

Case Report Rapport de cas

Corynebacterium pseudotuberculosis liver abscess in a mature alpaca (*Lama pacos*)

Philippa Sprake, Jenifer R. Gold

Abstract – A mature female alpaca was evaluated for weight loss and a 10-day history of anorexia, diarrhea, abdominal distension, and ventral edema. Ultrasonography revealed a hepatic mass, culture of which identified *Corynebacterium pseudotuberculosis*. This is the first reported case of an internal caseous lymphadenitis lesion resulting in clinical disease in a camelid.

Résumé – Abscès hépatique à *Corynebacterium pseudotuberculosis* chez un alpaga adulte (*Lama pacos*). Un alpaga femelle adulte a été évalué pour une perte de poids et une anamnèse de 10 jours d'anorexie, de diarrhée, de distension abdominale et d'œdème ventral. L'échographie a révélé une masse hépatique et la culture a identifié *Corynebacterium pseudotuberculosis*. Il s'agit du premier cas signalé d'une lésion lymphadénite caséueuse interne produisant une maladie clinique chez un camélidé.

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An 8-year-old female huacaya alpaca with a 2-month-old cria at foot was presented to the Veterinary Medical Teaching Hospital at Texas A&M University. The alpaca had lost weight since parturition, and had a 10-day history of anorexia, diarrhea, and ventral edema. Treatment with flunixin meglumine (dosage unknown) by the owner briefly improved her appetite 2 d prior to admission. The herd was routinely tested for bovine diarrhea virus and determined to be negative. This alpaca was purchased as an adult with other alpacas several years previously from Oregon.

Case description

On presentation, the alpaca had a body condition score of 2/10 and weighed 78.6 kg. She was depressed and exhibited weakness during the physical examination with unwillingness to stand for more than a few minutes at a time. Caudal ventral and udder edema was present with poor udder development and minimal milk production. She had a distended abdomen and a fluid thrill was palpable on ballottement of the abdomen. The alpaca passed brown-colored watery feces with no evidence of tenesmus, blood, or mucus. Tachypnea was evident (40 breaths

per min) with nostril flare. Bilaterally increased lung sounds were auscultated in the dorsal lung field. Mucous membranes were pale and moist. Ultrasonography of the abdomen was performed using a 3.5 MHz curvilinear probe, which revealed an increased amount of anechoic peritoneal fluid, with minimal fibrinous adhesions cranially. Marked liver enlargement, with a well-defined parenchymal mass that appeared to be adhered to the diaphragm, was visible on the right side at intercostal space 11 (Figure 1).

Ultrasound of the thorax revealed anechoic pleural effusion with fibrin deposition on the surface of the diaphragm and parietal surface of the pleura bilaterally. The cranial mediastinum had a similar appearance. No pericardial effusion was present and all structures of the heart appeared to be within normal limits.

A chest tube (24 French, Deknatel, New York, USA) was placed aseptically in the right thorax at intercostal space 7; 2 L of pleural fluid were obtained. The tube was removed after drainage due to aspiration of air into the pleural space. Abdominocentesis was also performed under aseptic conditions using an 18-gauge 1.5-inch needle in the ventral abdomen right of the midline. Samples of the pleural and peritoneal fluid were submitted for cytology and culture. Analysis of the pleural effusion revealed a clear colorless fluid, with total protein concentration of 21 g/L and a total nucleated cell concentration (TNCC) at 336/ μ L. Cytology identified 57% non-degenerate neutrophils, 42% macrophages, and 1% small lymphocytes, which suggested a transudate with increased numbers of non-degenerate neutrophils and monocytes, indicating a possible inflammatory process. The peritoneal fluid was characterized as a modified transudate: total protein concentration of 31 g/L [reference interval (RI): < 30 g/L] and TNCC of 1180/ μ L

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Figure 1. Ultrasonographic image of the liver parenchymal mass as viewed from the right 11th intercostal space.

(RI: 2000 to 5000/ μ L) with 74% large mononuclear cells (macrophages and mesothelial cells), 25% nondegenerate neutrophils, and 1% small lymphocytes.

