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MASTITIS: A MAJOR CHALLENGE IN DAIRY HERD

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ABSTRACT

Mastitis caused by *Streptococcus agalactiae, Staphylococcus aureus*, *Corynebacterium bovis, Mycoplasma sp, and Streptococcus dysgalactiae* leads to severe economic losses, both direct and indirect. It is not only an economic concern but also a significant animal welfare issue because it causes pain, inflammation, and reduced productivity. Milk produced by a mammary gland affected by clinical mastitis is useless in dairy production and should be discarded. It is responsible for approximately 60–70% of antimicrobial drug use in dairy animals. These bacteria multiply and produce toxins that cause injury to the milk-secreting tissue, in addition to physical trauma and chemical irritants. External infections weaken the internal udder defence. Current mastitis treatment relies on antibiotics. Effective vaccines can also reduce the incidence of mastitis, thereby effectively reducing antibiotic use. This article highlights the epidemiology of mastitis, risk factors, and economic impact of mastitis in dairy animals with emphasis on prevalence and management practices.

Keywords: Mastitis, dairy cattle, somatic cell count, antibiotic resistance, udder health

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Introduction

Mastitis, the inflammation of the mammary gland, is one of the most frequent diseases affecting dairy cows worldwide. Major causative agents of mastitis are bacteria like *Streptococcus agalactiae, Staphylococcus aureus*, *Corynebacterium bovis, Mycoplasma sp, and Streptococcus dysgalactiae*. It is responsible for approximately 60–70% of the use of antimicrobial drugs in dairy animals; as a result,t antimicrobial resistance has also emerged over the last few decades. It leads to severe economic losses, both direct and indirect [1, 20]. Mastitis can be classified, according to its aetiology, into environmental and contagious or, according to symptoms, into clinical and subclinical [2].

Being a multifactorial disease, mastitis susceptibility is given by multiple factors such as age, parity, lactation stage, milk yield, and udder anatomical dispositions. Two of the main factors are immunological condition and reactivity of the mammary gland. Consequently, the clinical manifestations as well as their further course depend on the interplay between the innate resistance and adaptive immunity of the dairy cow and the type, concentration, and virulence of udder pathogens. These pathogens are discussed relative to prevalence, virulence factors, pathology, and control. These control measures include milking time hygiene, segregation, culling, vaccination, and treatment. Environmental mastitis pathogens include a wide range of organisms, such as coliforms *(Escherichia coli, Klebsiella, Enterobacter*, and *Citrobacter), environmental streptococci (Streptococcus uberis and Streptococcus dysgalactiae), Trueperella pyogenes, non-aureus staphylococci* (NAS), such as *Pseudomonas, Proteus, Serratia, Aerococcus, Listeria, yeast, and Prototheca.* Bovine mastitis can be divided into clinical and subclinical types. Clinical mastitis (CM) is a severe condition with local symptoms visible in the mammary gland and systemic symptoms, e.g., redness and inflammation of affected areas, pain, loss of appetite, elevated body temperature, reduced milk yield, and changes in the milk composition. In severe cases, abnormal teat discharge can be noticed, e.g., floccules and watery milk or blood in the milk [3].

In the clinical form, we can see changes in milk, udder, and the cow herself. It can be further classified as subacute, acute, and hyperacute depending on the severity of symptoms. In subclinical mastitis (SCM), the appearance of milk does not change. One of the indicators of subclinical mastitis is the possible development of inflammation, which is the increased somatic cell count [4, 20].

The etiological agents of mastitis are mainly bacteria, especially *staphylococci and streptococci,* as well as *mycoplasmas,* fungi, algae, protozoa, and viruses. Inflammation may also develop upon physical trauma to the udder, contact with irritating chemicals triggering an immune response, or any physiological damage. Bacterial mastitis is highly important due to its physiological complications and economic consequences [5]

Depending on the etiological factor (mode of infection), two categories of mastitis have been distinguished: that caused by infectious agents and that caused by environmental agents, although two veterinary researchers, Klaas and Zadoks, suggest that this division may be misleading in some cases.[6]

Mastitis is not only an economic concern but also a significant animal welfare issue because it causes pain, inflammation, and reduced productivity. Improper antimicrobial use for mastitis treatment may contribute to antimicrobial resistance and milk residue concerns. This review summarises the epidemiology, aetiology, pathogenesis, diagnosis, treatment, and prevention of bovine mastitis

Diagnosis

SCC and CMT are usually used for subclinical mastitis, and clinical mastitis is observed by udder and milk deviation

EFFECTS ON DAIRY PRODUCTION

Substantial losses in milk production occur as a result of both clinical and subclinical mastitis. Although antibiotic treatment prevented subclinical mastitis from progressing to the clinical form during lactation, the subclinical form endures even after treatment.[7]

Milk produced by a mammary gland affected by clinical mastitis is useless in dairy production and should be discarded. Economic losses due to discarded milk are much greater than losses due to the diminished milk production because the discarded milk is produced by cows and, thus, has incurred the associated feeding costs that are accounted for in the loss. This means that economic loss due to 10 kg of discarded milk is greater than the loss for 10 kg of decreased milk production.[8]

