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Mastitis in cattle

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Review Article

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Abstract

Mastitis ranks among the most serious diseases affecting the worldwide dairy production sector. Mastitis is a complex condition involving multiple infections, making treatment challenging. Mastitis has an impact on dairy farm profitability and animal welfare along with the animals' health. Mastitis is seen as being extremely important in terms of health of the public because it is linked to numerous zoonotic illnesses where milk acts as a vector for infection. Among different udder problems, subclinical mastitis is receiving a lot of attention since it causes both subpar milk supply and the spread of certain diseases to humans. Bovine mastitis can be classified into clinical, subclinical, or chronic forms based on the degree of inflammation present. Clinical bovine mastitis is easily identified through outward symptoms, like fever and a red, swollen udder in dairy cows. Bacterial mastitis occurs when a bacterial organism disrupts the breast microbiome. Previously believed to happen when bacteria penetrate through damaged nipple skin, recent research indicates that mammary dysbiosis, caused by a variety of factors, including medical conditions and genetics passed down from parents, antibiotic exposure, probiotic use, frequent use of breast pumps, and Cesarean deliveries.

***Keywords**: Mastitis, bacteria, diagnosis, subclinical mastitis*

Introduction

Bovine mastitis, which results in mammary gland infection, is a serious illness that affects dairy animals all over the world. There are three categories for the severity of inflammation: sub-clinical, clinical, and chronic. Subclinical mastitis has significant financial ramifications and is challenging to diagnose because there are no outward signs. Since somatic cell counts (SCCs) determine milk quality payments, higher levels equate to lower compensation. This has a substantial impact on farm revenues along with lowering milk quantity and medical expenses **(Yalcin, 2000).**

Clinical mastitis is further splitted into acute, sub-acute, and per-acute categories depending on the level of inflammation **(Kibebew, 2017).** Moreover, serious cases of clinical mastitis can lead to death **(Gruet *et al.*, 2001).**

Types of Mastitis

Mastitis can be categorized in a number of ways; however, a straightforward classification divides it up into two main groups:

A. Bacteria that reside inside the udder and on the teat's skin cause contagious mastitis. During milking, it can spread from cow to cow.

B. Environmental mastitis: A cow that comes into contact with a contaminated environment can have certain organisms, such as E-coli, which normally do not inhabit the skin or udder, enter the teat canal and cause environmental mastitis **(Kitchen, 1981).** This comprises disease-causing agents that are often found in feed, bedding, and excrement. Rarely do environmental mastitis cases account for more than 10% of all herd mastitis cases.

Three categories of contagious mastitis can be distinguished (Cobirka *et al.,* 2020):

1. Clinical Mastitis.
2. Subclinical Mastitis.
3. Chronic Mastitis.

1. Clinical Mastitis: Clinical mastitis is characterized by the presence of severe inflammatory symptoms, including swelling, heat, redness, and pain. The three kinds of clinical mastitis are as follows: **(Ganguly, 2016)**:

a) Per acute Mastitis is typically distinguished by systemic symptoms (fever, sadness, shaking, appetite loss and weight reduction), obviously severe inflammation, and disrupted functions (reduction in milk production, alterations in milk composition).

b) Acute mastitis is identical to per acute mastitis; however, it has mild depression and fever as systemic symptoms.

c) Sub-Acute Mastitis, there are no obvious systemic symptoms and only mild evidence of inflammation in the mammary glands.

2. Sub-clinical mastitis, which is defined by a shift in the content of milk without any obvious symptoms of abnormalities or severe inflammation. Special diagnostic tests can identify changes in the composition of milk.

3. Chronic Mastitis is an inflammatory condition that may persist for months and can carry over from one lactation period to another. The majority of the time, chronic mastitis is subclinical, but it can sometimes flare up in a brief, acute, or subacute form.

Epidemiology

Mastitis is seen to be a prime illustration of a complicated illness, which is recognized as the result of the interplay between three biosystems: the pathogen, the habitat of the animal, and the animal itself. Examples of host factors include the presence of a teat lesion, the anatomy of the teat canal, the tone of the sphincters, the physiological state of mammary gland, and breed. Examples of agent factors include the ability to inhabit the animal's immediate environment, colonize the teat duct, adhere to the mammary gland epithelium, and withstand being washed away by milk flow. According to **Makovec and Ruegg (2003),** environmental factors include bedding, living arrangements, and milking practices.

