Streptococcus equi subsp zooepidemicus pleuropneumonia and peritonitis in a dromedary camel (Camelus dromedarius) calf in North America

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Case Description—A 12-week-old female dromedary camel (Camelus dromedarius) calf was evaluated at the Texas A&M Veterinary Medical Teaching Hospital in late spring because of inappetence and lethargy of < 24 hours’ duration. The calf had been acquired 2 weeks previously from a farm in Michigan. The calf was transported to Texas, where it was housed with multiple species of livestock, including horses, as part of a circus. The cow had rejected the calf, which was being bottle-fed lamb milk replacer 3 times daily (exact volumes absent. The calf was considered mildly thin with a body condition score of 2 on a scale from 1 to 5. All other physical examination parameters were normal.

On physical examination, the calf was lethargic, sternally recumbent, and reluctant to move. Cardiothoracic auscultation indicated decreased bronchovesicular sounds bilaterally, tachycardia (40 beats/min; reference range, 32 to 36 beats/min), and tachypnea (20 breaths/min; reference range, 5 to 12 breaths/min) suggestive of pneumonia. The rectal temperature was 38.9°C (102.0°F; reference range, 36.0°C to 40.5°C [96.8°F to 104.9°F]). On abdominal auscultation, borborygmi were absent. The calf was considered mildly thin with a body condition score of 2 on a scale from 1 to 5. All other physical examination parameters were normal.

Results of hematologic analysis indicated leukopenia (3.4 × 10^3 leukocytes/µL; reference range, 6.0 × 10^3 to 13.5 × 10^3 leukocytes/µL), degenerative left shift (neutrophil count, 0.17 × 10^3 neutrophils/µL [reference value, 3.0 × 10^3 neutrophils/µL to 8.1 × 10^3 neutrophils/µL]; band neutrophil count, 1.3 × 10^3 band neutrophils/µL [reference value, 0 band neutrophils/µL]), metamyelocytosis (0.68 × 10^3 cells/µL; reference limit, 0 cells/µL), monocytopenia (0.2 × 10^3 monocytes/µL; reference range, 0.5 × 10^3 to 1.4 × 10^3 monocytes/µL), and hyperfibrinogenemia (800 mg/dL; reference range, 250 to 400 mg/dL). Results of serum biochemical analysis indicated hypoproteinemia (5.7 g/dL; reference range, 6.17 to 8.02 g/dL) characterized by hypoalbuminemia (2.2 g/dL; reference range, 3.0 to 4.5 g/dL) and hypoglobulinemia (2.3 g/dL; reference range,
The thoracic and abdominal ultrasonography revealed severe hypoechoic bivacitary effusion. Moderate pleural roughening, peripheral lung consolidation, and prominent fibrin strands were identified bilaterally in the cranioventral pleural cavity, which confirmed the diagnosis of pneumonia. The large quantity of hypoechoic peritoneal effusion was accompanied by numerous hypomotile to amotile large and small intestinal segments. A thoracic tube (14F; sixth intercostal space, ventrally) was placed for drainage of pleural fluid from the right hemithorax, and approximately 400 mL of nonfetid, turgid, straw-colored pleural fluid was evacuated. The thoracic tube was secured by means of a Chinese finger-trap suture with 2-0 nylon, and a Heimlich valve was attached for continual passive drainage. Cytologic evaluation of a pleural fluid sample revealed a septic, suppurative exudate (43.4 $10^3$ WBCs/$\mu$L; reference range, 0.07 $10^3$ to 0.13 $10^3$ WBCs/$\mu$L) with intra- and extracellular gram-positive cocci. Abdominocentesis was performed via a left paracostal approach with a No. 12 scalpel blade for insertion of a 14-gauge (6-cm) teat cannula. A nonfetid, turgid, straw-colored peritoneal fluid sample was collected and found to be a septic suppurative exudate (29.6 $10^3$ WBCs/$\mu$L; reference range, 0.07 $10^3$ to 0.13 $10^3$ WBCs/$\mu$L). There were intra- and extracellular gram-positive cocci on cytologic analysis, similar to findings for the pleural fluid.

