Mycoplasma bovis-associated pneumonia and arthritis complicated with pyogranulomatous tenosynovitis in calves

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- Infections with Mycoplasma bovis are refractory to treatment with antibiotics, and may cause pneumonia and arthritis in calves, polyarthritis in feedlot cattle, and polyarthritis with meningitis in suckling calves.
- Using monoclonal antibodies, avidin-biotin enhanced immunoperoxidase staining can detect M. bovis in tissue sections of tendon sheaths and joint capsules from affected cattle.
- In calves with pneumonia and arthritis, tendon sheaths and synovial bursae should be examined, in addition to joint capsules, for possible lesions.

A herd of 110 Charolais calves that had been purchased in the fall from a sale barn in Kentucky developed respiratory distress, pyrexia (40.5 to 41.5 C), and anorexia 2 weeks after arriving at a farm in eastern Iowa. The typical weight of each calf was 180 kg. In all, 30 calves (27.3% of the herd) were affected; however, auscultation of the thorax did not indicate fluid accumulation in the lungs, and there was not excessive nasal discharge. A few of the affected calves were lame, with swollen joints and concurrent respiratory distress.

Affected calves were treated by the attending veterinarian once with spectinomycin (15 mg/kg of body weight, IV) and with benzathine penicillin G and procaine penicillin G (15,000 U/kg, SC). This was followed by a second treatment with tilmicosin (10 mg/kg, SC) and sulfadimethoxine (83 mg/kg, PO). There was not any improvement observed by the owner after administration of these treatments.

Within 6 weeks after onset of respiratory tract disease, 8 of the calves died with severe bronchopneumonia and a few calves were culled. Because of the calves' poor response to treatment, 1 calf was examined after death, which revealed severe bronchopneumonia, with abscesses affecting the anteroventral lobes of both lungs. Liver and spleen appeared normal. Swollen limb joints had severely thickened joint capsules and synovial fluid was dark and extremely viscous.

Specimens of fresh and formalin-fixed lung, liver, spleen, and the shoulder and elbow joints from 1 calf were submitted to the veterinary diagnostic laboratory. Using aerobic and anaerobic bacteriologic culture techniques, Pasteurella multocida was isolated from lung specimens. Mycoplasma bovis was isolated from the lung and joint capsule specimens, using special culture conditions. The M. bovis isolate had minimal inhibitory concentration values of 20 μg of tilmicosin/ml (resistant), 10 μg of spectinomycin/ml (moderate resistance), 2.5 μg of tylosin/ml (sensitive), 2.5 μg of lincomycin/ml (sensitive), and 0.6 μg of tetracycline/ml (sensitive), indicating that the previous administration of spectinomycin and tilmicosin had not been of benefit in the affected Charolais calves. Using monoclonal antibody-based immunohistochemistry, M. bovis was confirmed from the lung and joint capsule. Mycoplasma antigen was specifically localized in microabscesses in both tissues, and on bronchial epithelium in less severely affected lung parenchyma.

Results of viral isolation of bovine viral diarrhea (BVD) and infectious bovine rhinotracheitis (IBR) viruses from lung specimens were negative. Moreover, direct fluorescent antibody examination failed to detect BVD, IBR, bovine respiratory syncytial, or parainfluenza 3 viral antigens in lung specimens.

On the basis of these laboratory findings, it was apparent that M. bovis was the major pathogen, and surviving calves that were clinically affected were treated with tylosin (5 mg/kg, IV, q 24 h, for 3 days). Tylosin was added to the ration at 1 g/head/day for 30 days as the clinical condition resolved.

A second herd of affected calves involved 25 Angus calves raised on a farm in northeastern Iowa. Calves were weaned in the fall, and had cross-fence contact in early winter with a group of 10 crossbred beef calves purchased from a local auction barn. Exact origin of the crossbred calves was unknown; they typically weighed 120 kg and developed a severe respiratory tract disease 7 to 10 days after arriving on the farm. Crossbred calves had been treated unsuccessfully by the farmer with a variety of antibiotics, including oxytetracycline, tilmicosin, and procaine penicillin G. Six of the crossbred calves died from apparent respiratory disease, and the remaining 4 were culled.

Approximately 2 weeks after coming in contact with the crossbred calves, the Angus calves, typically weighing 180 kg, developed signs of respiratory tract disease. Calves also had swollen joints within 5 days of developing clinical signs. Affected calves became anorectic and were reluctant to move. The owner treated the affected Angus calves with the same antibiotics used to treat the crossbred calves, with some favorable responses; 3 calves, however, remained severely affected.

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The 3 severely affected calves were treated by the attending veterinarian with erythromycin (8.8 mg/kg, IV), dexamethasone (44 μg/kg, IV), and phenylbutazone (4.4 mg/kg, IV). Initial treatment was followed by erythromycin (8.8 mg/kg, IM, q 24 h) for 3 days; however, there was no any response to treatment.

One of the severely affected Angus calves (calf 1) died a few days after treatment and, on examination, had bronchopneumonia with abscesses of the lung and extensive pleural adhesions. The entire lung and a forelimb from the calf were submitted to the veterinary diagnostic laboratory for further postmortem examination, which revealed a severe anterolateral bronchopneumonia with abscesses involving approximately 70% of the lung. Abscesses were 0.5 to 2 cm in diameter and contained caseous exudate. Joint capsules of the elbow and shoulder joints were severely thickened and contained caseous exudate (Fig 1). Histologic examinations of sections of lung revealed pulmonary abscesses with thick fibrous connective tissue capsules and central coagulative necrotic debris surrounded by large number of neutrophils and activated macrophages. There was severe fibrinopurulent and histiocytic synovitis, with multiple coagulative necrotic loci affecting the synovial membranes of the joints.

