate axons within a nerve bundle. Myelin sheaths are in various stages of degeneration with vesicular splitting and coiling of myelin lamellae. Affected axons vary from intact to vacuolated and degenerate with loss of neurofilaments and collapse. Uranyl acetate and lead citrate (scale 5.0 μm. Bar = 5,000 nm).

Four of the necropsied alpacas had shown signs of ataxia or tetraparesis on initial examination. Two of these 4 alpacas had cervical and lumbar spinal cord lesions. One had lumbar lesions associated with Wallerian degeneration and the 4th alpaca had multiple nerves showing signs of degeneration. Changes observed in the diaphragmatic muscles of 2 alpacas included widespread moderate to severe myofiber atrophy and multifocal, mild, nonsuppurative perivascular myositis in 1 of these alpacas.

Electron microscopy of phrenic nerves from 2 alpacas detailed both distal myelin and axonal degeneration. The report from 1 alpaca confirmed the peripheral polyneuropathy with severe axonal and myelin degeneration, particularly in the large diameter fibers of most sections examined. In the 2nd alpaca (Fig 2), degeneration was primarily centered on myelin with early degeneration of Schwann cells and late degeneration of axons.

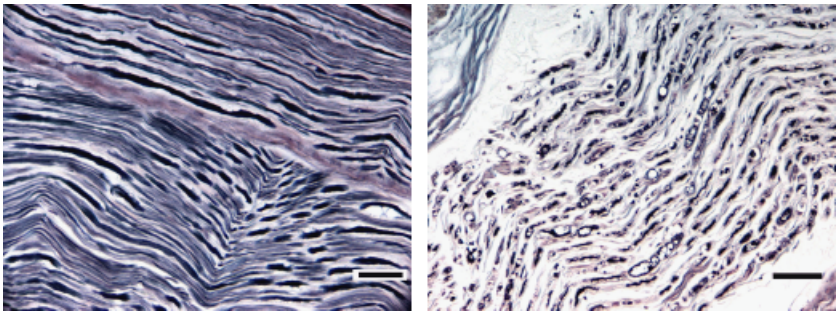

Fig 1. The image on the left is a normal radial nerve. The image on the right is from the distal phrenic nerve of an alpaca diagnosed with diaphragmatic paralysis. Note severe swelling and vacuolation of axons. Both sections are stained with Bielchowsky stain. ×200, bar = 40 μm.

Discussion

The 11 alpacas reported in this study demonstrated varying degrees of dyspnea and respiratory distress. Respiratory disease is relatively uncommon in alpacas, and other causes of the respiratory signs such as choanal atresia, airway obstruction, depression of the respiratory center, restrictive airway disease, pneumonia, or lung compression were ruled out based on diagnostic testing. Fluoroscopic examination revealed failure of normal diaphragmatic contraction in the 8 alpacas examined. The other 3 alpacas were highly suggestive of diaphragmatic paralysis based on clinical signs, and a definitive diagnosis was confirmed by thoracic ultrasound or histopathology showing phrenic nerve degeneration. One alpaca was found to have signs of pneumonia on necropsy; however, antemortem blood work did not indicate an infectious or inflammatory process. Two others had pulmonary atelectasis, which was thought to be secondary to the diaphragmatic paralysis and decreased inspiratory ability.

Diaphragmatic paralysis is more widely described in humans than in animals and bilateral paralysis is relatively uncommon.[6] Bilateral diaphragmatic paralysis in humans can develop after various medical conditions that cause lesions in the brain or spinal cord, cervical nerve roots, peripheral nerves, or the diaphragmatic muscle.[6] Cervical or thoracic trauma from surgery, injuries, or diseases such as neoplasia and aberrant parasite migration, such as by *Parelaphostrongylus tenuis*, can damage the cervical spinal cord, nerve roots, or the phrenic nerve. Inflammatory conditions such as pneumonia can affect the phrenic nerve passing through the thoracic cavity. Central or peripheral neuropathies such as viral, metabolic, toxic, or immune-mediated diseases can lead to degeneration of the spinal cord, nerve roots, or phrenic nerve. Finally, myopathies can directly affect the diaphragm muscle or accessory respiratory muscles leading to respiratory weakness.

