Clinical Management of Septic Arthritis in Cattle

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KEYWORDS

• Cattle • Septic arthritis • Surgery • Joint disease • Diagnostic

KEY POINTS

- Synovial fluid, ultrasound, and radiographic imaging are common diagnostic tools for septic arthritis.
- Mycoplasma septic arthritis is suspected in calves with clinical signs of otitis and pneumonia.
- Commonly affected joints are carpus, stifle, and tarsus.
- Treatment strategy must include long-term antibiotics, anti-inflammatories, and joint lavage.
- Knowledge of communication and boundaries for commonly affected joints is essential to perform joint lavage and arthrotomy.

Lameness in cattle originates from the digits. The second most common cause of lameness is from the joint; 47.0% to 72.2% of all lameness other from the foot is located to the joint and ligament.[1,2] The most common lesions affecting the joint are of traumatic origin, developmental (osteochondrosis), and septic. Few data are available on the importance of septic arthritis (SA) in cattle. In Israel, arthritis accounts for 13.8% of lameness cases.[3] In American feedlots, swollen joints are linked to 12% of lameness. The most common joints infected are front fetlock, hock, and elbow.[4] A Canadian survey in feedlot calves from Saskatchewan showed that 1.3% became chronic; 39% of these calves had a diagnosis of polyarthritis.[5] In Sweden, the incidence rate of arthritis in dairy calves was reported to be 0.002 cases per calf-months at risk.[6] In a study in veal calves in Belgium, the incidence rate of SA was 0.11 cases per 1000 calf days at risk.[7]

Although not as common as claw diseases, the consequences of SA are dramatic if left untreated, with potential irreversible joint function. It is a painful disease demanding a rapid medical decision. It also can be the first sign of a contagious disease like *Histophilus somni*[8,9] or *Mycoplasma bovis*.[10–13]

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Vet Clin Food Anim 30 (2014) 177–203
http://dx.doi.org/10.1016/j.cvfa.2013.11.006 **vetfood.theclinics.com**
0749-0720/14/$ – see front matter

PATHOPHYSIOLOGY

Bacterial arthritis is the most common form of SA in cattle.[14] There is anecdotal report of virus infection.[15] Bacterial arthritis is certainly the most damaging joint pathology. The origin of the bacterial infection is from direct trauma or contamination, adjacent infection, or hematogenous seeding. The distal joints like the interphalangeal joints and the fetlock are mostly affected by direct trauma. Iatrogenic infection following intra-articular (IA) injection is also possible, but rare in cattle compared with horses.[16] Infection adjacent to a joint is another cause of septic joint. A solar or bulbar abscess can expend and contaminate the distal interphalangeal joint.[17,18] Finally, the systemic or remote infection has to be considered when a calf is diagnosed with SA, especially if more than one joint is affected and no wound can be seen. The umbilicus is a very common route of infection. Inadequate hygiene and disinfection of the umbilicus after birth and passive immunity transfer failure are the most important factors contributing to umbilical infection. Other frequent newborn infections have to be considered if the umbilicus is normal at the physical or ultrasound examination: pneumonia, diarrhea, and septicemia. Because passive immunity transfer failure is the most important risk factor for all these diseases, it should also be considered Systemic origin rather than local trauma will increase the probability of more than one joint being infected.[19] In adult endocarditis, Lyme disease, pneumonia, or mastitis can be the source of SA.[10,20,21]

Even if the synovial membrane has a relative effectiveness on the control of the bacteria, multiple villosities support the establishment and the attachment of microorganisms. Bacteria will take action on the cartilage, the synovial membrane, and fluid but the most perverse effects are of immunologic origin. First, microorganisms are destroyed by neutrophils and their enzymes: elastase, cathepsin, gelatinase, and the collagenase. These enzymes destroy not only the bacteria but also the cartilage and its components. Moreover, the neutrophils and the inflamed tissues release free radicals, which have the same harmful effects on articulation. The inflammation will increase the permeability of the capillaries and let other mediators arrive at the site of infection (kinine, factor of coagulation, cascade of the complement, fibrinolytic system). These mediators will stimulate the synoviocytes and the chondrocytes. The chondrocytes will release mediators as the MMP (matrix metalloproteinase), which will decrease the production of proteoglycan. The reduction in production and the degradation of the proteoglycans deteriorate the physical properties of the cartilage, thus decreasing its potential of compression. The cartilage is therefore more fragile. The presence of fibrin on the cartilage and the synovial membrane decreases the nutritive effectiveness of the synovial fluid and the diffusion of antibiotics used in the treatment of SA. If left untreated, this fibrin will form a pannus covering all surfaces of the joint cavity.

PATHOGEN

Numerous bacteria have been isolated from infected joints in cattle. To our knowledge, only a few studies are available on the prevalence of the different pathogens isolated for septic joints. In our hospital, we have performed a retrospective study of joint bacterial culture in 172 cases of SA (between 1980 and 2000).[14] No bacterium was cultured in 40% of cases (n = 69), 1 bacterium in 47% (n = 80), 2 bacteria in 9% (n = 16), and more than 3 in 4% (n = 7). The proportion among the different bacteria cultured is presented in **Table 1**. *Truepurella pyogenes* was by far the most frequent bacterium cultured. Because their specific culture was not requested for all cases, the importance of anaerobic bacteria and *Mycoplasma* spp is probably