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Mycoplasma capricolum in an Outbreak of Polyarthritis and Pneumonia in Goats

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Bölske, G., H. Msami, N. E. Humlesjö, H. Ernø and L. Jönsson: *Mycoplasma capricolum* **in an outbreak of polyarthritis and pneumonia. Acta vet. scand. 1988, 29, 331–338.** – An outbreak of polyarthritis, pneumonia and mastitis was encountered in a herd of goats in northern Sweden. The most obvious clinical sign was lameness, progressing to recumbency. Necropsy revealed fibrinous and suppurative polyarthritis and in some kids interstitial pneumonia. Mycoplasmas were isolated from joints and lungs and also from the udder of a mastitic goat. The strains were identified as *Mycoplasma capricolum*, although extensive cross-reactions with antiserum against mycoplasma strain F38 was noted.

mycoplasmas; F38-like group; arthritis; caprine.

Introduction

Infection with *Mycoplasma capricolum* was first described in a dairy goat herd in California (*Cordy et al.* 1955). The disease was characterized by severe polyarthritis and showed a high morbidity and very high mortality in the kids. The new species was characterized and named *M. capricolum* in 1974, when *Tully et al.* examined a number of unidentified goat mycoplasmas from joints.

The organism has been reported from outbreaks of goat mastitis and arthritis in France (*Perreau & Breard* 1979, *Picavet et al.* 1983), arthritis in sheep in Zimbabwe (*Swanepoel et al.* 1977) and vulvovaginitis of sheep in England (*Jones et al.* 1983). It has also been isolated in Australia (*Littlejohns & Cottew* 1977), Egypt (*Al-Zeftawi* 1979), Spain (*Talavera Boto* 1980), and India (*Banerjee et al.* 1979).

Experimental studies have demonstrated *M. capricolum* to be highly pathogenic causing polyarthritis, mastitis, and pneumonia (*Cordy et al.* 1955, *Da Massa et al.* 1983, *Da Massa et al.* 1984).

M. capricolum is closely related to and may give serologic cross reactions with the F38-like group of mycoplasmas (*Christiansen & Ernø* 1982, *Ernø et al.* 1983). The F38-like group is believed to cause contagious caprine pleuropneumonia (CCPP) (*Mc Martin et al.* 1980). Despite the close relationship the clinical and pathological features of the disease associated with *M. capricolum* differs in many respects from CCPP caused by F38-like group (*Mc Martin et al.* 1980, *Mac Owan* 1984). The latter is a highly contagious disease which involves lung and pleura exclusively and without septicaemia.

In this report we describe a disease outbreak in a Swedish goat herd from which we isolated strains of *M. capricolum* showing extensive cross-reactions to mycoplasma strain F38.

Material and methods

Animals

The herd was composed of 38 dairy goats, 1 buck and 13 kids of Swedish landrace breed. Most of the kidding occurred in the middle of January. In previous November there were 2 cases of mastitis and 1 abortion. During the kidding and until the beginning of March the herd health was good. At that time the kids were by mistake fed mouldy hay for 1 day. Two of the kids reacted with tympany but recovered. A couple of days later one kid became paretic and was euthanized. During March, 9 kids and 3 does became ill. Of those 7 kids and 1 doe were euthanized in a moribund state or when they were unlikely to recover completely. The animals were sent for necropsy.

Pathology

Complete necropsies were performed on sacrificed goats. All tissues for histological examination were fixed in neutral buffered 10 % formalin. Sections were cut at 4 µm and stained with haematoxylin and eosin. Special stains used were van Kossa stain for calcium, Van Gieson, Gram and Weigerts stain for fibrin.

Microbiology

At necropsy, samples were collected aseptically from joints, lungs, udder, nasal cavity, spleen and lymph nodes for mycoplasma isolations. Mycoplasma isolations were performed essentially as described by *Friis* (1975) but 0.1 % ampicillin was used in the medium as a bacterial inhibitor and 20 % horse serum was used instead of 10 % horse serum and 10 % swine serum.

