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Bovine Mastitis: Epidemiology, Diagnosis, and Control Strategies in Dairy Sectors of Bangladesh

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Abstract

MASTITIS is a common and highly-cost disease of various animals, which poses substantial challenge and serious implications to dairy sectors of different countries including Bangladesh. This review describes the effects of bovine mastitis among both clinical and subclinical forms, which leading to udder illness, decrease in milk quantity and quality, and severe economic losses. Subclinical mastitis is frequently overlooked due to the absence of visible clinical symptoms but poses significant challenges as it lead to considerable economic and productivity losses. In Bangladesh, the disease is intensified by elements like insufficient farmer awareness, poor hygiene practices, and the population of high-yielding crossbred cows, which exhibit greater vulnerability to infections. Investigations indicate a notable occurrence of subclinical mastitis in different areas of the country, with infection rates differing according to factors like lactation stage, parity, and breed. The economic impact encompasses diminished milk output, heightened veterinary expenses, early culling of livestock and public health issues associated with antibiotic residues and the transmission of pathogens via milk. Effective control strategies include enhanced management practices, routine screening through methods such as the California Mastitis Test, implementation of biosecurity measures, and focused treatments. Furthermore, the implementation of vaccination initiatives and the education of farmers are essential for the effective management of disease sustainability. This review highlights the critical necessity for a unified strategy in addressing mastitis control, merging scientific progress with effective on-farm practices to protect Bangladesh's dairy sector from this ongoing challenge.

Keywords: Milk, Cow, Udder health, Lactation, Prevalence

Introduction

Mastitis is a disease characterized by swelling, heat, redness, hardness, pain, and aberrant milk due to physiological, chemical, and cellular changes in milk and pathological changes in udder tissue [1, 2]. The most common dairy production disease is mastitis. According to the livestock economy assessment of 2023-2024, livestock can provide 1.82% of our gross domestic product (GDP) and mastitis causes losing millions of litters of milk. It estimated that each individual must drink 250 ml/day of milk, but the supply is reduced to 193.38 ml/day if the mastitis uncontrolled [3]. Untreated mastitis in dairy herds has substantial economic effects due to decreased milk quantity and quality, and hygiene. Mastitis also negatively impacts the days of first heat after parturition and calving interval [4, 5]. Clinically, there were clinical and subclinical mastitis. Clinical mastitis characterizes by clinical signs, while subclinical is not, making it more economically important. Milk pockets like Sirajganj (42.7%) have a higher mastitis rate than non-milk pockets [6, 7]. Mastitis affects the economy greatly. Epidemiological studies suggested that mastitis costs INR1390 per lactation, with 49% related to milk supply reduction and 37% to veterinary expenses in

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India. Crossbred cows and buffaloes lost INR 700 and INR 364 due to milk output reduction [8, 9]. Livestock GDP is 82,014 Crore Taka, and 20% and 50% of people depend on it directly and partly, respectively [3]. We will not meet our milk and nutrition needs if we cannot control economic diseases like mastitis. This review underlines the essential need for an organised strategy in mastitis control, integrating scientific advancements with efficient on-farm approaches in order to protect Bangladesh's dairy industry from this persistent issue.

Methodology

Information Collection

To put together this review, we gathered data from a wide range of reliable sources. These included scientific journals, reports from government and research organizations, and key guidelines from the World Organization of Animal Health (WOAH). We also used popular databases like PubMed, Scopus, Web of Science, and Google Scholar to dig deeper into the topic. For our searches, we combined keywords like "Bovine Mastitis," "Epidemiology and Diagnosis of Bovine Mastitis," "Control Measures for Bovine Mastitis," "Pathogenesis of Bovine Mastitis," and "Public Health Implications of Bovine Mastitis." Our main focus was on studies published between 2000 and 2024 to make sure the information we included was up-to-date and relevant.