Epidemiology and risk factors

Mastitis is considered to be a typical example of a complex disease, known to be established as a result of the interactions of three bio-systems, namely the causative agent (pathogen), the animal (host) and the environment in which the animal lives. Host factors include breed, physiological state of the mammary gland, and anatomy of teat canal, sphincter tone and presence of teat lesion. Agent factors include the ability to survive in the immediate environment of the animal, the ability to colonise the teat duct, the ability to adhere to the mammary epithelium, and not to be flushed out with milk flow. Environmental factors include milking practice, housing system and bedding. Sandholm and Korhonen reported that the primary and secondary body defence mechanisms prevent pathogenic microbes from entering the mammary gland through the teat canal orifice. They also indicated that the concentrations of the antibacterial factors in the udder secretion are under genetic control and depend on the lactation stage and udder health. Environmental factors such as management, feeding, hygienic status, bedding, milking and the virulence of the organism contribute to the disease. [9].

Pathogensis

Mastitis in dairy animals occurs when the udder becomes inflamed, and bacteria invade the teat canal and mammary glands. These bacteria multiply and produce toxins that cause injury to the milk-secreting tissue, in addition to physical trauma and chemical irritants. These cause an increase in the number of leukocytes, or somatic cells, in the milk, reducing its quantity and adversely affecting the quality of milk and milk by- products. The teat end serves as the first line of defence against infection. From outside, a sphincter of smooth muscles surrounds the teat canal, which functions to keep the teat canal closed. It also prevents milk from escaping and bacteria from entering the teat. From inside, the teat canal is lined with keratin derived from stratified squamous epithelium. Damage to keratin has been reported to cause increased susceptibility of the teat canal to bacterial invasion and colonisation. The fibrous proteins of keratin in the teat canal

bind electrostatically to mastitis pathogens, which alter the bacterial cell wall, rendering it more susceptible to osmotic pressure. Inability to maintain osmotic pressure causes lysis and death of invading pathogens. The keratin structure thus enables trapping of invading bacteria and prevents their migration into the gland cistern. During milking, bacteria present near the opening of the teat find opportunity to enter the teat canal, causing trauma and damage to the keratin or mucous membranes lining the teat sinus. The canal of a teat may remain partially open for 1-2 hours after milking, and during this period the pathogens may freely enter the teat canal [10, 19].

Bacterial pathogens which are able to pass the opening of the teat end by escaping antibacterial activities establish the disease process in the mammary gland, which is the second line of defence of the host. In dairy animals, the mammary gland has a simple system consisting of teats and udder, where the bacteria multiply and produce toxins, enzymes and cell-wall components which stimulate the production of inflammatory mediators attracting phagocytes. Neutrophils are the predominant cells found in the mammary tissue and mammary secretions during the early stage of mastitis and constitute >90% of the total leukocytes. The phagocytes move from the bone marrow toward the invading bacteria in large numbers, attracted by chemical messengers or chemotactic agents such as cytokines, complement and prostaglandins released by damaged tissues. The neutrophils exert their bactericidal effect through a respiratory burst and produce hydroxyl and oxygen radicals that kill the bacteria. During phagocytosis, bacteria are also exposed to several oxygen-independent reactants such as peroxidases, lysozymes, hydrolytic enzymes and lactoferrin. In addition to their phagocytic activities, neutrophils are a source of antibacterial peptides called defensins, killing a variety of pathogens that cause mastitis. Masses of neutrophils pass between the milk-producing cells into the lumen of the alveoli, thus increasing the somatic cell counts and also damaging the secretory cells. Clots are formed by aggregation of leukocytes and blood clotting factors, which may block the ducts and prevent complete milk removal, resulting in scar formation with proliferation of connective tissue elements. This results in a permanent loss of function of that portion of the gland. The milk ducts remain clogged, secretory cells revert to a non-producing state, and alveoli begin to shrink and are replaced by scar tissue. This helps in the formation of small pockets, making it difficult for antibiotics to reach there and also prevents complete removal of milk. Macrophages are the predominant cells found in milk and tissue of healthy involuted and lactating mammary glands [11].

Sign and symptoms

In acute mastitis, there is a red, swollen, and painful breast. Targeted ultrasound can be performed to evaluate the extent of infection and for an underlying abscess. Noncomplicated mastitis or a small fluid collection may respond to oral antibiotics without further intervention, but a larger or more complex abscess may require single or serial percutaneous aspiration. Breast infections, particularly those complicated by an abscess, can have a prolonged clinical course, and close follow-up is required. Since the clinical presentation and imaging features of acute infectious mastitis can overlap with other etiologies, such as inflammatory breast cancer and idiopathic granulomatous mastitis, a percutaneous biopsy may be indicated to accurately diagnose patients [12].