A 10 % suspension was made from the tissue by grinding with sterile quarts sand in broth in a mortar. From this suspension a plate was inoculated and 10-fold dilutions to 10^{-4} or 10^{-6} were made in broth. Broth cultures were incubated at 37°C and observed daily for indicator colour shift for 14 days before they were regarded as negative. If a colour shift occurred, subcultures were made to broth and to an agar plate. The plates were incubated at 37°C with 5 % CO_2 for 7 days. Mycoplasma isolates were identified by the growth inhibition test (GI) (*Black* 1973) and immunofluorescence on unfixed agar colonies (IF) (*Rosendal & Black* 1972). The biochemical tests were performed according to *Freundt et al.* (1979). Specimens submitted for bacteriological examination were cultured on 5 % horse blood-agar and on lactose agar. Some samples were also cultured for Chlamydia.

Results

Clinical signs

The first signs in the kids were swollen joints and lameness. In terminal stages the animals showed severe lameness, which rapidly progressed to recumbency. During most of the illness, which lasted about 10 days, the kids were remarkably alert and feeding well.

At the end of March 1 doe showed lameness in front legs, reduced appetite and mastitis. The general body condition got worse day by day and the animal became recumbent. Two other does also became lame but without any swollen joints. These 2 animals were treated with Spiramycin for 5 days and recovered.

Pathology

Gross pathology. The pathological alterations in the kids were mainly confined to the joints and lungs. There was generalized but variable swelling of the joints which were filled with large yellowish fibrinous masses often mixed with pus. Several joints showed local destruction of the joint cartilage and the joint capsule appeared to be thickened with proliferating synovial mem-

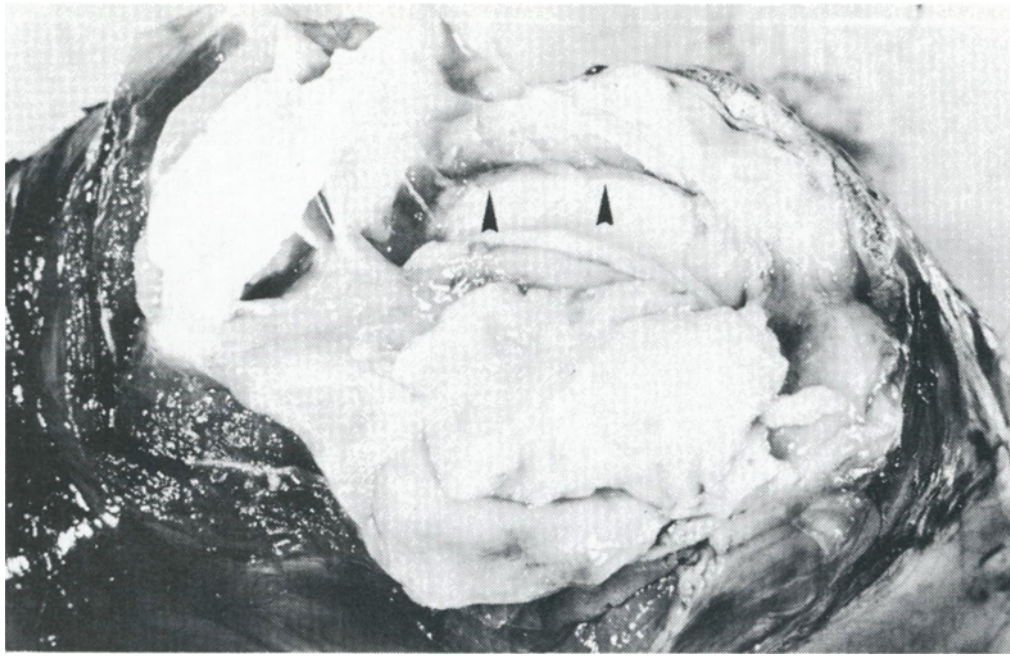

Figure 1. Stifle joint filled with fibrinous masses (goat kid 530). There is periarticular oedema and local destruction of joint cartilage (arrowheads).

brane (Fig. 1). Periarticular fibrous outgrowths were visible in some joints and 1 kid showed evidence of bursitis over the dorsal aspect of the carpal joint. The lungs of 3 kids showed acute pneumonia affecting the apical and cardiac lobes. These parts of the lungs were consolidated and dark red with a dry cut surface.

The doe showed fibrious arthritis in several joints, chronic fibrino-purulent mastitis and parasitic pneumonia.

Histopathological findings. The histopathological changes of the joints were those of acute to subacute fibrinous or fibrino-purulent arthritis. The joint capsule showed hyperemia and oedema. There was heavy infiltration of mononuclear cells especially lymphocytes and plasma cells but also macrophages (Fig. 2). Some vessels of the joint capsule showed thrombosis and fibrinopurulent exudate was either free in the joint space or attached to the synovial membrane. In some joints the exudate appeared stratified with the outermost layer showing hemorrhages. There was slight calcification in some of the exudates.