Serum biochemical abnormalities included hyperglycemia (10.5 mmol/L, RI: 4.5 to 8.7 mmol/L), increased blood urea nitrogen (24.6 mmol/L, RI: 4.6 to 13.6 mmol/L), hypoproteinemia (43 g/L, RI: 52 to 74 g/L) characterized by hypoalbuminemia (17 g/L, RI: 29 to 52 g/L), increased gamma glutamyl transferase (GGT) (113 U/L, RI: 8 to 30 U/L). Electrolyte abnormalities included hyponatremia (130 mmol/L, RI: 148 to 158 mmol/L), hypochloremia (96 mmol/L, RI: 97 to 117 mmol/L) and hypocalcemia (7.0 mmol/L, RI: 7.6 to 10.1 mmol/L).

Hematological abnormalities included leukocytosis (white blood cells 30 200/ μ L, RI: 7800 to 19 900/ μ L) with a neutrophilia (22 348/ μ L RI: 3650 to 13 826/ μ L) and toxic left shift (absolute bands 1208/ μ L, RI: 0 to 145/ μ L) and monocytosis (absolute monocytes 4228/ μ L, RI: 0 to 977/ μ L). The interpretation was that there was a chronic inflammatory process. There was also a non-regenerative anemia (packed cell volume 14%, RI: 26% to 47%).

Treatment was initiated with flunixin meglumine (Banamine; Intervet/Schering-Plough Animal Health, Union, New Jersey, USA), 0.5 mg/kg body weight (BW), IV, q12h, prior to a 256-mL IV bolus of llama plasma (Triple J Farms, Bellingham, Washington, USA). Following the plasma transfusion, intravenous lactated ringer's fluids (Baxter, Deerfield, Illinois, USA) with an additional 50 mL 23% calcium gluconate (Vedco, St. Joseph, Missouri, USA) per 5 L and 10 mL B Vitamin Complex (Vedco) per 5 L were administered at 1.6 mL/kg BW per hour. Antimicrobial therapy included procaine penicillin G (Pen-aqueous; Agripharm, Westlake, Texas, USA) 22 000 IU/kg BW, SC, q12h and ceftiofur sodium (Naxcel; Pfizer, New York, USA) 5 mg/kg BW, IV, q6h.

During the first night of hospitalization the alpaca became hypothermic (temperature 35.4°C). A heat lamp and blankets were added and her temperature returned to normal range in 8 h. The cause of the hypothermia was unclear. On the second day, repeated serum biochemistry revealed a more severe hypoproteinemia (33 g/L) with a further decrease in albumin concentration (12 g/L). Gamma glutamyl transferase activity

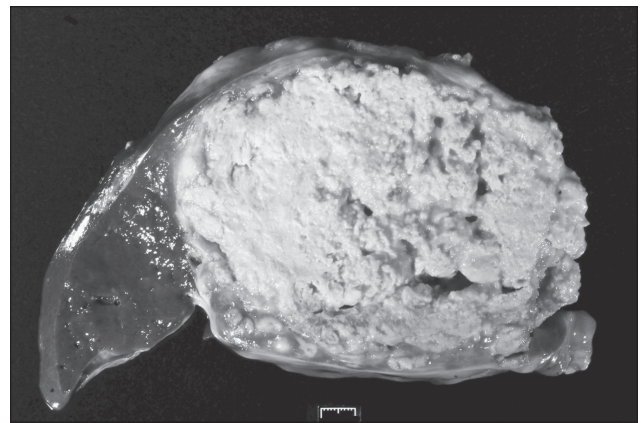


Figure 2. Cross section of the liver abscess at necropsy.

had decreased to 66 U/L. Abdominal radiographs did not reveal evidence of a metallic foreign body. Thoracic radiographs were consistent with biventricular effusion, with an alveolar pattern present in the right cranial lung lobe. There were several ovoid gas opacities within the plane of the heart and lungs ventrally, which suggested pulmonary abscessation or bullae. An abdominal drain (22 French, Deknatel) was placed in the right cranial abdomen and 12 L of fluid were recovered. Hetastarch (Texas Parenteral Medicine, Irvine, California, USA), 10 mL/kg BW, IV was added as further treatment following drainage of the abdominal fluid. Abdominal ultrasound was repeated and aspirates and biopsies were obtained from the liver mass under ultrasound guidance. Cytology of the aspirate revealed a mixed cell inflammation with bacterial sepsis and many extracellular bacteria. The cell population consisted largely of intermediate to large lymphocytes. Interpretation of the cytology was inconclusive but differentials included a neoplastic lymphoid cell population with secondary necrosis and sepsis, or a primary inflammatory process (abscess). Samples were also submitted for culture.

