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Bovine Mastitis and Therapeutic Management

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Summary

Mastitis is inflammation of the mammary gland usually caused by pathogens, mainly bacteria which have entered the teat canal. Preventive measures include pre and post dipping of teats. The most critical period of acquiring infection is the dry period Treatment includes dry therapy and lactational therapy by intramammary route and in severe case of mastitis: intramammary and systemic route together.

Introduction

Intramammary infection (IMMI) usually occurs as an immune response of animal to bacterial invasion to eliminate invading pathogen. Bacterial presence within the udder results in the movement of white blood cells into the gland to help fight the disease. An uninfected mammary gland will maintain a low somatic cell count (<200,000 cells/ml of milk). Once gland tissue becomes infected, numerous neutrophils will be drawn to the mammary gland, resulting in increased somatic cell counts.

Mastitis can be divided into two major categories on basis of source of infection: contagious mastitis and environmental mastitis. Contagious mastitis can spread from infected gland to healthy gland during milking process by contact with infected milk, splashes or aerosols of milk during milking, milkers' hands or milking equipments. The most common bacteria causing contagious mastitis are Staphylococcus aureus, coagulase negative staphylococci (CNS) and Streptococcus agalactiae. The primary habitat of bacteria causing contagious mastitis is inside udders or on teat skin. Contagious mastitis is usually a chronic or subclinical mastitis.

The primary habitat of bacteria causing environmental mastitis is manure, bedding and soil. It can occur during environmental contact of the teats at milking time or between milkings. The major organisms causing environmental mastitis include the coliforms (*Escherichia coli*,

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Klebsiella), the environmental Streptococcal species (*Streptococcus uberis*, *Streptococcus dysgalactiae*, *Enterococcus)* and *Pseudomonas* species. Most environmental mastitis cases are seen in the period immediately before, to a few weeks after, calving when animals are very susceptible to infection because their natural defence mechanisms are low. The environmental mastitis is usually an acute clinical mastitis but subclinical cases do occur too. On the basis of severity, mastitis is commonly categorised as clinical and subclinical, depending on the bacteria causing the infection and the immune response of the animal towards bacteria. Clinical mastitis is characterized by abnormalities in the milk or the udder that can be seen such as flakes, clots, and a watery appearance in milk, hardness and swelling of udder. Clinical mastitis can be acute clinical mastitis characterized by a sudden onset of symptoms and shows severe signs. Chronic clinical mastitis persists for a long time but is not severe. Subclinical mastitis is characterized by an udder infection that shows no external changes in udder and milk but significant changes in milk composition. Subclinical mastitis can only be detected with somatic cell count (SCC), California mastitis test or microbiological culture of milk. Infected animal with subclinical mastitis serves as reservoirs and can infect other animals.

Table1: Major differences between contagious and environmental mastitis (Blowey R & Edmondson P, 2010)

	Contagious mastitis	**Environmental Mastitis**
Source of Infection	Teat and udder	Contaminated environment
Transfer of infection in to udder	During Milking	Between milking and during dry period
Clinical mastitis	Most cases are subclinical	Higher proportion are clinical
Control by	Post milking teat dipping, dry cow therapy, milking hygiene and culling of chronic cases	Environmental hygiene, predipping, dry period teat sealant

Principles to control mastitis:

1. Elimination of existing infections in udder: Antimicrobial therapy during dry period is a method of choice. Dry period antibiotic treatment is a formulation of antibiotics prepared for

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administration into the udder immediately after the last milking of lactation to reduce new infections and elimination of existing infections. In India, ceftiofur hydrochloride 500mg/10ml or Cefalonium 250mg Intramammary suspension are available for the treatment of sublinical mastitis at time of dry off mainly associated with Staphylococcus aureus, Streptococcus dysgalactiae and Streptococcus uberis.