Pathogenesis

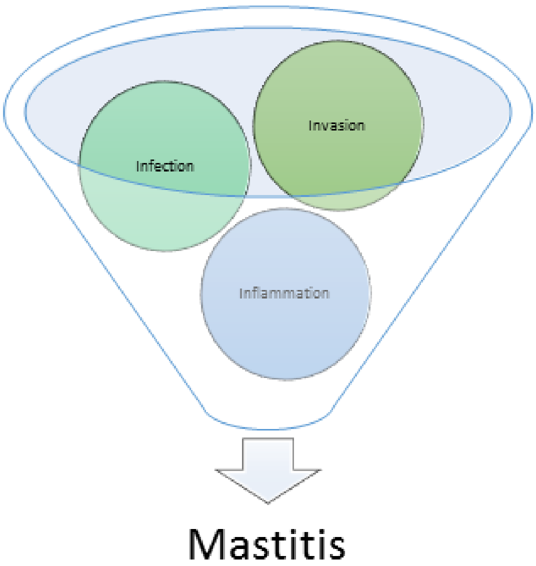
Various bacterial strains primarily cause mastitis; however, there have also been documented cases related to viruses, algae, and fungi. **(Eriksson, 2005 and Awandkar *et al.,* 2021).** Bacteria are the most widespread causative agents associated with mastitis. More than 150 Gram-positive and Gram-negative bacteria have been recognized as mastitis pathogens, which can be categorized as environmental (present in the surrounding environment) or contagious (transmitted from other infected quarters) **(Ndahetuye *et al.,* 2019; Cobirka *et al.,* 2020).** *Staphylococcus aureus* can generate numerous potential virulence factors (such as the *coa* and *clfA* genes), which include various exotoxins and proteins associated with the cell surface. This could contribute to an increased udder pathogenicity of the organisms and have implications for food hygiene, especially in instances of subclinical mastitis caused by *S. aureus.* **(Kalorey *et al.,* 2007).**

According to numerous studies, the most common Gram-negative coliform responsible for environmental clinical mastitis is *E. coli.* When it invades mammary tissue, it produces a number of virulence factors **(Alawneh *et al.,* 2020; Campos *et al.,* 2022).** The association between this pathogen virulence factors and mastitis has been established **(Guler and Gunduz, 2007)**. Humans are known to suffer from severe acute illness and chronic side effects when exposed to Shiga toxin-producing *E. coli* (STEC) **(Chern *et al.,* 2004).** *Streptococcus uberis* is responsible for approximately one-third of all instances of intramammary infections in cows, which invades the teat channel following injury or milking **(Abureema *et al.,* 2014; Monistero *et al.,* 2021)**. According to **Cvetni´c *et al.* (2022),** a research study that pinpointed Mycobacteria as the source of mastitis uncovered two strains resistant to clarithromycin: Mycobacterium fortuitum II and Mycobacterium margeritense.

Invasion, infection, and inflammation are the three phases that make up the development of mastitis. The phrase "invasive stage" denotes the portion of the teat canal extending from the end of the teat to the milk. The stage of infection is when the germs proliferate quickly and infiltrate the tissue of the breast. During the inflammatory stage, the milk exhibits both gross and subclinical abnormalities. Additionally, there are mild to severe systemic effects and varying degrees of clinical abnormalities in the udder **(Radostits *et al.,* 2007)**.

The entry of bacteria into the mammary gland triggers the inflammatory response, which serves as the body's second line of defense. The intensity of the inflammatory response can be affected by various factors, including the pathogen responsible for the infection, the cow's age and lactation stage, her immune status, genetic background, and nutritional condition. These bacteria reproduce and generate toxins, enzymes, and cell wall components that prompt inflammatory cells to release additional mediators of inflammation **(Harmon, 1994)**.

