Case Report  Rapport de cas

*Mycoplasma hominis* ssp. associated endocarditis with myocardial necrosis in an alpaca (*Vicugna pacos*) in Manitoba in 2011

Krzysztof M. Tomczyk, Shelagh Copeland, Rosemary Postey, Musangu Ngeleka

**Abstract** — Severe endocarditis with myonecrosis, moderate to severe pleural and pericardial effusions, and mild ascites were found on necropsy in 3 alpacas. *Mycoplasma hominis* ssp. was detected on polymerase chain reaction (PCR) of fresh affected endocardial tissue in 1 alpaca.


Can Vet J 2015;56:141–143

**Case description**

Alpacas from Case 1 (8-year-old, female), Case 2 (4-year-old, female), and Case 3 (6-year-old, male) originated from 2 unrelated farms (1 and 2 from the same farm). The 3 animals had a short history of respiratory signs interpreted as pneumonia and weight loss followed by sudden death. They were unresponsive to supportive and antimicrobial treatment (Case 1 — Baytril, Case 2 — no information, Case 3 — penicillin). Common clinical findings were: sternal recumbency, respiratory distress, tachypnea, and tachycardia. Alpacas 1 and 2 were additions to an existing herd in May 2010. Alpaca 3 was from a closed herd. All animals died naturally and the time between death and postmortem examination varied between 12 and 24 h.

Gross physical examination of the carcasses showed alpaca 1 to be in moderate body condition with subcutaneous edema extending from the sternum caudally to the udder, and alpacas 2 and 3 to be in poor body condition with no subcutaneous fat and mild muscle wasting. These latter cases both presented in late winter. Gross necropsy revealed that all alpacas had endocarditis and multifocal myocardial pallor (Figure 1), moderate to severe pleural and pericardial effusions and mild ascites (Figure 2).

Alpaca 1 had 1.5 cm to 2 cm thick green/yellow fibrinous material subjacent to the valves, which was a part of the endocarditis plaque and firmly adhered to the entire endocardium which obstructed most of both ventricular lumens (Figure 1). Histologically, the endocardium was replaced by necrotic material infiltrated by mild to moderate numbers of neutrophils, macrophages, lymphocytes, plasma cells, and cell debris. Multifocally, myofiber degeneration and focal myonecrosis were randomly distributed in the deeper layers of the myocardium of both right and left ventricles and the septum. Fibrinous exudate associated with the myocardium contained multiple sidero-macrophages, bacterial colonies, neutrophils, occasional giant cells, and foci of basophilic non-birefringent material (mineral) (Figure 3).

Alpaca 2 had bilateral accumulations of light green caseous material adhered to the endocardium below the atrioventricular...
AV valves. Histologic lesions in the endocardium were similar to those in Case 1. Serous atrophy of fat was present at the base of the heart and in the femoral bone marrow.

Alpaca 3 had friable material adhered to the right ventricular endocardium with involvement of the tricuspid and pulmonary valves. The right ventricular wall was as thick as the left. Histologically, marked endocardial fibrosis and moderate neovascularization with multifocal hemorrhage, numerous sidero-macrophages, and small to moderate numbers of macrophages, plasma cells, and lymphocytes were observed in the right ventricular wall. In the myocardium there were small areas of focal myonecrosis.

Except for changes caused by flukes in Case 1, hepatic lesions were similar in all animals. The livers had severe multifocal congestion, hemorrhage, and loss of hepatocytes with focal areas of centrilobular coagulation necrosis of hepatocytes. Portal triads had mild to moderate fibrosis, bile duct proliferation, infiltration of lymphocytes, plasma cells and sidero-macrophages. Multifocally, canaliculi were dilated with bile. Scattered throughout the parenchyma were large foci of liquefactive necrosis bordered by a thick wall of connective tissue, karyorrhectic and pyknotic cellular debris.

Lungs contained mild to moderate accumulations of sidero-macrophages and low protein fluid in the alveolar spaces. Occasional pulmonary vessels contained organized fibrin thrombi in Case 1. Diffuse congestion and multifocal atelectasis and fibrosis were seen in the other animals. Mild membranous glomerulonephritis was observed in alpacas 2 and 3.

Gross and histological examination was consistent with heart failure caused by severe endocarditis with myonecrosis.