Pasteurella haemolytica and P multocida were isolated from the lung specimens. Bacteria were not isolated from the joint lesions. Acid-fast staining for mycobacterium organisms was negative. Direct fluorescent antibody examination failed to detect BVD, bovine respiratory syncytial, or IBR viral antigens in lung specimens. Mycoplasma bovis was isolated from specimens of abscessed lung and joint capsule exudates. Moreover, coagulative necrotic loci in lung and joint capsules stained intensely for M bovis antigens, using immunohistochemistry.

Approximately 1 month later, 6 of the remaining 23 Angus calves had a persistent cough and 5 had joint swellings affecting carpal, elbow, shoulder, hock, and stifle joints. On the basis of signs of respiratory tract disease (with or without swollen joints), specific morbidity at this time was 32% (8/25) in the herd of Angus calves. From 2 lame calves, synovial fluid was aspirated aseptically from radiocarpal and tibiotalar joints and was clear to slightly turbid. Synovial fluid samples, as well as samples of frozen lung, forelimb, and hind limb that had been obtained from an affected calf euthanized 4 days earlier (calf 2), were submitted to the veterinary diagnostic laboratory for further examination. Using synovial fluid, bacteriologic culture results for aerobic/anerobic bacteria and mycoplasmas were negative, whereas frozen lung samples yielded overgrowth with environmental bacteria. Approximately 90% of the left lung and 60% of the right lung from the euthanized Angus calf had multiple abscesses. Radiocarpal, elbow, and tibiotalar joints were swollen. Tendon sheaths of extensor muscles (in particular, extensor carpi radialis and common digital extensor muscles) and the subtendinous bursa of the extensor carpi radialis muscle were carefully dissected and were found to be markedly distended. On extrusion, the cut tendon sheaths contained caseous exudate, and pyogranulomas were observed lining the sheaths (Fig 2). Similar lesions were found at the elbow joint. The prescapular lymph node was large and, on cut section, had minute subcapsular pyogranulomas. Mycoplasma bovis was isolated from the lung abscess and from exudates in extensor tendon sheaths, extensor bursa, and joint capsule. Mycoplasma bovis also was detected in these tissues by use of immunohistochemistry (Fig 3). Mycoplasma bovis was not detected, however, in the prescapular lymph node. Results of histologic examination of the abscessed lung and joint tissues were as described for calf 1.

Mycoplasma bovis is an etiologic agent of pneumonia and arthritis in calves that are refractory to antibiotic treatment, of polyarthritis in feedlot cattle characterized by joint swellings and distention of tendon sheaths, and of polyarthritis with meningitis in suckling calves. In neonatal calves, intratrancreal inoculation of M bovis in calves produces massive fibrinopurulent synovitis and tenosynovitis, and intratracheal inoculation of gnotobiotic calves results in pneumonia and severe lameness. There is, therefore,
a strong etiologic role for *M. bovis* in calf pneumonia-arthritis syndrome. To the best of our knowledge, this is the first report of naturally developing pulmonary abscesses, concurrent with arthritis and tendon sheath abscesses, in calves in the United States.

Involvement of *M. bovis* was confirmed in the earlier studies on the basis of serologic evaluation or isolation of a mycoplasma and identification of its colonies by use of immunofluorescence testing or electron microscopy. With the use of monoclonal antibodies, avidin-biotin enhanced immunoperoxidase staining of lung tissue successfully detects *M. bovis* in pneumatic calf lungs. The test is highly reliable in detecting abscess-associated and nonabscess-associated strains of *M. bovis* in lung tissues. In the calves reported here, the test was successfully adapted to detect *M. bovis* in joint tissues. Tendon sheaths, bursae, and joint capsules were fixed in neutral-buffered 10% formalin for not more than 2 to 4 hours and then stored in 70% ethyl alcohol for further processing.

Although lung lesions in the 2 outbreaks reported here were indicative of infection with a chronic pyogenic bacteria such as *Actinomyces pyogenes*, the only bacteria cultured from lungs (apart from *M. bovis*) were *P. multocida* and *P. haemolytica*, which are involved in acute pyogenic lesions. Failure to isolate additional pathogenic bacteria could be related to previous antibiotic treatment of affected calves. However, using immunohistochemistry, the mycoplasma was found specifically concentrated in granulomatous lesions in the lungs, tendon sheaths and bursa, and joint capsules. Specific localization of antigen, therefore, provided evidence in the calves of this report that *M. bovis* was involved in production of abscesses.

Lung abscesses associated with *M. bovis* were reported in a study of calf lung specimens obtained from the midwestern United States. Findings in that study indicated that abscess formation by *M. bovis* might be strain related because abscesses were not found in every lung specimen in which *M. bovis* was detected. In contrast, abscess-associated strains of *M. bovis* were involved in the 2 outbreaks of disease reported here. By comparison, similar strain variation exists among isolates of *Mycoplasma synoviae*, the causative agent of infectious synovitis in chickens and turkeys. Interestingly, exudative synovitis, tenosynovitis, and bursitis also have been reported with *M. synoviae* infection. Because immunohistochemistry is now available as a diagnostic tool, it should be useful in further investigation of strain-related properties of *M. bovis*.

On the basis of the findings in the calves reported here, it is suggested that tendon sheaths and bursae be routinely examined, in addition to joint capsules, for possible lesions in calves with fatal pneumonia and arthritis. It is concluded that *M. bovis* is a serious pathogen of calf pneumonia-arthritis syndrome, and that clinical manifestation could be aggravated in some calves by abscess formation in the lungs, and in tendon sheaths, bursae, and joint capsules.

References