Literature Review

We took a systematic approach to reviewing the available literature, aiming to cover as many aspects of bovine mastitis as possible. This included looking into epidemiological data, lab studies, case reports, and research on how the disease develops. We also reviewed national and international outbreaks, surveillance data, diagnostic tools, and control methods. By doing this, we were able to map out trends, identify recurring patterns, and highlight any unexpected findings in how bovine mastitis spreads, progresses, and can be managed. This approach helped us pull together a clear and balanced view of the disease, both in areas where it's common and in regions where it's not as prevalent.

Inclusion and Exclusion Criteria

To ensure our review stayed focused and relevant, we followed specific criteria for what to include and what to leave out. We included studies that directly discussed bovine mastitis, including its transmission, clinical signs, and economic impact. On the flip side, we excluded studies that weren't in English, duplicate reports, or anything not directly linked to bovine mastitis. We prioritized more recent studies (2000–2024) so that we could present the latest findings and developments in the field.

Importance of Mastitis in Dairy Industry

Bangladesh's dairy self-sufficiency is threatened by mastitis, a severe production-related disease of dairy cattle [10]. Intramammary infections of dairy cows with Gram-positive bacteria like *Staphylococcus aureus* (a major cause of mastitis) have garnered attention due to their financial impact on the dairy farm due to production losses caused by a rise in somatic cell count [11,12]. Mastitis, one of the most frequent diseases in dairy cows, lowers milk production and necessitates early culling, causing major economic losses [13]. Mastitis is the most common cause of early dairy animal slaughter, sometimes before peak production age [14,15].

Economic losses

Mastitis is a substantial economic loss for production animals. Decreased milk production, quality, abandoned milk, premature culling, mortality, labor, veterinary services, diagnosis, and treatment are important. Consider other costs like danger of other diseases and materials and investments for mastitis management. Mastitis economic losses have been estimated using several methodologies, however modeling approaches, farm variability, region, and inputs have led to differing reported statistics. Huijps *et al.* [16] built a statistic tool utilizing data from 78 Dutch farmers to calculate mastitis cost factors. The lactation month affected the average economic loss per mastitis case, which was €210. Production losses made up 71%, animal culling 16%, and veterinary activities 1%. Results ranged from €65 to €182 per animal and year. Farmers assessed economic losses at €78/cow per year, showing they overestimated mastitis' economic impact [16,17]. In another study, Bar *et al.* [18] used a dynamic programming model with data from 5 big New York herds (600–1,200 milk cattle). The animals were monitored for 24 months (2004–2006). Results showed that mastitis cases cost $179 and $71/animal per year, with 64.2% going to milk losses, 7.8% to mortality, and 27.9% to treatment. Nielsen *et al.* [19] studied a Swedish herd of 150 animals using a biodynamic model and found a €278 average cost per clinical mastitis case. Recently, Rollin *et al.* [20] estimated the impact of clinical mastitis in the first 30 days of breastfeeding using a deterministic partial budget model. Model inputs used a 1,000-animal US herd and 2012–2014 timeframe. Clinical mastitis cost $435 on average. The cost of future production losses was 28.7%, early culling and animal replacements 41.8%, treatment 8.3%, mortality 7.4%, discarded milk 5.7%, labor 4.8%, and veterinary services and diagnostics 3.2% [20]. Results show that mastitis has a global economic impact, notwithstanding some variances. Mastitis reduces dairy productivity in Bangladesh. Mastitis costs Bangladesh's dairy industry Tk. 122.6 (US $2.11) million annually [21]. Many studies have examined Bangladesh's SCM status and that of India, Sri Lanka, and Pakistan.

SCM is found in 28.5–61.3% of crossbred dairy cows in Bangladesh [22].

What is Mastitis

Mastitis means udder inflammation and comes from the Greek word "mastos" for udder. Mastitis is mammary gland inflammation that causes physical, chemical, and bacteriological alterations in milk and pathological changes in glandular tissue [22]. Mastitis, caused by physical, chemical, or infectious agents, affects milk production and causes pathological changes in the udder, such as swelling, pain, edema, and fibrosis, as well as changes in milk's physiological, chemical, and bacteriological properties [23]. The pathogen and host parameters such lactation stage, age, cow immunity, genetics, and nutrition determine mastitis severity. Coagulase-negative staphylococcus *Staphylococcus aureus* and other bacteria lower lactose, fat, protein, and milk urea nitrogen in mastitis milk. Immunological reactions to pathogen invasion cause intramammary infection and inflammation [24].