Infectious polyserositis was suspected on the basis of the septic bicavitary effusion and clinicopathologic indicators of infection including leukopenia and a decrease in total protein concentration. Initial treatment included IV fluid therapy (lactated Ringer’s solution with 1% dextrose, 2.5 mL/kg/h [1.14 mL/lb/h]), empirical antimicrobials (ceftiofur sodium, 5 mg/kg [2.27 mg/lb], IV, q 6 h), and penicillin (22,000 U/kg [10,000 U/lb], IV, q 12 h), and flunixin meglumine (1 mg/kg [0.45 mg/lb], IV, once, then 0.5 mg/kg [0.227 mg/lb], IV, q 12 h). Bacteriologic culture and antimicrobial susceptibility testing of the pleural and peritoneal effusions yielded pure growth of Streptococcus spp. The organism was susceptible to multiple antimicrobials, including cephaflor and penicillin, confirming the appropriateness of the initial empirical antimicrobial choice.

On day 2 of hospitalization and treatment, the calf was ambulatory, bright, alert, and responsive, and its appetite had improved. Cardiothoracic auscultation revealed a grade 5/6 holosystolic heart murmur that was louder on the right side of the thorax, near the chest tube location. No arrhythmias or pulse deficits were identified. Echocardiography revealed cross-sectional enlargement of the pulmonary artery, suggestive of pulmonary hypertension secondary to lung consolidation. No valvular abnormalities were identified to explain the acute cardiac murmur. The right thoracic tube remained patent and had been draining slowly overnight. Repeated ultrasonography of the thorax suggested a reduced volume of pleural fluid in the right hemithorax. Peripheral lung consolidation remained, and bilateral hepatopatination of the cranioventral lung fields was identified. The fibrin accumulation appeared increased and thus compartmentalized some of the pleural fluid. A moderate amount of pleural fluid remained in the left hemithorax. Therefore, pleural drainage of the left hemithorax via a 14F thoracic tube (sixth intercostal space, ventrally) was performed, and 400 mL of nonfetid, turgid, straw-colored fluid was obtained. The fluid appeared grossly very similar to the previous pleural fluid obtained from the right hemithorax on day 1. The left thoracic tube was secured with a Chinese finger-trap suture (2-0 nylon) and left in place with a Heimlich valve for continual passive drainage. On abdominal ultrasonography, the amount of peritoneal fluid was estimated to be moderate, and gastrointestinal borborygmi appeared improved for both the large and small intestines. Abdominal auscultation also revealed normal borborygmi. Bottle-feeding of approximately 500 mL of goat milk replacer every 6 hours was initiated, then increased to approximately 500 mL every 2 hours. The milk supplementation was changed from lamb milk (X-Antadrink) to goat milk because its nutritional qualities are more similar to camel milk. The initial feeding regimen was conservative and started at approximately 3.5% of body weight (2 L/d). The regimen was gradually increased to 10% of body weight (6 L/d). A small amount of self-limiting diarrhea developed (resolved in approx 48 hours), and results of a McMaster fecal egg count were negative. The diarrhea was attributed to the diet change and stress.

On day 3 of hospitalization, the calf continued to clinically improve. Repeated thoracic and abdominal ultrasonography revealed substantial reduction in the amount of free pleural and peritoneal fluid. Additionally, the appearance of fibrin in the thorax continued to increase bilaterally, and pleural lavage with 50 mg of cefiotiofur sodium in 200 mL of lactated Ringer solution was performed bilaterally once. The thoracic tubes did not remain patent and were both removed. The leukopenia had resolved, but the hyperfibrinogenemia (700 mg/dL), band neutrophilia (2.1 $10^3$ band neutrophils/μL), and metamyelocytosis (0.51 $10^3$ cells/μL) remained. Serum biochemical analysis revealed slight worsening of the hypoproteinemia (5.0 g/dL). The decrease in total protein concentration was attributed primarily to the improvement in hydration. On day 4 of hospitalization, thoracocentesis was performed in the right craniothorax and revealed a septic suppurative exudate similar to that seen on day 1. However, subjectively, there were fewer bacteria evident than on the sample obtained the day of admission. The calf’s diarrhea had resolved and appetite remained normal. The daily feeding plan included 6 L of goat milk replacer/d via equally divided bottle-feedings every 2 hours.