There are three phases of dry period namely early, mid and late dry period. In the first two weeks (early dry period); keratin plug forms a teat seal and active involution of udder tissue occurs. In the mid dry period, immunoglobulins and natural inhibitory substances like neutrophils and lactoferrins are formed. In the last two weeks before calving (late dry period), keratin plug slowly dissolves and colostrogenesis occurs. Animals are most susceptible to infection during first and last two weeks of dry period because teat plug is forming and dissolving respectively. Further depressed immune system of dam with approaching parturition contributes to new infections. The persisted subclinical infections during dry period may flare into clinical cases after calving. Most new cases of mastitis occur first four weeks of lactation and 60% of clinical cases by environmental pathogens originate from the infections established during early and late dry period (Blowey R & Edmondson P, 2010).

2. Prevention of new infection: Pre-milking and post milking teat disinfection are the most effective mastitis control practice in lactating animals. Pre-milking teat disinfection with chlorhexidine in association with post milking teat disinfection reduces new intramammary infection. Post milking teat disinfection is regarded as the single most effective control practice in lactating animals. Iodophores teat dips with 0.1 to 1% available iodine can be used as post dip. Post milking teat disinfection removes bacteria deposited during the milking process and therefore it is an extremely important control measure against contagious mastitis. Post dip should be applied as soon as milking is over. Teat must not be wiped dry after post dip. Use of betadine (Povidone Iodine 5%, without dilution) with 7% glycerine can be suggested to farmers as a post dip. Pre-milking teat disinfection is aimed at reducing the incidence of environmental mastitis.

3. Monitoring udder health status: Implementing and effective system of monitoring udder health involves monitoring at herd and individual level. Use of animal side diagnostic test like California mastitis test - somatic cell count and milk bacteriological culturing are important for udder health and milk quality.

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Five points mastitis control programmes promoted by The National Mastitis Council (NMC) is as follows:

Udder hygiene and milking management

Milking equipment's maintenance

Dry animal therapy

Appropriate therapy of mastitis during lactation

Culling chronically infected animals

Udder hygiene includes pre-milking udder preparation by washing teat in water and drying of teat with paper towel followed by use of 0.25% iodine pre-milking teat disinfectant (Dipping is better than spraying). The foremilk stripping is checked for clinical mastitis (clot, watery or stringy milk) using a strip cup test. The early recognition and treatment of clinical cases is important part of mastitis control programme (Radostits, 2000).

Therapeutic management of different forms of bovine mastitis

Mastitis is the most frequent cause of antibacterial use on dairy farms and contributes to a substantial portion of total drug and veterinary costs incurred by the dairy industry (Erskine *et al*.2003). Knowledge on the microbiological profile of mastitis by clinical and laboratory diagnosis (isolation, identification and antibiogram) is one of the basic pillars for the rational use of antimicrobial agents (ATM). The aim in selecting the best antimicrobial treatment regimen for mastitis is administering the drug at a dose and site that will allow accumulation in the mammary gland to maintain an effective drug concentration. There are three pharmacological compartments. The most common target compartment milk and epithelial lining of duct and alveoli of mammary gland. The Strep. agalactiae, Strept. dysgalactia, Coagulase negative staphylococci pathogen tend to locate in duct areas of the udder where antibiotics are effective. Strep. agalactiae are very sensitive to penicillin, so treatment has a relatively high cure rate by administering intramammary administration (Erskine *et al*. 2003). Second compartment is deep tissue of the mammary gland. Staph. aureus can penetrate into udder tissue and form micro abscesses that are protected from Antimicrobials (ATMs) by scar tissue. They are difficult to cure, especially during lactation, so prevention is essential. Coliform mastitis involve animal itself, third compartment. Bacteraemia can occur and respond to systemic therapy. Mild cases of clinical coliforms mastitis generally are self-limiting and the animals own defense mechanisms can successfully clear the infection from

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the udder, and though antibiotics are not required at all; serious cases support the use of systemic administration of antibiotics. Pseudomonas infection is impossible to treat so animal must be culled. In mastitis caused by penicillin-susceptible Staph. Aureus strains, best results were achieved using combination of systemic and intramammary (IMM) treatment with penicillin G. In infections of the milk compartment such as streptococcal mastitis, there is probably no advantage of systemic administration indeed the concentration of penicillin G in milk remains 100-1000-fold lower than when given intramammarily.