Types of Mastitis

Clinical (per-acute, acute, and sub-acute) and subclinical bovine mastitis exist. Today, specialists divide mastitis into three types: sub-clinical, clinical, and chronic, depending on causative organisms, breed, age, immunity, and lactation stage. Histopathological results divided mastitis into seven patterns: mixed, lymphoplasmacytic, suppurative, pyogranulomatous, Abscedative, Necrosuppurative, and granulomatous [13].

Clinical mastitis

Clinical mastitis symptoms include redness, heat, inflammation, pain, loss of appetite, increased body temperature, swollen quarters, decreased feed intake, milk yield reduction, milk composition changes, and other behavioral abnormalities [25]. In severe cases, teat secretions include flakes or blood clots. Clinical mastitis infections affect profitability because to treatment costs and productivity losses [26].

Subclinical mastitis

Subclinical mastitis is an infection without local or systemic inflammation. Sub-clinical mastitis (SCM) still costs the dairy industry. Parity, farming system, milking area, geography, and herd strongly affect subclinical mastitis prevalence [27]. SCM causes lower milk quality, particularly in young animals, poor product hygiene, and undesired milk composition changes, which contribute to animal suffering. Sub-clinical mastitis affects milk output and hinders livestock economics worldwide. Farms struggle with subclinical mastitis, which can be 20 times more common than clinical mastitis because the milk, udder, and cow appear normal. Thus, standard eye monitoring cannot detect subclinical mastitis [28]. The economic impact of subclinical mastitis is difficult to evaluate because many of its effects are indirect and not visible. Thus, subclinical mastitis can greatly impact profitability due to the following: (i) it often goes unnoticed for a long time; (ii) it lowers a cow's productive potential by gently decreasing milk yield during and after an infection; (iii) it can increase bulk tank SCC if enough cows are affected; and (iv) it is often caused by contagious pathogens that can spread easily from cow to cow. Subclinical mastitis, an expensive ailment, affects all dairy herd sizes. Targeted milk culture tests, routine SCC monitoring, and excellent milking procedures can reduce its effects on the herd. Lakew says many germs can cause cow mastitis. Contagious and environmental mastitis are bacterial diseases that differ in origin [29].

Contagious mastitis

Contagious mastitis can spread between cows, especially while milking. Contagious bacteria including *Staphylococcus aureus* and *Streptococcus agalactiae* and rare species like *Mycoplama bovis* and *Corynebacterium infiltrate* and develop into the teat canal on the cow's udder and teat skin [30]. These can produce subclinical infections that raise SCC. SCCs, which contain leukocytes like neutrophils, macrophages, lymphocytes, and erythrocytes and epithelial cells, indicate IMI infection. Reducing reservoir-uninfected cow contact reduces infectious diseases. Maintaining milking equipment, disinfecting teats after milking, culling, and dry cow therapy (DCT) minimize infectious diseases [31].

Environmental mastitis

Streptococcus spp. (such as *Strep. uberis*), coliforms (*E. coli, Klebsiella, Enterobacter*), *Pseudomonas*, and others can cause environmental mastitis [32]. Environmental pathogens, unlike infectious diseases, live in herd bedding and housing rather than cow udders and teats. Opportunistic pathogens wait for a host to infect. They can enter the teat if the liner slips during milking or the cow's immunity is inadequate, causing clinical mastitis. Environmental pathogens including *E. coli* and *Streptococcus uberis* enter and thrive in the cow's udder, triggering an immune response and being promptly eliminated by the immune system. Environmental infection can be controlled by reducing teat end exposure to pathogens and boosting cow IMI resistance through antibiotics and immunization [33].

Epidemiology

The following points are some causes that might lead to mastitis in animals [34]:

- Age: Infection rates rise with age, reaching a peak at 7 years old.
- Herd size: larger herds have a higher incidence.

