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Short Communication

Atypical non-progressive pneumonia in goats

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ABSTRACT

An outbreak of severe respiratory disease in a goat herd was associated with *Mycoplasma ovipneumoniae*, *Mycoplasma arginini*, *Mannheimia haemolytica* and *Pasteurella multocida* with mortality rates exceeding 20% in kids. Post mortem features in affected kids included severe pleuropneumonia, lung consolidation, large quantities of pleural fluid and pericarditis. This is the first report of atypical proliferative pneumonia in goats in Portugal.

Mycoplasma infections in goats are reported frequently and usually involve the *Mycoplasma mycoides* cluster organisms, including *Mycoplasma capricolum* subsp. *capripneumoniae* and *Mycoplasma mycoides* subsp. *capri*, the causes of caprine pleuropneumonia (Gonçalves, 1982; Nicholas, 2002). The role of other mycoplasmas is less well understood. *Mycoplasma ovipneumoniae* has been reported in sheep but is often overlooked because of apparent greater involvement of pasteurellosis (Ayling and Nicholas, 2007). There are few reports incriminating *M. ovipneumoniae* as a cause of severe respiratory disease in goats. Respiratory disease associated with *M. ovipneumoniae* has been referred to as atypical pneumonia of sheep, mycoplasma pneumonia, non-progressive (atypical) pneumonia of sheep, proliferating exudative pneumonia, summer pneumonia, though the preferred scientific name is atypical non-progressive proliferative pneumonia (CABI, 2006). We report here an outbreak of a severe respiratory disease in kids from a goat herd associated with the isolation of *M. ovipneumoniae*, *M. arginini, Mannheimia haemolytica* and *Pasteurella multocida* in the Alentejo region of Portugal.

In the late spring of 2007, an outbreak of a severe respiratory disease was seen in kids from primipara goats of the Saanen breed in which 21/91 kids died and there was a morbidity rate of 34%. Before parturition, the goats were vaccinated against contagious agalactia because there was a history of mycoplasmosis in the herd. Caseous lymphadenitis (CLA) had also been seen in some animals. Subsequently the kids were allowed to suckle their mothers. Two weeks after birth the kids developed enteric disease caused by *Escherichia coli* which was treated with a macrolide antibiotic. Almost 1 month later the young kids presented with severe respiratory signs, which preceded death by dyspnoea.

Clinical observations throughout the outbreak showed difficult breathing with dyspnoea, followed by death in newborn kids and those up to 2 months of age. At necropsy the following pathological features were seen: severe congestion of the nasal sinus with catarrhal mucus, a bilateral pneumonia with pulmonary congestion of cranial lobes with progression to the caudal lobes, fibrinous pleurisy, adhesions to the chest, pericarditis with hydropericardium and abundant pleural fluid (Figs. 1 and 2). Cut sections of both affected lungs showed mild to severe hepatisation with red-grey areas of consolidation. Adults appeared unaffected throughout the outbreak.

Histological sections of the lung stained with haematoxylin and eosin (H&E) revealed a suppurative bronchopneumonia with alveoli and bronchioles filled with variable proportions of neutrophils, macrophages, serofibrinous exudation, degenerated leukocytes and necrotic debris, and multifocal presence of aggregates of bacteria. Fibrinous pleuritis and severe distension of the interlobular septa by fibrin and oedema was also observed (Fig. 3), and occasional peribronchiolar lymphocytic accumulation (Fig. 4).

Contagious caprine pleuropneumonia (CCPP) was initially suspected because of many similar characteristics observed at

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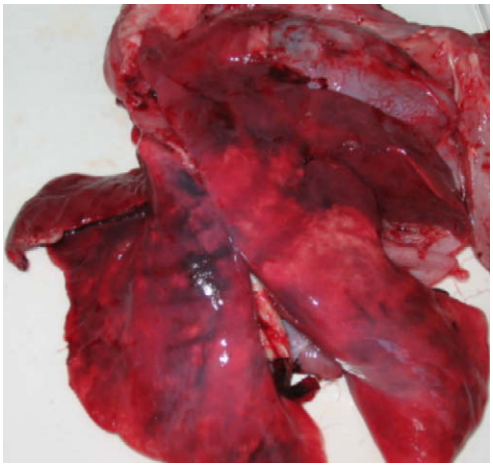

Fig. 1. Pneumonic lung and pericarditis from a kid.

