CONTAGIOUS CAPRINE PLEUROPNEUMONIA OUTBREAK IN CAPTIVE WILD UNGULATES AT AL WABRA WILDLIFE PRESERVATION, STATE OF QATAR


Abstract: Contagious caprine pleuropneumonia (CCPP) caused by Mycoplasma capricolum subsp. capripneumoniae is a highly contagious and serious respiratory disease of domestic goats, characterized by coughing, severe respiratory distress, and high mortality rates. The lesions at necropsy are mainly a fibrinous pleuropneumonia with increased straw-colored pleural fluid. An outbreak of CCPP in wild goat (Capra aegagrus), Nubian ibex (Capra ibex nubiana), Laristan mouflon (Ovis orientalis laristanica), and gerenuk (Litocranius walleri) occurred at Al Wabra Wildlife Preservation in the State of Qatar. The disease was suspected because of the clinical symptoms and the necropsy findings and was confirmed by the isolation and identification of the causative organism. This new finding indicates that CCPP should be considered a potential threat to wildlife and the conservation of endangered ruminant species, especially in the Middle East, where it is enzootic because of its presence in chronic carriers. Susceptible imported animals should be quarantined and vaccinated. The preferred samples for diagnosis are the pleural fluid, which contains high numbers of Mycoplasma, and sections of hepatized lung, preferably at the interface of normal and diseased tissues. Samples must be shipped to diagnostic laboratories rapidly, and appropriate cool conditions must be maintained during shipping.

Key words: Contagious caprine pleuropneumonia, Mycoplasma capricolum subsp. capripneumoniae, wild goat, gerenuk, Nubian ibex, PCR.

INTRODUCTION

Contagious caprine pleuropneumonia (CCPP) is a highly contagious disease of domestic goats and small ruminants caused by Mycoplasma capricolum subsp. capripneumoniae (Mcp). It is characterized by fibrinous pleuropneumonia with increased straw-colored pleural fluid. Morbidity and mortality rates may reach 100% and 80%, respectively. Although it is considered primarily a disease of goats, there are reports of the isolation of Mcp from both healthy and sick sheep in Kenya that were in contact with diseased goats. Exposure to Mcp has been detected by complement fixation in African buffalo and camels. Although some animals had very high titers, no clinical signs were observed and mycoplasma cultures were negative. The possible occurrence of CCPP in wild herbivores in contact with livestock has been investigated in Kenya.

Isolation of mycoplasmas other than Mcp has been reported in other wild species such as the Rocky mountain big horn sheep (Ovis canadensis), mouflon (Ovis musimon), aoudad (Ammotragus lervia), and Vaal rehobok (Pelea capreolus). Experimential infection of a single Thompson’s gazelle (Gazella thomsoni) with Mycoplasma mycoides subsp. capri resulted in serologic positivity but no clinical signs. More recently, pulmonary mycoplasmosis caused by Mycoplasma bovis in farmed white-tailed deer (Odocoileus virginianus) has been reported. Transmission of Mycoplasma conjunctivae from sheep to chamois (Rupicapra rupicapra) was also documented in Switzerland.

In this report, we document for the first time an outbreak of CCPP in captive wild goat (Capra aegagrus), Nubian ibex (Capra ibex nubiana), Laristan mouflon (Ovis orientalis laristanica), and gerenuk (Litocranius waller) at the Al Wabra Wildlife Preservation (AWWP) in the State of Qatar.

CASE REPORT

In March and April 2004, an outbreak of a highly fatal respiratory disease characterized by rapid respiration and violent coughing spread among a group of wild goats, Nubian ibex, Laristan mouflon, and gerenuk. Within 6 wk, 20 wild goats (83% of the population) and 11 Nubian ibex (58%) died with respiratory distress. In May and June 2004, one old male gerenuk and four Laristan mouflon in an adjacent enclosure died with similar symptoms and necropsy findings. An additional 40 animals displayed mildly acute signs, but of this group, only 22 recovered after antibiotic treatment and vaccination. Four Nubian ibex kids that had direct con-
tact with affected animals showed no signs of illness.

The disease had peracute, acute, and chronic forms. In the peracute form, the affected animals isolated themselves from their penmates, were unwilling to eat or drink, had rapid respiration, appeared stiff when they moved, and died suddenly. In the acute form, the onset was rapid. The animals were lethargic and anorexic, sought shaded areas, and isolated themselves from penmates. The affected animals exhibited an abnormal posture, with head and neck extended and froth observed coming from the nostrils and mouth of some animals. Respiration was labored and appeared painful when the animals were forced to move. There was a percussive dullness over affected areas of the lungs. Moist rales and rough friction sounds could be heard over the ventral areas of the lungs. In the chronic form, some animals had nasal discharge, salivation, and coughing on exertion. A few animals were hyperthermic with rectal temperatures ranging from 40.0 to 41.2°C. Animals with the subacute and chronic forms had rectal temperatures within the normal range; but they appeared weak and coughed when excited.

The morbidity and mortality rates among the affected groups of wild goats were 100% and 82%, respectively, and in the Nubian ibex they were 83% and 58%, respectively. These findings are similar to those reported in vaal rhebok13 and in domestic goat (Capra hircus).12,20

All animals that died were necropsied. Gross pathologic lesions were mainly restricted to the lungs. The affected lungs showed partial or total hepatization, some with thickening of the interlobular septa. Serofibrinous pleuropneumonia and profuse pleural fluid were observed (Fig. 1). The pleural fluid was straw colored with fibrin flakes, sometimes filling the thoracic cavity and forming a gelatinous membrane covering the affected lungs and adhering to the costal thoracic wall. Similar to other reports, a thickened pericardium was sometimes seen.8,15,22 In the more advanced cases, there was a black discoloration of the lung tissues and sequestration of necrotic areas with some interlobular edema of the lungs.10 Histologic examination of the lungs showed a fibrinopurulent pleuropneumonia with diffuse, mild vascular and parietal alveolar congestion and diffuse fibrinous deposits in the alveolar septa.