- Breed: Crossbred and exotic cows are more likely to be affected than zebu cows. The incidence has been observed to be higher in Holstein Friesian cows than in Jersey cows.
- Stage of breastfeeding: The first and last stages of lactation have higher infection rates.
- High-yielders of milk are more frequently impacted than low-yielders.
- Nutrition: A high-protein diet may operate as a risk factor. Selenium, vitamin A, and vitamin E could all play a role in mastitis resistance.
- Milking frequency and udder structure: An increased risk of intramammary infection has been linked to a high milking frequency and a large teat canal diameter. Differences in mastitis are also assumed to be related to variations in udder depth, teat length, shape, and orifice morphology.
- Hygiene: Poor sanitation and hygiene contribute to the growth of microorganisms.

Transmission of Bovine Mastitis

Sharing milking equipment or direct touch spreads bovine mastitis pathogens across cows. Environmental contamination from bedding, manure, water, or polluted intramammary preparations is another prominent source. Poor farm cleanliness and handling cause epidemics. Soil-borne viruses like Nocardia can infect cows through contaminated preparations or ambient exposure. Environment-borne bacteria can enter the mammary gland (Figure 1) [13]. Dogs, cats, and even humans can spread the disease to cows [35].

Etiology

Although many infectious agents like bacteria, mycoplasma, fungus etc. have been implicated as causes of mastitis but the only significant cause of mastitis is bacteria (Table 1).

Microbiome diversity and bovine mastitis

The current microbiome study used bioinformatics to show significant differences between CM and H milk's microbial communities. PS and MR studies showed that Proteobacteria, Bacteroidetes, Firmicutes, and Actinobacteria made up 96.51% of the sequences (U test). The top 40 bacterial species' relative abundances were compared between the CM and H cohorts using PS and MR. The most diverse phylum was Proteobacteria, which includes *Acinetobacter, Pseudomonas, Escherichia, Vibrio, Erwinia*, and *Pantoea*. *Streptococcus, Enterococcus, Staphylococcus*, and *Bacillus* dominated *Firmicutes, Chryseobacterium, Porphyromonas*, and *Prevotella* dominated Bacteroidetes, and *Corynebacterium* dominated *Actinobacteria* [38]. *Acinetobacter* (60.14%), *Campylobacter* (10.93%), *Pantoea* (0.66%), *Klebsiella* (0.63%), *Kluyvera* (0.42%), *Salmonell*a (0.31%), *Enterobacter* (0.30%), *Shewanella* (0.30%), *Escherichia* (0.28%), *Citrobacter* (0.15%), and *Bacillus* (0.10%) had higher mean relative abundance in CM samples, while the other genera had lower mean abundances (<0.10%). The H milk metagenomes showed higher mean relative abundances of Acinetobacter (52.90%) in PS and MR pipelines, followed by Pseudomonas (22.81%), Micromonospora (10.57%), Eubacterium (5.37%), Catenibacterium (2.12%), and Ralstonia (0.12%). The remaining genera had much lower abundances (<0.10%). MR found more microbial genera than PS, however both techniques found 98.00% of the overall microbial abundance as shared genera [39].

Pathology of Mastitis

Histopathological features characterized mastitis as mixed, lymphoplasmacytic, suppurative, pyogranulomatous, Abscedative, Necrosuppurative, and granulomatous [13]. *S. aureus* and *Nocardia* spp. destroy the secretory mammary epithelium and replace it with fibrous connective tissue (Table 2).

Global distribution of subclinical and clinical mastitis

The total prevalence of SCM and CM was 42% and 15% worldwide, 45% and 18% in India. SCM was more common in North America and Europe, whereas Uganda and the UK had higher SCM rates. Buffaloes have a greater SCM and CM frequency than cattle. Ethiopia had the most SCM and CM prevalence studies (63 and 55), followed by Bangladesh, Pakistan, Egypt, and the US [40]. SCM has the greatest frequency estimated at 42% worldwide, followed by CM at 15%. This showed that dairy cattle and buffaloes' worldwide value SCM above CM. The meta-analysis of 1995–2014 research in India found a prevalence of 46.35% [41], which was higher than the present study. The SCM prevalence in cows between 15 to 75%, which supported the present findings. The SCM prevalence estimate matched Bangladesh [42] and Sri Lanka [43]. As previously reported, SCM prevalence in Bangladesh was 29.5% and 20.2%, which was low for this study [44].