From the endocardial lesions, Pseudomonas sp., Streptococcus intermedius, Staphylococcus capitis, and Aspergillus sp. were isolated from Case 1, and Bacteroides sp. and Clostridium perfringens type A from Case 2. Bacteria were not isolated from the heart in Case 3. For Case 3, polymerase chain reaction (PCR) tests on fresh affected endocardium were positive for Mycoplasma sp. and negative for Bartonella sp. and Mycoplasma bovis. Fresh tissue from the test-positive alpaca was negative for mycoplasma on culture. However, a technique used to characterize the Grey Lung agent (GLA), a mycoplasma associated with Grey Lung disease in mice (1), was used to isolate and sequence the complete genome of the mycoplasma in tissues from alpaca 3. The GLA is a species of Mycoplasma located phylogenetically in the hominis group of mycoplasmas. Complete DNA sequencing placed Case 3’s Mycoplasma agent in the Mycoplasma hominis group, which showed a 92% convergence with the GLA agent. Immunohistochemistry tests on Cases 1, 2 and 3 performed on paraffin blocks containing formalin fixed tissue were negative for Mycoplasma spp.

Discussion

Cardiovascular disorders in South American camelids are infrequently described in the veterinary literature (2). When endocarditis does occur in alpacas, it may have a predilection for mural sites making diagnosis difficult due to the lack of a heart murmur (3). The right ventricle may be a more common site, as in Case 3, but biventricular involvement is also reported as in Cases 1 and 2 (3). In other species such as cattle, the endocarditis is usually valvular and mainly caused by Trueperella (Arcanobacterium) pyogenes or streptococci (4).

The agents associated with bacterial endocarditis in camelids include Escherichia coli, Corynebacterium pseudotuberculosis, Actinobacillus suis, Listeria monocytogenes, and C. perfringens (5). Of these agents, only C. perfringens type A was isolated from 1 animal. None of the animals had evidence of muscular changes as reported in the 1 case with C. perfringens (6) or consistent with other types of clostridial myonecrosis (7). The Clostridium and the Bacteroides isolated from Case 2 are interpreted as post-mortem invaders and/or contaminants.

The Aspergillus isolated from Case 1 was also likely due to postmortem invasion and/or contamination as no fungal elements were found histologically in affected tissue. The histology does support one or more of the bacteria isolated having been involved in the endocardial lesion. This animal had liver flukes but the finding is interpreted as incidental since the remaining 2 alpacas had no flukes at postmortem.
No bacteria were isolated from alpaca 3 but PCR of fresh affected endocardial tissue was positive for *Mycoplasma hominis* ssp. The PCR on paraffin-embedded formalin-fixed affected tissue from all 3 cases was negative, indicating that the mycoplasma was a contaminant or use of formalin-fixed paraffin embedded tissue hindered detection.

Mycoplasmas are obligate intracellular parasites that tend towards host species specificity and adaptation. Most researched mycoplasma in the American Camilidae is *Candidatus Mycoplasma haemolamae*, which is a highly endemic hemotropic mycoplasma that affects red blood cells of llamas and alpacas (8). Clinical cases are associated with anemia; however, most infections are subclinical. Development of a PCR assay has facilitated detection of this infection in llamas and alpacas in the United States and other countries (9).

*Mycoplasma hominis* is a cluster in a group of related but poorly characterized species of non-hemotropic mycoplasmas that are frequently commensal in the urogenital tract of sexually active women, and less often in men. *Mycoplasma hominis* has been related to infectious pathology such as pelvic inflammatory disease, postabortion and postpartum bacteremia, amnionitis, and surgical wound infections in humans (4,8,10–15). With a minimal set of 537 annotated genes, *M. hominis* appears to have a predilection for causing artificial heart valve associated endocarditis rather than native valvular endocarditis, suggesting that the agent is not cardiomyotropic in humans (13). Atypical growth characteristics in routine bacterial culture, negative repeat blood culture and an inability to demonstrate the organism using Gram staining can lead to a delayed diagnosis of *M. hominis* infections in people. The organism is thought to be often missed during investigation (10).

*Mycoplasma hominis* clusters have been successfully isolated from dogs (vagina), horses (nasophaynx and genital tract), geese (phallus), and birds of prey (trachea of saker falcon and griffon vulture), and most isolates have been considered commensals or non-pathogenic (16,17).

The gross and histological lesions in the hearts in this series of alpacas had morphological traits similar to those described by Firshman et al (3), and appear unusual compared to other species. Pathogenesis of endocarditis usually involves implantation of bacteria to a damaged endocardium which most commonly involves the valves. Such trauma exposes collagen and leads to platelet binding and activation of the extrinsic coagulation cascade with deposition of fibrin and the formation of sterile platelet-fibrin deposits (18). Non-infectious endocarditis has been discussed by Firshman et al (3) along with an association with fluke infection but remains speculative.

Further investigation of causative etiological agents remains open and may be multifactorial. A diagnosis of *M. hominis* endocarditis is rare and difficult (10). Identification of a variant of *M. hominis* in this report from one animal cannot be ruled out from contamination but its finding may be of interest to others trying to determine factors involved in mural endocarditis of alpacas.

*References*