On day 5 of hospitalization, the calf developed signs of colic and had reduced fecal output with mild abdominal distention (suspected gas or spasmodic colic). Bottle feedings were discontinued for 24 hours, and treatment included orogastric administration of magnesium sulfate (0.5 g/kg, PO, once) in 1 L of water as a laxative. The calf passed normal feces that same day, and signs of colic resolved without further treatment.
Feedings were gradually introduced over a 24-hour period, and pantoprazole (0.65 mg/kg [0.30 mg/lb), IV, q 24 h) was administered empirically to address potential third-compartment ulceration. A CBC indicated resolution of the metamyelocytosis and mature neutrophilic leukocytosis (19.1 × 10^3 cells/µL). The neutrophilia was attributed to the ongoing inflammatory response associated with pneumonia, fibrinous pleuritis, and peritonitis.

Over the next week, the pleural and peritoneal effusions resolved, and there was evidence of consolidation of the pleural fibrin on repeated ultrasonography. Additionally, the idiopathic heart murmur resolved. The calf’s body weight had increased from 61 kg at admission to 64 kg (140.8 lb), and antimicrobial treatment was changed to ceftiofur hydrochloride (2.5 mg/kg, IM, q 12 h). The patient was discharged from the hospital 11 days after initial evaluation and prescribed 2 weeks of continued antimicrobial treatment (ceftiofur hydrochloride; 2.5 mg/kg, IM, q 12 h) at home. Follow-up 4 weeks after discharge (approx 6 weeks after initial evaluation) from the hospital revealed that the calf had no clinical signs of relapse or reported complications. Long-term follow-up was attempted in this case but could not be accomplished because of poor owner compliance. We do not know whether this calf was able to fulfill its intended purpose of being part of a circus show.

**Discussion**

The case described in the present report illustrated that *S equi* subsp *zooepidemicus* may cause polyserositis in Old World camels (eg, dromedary camels) in North America. The patient’s rapid response to medical treatment suggested that *S equi* subsp *zooepidemicus*-induced polyserositis in dromedary camels may respond favorably to appropriate treatment. *Streptococcus equi* subsp *zooepidemicus* is the etiologic agent for one of the most important diseases in the South American camelids of Altiplano.1,2 In Peru, this condition is also known as alpaca fever.3 The morbidity rate in infected New World camelids (eg, alpacas and llamas) may be as low as 5% to 10%, but the mortality rate of those affected varies from 50% to 100%.4 Transmission may be via inhalation or oral ingestion from contaminated objects or via direct contact with other animals. The systemic forms are frequently associated with stressors such as travel, inclement weather, or malnutrition.4,5

This calf of the present report had recently traveled from Michigan to Texas and was being housed with a group of other livestock. In New World camelids, signs of acute disease typically develop approximately 24 hours after transport or experimental infection.6 However, for the calf of this report, a history of travel within several weeks before onset of clinical signs was similar to that identified in a 2009 outbreak of alpaca fever in alpacas.7 The stress of this calf being rejected by the cow and requiring nutritional supplementation may also have resulted in some degree of malnutrition. The evidence for undernourishment was the lower body condition score identified on initial evaluation and insufficient milk supplementation. Therefore, we considered that multiple stressors, including travel, malnutrition, and maternal rejection, were likely major contributors in the development and progression of the infection.