The lungs showed interstitial pneumonia and purulent bronchopneumonia. Interalveolar septa were broadened due to oedema and cellular infiltration mainly by mononuclear cells. The alveoli contained increased number of alveolar macrophages and there was marked peribronchial lymphoid hyperplasia (Fig. 3). Some alveoli and bronchioles were filled with polymorphonuclear cells, and desquamated epithelial cells. The lungs of the doe showed, in addition to interstitial pneumonia, a verminous

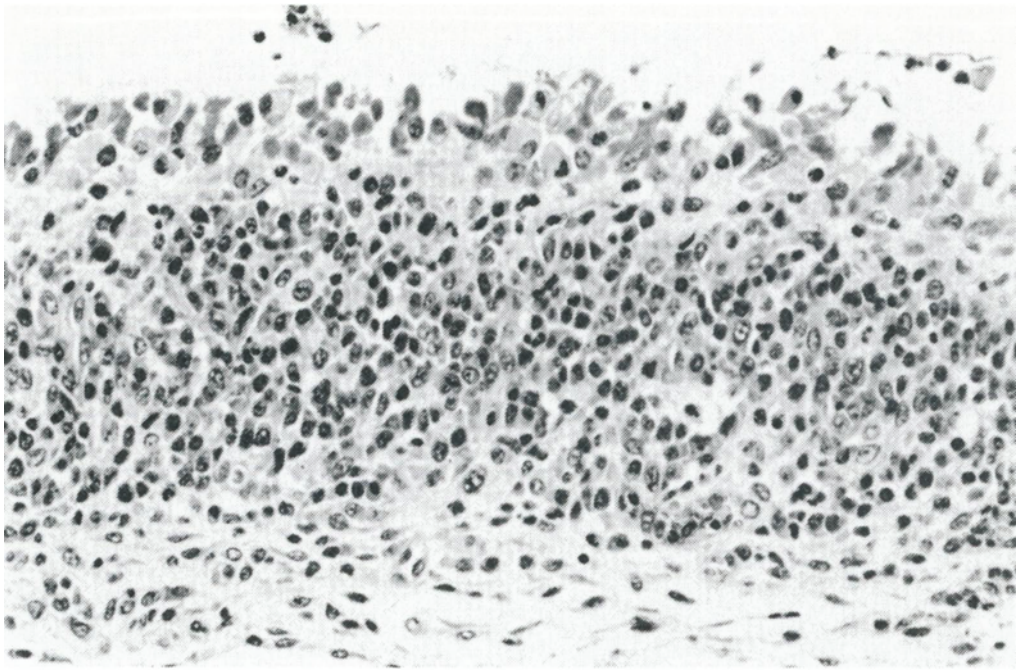

Figure 2. Synovial membrane from carpus of goat kid 685, showing proliferation of synovial lining cells and subsynovial infiltration by lymphocytes, plasma cells and macrophages. HE × 240.