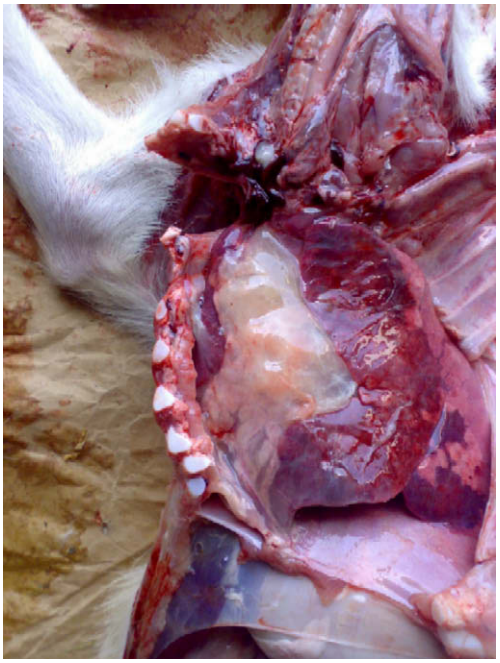

Fig. 2. Bilateral pneumopathy with fibrinous pleurisy and adhesion.

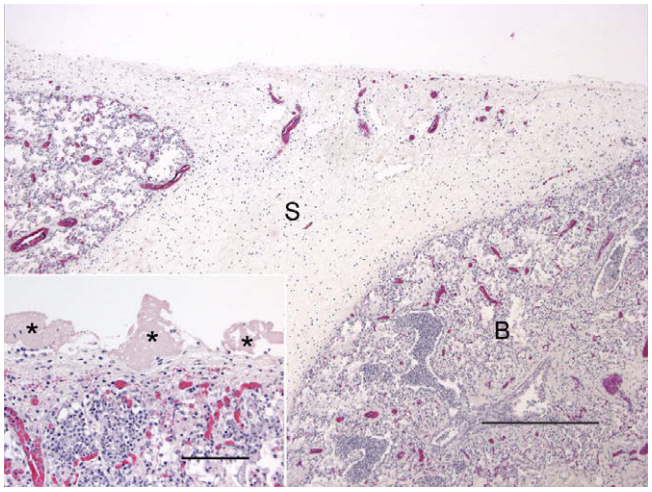

Fig. 3. Lung. Suppurative bronchopneumonia (B), fibrinous pleuritis and lobular septa expanded by fibrin and oedema (S). H&E. Bar: 500 μm. Insert: Detail of fibrin deposition on the pleural surface (*). H&E. Bar: 100 μm.

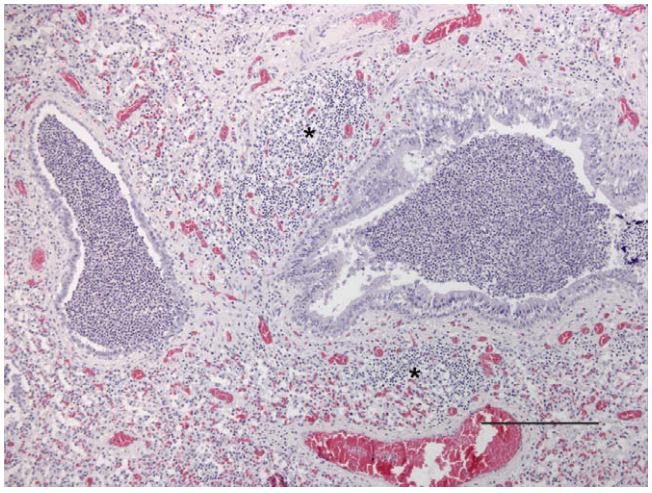

Fig. 4. Lung. Suppurative bronchiolitis with attenuation of the bronchial epithelium (left bronchiole) and mild lymphocytic peribronchiolar accumulation (*). H&E. Bar: 250 μm.

necropsy and reported above. Consequently, milk and serum from a live adult goat and respiratory tracts from two kids as well as pleural and joint fluids from one of these kids were submitted for isolation of *Mycoplasma*. Also, samples of lung, pleural fluid, spleen and brain from another kid were submitted for routine bacteriology. Samples of lung, mediastinal lymph nodes, pericardial, pleural and joint fluids, and milk were cultured in Eaton's broth medium and plated later on in Eaton's agar (Nicholas and Baker, 1998). DNA was extracted either directly from the samples, or after enrichment in culture media by incubation at 37 °C and 5% CO_2.