Treatment

Current mastitis treatment relies on antibiotics and is the most important reason for antibiotic use in dairy cows. However, the emergence of drug-resistant strains is threatening the viability of antibiotics for mastitis treatment. Antimicrobial resistance (AMR) occurs when pathogens can overcome the effects of antibiotics that were originally effective. It was reported that AMR was first detected in penicillin (inhibits cell wall synthesis by binding to enzymes on the bacterial cell wall) resistance of *Streptococcus pneumoniae.* Consequently, there is a global focus on finding alternatives to treat bacterial diseases. Finland substantially reduced macrolide use, resulting in nearly a 50% decrease in erythromycin (a protein synthesis inhibitor) resistance. This was proof of concept that reducing antibiotic use can reduce AMR.[13]

Intramammary antibiotic therapy is generally recommended only for infections caused by Gram-positive bacteria (have a thick peptidoglycan layer in the cell wall) such as *S. aureus*, *S. agalactiae* and environmental *Streptococci* spp. In contrast, most Gram-negative infections are cleared by the cow's own immune system. Therefore, antibiotics approved for use in the udder of dairy cows are effective in only 20–50% of clinical mastitis [14, 21].

The specific mechanism of action of non-steroidal anti-inflammatory drug (NSAIDs) is inhibition of cyclooxygenase (COX), reducing production of prostaglandins (an inflammatory mediator). Carprofen, like meloxicam, is a COX-2 selective, single-dose, long-acting NSAID to treat bovine mastitis.

Many herbal medicines have antibacterial ability. For example, *Red ginger* had good bactericidal effects on *Staph epidermidis*, *S. aureus*, and *S. agalactiae* derived from bovine mastitis.

Antimicrobial peptides are another promising replacement for antibiotics. Nisin, a natural antimicrobial peptide produced by *Lactococcus lactis*, had excellent antimicrobial activity against Gram-positive bacteria isolated from mastitis in dairy cows. In a bovine mastitis trial, there was no difference between Nisin and an antibiotic group for rates of bacteriological or clinical cure [15, 21]

Vaccination

Effective vaccines can reduce the incidence of mastitis, thereby effectively reducing antibiotic use. Vaccines have been developed for some pathogens causing clinical mastitis, e.g., *E. coli*, *S. aureus*, and *Streptococcus* spp. Among them, J5 mutant strain-based vaccines represent a breakthrough in *E. coli* vaccine development. In clinical trials, *E. coli* J5 vaccination reduced the incidence of gram-negative mastitis in dairy cows, with protection lasting up to the third month of lactation. However, the number of pathogenic bacteria causing mastitis in cows far exceeds bacteria targeted by existing vaccine development. Furthermore, pathways and mechanisms of infection for these pathogenic bacteria are not uniform, posing challenges to developing effective vaccines for mastitis in cows [16, 19].

Prevention

Milking Machines Management

As milking machines became popular, defining appropriate milking procedures was an important priority. Early mastitis workers had studied physiological mechanisms of milk secretion and ejection, and "pituitrin" (oxytocin) was identified as a substance that could positively stimulate milk flow. As milking machines were adopted, factors that could influence milk ejection were studied. In one experiment, the effect of fright on milk ejection was evaluated by placing a cat on the back of a cow and exploding paper bags every 10 s for 2 min (the authors noted that "later the cat was dispensed with as unnecessary". This work clearly demonstrated that fear had a significant effect on reducing milk ejection. This was a potentially important finding because incomplete milking of cows chronically infected with Strep. Agalactiae was soon shown to result in increased occurrence of clinical mastitis. This study had a lasting effect on milking [17]

Dry cow therapy

Dry cow therapy treats existing inflammatory infections and prevents new infections during the dry period [18].

Supplementation with Vitamin E and Selenium

The role of nutritional management in the development of mastitis has long been controversial and difficult to separate from other confounding effects. It is suggested that feeding high-concentrate diets was a risk factor for mastitis, but direct effects of nutrition on mastitis were not reported until experiments were performed that demonstrated that dietary deficiencies of selenium and vitamin E increased incidence and duration of clinical mastitis. Initial experiments were supported by later field studies that demonstrated increased subclinical and clinical mastitis in selenium-deficient herds. Researchers performed experiments that demonstrated the essential role of these nutrients in maintaining effective neutrophil function. The important role of vitamin E and selenium in maintaining udder health is now well established, and this work contributed to both dietary modification and important knowledge about neutrophil function.

Conclusion

Mastitis continues to be one of the most significant diseases affecting the dairy industry, reducing milk production, compromising animal welfare, and causing substantial economic losses. However, it is largely preventable through good herd management, proper milking hygiene, regular health monitoring, and early diagnosis. Advances in diagnostic techniques and improved treatment strategies have made it easier to detect and control the disease before it causes severe damage. At the same time, responsible use of antibiotics, along with preventive measures such as vaccination, selective dry cow therapy, and enhanced farm biosecurity, is essential to limit antimicrobial resistance and ensure sustainable dairy farming. The future of mastitis control lies in combining modern technologies with sound management practices and educating farmers about disease prevention. By adopting these approaches, dairy producers can improve milk quality, protect animal health, increase farm profitability, and contribute to a more sustainable and productive dairy industry.

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