Polymorphonuclear neutrophils (PMN), as both leukocytes and phagocytes, exit the bone marrow toward the invading bacteria, attracted in large numbers by chemical signals (chemotactic agents) from damaged tissues. PMN masses moving from milk-producing cells into the alveolar lumen can cause damage to secretory cells and an increase in the somatic cell count (SCC). The majority of somatic cells are PMN. At the location of the infection, PMN encircles the bacteria and discharges enzymes capable of destroying the organisms. The leukocytes in milk generate specific substances to draw additional leukocytes to the site of infection as a means of fighting it. Once the bacteria are eliminated, a considerable number of somatic cells persist until the gland heals. Leukocytes and blood clotting factors can come together to create clots that obstruct small channels and hinder the complete evacuation of milk. In certain instances, harm to epithelial cells and blockage of small channels may lead to scar tissue development, permanently impairing the gland ability to function. In other situations, tissue repair, inflammation reduction, and function restoration may take place during that lactation or the one after **(Harmon, 1994)**.



Diagnosis

Qualitative Milk examination: Quinn *et al.* (1994) state that the presence of pus (yellow) or blood (red or brownish) can alter milk color. A thicker, "sticky" milk could be produced by increasing the consistency, or it could be more watery than usual. Always abnormal are clots and flakes. Mastitis can also change the secretion's quality of smell. Additionally, constant evaluation (visualization and palpation) of the udder, particularly while milking, is the main way to prevent mastitis. When a person has clinical mastitis, their udder may appear red, firm, and warm to the touch. The cow may experience pain when the udder is palpated. According to **Mulugeta and Wassie (2013),** these symptoms demonstrate how the gland's vascularity and blood flow alter when it becomes inflamed. It is also essential to visually examine the milk while it is being extracted to detect any significant alterations in its composition, including flakes, clots, or serous, blood, or watery substances. Moreover, it serves as the conventional approach for identifying clinical mastitis when these changes are evident. At the start of milking, the first few squirts of milk from each quarter are collected in a strip cup to check for any flakes or clots. During a dry spell, milk becomes extremely fluid, and its composition changes as well. The appearance of udder discharge is typically irregular at times in chronic cases. Acute mastitis causes a significant alteration in the secretion **(Chiara *et al.* 2023).**

Field test:

1. California Mastitis Test (CMT): The CMT is a straightforward, reliable screening tool for subclinical mastitis that can be used in all situations. It was designed to assess quarter milk samples, both individual and bulk. To obtain accurate values, the CMT can analyze fresh milk that has not been refrigerated for a maximum of 12 hours and refrigerated milk for a maximum of 36 hours. The test merely indicates whether the cell count is high or low and aids in determining the level of infection in each quarter, rather than providing an overall result for the udder. As part of the test procedure, approximately 2 milliliters of milk is to be squeezed from each quarter, and an equal volume of CMT solution is then to be added to each cup in the plastic CMT paddle as outlined by **National Mastitis Council (1990).** Once the ingredients are fully combined by circularly rotating the paddle, the CMT reaction must be scored within 15 seconds; otherwise, weak reactions will vanish. The CMT employs a detergent and a pH indicator known as bromoserol purple as reagents. The number of somatic cells in milk is determined by the intensity of the reaction between the detergent and the DNA of cell nuclei.

2. Strip Cup or Plate Test: This supplementary cow-side examination can be used to detect clinical mastitis by searching for visible milk particles. It is a useful and efficient way to determine which cows have clinical mastitis. This strip cup can be used by any layperson. In order to make it easier to view the milk flakes when the cups are tilted at an angle, the bottom of the enamel plate containing the four strip cups is black. Runny, discolored, or containing clots, flakes, or shreds are typical characteristics of dysfunctional milk **(Radostits *et al.* 2007).**

Treatment

To be effective, mastitis control programs should incorporate the strategic use of antimicrobials to lower antibiotic residues in milk and combat antimicrobial resistance. They should also focus on early infection detection through an understanding of pathogenesis, the creation of new sensitive early screening tests, and the implementation of good management practices to minimize transmission and protect uninfected individuals **(Ruegg *et al.* 2017a)**.