Bovine mastitis risk factors

Pathogen, host, and environmental variables affect bovine mastitis risk. Mastitis management strategies considered all these factors and detailed descriptions in Table 3.

(a) Factors Associated with Clinical Mastitis

Milk output is linked to teat canal diameter and stretch. The higher teat canal of high-yielding cows stays open longer after milking, increasing the risk of mastitis caused by environmental pathogens like *E. coli*. Local-breed cows show lower mastitis affinity than higher-yielding cross-breeds in tropical regions (high temperature and humidity) and intensive farming practices [66]. According to Sinha *et al.* [6], lactating cows producing 1-5 and 6-10L milk had considerably greater milk output than those producing 16-20L or more milk. In cows with reproductive and periparturiant disorders, mastitis was more common. Mastitis prevalence is greater in rainy season than winter or summer (3.47%, 11.52%) and 5% in winter [42]. Lactating calves with BCS scores >2.5–3 are more susceptible to SCM than weak bodies [67]. Other farm-level variables including cow hygiene, farmer knowledge, and herd size can also cause mastitis.

(b) Factors Associated with Sub-Clinical Mastitis

The most common farm-level SCM risk factors include unhygienic conditions, big udders, teat injuries, udder wounds, filthy milkers' hands, and inappropriate milking machine management. Breeding, teat shape, and body condition score (BCS) are controllable risk factors, however age, parity, lactation stage, and housing are not. Other sources say the biggest risk factors for mastitis include teat end-to-floor distance, pregnancy, milk output, milking stimulation, milk leakage, and floor type [66,67].

Clinical signs of clinical mastitis

Clinical mastitis causes aberrant milk (color, clots, flakes), mammary gland (tissue color, swelling), and animal state [68].

Clinical signs of sub clinical mastitis

Clinical symptoms are absent in subclinical mastitis. Milk quality and production yields frequently suffer [68]. In subclinical mastitis, an inflammatory reaction can only be detected by testing, and the milk has a high somatic cell count without any obvious abnormalities. As this type of mastitis appears normal with no obvious abnormalities, such as udder swelling, hardness in the affected area, soreness, or watery milk, the milk undergoes physical and chemical changes that help diagnose SCM by several diagnostic procedures [69].

Clinical signs of gangrenous mastitis

Gangrenous mastitis is caused by *Staphylococci, Bacilli, Clostridium perfringes*, and *E. coli*. Gangrenous mastitis begins with severe acute inflammation, heat, redness, swelling, and pain, and progresses to necrosis with coldness, blue-black color, fluid exudation, and crepitation (Figure 2) [70].

Methods of Diagnosis of Mastitis

Mastitis can be clinical or subclinical depending on inflammation degree. Clinical mastitis causes aberrant milk (color, clots, flakes), mammary gland (tissue color, swelling), and animal state. In contrast, subclinical mastitis has no symptoms. Milk quality and production yields frequently suffer [68]. Detailed diagnostic outlines are described in Figure 3.

Physical Examination of Animal and Milk Sample

Udder condition, body temperature, pulse rate, hunger, and posture are frequently visually evaluated in physical examination. Kelly [71] and Samad [22] outline procedures for examining suspicious cow bodily parts and systems.

Inspection of udder

A naked inspection and probing of suspected farm cows' udders can reveal skin abrasions, hair loss, fibrosis, mammary teat consistency, supra-mammary lymph node enlargement, dermatitis, traumatic, udder cleft, open wound, etc. Lactating cows' udder lesions are thoroughly evaluated in selected herds. Cows are assessed by udder lesion increase. The following grading criteria are utilized for ventral abdomen observation. Grade 0: No udder lesions; Grade 1: Discoloration or moist appearance; Grade 2: Hair loss or skin abrasion; Grade 3: Closed skin crusts or Papillomatosis; Grade 4: Open wound, no skin; and Grade 5: Open wound and bloody, serous, or purulent exudates. Naked eye and organoleptic testing were performed on suspicious milk samples immediately after collection.

