PEER REVIEWED

Mycoplasma Infection in Cattle. II. Mastitis and Other Diseases

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Abstract

Mycoplasma spp. have been associated with many disease entities in cattle including mastitis, keratoconjunctivitis, otitis media, decubital abscesses, meningitis, reproductive problems and the pneumonia-arthritis symdrome. Since there are no pathognomonic signs to aid in making a clinical diagnosis, diagnosis of Mycoplasma infections presents a challenge to practitioners. Culture with identification of the organism and other ancillary tests are required for definitive diagnosis. Failure to respond to treatment is common. Isolation/segregation and culling infected or carrier animals are the best recommendations for control and prevention of Mycoplasma infections in a herd. In a companion paper, the pneumonia-arthritis syndrome was discussed. This paper reviews other disease entities caused by *Mycoplasma* spp.

Résumé

Le syndrome de pneumonie-arthrite relié aux mycoplasmes est présenté en détail dans l'article « Mycoplasma Infections in Cattle I ». Les mycoplasmes ont été associés avec plusieurs types de maladies chez le bétail incluant les mammites, la kératoconjonctivite, l'otite moyenne, les abcès du décubitus, la méningite et les problèmes de reproduction. Comme il n'existe pas de signes pathognomoniques pour aider à faire le diagnostic clinique, le diagnostic des infections à mycoplasmes représente un défi pour les praticiens. Une culture identifiant l'organisme et d'autres tests secondaires sont requis pour un diagnostic final. L'absence de réponse au traitement est fréquemment rencontrée. L'isolement et l'élimination des individus porteurs ou infectés sont les deux meilleures recommandations à faire pour le contrôle et la prévention des infections à mycoplasmes dans un troupeau.

Introduction

A companion paper reviewed and discussed the Mycoplasma pneumonia-arthritis syndrome. This paper will review and discuss other clinical entities associated with *Mycoplasma* spp. infections. Mastitis is a significant problem in the dairy industry. When the somatic cell count (SCC) of milk exceeds an established level, usually secondary to a mastitis outbreak on a farm, the processing plant does not pay the producer the maximum premium for the milk. Additionally, cows suffering from mastitis will have decreased milk production, hence, less product for the producer to sell. Decreased premiums and quantity of milk sold means less revenue for the producer.

Mycoplasma mastitis was first reported in Sweden in 1955,when pleomorphic organisms were isolated from bovine milk. These organisms were considered variants of bacteria.[26] *M. bovis*, *M. bovigenitalium*, *M. alkalescens*, *M. canadense*, *M. capricolum*, *M. californicum*, *M. bovirhinis*, *M. arginini*, *M.* sp group 7, F-38, *M. dispar*, *Acholeplasma laidlawii*, and *Ureaplasma urealyticum* have all been associated with cases of naturally occurring or experimentally induced bovine mastitis.[10,11,22,25,29] Of these species, *M. bovis* is considered the most pathogenic.[21,22,30] Treatment and control programs for Mycoplasma mastitis are a major challenge for producers and veterinarians.

The first report of a severe outbreak of mastitis caused by a *Mycoplasma* species in the United States occurred in Connecticut in 1961.[17] Since then, the disease has been reported nationwide, but primarily affecting intensified dairy areas.[8,20,25,30,40] In a paper summarizing *M. bovis* mastitis, the incidence of the disease in US herds ranged from 6 to 77%, and the average cull rate was 33%.[8]

In a recent California epidemiological study, *M. bovis* was the most common *Mycoplasma* species iso-

lated from milk samples. The samples were collected monthly from 260 dairies over a six-year period, cultured for *Mycoplasma* spp., and then speciated. The prevalence of positive samples for all *Mycoplasma* spp. isolated varied from 1.8 to 5.8%, and the prevalence for known mastitis-producing species ranged from 1.2 to 3.1% during the study period. Three species - *M. bovis, M. californicum* and *M. bovigenitalium* were consistently isolated.[29]

In an Ontario outbreak of bovine mastitis caused by *M. bovis* (formerly *agalactiae* subspecies *bovis*), herd incidence ranged from 15 to 40%, with the exception of one closed herd which reported a high incidence of 61%.[35]

Besides the more commonly recognized pneumonia-arthritis and mastitis syndromes caused by *Mycoplasma* spp. infections, these organisms cause severe keratoconjunctivitis, otitis media, decubital abscesses, meningitis and infertility problems.[2,14,27,28,40]