While not all herds achieve lower SCC and high milk production, treating heifers prepartum provides a significantly improved cure rate, no milk loss, and minimal risk of antibiotic residues **(Borm *et al.* 2006).**

Effective treatment of clinical mastitis requires antimicrobial therapy, identification of the causative agent, consideration of parity and lactation stage, assessment of previous SCC history, and evaluation of clinical mastitis and other systemic disorders **(Steeneveld *et al.* 2011).**

The cornerstone of mastitis treatment is antibiotic therapy, which includes both therapeutic and preventive measures. But newer methods of treating mastitis include using natural remedies like propolis and zeolites, which may be a better option than antibiotic treatment **(Benić**

***et al.* 2018)**.

Antibiotic therapy: The selection of antibiotics for mastitis treatment should rely on sensitivity and culture results rather than on empirical therapy **(Tiwari *et al.* 2013).** In the currently approved methods for treating clinical mastitis caused by Gram-positive agents, targeted antimicrobial therapy focuses on particular organisms. The treatment plans allow the remaining instances sufficient time for self-healing **(Ruegg 2017b).** The clinical cure rate improves with combination therapy involving antibiotics administered through multiple methods, including systemic and intramammary **(Wei and Sung 2020).** This could be because milk and mammary tissues have higher concentrations of antibiotics **(Lima *et al.* 2018).**

Current directions in non-antibiotic research: In recent years, there has been a considerable uptick in research focused on identifying non-antibiotic treatments for bovine mastitis **(Lara *et al.,* 2024).** As per the literature, most of them utilize biomimetic techniques, taking cues from the methods organisms use to maintain homeostasis, defend themselves, or survive. Some of these options include probiotics, bacteriocins, phytochemicals (such as plant extracts and essential oils), and bacteriophages. Moreover, recent studies have explored new options such as nanoparticles and antimicrobial peptides that do not originate from bacteria **(Angelopoulou *et al.* 2019 and Zaatout, 2022).**

Control and prevention

It has been suggested that mastitis management and therapy should adhere to the same guidelines that a surgeon would when performing surgery. Therefore, the following essential components must be taken into account: cuddling, treating mastitis during the non-lactating period, teat dipping after milking, using proper husbandry techniques and hygiene, and culling chronically sick animals. This method involves several specific steps: hand washing with soap and water, sanitizing the udder and teats, using individual towels to ensure complete drying, dipping the teats in an effective germicidal teat dip for 30 seconds before removing it with a separate towel, and cleaning the teat end thoroughly with a cotton swab soaked in alcohol. If all four quarters are being managed, the teat that is farthest from the closest should be cleaned first. To avoid contaminating the clean teat ends, start with the teats closest to the milkers, then move on to those that are farthest away. After therapy, immerse the teats in a teat dip with effective germicidal properties **(Smith and Hogan, 1995).**

Management

Genetic selection: Due to the considerable genetic variation among individual cows, mastitis resistance can be enhanced through genetic selection. Consequently, sire selection can be used to increase resistance, improving herd health over the long run **(Weigel and Shook 2018).**

Nutrition: The animals diet and the mammary tissues ability to withstand infection are more closely related. This is attributed to nutrients' capacity to provide antioxidant function, which strengthens the immune system defenses against infections **(Erskine 1993).** Trace elements such as selenium, copper, and zinc, as well as vitamins like vitamin A/b-carotene and vitamin E, can affect udder health **(O'Rourke 2009).**

Dry cow therapy and lactation therapy: The two methods of antibiotic treatment for mastitis are dry cow therapy and lactation therapy. Using antibiotics to treat mastitis during the lactation phase is known as lactation therapy **(Tiwari *et al.* 2013).**

Conclusion

Programs for controlling mastitis that are effective focus more on prevention than on therapy. As of right now, mastitis management regimens still include antibiotic treatment as a standard component. Although antibiotics are often used alongside other medications, their effectiveness remains inadequate. Consequently, it is crucial to seek out new treatment alternatives. A variety of natural substances from plants, animals, and microbes have been researched and demonstrated to be effective in treating bovine mastitis. Before any commercial applications, field research should be considered to verify the efficacy of the alternative medications.

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