A fecal Wisconsin sugar floatation examination revealed thousands of *Eimeria punoensis* oocysts. On the third day, the alpaca was markedly weaker and continued to be anorexic. Due to the poor prognosis the owners elected humane euthanasia and necropsy. The results of histopathology of the liver mass biopsy were obtained just prior to necropsy and identified a severe chronic suppurative hepatitis with necrosis, consistent with liver abscessation. No neoplastic cells were identified. Pre-mortem culture of the liver biopsy obtained a pure growth of *Corynebacterium pseudotuberculosis*. Gross necropsy revealed marked subcutaneous edema, pleural effusion, and ascites. There were numerous 2 to 4 cm diameter firm nodules with caseous material on cut section (abscesses) in the lungs. The liver had numerous similar nodules ranging from 2 cm to a single large abscess measuring 13 × 9 × 7 cm. The largest abscess involved the caudal vena cava, resulting in 5 cm of the vena cava exiting the liver with coarsely nodular fluctuant thickened wall containing caseous material (Figure 2). Multiple lymph nodes exhibited hyperplasia, congestion, and edema. Histopathology and culture from the liver abscess at necropsy confirmed caseous lymphadenitis caused by *C. pseudotuberculosis*.

Discussion

Liver disease in camelids is commonly reported in the USA and is associated with vague clinical signs including weight loss, inappetance, lethargy, colic, depression, and recumbency (1,2). The most common presentation of liver disease is hepatic lipidosis secondary to decreased protein intake and stress (3). Unlike the situation in cattle, the diagnosis of primary liver abscessation is rarely reported in the literature. Pathogens that have been isolated from liver lesions at necropsy include *Rhodococcus equi* in 4 camels from the same farm, all of whom exhibited primary lung lesions (4), *Listeria monocytogenes* from the liver and ileum in an adult male alpaca with histopathological evidence of necrosis in these organs (5), and *Mycobacterium microti* in caseous nodules of multiple lymph nodes and organs, including the liver (6). Initial ultrasound images of the hepatic mass could not differentiate between neoplasia and abscessation. Hepatic neoplasias documented in camelids include lymphosarcoma (7–9), congenital hepatoblastoma (10), biliary carcinoma (11), adenocarcinoma (11) and liver metastases from a primary pulmonary carcinoma (12). Hepatoma and hemangiosarcoma have each been reported in 1 llama (8). In this alpaca, the presence of a large liver abscess obstructing the caudal vena cava resulted in bicavitary effusion, ventral edema, and diarrhea due to increased hydrostatic pressure. *Eimeria punosensis* has been associated with clinical coccidiosis in alpacas (13) and may have contributed to the mild diarrhea and low albumin and total protein in this alpaca.

Caseous lymphadenitis (CLA) caused by the gram-positive facultative intracellular pathogen *Corynebacterium pseudotuberculosis* is an important disease of small ruminants worldwide (14), leading to economic losses due to carcass trimming and clean wool loss (15). *C. pseudotuberculosis* has been cultured from multiple species including sheep, goats, cattle, buffalo, horses, zebras (16), and deer (17). The prevalence in small ruminants in the western USA has been documented to be as high as 42.5% at slaughter (15). The disease prevalence is higher in female small ruminants, possibly due to females being kept until older ages (18). The prevalence of CLA in South American camelids has not been determined but infection has been documented in both North and South America (19,20). Braga et al (19) cultured *C. pseudotuberculosis* from 45 of 84 animals with abscesses from 2 herds in the Peruvian highlands. Of those culture-positive animals with external abscessation of the superficial lymph nodes or mammary glands (or both) that had postmortem examinations performed, 4% had evidence of liver abscessation. External abscessation is more common in goats, in particular of the lymph nodes of the head and neck (15), whereas internal (21) and superficial lymph node abscesses of the torso occur more frequently in sheep (15,21). In alpacas, abscessation of renal lymph nodes and superficial lymph nodes in adults and juveniles, respectively, has been observed (15). Superficial lymph nodes in 5 young alpacas from a single farm in North America had external abscesses positive for *C. pseudotuberculosis* on culture (20). None of these alpacas showed signs of systemic disease.