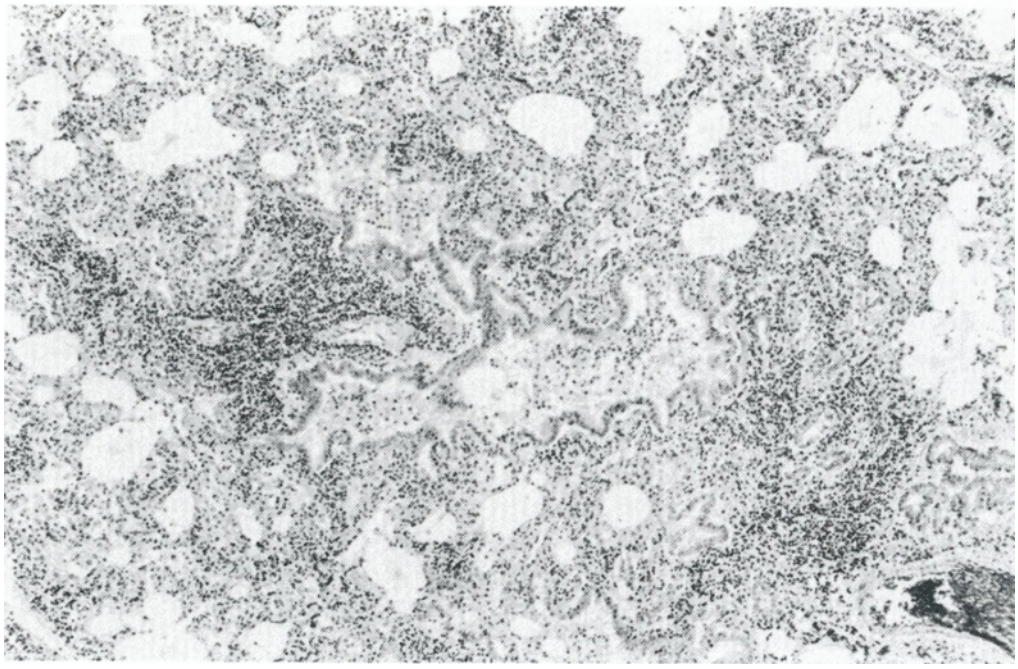

Figure 3. Interstitial pneumonia of goat kid 528 with pronounced focal proliferations of lymphocytes and several air spaces filled with neutrophils. HE × 60.

Table 1. Recovery of Mycoplasmas from the tissues.

	Tissues					
Animal no.	Joint	Lung	Spleen	Lymph node	Nasal cavity	Other
530 Kid	R. stifle M.C. 10^4 L. stifle M.C. $> 10^4$ Elbow M.C. $> 10^4$	M. ovipn $> 10^6$	M.C. 10^2	M.C. 10^2	M. ovipn. 10^5	
529 Kid	neg	neg	neg	M.C. 10	M. ovipn. 10^4	
528 Kid	R. stifle M.C. 10^2 L. stifle neg.	M.C. 10^2	M.C. 10	neg	M. ovipn. 10^5	
685 Kid	R. elbow M.C. $> 10^4$ R. carpal M.C. $> 10^4$ Left hock M.C. $> 10^4$					
492 Kid	R. stifle M.C. 10^4	M.C. $> 10^4$				
493 Kid	R. stifle M.C. 10^6					
494 Kid	L. stifle M.C. $> 10^6$	M.C. $> 10^4$				
434 Doe	Carpal M.C. $> 10^4$					Udder M.C. $> 10^6$ Brain: neg

Growth of Mycoplasma is given in number of organisms per gram.
neg = mycoplasma growth not found; M. ovipn. = *Mycoplasma ovipneumoniae.*
M.C. = *Mycoplasma capricolum.*

pneumonia with compensatory alveolar emphysema.

The mammary gland showed inflammatory reactions consisting of purulent exudate and necrosis. There was increased interalveolar connective tissue. The brain of most cases exhibited oedema and congestion without inflammatory reaction. One kid showed renal lesions characterized by focal interstitial nephritis.

Microbiology. The recovery of mycoplasmas from various tissues is presented in Table 1. Mycoplasmas were isolated from all 8 cases investigated. One strain from a lung and all from noses were identified as *Mycoplasma ovipneumoniae.* All the other strains were similar: a fast growing mycoplasma which produced acid in the medium. In IF they were positive to antiserum to *M. capricolum* and weakly positive to

M. mycoides subsp *mycoides* and subsp *capri.*

In GI the strains were positive with antiserum to *M. capricolum,* but not to *M. mycoides* subsp *mycoides* or subsp *capri.* Two strains, G5 and G6, were cloned 3 times and investigated further. They showed ability to ferment glucose and catabolize arginine. They were sensitive to digitonine and positive in the phosphatase test. They reduced tetrazolium both aerobically and anaerobically and were positive to the serum digestion test. In IF and GI tests, G5 and G6 were positive to antiserum against *M. capricolum* and strain F38. In IF they reacted weakly against *M. mycoides* subsp *mycoides* and subsp *capri,* but in GI they did not. Antisera against *M. conjunctivae, M. putrefaciens* and *M. ovipneumoniae* were negative in the IF and GI tests. Based on the biochemical results, especially arginine catabolism, the strains were classified as *M. capricolum* and not F38 group.

In the samples for bacteriological investigation from joints of kids 492, 493 and 494, pinpoint colonies were seen but could not be characterized by ordinary bacteriological methods. These were later shown to be *M. capricolum.* The lung from 492 gave also pinpoint colonies and the lung from kid 494 gave moderate numbers of E. coli and β-haemolytic streptococci. Samples from 685 and doe 434 were negative or yielded an insignificant mixed bacterial flora.