It is always desirable to treat mastitis according to the antibiogram pattern of the causative agent. As it takes time to do sensitivity test, broad-spectrum antibiotics can be given initially. However, the basic rule in selecting the drug is to opt for one with as narrow a spectrum as possible, to focus treatment on a specific pathogen and minimize side-effects. In general, narrow-spectrum antibiotics are bactericidal and those with a broad spectrum are bacteriostatic (Wilson, 1980).

Intramammary administration of drugs is the route of choice in subclinical to moderate mastitis and is used along with parenteral administration in severe mastitis. The disadvantage of local application of antimicrobials is the slow and uneven distribution of certain drugs in the infected udder. In severe mastitis, parenteral route of drug may overcome problems of blockage of milk ducts by debris, although it is usual to administer agents concurrently by the intramammary route. Severely inflamed udders should be milked out frequently, with the aid of oxytocin if necessary. Treatment at drying-off with a dry-cow antibiotic preparation is practical and inexpensive for subclinical mastitis. Subacute clinical mastitis is the most prevalent form of clinical mastitis in bovines. Intramammary treatments are usually administered 4 times at 12-hourly intervals with rapidly absorbed drugs or 3 times at 24-hourly intervals with slowly absorbed drugs. Parenteral antibiotic therapy is not routinely advised. Parenteral antibiotic therapy is preferred due to poor or uneven distribution of the drug in the inflammed udder parenchyma in acute clinical mastitis. The intravenous route must be used to achieve maximum parenchymal diffusion. In chronic mastitis, it is usually necessary to cull the animal or to destroy the affected quarter by means of an infusion of 25–40 mL of concentrated ether to eliminate an important potential source of bacterial infection for healthy quarters. Parenteral and intramammary antibiotic treatment for 3–5 days may be used but the prognosis remains poor.

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Reasons for treatment failure

Reasons for treatment failure include lack of contact between bacteria and antibiotics due to scar tissue formation, protection within leukocytes, poor drug diffusion, and inactivation by milk and tissue proteins; microbial resistance to antibiotics; improper treatment procedures like stopping the therapy too soon.

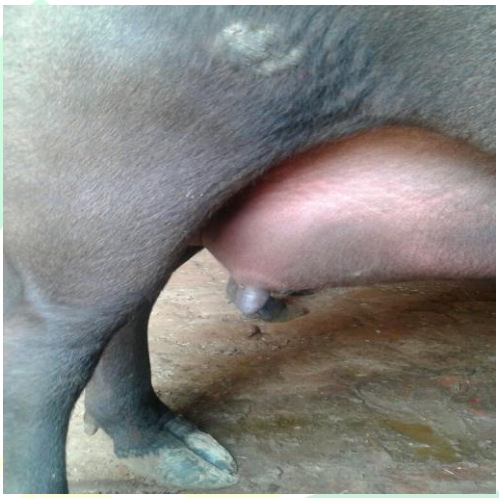

Fig 1 Acute clinical colliform mastitis in buffalo

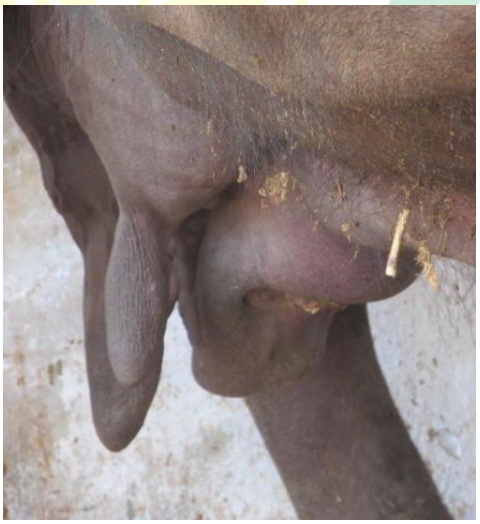

Fig 2 Chronic Staphylococcus mastitis