The Organism and Mastitis

Mycoplasma spp. are unique organisms belonging to the family *Mycoplasmataceae* and the genus *Mycoplasma*. Although *Ureaplasma* spp. are members of the same family, *Ureaplasma* spp. produce a urease enzyme while *Mycoplasma* spp. do not. *Acholeplasma* spp. are in yet a separate family, *Acholeplasmataceae*, and do not require sterol for growth.[16,31] The *Mycoplasma* spp. are relatively host-specific; they can infect other animals, but primarily produce disease in a particular host. Those that infect cattle are classified into groups 1 to 8. *Acholeplasma laidlawii*, formerly known as *Mycoplasma laidlawii*, is in group 6.[31]

These microorganisms have unique features and characteristics, including a small genome and lack of a characteristic cell wall, compared with commonly encountered pathogenic bacteria of cattle. Instead of a typical cell wall, they possess a limiting membrane. Special culture media, growth substances and conditions are necessary to isolate these microorganisms. Culture of *Mycoplasma* spp. requires longer incubation times than other bacterial pathogens. In a laboratory, typical colony growth on agar is described as having a "fried egg" appearance. Laboratories must use serological tests to perform species differentiation on the isolated colonies. Species identification determines the organism's potential significance in the disease process. Because of the special conditions involved, the practitioner must wait longer for results of *Mycoplasma* spp. cultures than for routine bacterial cultures.

Amies or Modified Stuarts transport media culture swabs are recommended for submission of antemortem samples. Aseptic collection of postmortem tissue specimens is necessary to minimize contamination. For best results, specimens should be refrigerated and submitted with ice packs to the laboratory as soon as possible after sample collection.[21,22] Since most diagnostic laboratories do not routinely culture for *Mycoplasma* spp., practitioners must specifically request these tests.

Clinical Signs of Mycoplasma Mastitis

An accurate, complete history may suggest a *Mycoplasma* spp. mastitis. Frequently, infected animals or replacements from unknown mastitis-status herds have recently been added to a client's herd that lacks a biosecurity program.[8,21,30]

A variety of clinical signs have been described in herds that have experienced Mycoplasma mastitis outbreaks. Cases can be mild, with normal-appearing milk and decreased production, with or without a high cell count.[7,8,21,30] Observant producers have noticed some cows early in the stages of infection may be "slow milkers."[7,8,20] The infection usually begins in one quarter, and then spreads to other quarters.[7,8,10,17,21,22,24,25,30] The udder and teats can be swollen and firm, but usually are neither warm nor painful when touched. In more severe cases, the milk is brown to tan in color, with watery consistency and flakes. Fibrinous plugs may also be observed. These secretions may last several weeks.[7,8,20,21,22,24,25]

Mycoplasma mastitis does not respond to routine antimicrobial therapy and can spread rapidly in a herd.[10,18,20,21,22,24,25,30] Clinical signs are generally localized and restricted to the udder, although arthritis has been reported in some herds.[7,20] As the disease progresses, herd milk production drops dramatically.[21,25,30] An affected cow may not return to normal or near-normal production until the next lactation.[20,21,22,25] Cows in all stages of lactation, as well as dry cows, may develop mastitis.[2,4,7,10,21,22,25,35]

Pathogenesis and Transmission

The disease is transmitted primarily through fomites. Improperly sanitized milking machines, contaminated teat dips, and contaminated human hands are believed to play an important role in spreading the organism to different quarters of an infected cow and to other cows.[21,30] Like the pneumonia-arthritis syndrome, *Mycoplasma* spp. can gain entry to the systemic circulation.

Besides milk, the organism has been isolated from the nose, vagina, synovial fluid, respiratory tract secretions, ocular fluid, urine, feces, skin, middle ear and uterus.[18,27-29,35,40] In infected herds and carrier animals, aerosol droplet transmission is believed to play a role in perpetuation of infections. The organism also has been isolated from barn air.[18] After resolution of clinical signs, cows can shed the organism intermittently and be a source of continual exposure to other animals.[7,8,20,21,22,23,39]

Researchers in Ontario isolated *M. bovis* (formerly *agalactiae* subspecies *bovis*) 8 to 12 months after initial infection, and found the severity of disease corresponded to the stage of lactation. Milk production of cows in early lactation (less than 8 weeks) gradually increased to 70 to 85% of expected production by the end of lactation. In contrast, cows infected after 12 weeks of lactation frequently dried up.[35]

Research comparing susceptible quarters to resistant quarters found previously infected quarters to be resistant to intramammary challenge with *M. bovis* for six months. Quarters from the same cows not previously infected were susceptible to infection. Cows challenged more than one year after previous infection were also susceptible to new infection.[3]