At the beginning of the outbreak, the animals showing respiratory signs were treated unsuccessfully.
fully with different antibiotics, including chloramphenicol (Albrecht, Aulendorf, Germany) 10 mg/kg and tetracycline (Pfizer, Karlsruhe, Germany) 20 mg/kg i.m. However, the surviving pen-mates were treated with 20–40 mg/kg tylosin i.m. (Selectivet, Holzolling, Germany) and with 14 mg/kg dexametason i.m. (Essex Tierarznei, Munich, Germany) for five consecutive days. The use of anti-inflammatory drugs might increase the efficacy of the antibiotic treatments and might prevent animals from succumbing to shock.

Because CCPP was suspected, the surviving 40 wild goats, laristan moufflon, and Nubian ibex were vaccinated with a freeze-dried attenuated strain of M. mycoides subsp. capri strain BQT (Pulmovac®, Vital Veterinary Vaccines Production Co., Turkey; 0.2 ml s.c. in the back of the ear). This had no obvious effect on the course of the disease, and the 22 survivors were given a vaccine for contagious caprine pleuropneumonia containing Mecp antigen (Caprivax®, Kenya Veterinary Vaccine Production Institute, Nairobi, Kenya; 1 ml s.c.), after which no more animals died.

The clinical signs and gross lesions seen at necropsy suggested contagious caprine pleuropneumonia. Portions of affected lung tissue and aliquots of pleural fluid were placed in paired sterile containers (one of which contained a few drops of ampicillin), frozen, and sent for microbiologic examination at various laboratories. Most samples were negative for mycoplasma until samples sent to CIRAD; France (an OIE reference laboratory) produced growth on a mycoplasma medium enriched with sodium pyruvate. With the use of a polymerase chain reaction (PCR) assay and immunoblot technique with a specific monoclonal antibody, this culture was identified as Mecp. The isolate was further characterized by amplification and sequencing of a 2,400-bp-long DNA fragment found to be similar to the Mecp strain previously isolated in East Africa (GenBank AF 378154).

Immunohistochemistry of affected lung tissue detected antigen in macrophages and in neutrophils; no other bacteria were observed. Negative controls from goats reared in France did not show similar staining results.

**DISCUSSION**

*Mycoplasma capricolum* subsp. *capri pneumoniae* has been thought to produce disease only in domestic goats. On the basis of this report, it seems that Nubian ibex, wild goats, gerenuk, and laristan moufflon are also susceptible to disease caused by Mecp. It is not clear whether the infection at AWWP was introduced with three Nubian ibex imported in April 2003 or whether the disease was the result of an infection from domestic goats outside the conservation area. The imported ibex were penned adjacent to the other Nubian ibex, wild goat, and Laristan moufflon and faced the enclosure of the gerenuk. All three imported animals died during the outbreak. Once the disease was introduced, the continuing spread within the conservation area was probably from inhalation of infected aerosols from nasal discharges of resident wild ruminants. Because of the fastidious nature of Mecp, it was difficult to isolate the *Mycoplasma* responsible for CCPP. However, Mecp can be readily identified in pleural fluid samples by PCR on the basis of the 16S RNA gene, followed by specific identification of Mecp by restriction enzyme digestion. The samples of pleural fluid can be dried on filter paper and sent to reference laboratories without biological hazard.

The most important step for the control of CCPP is adequate vaccination, although serologic surveillance might be useful in detecting subclinical carriers. Some investigations have indicated that CCPP might become enzootic in Middle Eastern countries because of the presence of inapparent chronic carriers. This suggests that imported susceptible species should be vaccinated during quarantine. In AWWP, quarantined animals are vaccinated as soon as possible after arrival, given booster vaccines 4 wk later, then revaccinated annually.

This case illustrates the risks of importing ungulates into a new and unfamiliar environment without adequate isolation. *Mycoplasma* infections should be considered when animals are being screened for disease before being released into a collection. Unfortunately, *Mycoplasma* infections are difficult to detect because they do not induce long-lasting antibodies and because there are few specific serologic tests. It is also important to establish a buffer zone between collections of exotic ungulates and domestic animals. The use of PCR is a valuable and rapid diagnostic method for detecting mycoplasmosis in sick and carrier animals.

Further studies are required to assess the importance of CCPP in wildlife because this disease might be a threat to many endangered species that come in close contact with goats. It is doubtful that routine diagnostic laboratories are able to identify Mecp by isolation unless rapid and sensitive detection techniques are used.

**Acknowledgments:** This case is being reported with the approval of HE Sheikh Saud Bin Mohammed Al-Thani, the owner of Al Wabra Wildlife Preservation, without whose continuous support.
this investigation could not have been completed. We gratefully acknowledge the willing assistance of Al Wabra Wildlife Preservation staff, the help and advice of Dr. Tim Chayne, the work of Girard laboratory staff in France in isolating the Mcp, and Dr. Caroline Lacroux (Pathology Department of National Veterinary School of Toulouse France) and her colleagues for their effort of performing the immunohistochemistry and histopathology.

LITERATURE CITED

Received for publication 16 September 2005