Surf Field Mastitis Test (SFMT)

A cheaper and easier alternative to the animal-side subclinical mastitis diagnostic test is the SFMT. The Surf field mastitis test (SFMT) employs 3% Surf Excel (Unilever), a common household detergent. Surf Field Mastitis Test and Interpretation- Make a 3% Surf Excel (Unilever) solution. Mix 5–6 tablespoons Surf Excel powder with 1–2 liters of ordinary water. Seal the solution-filled plastic bottle and put it somewhere dark. Allow three months for this reagent. Transfer 10–15 ml of milk per teat to a tea cup. A Surf Field Mastitis Test paddle can collect milk from each cow and buffalo quarters, eliminating the requirement to collect milk samples in tea cups. Add 10-15 ml of 3% Surf solution to one teat's milk and stir it approximately equally. Rotate milk and Surf solution for 15–20 seconds. Check the mixture for thickening or other changes. If subclinical mastitis is present in the udder quarter, the milk-surf solution combination will gel within 15 seconds. If milk and Surf solution do not thicken, the udder has no subclinical mastitis [72].

White Side Test (WST)

WST serves as a method for detecting subclinical mastitis. Five drops of suspected milk samples are well mixed and placed on a glass slide with a dark background, followed by the addition of 20 µl of 4% NaOH solution. The mixture was stirred for 20-25 seconds using a sterile toothpick. For results, we can adhere to the protocol established by Kahir [73].

California Mastitis Test (CMT)

Following cultural isolation and SCC, CMT emerged as the test demonstrating the highest level of accuracy. In contrast to laboratory tests such as culture isolation and SCC, which require sufficient laboratory space and qualified personnel, CMT serves as a dependable diagnostic method for routine mastitis screening in the field Badiuzzaman *et al.* [74]. SCM was identified through the CMT, which indicated the presence and severity of the condition. Pyorala [75] outlines the methodology of CMT. A white plastic paddle featuring four shallow cups labeled A, B, C, and D is initially utilized, followed by the addition of approximately 3 mL of milk and CMT reagent. The paddle is rotated in a circular motion for approximately 10 seconds to ensure thorough mixing of the content. The formation of gel indicated the results. The reactions were observed and recorded. The results were interpreted according to the following criteria: N = Negative, No infection = No thickening of the mixture. T = Trace; Possible infection = Slight thickening of the mixture; Thickening resolves with continued rotation. 1 indicates a weak positive correlation. Infected: distinct thickening of the mixture; no gel formation. 2: distinct positive. Infection results in immediate thickening of the mixture and slight gel formation; a score of 3 indicates a strong positive response. Infection results in gel formation, leading to an elevation of the mixture's surface.

Histopathological Investigations

Mastitic cattle have greater neutrophil and total milk somatic cell counts than healthy cattle, but lower macrophage and lymphocyte counts. Mastitic animals had reduced alveolar diameter, number, and epithelial cell population. Infected udder parenchymal tissues demonstrate alveolar loss and fibrous tissue growth. Cellular infiltration is exclusively found in the teat cistern lining, gland cistern, and deep parenchyma. Dissecting the mastitic udder shows mild, moderate, or severe alveolar atrophy and cellular exudate in certain mastitis instances. Acute inflammation in 56% of mastitic patients and persistent inflammation in 44% [76, 77].

Somatic cell count

A single test may not accurately identify SCM, however, combining the CMT and SCC may be more efficient. Most typically, an SCC value of ≥200,000 cells/mL is used to indicate subclinical mastitis [75]. SCC values below 200,000 cells/mL in composite milk samples indicate IMI-free mammary glands [78]. However, this criterion assumes flawless culture testing and does not account for false-negative findings. Research comparing SCC and culture to identify IMI suggests a lower threshold of 150,000 cells/mL, which might misclassify culture. If differential cell counting is used to diagnose mastitis, an SCC value of 100,000 cells/mL may be better for each quarter [79].