The same researchers investigated systemic and local immune responses to *M. bovis* in dairy cows. Immunoglobulin (Ig) A and IgG levels were greater in milk whey from cows resistant or partially resistant to challenge than from susceptible quarters. They concluded that "locally secreted antibodies, IgA and IgG, are probably critical to the resolution of infection, resistance to re-infection, and reduction of severity in chronically infected quarters."[4] Other investigators have examined cell-mediated immune (CMI) responses in the mammary gland. While quarters from vaccinated cows exhibited antibody responses in serum and milk whey, mammary gland lymphocytes were unresponsive to mitogen and antigen stimulation.[6] Hence, the exact immune mechanism involved in Mycoplasma mastitis is unclear.

Diagnosis

History, clinical signs and possibly postmortem findings aid in the clinical diagnosis of Mycoplasma mastitis. Isolation of *Mycoplasma* spp. and species identification are still confirmatory for a diagnosis of Mycoplasma mastitis.[7,20,21,25,30] Milk samples collected aseptically from affected cows should be refrigerated or frozen, and delivered to the laboratory as soon as possible.[21,22] Laboratory culture requires special media and incubating conditions.[17,20,21,22,30]

At necropsy, abscesses in glandular tissue of the udder may be observed. The abscessation is similar to that observed with Mycoplasma pneumonias. A positive culture from the nasal passages of cattle is generally not helpful, since the organism can be found in normal animals.[5,15,18] Because of the unique features and characteristics of the organism, routine aerobic culture will not detect it. Practitioners may need to specifically request Mycoplasma culture from some diagnostic laboratories.

Other tests supporting a diagnosis of Mycoplasma include histopathological examination, cytology and immunohistochemistry (IHC) testing. Specific antibodies against *M. bovis* identify the antigen in affected tissue.[1,34] Immunohistochemistry techniques may be useful in cases treated with antimicrobials and negative on culture, or if cultures are overgrown by bacteria. Polymerase chain reaction (PCR) has received recent attention as another possible diagnostic tool for Mycoplasma, but most labs do not offer this test.

Research has shown that recent infection has produced an increase in serum indirect hemagglutinating activity (IHA) titers. However, these titers were not indicative of quarter immunity.[3] Serology may be helpful in identifying an infected herd, but is generally not very useful in identifying an individual infected animal.[7] If milk cultures are negative and tissue specimens are unavailable, a positive serologic test could support the diagnosis. However, serologic tests are usually performed only in research laboratories and can be expensive.[38]

Treatment and Control

Mycoplasma infections generally respond poorly to treatment.[8,18,20,21,22,25,30,33,35] In earlier studies, *in vitro* sensitivity tests indicated that *M. bovis* was susceptible to tetracyclines. However, treatment outcomes were unsatisfactory. Spontaneous recovery is reported, which makes evaluation of treatment response difficult. Recovered cows shed Mycoplasma organisms in normal-appearing milk, serving as a reservoir for infection for other cows.[22]

Because of the poor response to treatment, some suggest that infected animals should not be treated.[30] Sound biosecurity practices offer the best solution for prevention and control.[9,20,24,30,36,41] Herds that have experienced Mycoplasma mastitis apparently develop a degree of resistance over time. Infections are usually mild or inapparent.[25]

Dairy practitioners should encourage clients to obtain a herd mastitis history from herds where animals are purchased. A bulk-tank culture from the herd of origin is helpful, but not foolproof, to identify potential Mycoplasma problems. False-negative bulk-tank cultures are common in large herds with a low prevalence due to potential dilution effects in the bulk tank.[30] Additionally, because cows with latent or subclinical infections may not be shedding the organism at the time of sampling, multiple samples for culture may be necessary.[7,20,21,23,25,39] If the producer does not add mastitic milk to the bulk-tank, the chance of false-negative cultures are increased.[21,24] Once a positive sample for *Mycoplasma* spp. is obtained, the organism should be speciated to determine its potential role in mastitis.[21,29,30]

Mycoplasma infections can be transferred through secretions including milk, urine, feces, nasal discharge, and directly from udder to udder.[7,18,21] The organism remains viable on cloth towels for a prolonged period of

time. Individual clean, disposable udder towels are recommended to reduce the chance of horizontal transmission.[7] Purchased cows should be isolated from the main herd and have a negative milk culture before being added to the herd. All personnel should be thoroughly trained on proper milking procedures, especially since most transmission is believed to occur during the milking process.[9,20,21,22,24,25,30] Sanitizing hands or rinsing gloved hands with a disinfectant between each cow will minimize the spread of the organism.[8,20,21,22,25,30]