The source of infection could have included the environment, the equids housed in the pasture, or even other camelids. The most common opportunistic lung pathogen in equids is *S equi* subsp *zooepidemicus*. Similarly, there is evidence that *S equi* subsp *zooepidemicus* may be a normal inhabitant of healthy camelids.1,4 Consequently, this bacterium may have been an opportunistic pathogen that, in the setting of stress and immunosuppression, was capable of establishing a systemic infection.9 In addition, concomitant viral infections could have favored the development of the *S equi* infection.9 In 2 recent reports,5,10 the gross pulmonary lesions found in 300 camels at an abattoir in Ethiopia revealed a prevalence of 19.3% for streptococci. The various pathogens identified in those reports, including streptococcal species, were all identified in apparently healthy camels prior to slaughter. The finding that many clinically normal camels could be harboring this pathogen may make early diagnosis of alpaca fever and epidemiological studies challenging in camels.5,10 The role of viral infections in this patient was not evaluated and may be another mechanism by which this pathogen spread systemically. Ideally, an epidemiological investigation would have been performed to identify the source of the etiologic agent in the equids or other camelids located on the farm.

Although the camel population in North America does not rival that of the Horn of Africa and the Arabian Peninsula, their use in circuses or zoos remains fairly widespread. Therefore, it is important for a clinician to be able to recognize the clinical signs associated with this condition in camels. Often, the diagnosis of pneumonia in camelids is presumptive and based primarily on increased respiratory sounds and some degree of dyspnea.1 In this calf, the history and clinical signs were suggestive of pneumonia, which led to the decision to evaluate that specific body system further with ultrasonography. In a 2009 outbreak of alpaca fever in New World camelids, there was a survivor that had similar clinical signs of respiratory distress, recumbency, and colic.7 The only published reports of *S equi* subsp *zooepidemicus* infection in dromedary camels include a case of purulent rhinitis11 and septic peritonitis.12 In the report12 describing septic peritonitis in a young dromedary camel, the primary clinical sign was colic, and an exploratory laparotomy was performed. This was similar to the clinical signs described for a llama with septic peritonitis.13 Although dromedary camels are of a different genus than alpacas and llamas, it appears that both are susceptible to infections with *S equi* subsp *zooepidemicus*, and clinical signs consistent with polyserositis may be expected.1,2

The response to medical treatment in this dromedary camel calf was much better than that reported1 for New World camelids with alpaca fever. In 4 camel herds in Somalia, the streptococcal organisms described in that respiratory outbreak included both Lancefield group B and Lancefield group C organisms. The reported mortality rate for those 4 camel herds was relatively low (up to 5%) with on-farm treatment.7 This is contra-
dictory to what has been described for alpaca fever in New World camelids, in both North6,7 and South America.1 It is important to note that the dromedary camel calf described in our report required intensive medical care and almost 2 weeks of hospitalization. Further epidemiological studies are required to determine whether the mortality rates of alpaca fever are actually lower in Old World camelids.

In Kenya and Somalia, regional variation in strain pathogenicity was identified for *S equi* subsp *zooplaemicus* isolates collected from camels and camel milk.14 Given that we did not perform molecular characterization13,15 of the organism isolated from this calf, it is important to acknowledge that our treatment success may have been influenced by a less pathogenic strain. Although no controlled studies have been performed in dromedary camels to confirm or refute this hypothesis, dromedary camel farmers have reported high-morbidity respiratory infections in some dromedary camel herds that could be associated with *S equi* subsp *zooplaemicus*.9,16

The present report of a dromedary camel calf with alpaca fever further clarifies the importance of reducing stress (eg, travel and early weaning), overcrowding, and housing of equids with camelids. Although *S equi* subsp *zooplaemicus* infection in camels likely represents an opportunistic infection of a commensal organism, exposure to higher concentrations of this organism in the environment (eg, housing with equids) or exacerbation of subclinical lesions may contribute to the animal’s development of active infection. Local farmers, zoos, and circuses should strive to reduce stress and use caution when housing camels with equids. This report also demonstrates that *S equi* subsp *zooplaemicus* may cause polyserositis in Old World camelids with clinical signs that may be very similar to those seen in New World camelids. Further studies are needed to better assess the epidemiology of alpaca fever in dromedary camels in North America.

References