In sheep, inoculation in natural disease is thought to occur trans-cutaneously via wounds and abrasions, but has been

induced experimentally via intravenous, intratracheal, intravaginal, and intralymphatic routes (15). *Corynebacterium pseudotuberculosis* survives and replicates in macrophages, which transport the organism to locally draining lymph nodes, allowing for systemic dissemination (15). Intra-dermal experimental infection of *C. pseudotuberculosis* in alpacas induced a febrile response locally 96 h after inoculation and subsequently caused purulent drainage at the inoculation site (22). There were internal abscesses (primarily of the renal lymph node and liver) in all but 1 alpaca euthanized after day 58. Experimental infection induced an increase in antibody titer against a cell wall antigen on day 16 of infection (22); however, titers were not correlated to disease severity. Unlike in sheep, pulmonary lesions have not been associated with CLA in South American camelids (23).

This herd had no previous history of external abscessation, and no other herd members have been identified with *C. pseudotuberculosis* either internally or externally.

To our knowledge, this is the first report of internal abscessation caused by *C. pseudotuberculosis* in South American camelids in North America. Caseous lymphadenitis should be considered among other causes of weight loss and internal abscessation in alpacas.

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Book Review

Compte rendu de livre

Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Dermatology. 2nd edition

Rhodes KH, Werner AH. Wiley-Blackwell, Ames, Iowa, USA, 2011. 749 pp. ISBN 9780-8138-1596-1.

This book would be a great addition to any general practitioner's library. Arranged in the typical format of all Five-Minute Veterinary Consult textbooks, the reader can quickly access the information they need. Scanning through the bullet-points of a specific chapter will deliver the information for quick reference to provide a better understanding of the etiology/pathophysiology of a dermatological disorder or a quick reminder of the clinical features, differential diagnoses, diagnostics, or therapeutic options.

The main body of the book has 9 sections, but could be considered to have 3 general "categories." The first section and "category" appropriately titled "Basics" includes a well-organized, brief review of lesion descriptions and terminology, dermatology diagnostic methods, and zoonoses. This is a useful resource for both the veterinary student first learning the world of dermatology to the general practitioner brushing up on terminology. The chapter on "Lesional and Regional Differentials" is well done and is useful in helping to formulate differential diagnoses based on the characteristics, locations, and patterns of the skin lesions in an everyday clinical case.

Sections 2 through 8 could be grouped into a "second category" as they encompass the more common dermatological disorders of dogs and cats, including allergic and hypersensitivity dermatitis; endocrine dermatoses; immunologic and autoimmune disorders; infectious dermatoses; neoplasias, cutaneous/paraneoplastic dermatoses; parasitic disorders; and some miscellaneous topics (such as, otitis, acne, anal sac disorders). The multitude of colored photographs that accompany each dermatological disorder is impressive, and would be a useful for assisting a practitioner in making a diagnosis or as examples to show a client.

The ninth section rounds out the "third category" and very briefly details the common dermatological disorders of exotic pets (ferrets, guinea pigs, hamsters, hedgehogs, mouse/rat, and rabbits).

One potentially disappointing aspect of the book is the supplementary CD. I was expecting there to be an electronic version of the text on the CD; however, only copies of the client education handouts used by the authors in their own dermatological referral practice are provided. On the upside, these twenty-two client handouts are excellent resources written for the general public in an editable word document. As such, they can be used by a practitioner as handouts for their own clients.

The same client education handouts on the CD also appear in print in the appendix, which provides a convenient drug formulary and a handy alphabetical list of breed-predisposition dermatoses.

A particular strength of this book is the "comments" sections found at the end of most chapters. These sections provide useful information ranging from expected course and prognosis, to great tips and pearls of wisdom. In addition, a small section in the appendix is written about pruritic inflammatory skin disease and provides a brief, but good drug formulary resource for the clinical management of this common presenting complaint.

One of the authors sums up this book quite well in the preface: "...this new book [is] an informative and vital resource for your clinical practice. The text is not meant to replace the more comprehensive dermatology textbooks available but rather to augment your current library with a quick clinical resource of the more common dermatological disorders." The expediency of use provided in the Five-Minute Veterinary Consult format and lots of colored photographs make this book a great addition to a general practitioner's library.

Reviewed by Kevin MacAulay, DVM, BSc (Zoology), small animal veterinarian, Glamorgan Animal Clinic, Calgary, Alberta.