Material for Chlamydia isolations were collected from knee joints of kids 492, 493 and 494 and udder tissue from doe 434 and were inoculated and passed in eggs without detection of chlamydia.

Discussion

In this outbreak *M. capricolum* was consistently found in high numbers in joint lesions. There was no evidence of bacterial or Chlamydial involvement. Isolation of *M. capricolum* from spleen and lymph nodes confirmed the septicaemic nature of the disease. Of 3 pneumonic lungs that were cultured, *M. capricolum* was isolated from 2 and *M. ovipneumoniae* from 1. This may suggest *M. capricolum* as responsible also for pneumonia in accordance with the experimental studies (*Da Massa et al.* 1983, *Da Massa et al.* 1984). *M. ovipneumoniae,* which is widely distributed among apparently healthy goats, has also been recovered from pneumonic lesions in goats (*Ali* 1977, *Livingstone & Gauer* 1979). Experimental infection has caused pleuritis and pneumonia in a small portion of the animals (*Golz et al.* 1986). The possibility that *M. ovipneumoniae* contributed to the pneumonic lesions in this outbreak must therefore be considered.

The most consistent lesions in our study were polyarthritis and interstitial pneumonia in the kids. Occurrence of polyarthritis is in accordance with other reports (*Cordy et al.* 1955, *Littlejohns & Cottew* 1977, *Swanepoel et al.* 1977, *Perreau & Breard* 1979) but pneumonia has seldom been reported in spontaneous cases. In the investigation of *Banerjee et al.* (1978), *M. capricolum* was isolated from one pneumonic lung.

In the differential diagnosis, one should consider other infectious agents that are known to cause arthritis in goats, e.g. caprine arthritis-encephalitis virus (CAE) and chlamydia. Necropsy specimen from goats with CAE exhibit chronic hyperplastic synovitis, with subsynovial mononuclear cell infiltrates of lymphocytes, macrophages, and plasma cells. The changes are often combined with cellular necrosis mineralization and necrobiosis of collagen in the villi (*Crawford & Adams* 1981). The mycoplasma arthritis in the present outbreak was different with severe exudation and showed an acute fibrinous and suppurative polyarthritis. Chlamy-

dial polyarthritis could have similarities with mycoplasma infection. Therefore material for chlamydia isolation was collected but no microorganisms were detected.

All the kids investigated in this outbreak showed pneumonic lesions at autopsy although clinically there were no obvious signs of respiratory disease and some of the lesions were only seen microscopically. In the experiments of *Da Massa et al.* (1983) pneumonic lesions were consistently present in the young kids that were fed *M. capricolum* in normal goat milk. These authors attributed pneumonic lesions to the fact that oral administration of organism to young animals also exposes the respiratory passages through the formation of an aerosol during the suckling process. In this outbreak chronic active mastitis with high numbers of mycoplasmas in the milk was found in 1 doe. This case was diagnosed about 3 weeks after the first cases in the kids. Due to its chronic character it is possible that it persisted without detection for some time. As the kids were fed unpasteurized pooled colostrum from the does during the first weeks this may have been the way of transmission in the herd. The feeding of mouldy hay which led to a digestive disturbance in at least 2 kids may have been a predisposing factor for the disease outbreak.

The close relationship between *M. capricolum* and the F38-like group may lead to diagnostic problems. In this case the commonly used serologic identification methods failed to distinguish between them. The biochemical features (arginine positive, phosphatase positive, aerobic tetrazolium reduction) however indicated that they belonged to *M. capricolum* (*Ernø* 1983). The clinical and pathological findings in this outbreak were also consistent with earlier reports for *M. capricolum* disease and did not resemble those of CCPP.

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Sammanfattning

Mycoplasma capricolum – ett utbrott av polyarthrit och pneumoni hos getter.

Ett utbrott av polyarthrit, pneumoni och mastit ägde rum i en getbesättning i norra Sverige. Dominerande symtom var hälta. I senare stadier kunde djuren ej resa sig. Vid obduktion sågs fibrinös och suppurativ polyarthrit och i en del killingar interstitiell pneumoni. Mykoplasmer isolerades från leder och lungor och från juvret på en get med mastit. Stammarna identifierades som *Mycoplasma capricolum.* Kraftiga korsreaktioner med antiserum mot mykoplasma stam F38 förekom.

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