In an early study, seven teat dips and sanitizers were compared *in vitro* and *in vivo* to determine their Mycoplasmacidal activity. Teat dips evaluated were Bovadine[a], Chlorox[b], Kendall[a], Nolvasan[c] and Velvet Dip[a]. Roccal[b] and Rapidyne[a] were the two sanitizers compared. Three different isolates from Mycoplasma mastitis outbreaks were used in the tests. Results indicated that, except for Velvet Dip, the teat dips appeared to be of benefit when used at the recommended concentration. *In vitro* test results indicated that activity of Chlorox, Roccal, Velvet Dip and the iodophores were either partially or strongly inhibited when diluted by milk. Interestingly, this study found that the organism survived longer during humid, rainy weather. During sunny days, no *Mycoplasma* spp. could be recovered, even from controls.[19]

Teat dipping is useful to minimize the spread of Mycoplasma mastitis.[19,23] Teat dips should be fresh and applicators should be routinely replaced or sterilized. Maintenance and diagnostic testing to ensure all milking equipment is functioning properly should be performed on a regular basis. When administering local mastitis preparations or dry cow therapy, strict aseptic technique will help minimize or eliminate transmission to other cows. Only single-treatment preparations are recommended.[8,21,22,25,30]

If a bulk-tank sample or a clinical case is cultured positive for a pathogenic *Mycoplasma* sp., individual cow samples from the entire herd should be tested, including both lactating and non-lactating cows.[20-22,25] Cows with confirmed Mycoplasma mastitis should be isolated immediately. Some clinically normal cows can shed the organism and serve as a reservoir for herdmates.[20,21,22,25] Additionally, chronic carriers may be shedding only a few organisms, thus making it more difficult to obtain a positive culture.[21,22] Culling all infected animals remains the best control program, as control programs are of dubious efficacy when infected cows remain in the herd. However, if infected animals cannot be culled, they should be housed and milked separately from the remainder of the herd.[9,21,22,25]

Herd cultures should be performed at 2 to 4 week intervals until no new cases are found.[8,9,21,22] Mycoplasma-infected milk should not be fed to calves retained as replacements. Caretakers must be instructed on proper husbandry to eliminate the potential for fomite transmission.[7,8,9,21,25,32]

Other Disease Entities

In one report, *M. bovis* was isolated from an outbreak in a group of 20 cattle suffering from severe keratoconjunctivitis. The disease spread rapidly through the group, with clinical signs ranging from watery eyes to eyelid swelling and corneal ulceration. Three of the animals were blind for seven to 10 days. While most of the conjunctivitis resolved in approximately two weeks, some of the animals exhibited corneal scarring. Affected animals were treated with ampicillin systemically, and a powder containing chlortetracycline and benzocaine was administered topically. Response to therapy was poor.[28]

In a 600-cow dairy in Michigan, *M. bovis* was isolated from the tympanic bullae of five calves ranging in age from 19 to 29 days. Clinical signs included a drooped ear(s), head tilt, epiphora and recumbency. An exudative otitis media was found in all calves at necropsy. *M. bovis* was isolated from bulk-tank samples. The dairy also practiced feeding milk from mastitic cows to calves, which may have been the source of infection. The farmer estimated that 50% of affected calves that survived responded to treatment of antimicrobials used alone or in combination, including oxytetracycline hydrochloride, tylosin and procaine penicillin G.[40]

On a calf ranch in California, 50 Holstein calves developed decubital abscesses in the brisket, carpal and stifle areas. Cultures of material aspirated from 14 affected calves yielded *M. bovis*. There was no evidence of joint involvement, and cultures of joint fluid were negative for *Mycoplasma* spp. Since the abscesses corresponded to pressure sore areas on the animals, it was thought the organism could have gained entry through skin abrasions. Alternatively, the calves may have had a Mycoplasmaemia at some point and the organisms localized in the damaged tissue, resulting in abscess formation. Specific treatments for the affected calves were not described.[27]

Polyarthritis and meningitis have been reported in young calves three days to three weeks of age. *M. bovis* was isolated from the affected calves. The severity of clinical signs and mortality was not affected by treatment, which consisted of lincomycin-spectinomycin

[a]Unable to determine the active ingredients in these products or if they are still available
[b]Not approved by FDA as a teat dip
[c]Certain formulations of chlorhexidine are approved as a teat dip by the FDA

combination, tylosin and oxytetracycline. While investigators assumed the source of infection was semen, they could not rule out mastitic milk as the source.[37]