The alpacas in this study appeared to have a variety of lesions responsible for or contributing to the diaphragmatic paralysis. Two alpacas had findings associated with a diaphragmatic myopathic condition and multiple peripheral nerves showing signs of degeneration. The myopathies were variable with 1 alpaca showing signs of a neurogenic atrophy and the changes in the muscle of the 2nd alpaca were either too early to shows signs of neurogenic atrophy or attributable to another unknown process. Two alpacas had spinal cord lesions as well as phrenic nerve degeneration and 2 alpacas had degenerative lesions of only the phrenic nerve or multiple nerves. Reports in humans, dogs, and horses describe similar degenerative lesions as Wallerian degeneration or ''dying back'' neuropathies.[7–9] In horses, a dying back neuropathy is most often associated with the recurrent laryngeal nerve leading to laryngeal hemiplegia.[9] Similar recurrent laryngeal nerve lesions were not reported in these alpacas and clinical signs did not indicate laryngeal involvement. Currently, there are multiple proposed etiologies regarding these types of neuropathies in humans and animals and include inadequate transport of cellular products to the distal axons, inflammatory, nutritional, metabolic, toxic, and heritable conditions.[7,9] Ancillary testing and history essentially ruled out many disease processes as the etiology in these alpacas. There are no reported genetic or heritable conditions responsible for peripheral neuropathies in camelids. Three of the alpacas shared a grandsire but otherwise no hereditary link could be made.

Several of the alpacas were in poor body condition. This could have been attributable to poor animal management or secondary because of insufficient intake with the respiratory difficulty and increased metabolic activity by the accessory respiratory muscles. Three alpacas in Colorado were found to have low vitamin E levels, otherwise vitamin and mineral deficiencies or excesses (vitamin E, thiamine, and selenium) were not observed. Neuropathies caused by vitamin E deficiencies have been reported in humans, horses, dogs, and chickens.[9] Lesions vary by species and include cerebellar, spinal cord, myopathies, and retinopathies so diaphragmatic paralysis can be a manifestation for hypovitaminosis E in alpacas. Vitamin E deficiency appears to be a common regional problem because of year-round feeding of hay with insufficient supplementation; however, many alpacas are found to have vitamin E deficiencies without other clinical signs.[10,11] Additional vitamin supplementation did not alter outcome in most of the alpacas. The effect of vitamin E is unknown but might be because of the antioxidant benefits and the supportive role in neuron function.[12]

The etiology of the peripheral neuropathies was not determined and a common cause was not identified in this series of animals. Interestingly, 3 farms had more than 1 animal affected with similar clinical signs and were either confirmed or highly suspicious of diaphragmatic paralysis. Two farms continue to have affected alpacas (personal communication).

The combination of clinical signs and fluoroscopic imaging allows for a rapid and accurate determination of diaphragmatic paralysis in alpacas. Minimal respiratory stress occurs during the fluoroscopic procedures because the alpacas are maintained in sternal recumbency and oxygen can be provided as needed during the procedure. Thoracic radiographs can be difficult to interpret as it is difficult to obtain consistent inhalation or exhalation images and inhumane to restrict breathing in order to adequately access diaphragm position. Hemiparalysis often causes a characteristic increases in the affected side that is easily observed on thoracic films.[4,13] Ultrasound can be used but requires a skilled operator who is familiar with thoracic anatomy. Other diagnostic testing that can aid in diagnosing diaphragmatic paralysis in animals includes arterial blood gas measurements, respiratory inductive plethysmography and pneumotachography, nerve conduction studies, and electromyogram testing.[1,2,6] Transcranial magnetic stimulation is a relatively new tool for use in animals and might be helpful in evaluating nerve conduction abnormalities and localizing spinal cord lesions.[14]

Prognosis for bilateral diaphragmatic paralysis in alpacas appears guarded, particularly when both respiratory and neurologic deficits are observed. Medical

treatment did not appear to result in direct improvement of diaphragmatic function, but vitamin E supplementation might have been beneficial in some animals. Supportive care with oxygen supplementation and stall rest in a low-stress and low-activity environment is recommended until the animal can develop a satisfactory abdominal and intercostal respiratory function to become stable or nerve regeneration occurs. Four alpacas did survive the acute crisis associated with the diaphragmatic paralysis. Return to normal respiration occurred slowly over 4–6 months.

Surgical plication of the diaphragm is a potential treatment option.[3,15] Other treatment and supportive modalities in humans include phrenic nerve pacemakers and continuous positive airway pressure ventilation to reduce hypoxemia.[16] Further evaluation is required to determine the practicality and success in implementing these in alpacas with bilateral diaphragmatic paralysis.

Footnotes

[a] BO-SE, Intervet/Schering-Plough Animal Health Corporation, Whitehouse Station, NJ

[b] Tensilon, ICN Pharmaceuticals, Costa Mesa, CA

[c] Comparative Neuromuscular Laboratory, University of California, San Diego, CA

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