Colonies with a 'fried egg' appearance, typical for many *Mycoplasma* species, and centreless granular colonies were seen on solid medium after 4–6 days of incubation and were identified by denaturing gradient gel electrophoresis (DGGE) in mixed culture as *M. ovipneumoniae* and *M. arginini* (McAuliffe et al., 2005) (Fig. 5). The growth inhibition test using serum from the adult goat confirmed the presence of *M. ovipneumoniae*. *M. haemolytica* and *P. multocida* were also detected in the sample of lung submitted to the conventional bacteriological methods. Antibiotic treatment with enrofloxacin and florfenicol was administered for 5 days and clinical signs resolved within 10–14 days.

The use of *Mycoplasma*-specific primers with DGGE enabled the direct detection of mixed cultures of *M. ovipneumoniae* and *M. arginini* from clinical samples (McAuliffe et al., 2005). The isolation of these microorganisms from milk supports the view that the kids were infected directly by suckling from the adult females, which showed no clinical signs.

M. ovipneumoniae is the most commonly isolated mycoplasma from the respiratory tract of sheep often in association with *M. arginini* that may exacerbate disease signs (Ayling et al., 2004; Ayling and Nicholas, 2007). A high prevalence of *M. ovipneumoniae* in association with *M. haemolytica* has been reported in the lungs of lambs in Turkey (Haziroglu et al., 1994) and recently in Italy (Ettorre et al., 2007).

A very similar case to the present report was seen in the UK by Nicholas (2002), where imported French goats (also suffering from CLA) had been mixed with homebred goats. High levels of mortality and morbidity were seen with isolation of *M. ovipneumoniae*, *M. arginini* and *M. haemolytica*; an additional sign in that report

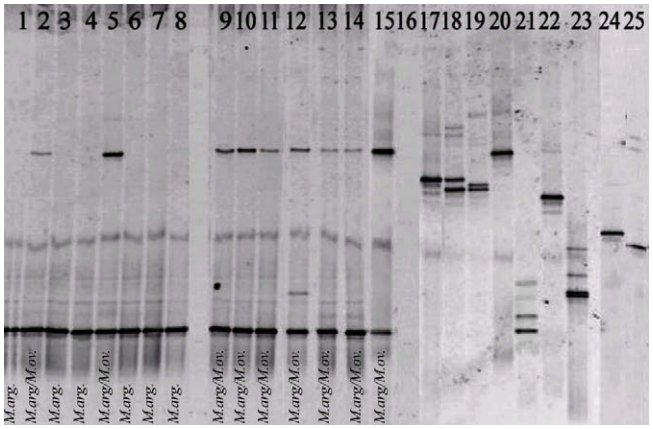

Fig. 5. DGGE of *M. arginini* and *M. ovipneumoniae* isolated from samples of G1 (kid 1), G2 (kid 2) and G3 (goat 3), directly from clinical material (lanes 1–8), after enrichment (lanes 9–15), and of several *Mycoplasma* species used as positive controls (lanes 17–26). Lanes 1 and 9, pericardial fluid G1; lanes 2 and 11, pleural fluid G1; lane 3, joint fluid G1; lane 4, lung G1; lanes 5 and 12, mediastinal lymph node G2; lanes 6 and 14, pericardial fluid G2; lanes 7 and 13, lung G2; lanes 8 and 15, milk G3; lane 10 mediastinal lymph node G1; lane 16, Negative control; lane 17, *M. mycoides* LC F30; lane 18, *M. capripneumoniae* F38; lane 19, *M. capripneumoniae* T9; lane 20, *M. ovipneumoniae* NCTC10151; lane 21, *M. arginini* NCTC10129; lane 22, *M. putrefaciens* NCTC10155; lane 23, *M. agalactiae* NCTC10123; lane 24, *M. bovis* NCTC10131; lane 25, *M. conjunctivae* NCTC10147.

was caprine arthritic encephalitis. *M. ovipneumoniae* facilitates lung colonisation by other organisms, such *M. haemolytica*, producing a more severe pathology (Ayling and Nicholas, 2007). Respiratory disease involving *M. ovipneumoniae* has also been reported in goats in Spain, Nigeria, Sudan (CABI, 2006) and Canada, where *M. ovipneumoniae* was implicated in a developing fibrinous pleuritis and pneumonia in an experimental infection but *M. arginini* did not appear to be a significant pathogen (Goltz et al., 1986).

Consideration should be given to the development of a vaccine for this serious but underreported disease, perhaps as a component of a pasteurellosis vaccine.

Conflict of interest statement

None of the authors of this paper has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

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