Several *Mycoplasma* spp. have been isolated from the bovine reproductive tract; *M. bovigenitalium* in particular has been isolated from mucopurulent vaginal discharges of naturally occurring diseased cows, as well as from experimentally exposed females. Infected female cattle experienced granular vulvovaginitis and infertility.[2,12-14,32] The organism has also been associated with genital infections in bulls, manifested as seminal vesiculitis, ampullitis, epididymitis and inflammation of the rete testis.[12-14] It has also been isolated from apparently normal bulls.[32,37] Both cows and bulls can shed the organism intermittently, making diagnosis difficult at times. Most practitioners recommend culling infected animals to prevent shedding of the organism to other members of the herd.

Like the Mycoplasma pneumonia-arthritis and mastitis syndromes, treatment of other disease conditions caused by *Mycoplasma* spp. is frequently frustrating and unrewarding. The best recommendation for control is to implement sound management strategies to reduce spread of the organism to other animals by culling infected and/or carrier animals and by reducing stress. However, if culling is not a viable option, strict segregation and isolation from other members of the herd can minimize problems.

Conclusions

Besides the pneumonia-arthritis syndrome, Mycoplasma organisms can cause other diseases in cattle including mastitis, keratoconjunctivitis, otitis media, decubital abscesses, meningitis and reproductive problems. Animals with Mycoplasma infections frequently fail to respond to treatment, and the organism is shed intermittently. Reducing stress and preventing other diseases will help to minimize Mycoplasma infections. Culling infected animals is an important part of a control program.

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Abstracts

Use of Valnemulin in the Control of *Mycoplasma bovis* Infection under Field Conditions

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In a blind trial, alternate calves in six consecutive production batches of calves (total 70), on a farm with a high incidence of respiratory and reproductive disease, were allocated to treatment with either valnemulin or a placebo premix added to the milk from four days of age. The calves were weighed at the beginning and end of a 21-day period of medication. Blood samples and nasal swabs were taken and examined for the presence of *Mycoplasma* and *Pasteurella* species, and antibodies to viral agents. Clinical condition, rectal temperature, respiratory and other signs and refusals of milk were recorded daily. Dead calves were examined postmortem. The calves medicated with valnemulin gained weight more quickly, had fewer cases of *Mycoplasma* infection and fewer respiratory signs, and required fewer treatments with antibiotics than those in the placebo group.

Comparison of the Measurement of total Carbon Dioxide and Strong Ion Difference for the Evaluation of Metabolic Acidosis in Diarrhoeic Calves

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Eighty-four calves with diarrhoea were treated with fluids and 13 apparently healthy calves of similar ages were sampled as controls. Their total blood carbon dioxide (TCO_2) was measured with a Harleco apparatus and 31 of the calves were treated with oral fluids and 53 with parenteral fluids. The oral fluid contained 118 mmol/litre Na^+, 25 mmol/litre K^+, 110 mmol/litre glucose, 108 mmol/litre bicarbonate (HCO_3^- as citrate), 43 mmol/litre Cl^-, 4 mmol/litre Ca^{++}, 4 mmol/litre Mg^{++} and 20 mmol/litre glycine, and the parenteral fluid contained 144 mmol/litre Na^+, 4 mmol/litre K^+, 35 mmol/litre HCO_3^- and 113 mmol/litre Cl^-. Both treatments resulted in significant improvements in acid-base status as demonstrated by an increase in TCO_2, and the treatment was successful in 27 of the 31 calves receiving oral fluids and in 45 of the 53 calves receiving parenteral fluids. Thirty-seven of the calves treated parenterally were very severely acidotic (TCO_2 <8 mmol/litre) initially and the received an additional 400 mmol HCO_3^- added to the first 5 litres of infusion. Treatment was successful in 33 of these calves. The decision to administer additional bicarbonate was made on the basis of their acid-base status as measured with a Harleco apparatus. The strong ion difference (Na^++K^+-Cl^-) (SID) of the calves was calculated retrospectively. There was a significant correlation between the SID and TCO_2 of the calves treated with oral fluids but not among the control calves or the calves treated parenterally. Furthermore, measurements of the change in SID during therapy gave little indication of the change in acid-base status as measured by the Harleco apparatus, with the SID decreasing (suggesting a worsening of acid-base status) in 16 calves in which the TCO_2 increased (suggesting an improvement in acid-base status). There was a significant correlation between the change in SID and the change in TCO_2 during treatment in the calves receiving oral fluids but not in the calves treated parenterally.