Prevalence of mastitis in different areas of Bangladesh

According to the California Mastitis Test (CMT), 20–44% of Bangladeshi cows have SCM. Mastitis prevalence in Bangladesh varies by geography [67].

Sirajgonj district: Mastitis prevalence in Ullapara upazila, Sirajgonj district, is 42.7%. The highest prevalence is in Sahiwal cross (47.4%), followed by Friesian cross (39.7%) and Local Zebu (37.6%). Cows in the 5th-7th parity have the highest prevalence (62.5%) compared to 1st-2nd parity. Lactating cows with 1-5 and 6-10L milk production had significantly higher ($P<0.05$) milk production than those with 16-20L or more milk [6].

Barishal: Subclinical mastitis is 28.50% in lactating cows, significantly higher in crossbreds (45.28%) than local zebu (22.45%), 32.50% in mid lactation (90-180 days), highest in 7th parity (42.86%), and most common isolates are Staphylococcus spp. (73.33%), Streptococcus (33.33%), and Escherichia coli (6.67%) [80].

Sylhet: Reproductive and periparturient diseases caused more mastitis in cows than normal individuals at Ullapara Jaintapur of Sirajgonj Sylhet, 7.15%. Mastitis prevalence is greater in rainy season than winter or summer (3.47%, 11.52%) and 5% in winter [42].

Rangpur, Mymensingh, and Satkhira: Total SCM prevalence was 20.2%. Mastitis was most common in cross-bred cows aged 4-8 years, with 49.5% affinity. Lactating calves with BCS scores >2.5–3 are more susceptible to SCM than weak bodies [67].

Brahmanbaria: CMT confirmed 28.75% SCM prevalence. The lactation stage affects prevalence. SCM detection is 34.14 percent in early lactation, 22.85 percent in mid-lactation, and 25.00 percent in late lactation. SCM prevalence varied with parity: 18.18% in first parity, 28.63% in second, 32.63% in third, 32.25% in fourth, 13.33% in fifth, 22.22% in sixth, and 20% in seventh [67].

Dhaka: 57% of cows had mastitis. SCM was most common (85%) followed by clinical mastitis (15%). Due to their genetic condition, high-yielding dairy cows are more susceptible to heat stress and pathogens due to weakened immune systems in hot, humid environments [81].

Southwestern part: SCM was 71.9% on farms, 67.9% on animals, and 29.5% on quarters. Farm and farmer demographics, sampled population characteristics, and management were described by descriptive statistics [22].

Mastitis vaccines in dairy cows

Mastitis treatment and prevention have been used for years to enhance dairy cow health, welfare, and output. Common udder pathogen vaccinations have been developed in recent decades. Most *E. coli* vaccines employ the mutant core antigen J5, although *S. aureus* and *S. agalactiae* vaccines use complete organisms (cellular lysates, inactive, and attenuated vaccines) or components (toxins, surface proteins, and polysaccharides) [82]. Immune cells from healthy, diseased, or vaccinated mammary glands are used to develop effective mastitis vaccines. *E. coli* J5 Mastitis Dry-season or early-lactation vaccination reduces clinical mastitis [83]. Vaccination around parturition boosts dairy cow immunity. The *E. coli* core antigen vaccination has decreased severe coliform mastitis after calving [84]. Recombinant *Staphylococcal enterotoxin* Type C mutant vaccine (MastaVac) produced measurable antibodies against experimentally induced mastitis in cows 4 weeks after inoculation. Vaccinated cows had lower SCCs. The MastaVac protects cows from S. aureus intramammary infection during lactation and improves udder health [82]. Immunity to a pathogen from vaccination. Vaccine quality and timely immunization of endangered populations improve vaccine prevention. Marketing, autogenous (herd-specific), and experimental mastitis vaccines were common. Vaccines were mono or polyvalent based on the number of targeted pathogens [85].

(a). Commercial Vaccines

The commercial polyvalent vaccination is Startvac, Hipra, Spain. Startvac-vaccinated dairy cows produce higher milk and milk solids and have less clinical mastitis. Startvac reduced clinical mastitis originating from *S. aureus* and coagulase-negative Staphylococci. MastaVac vaccination protects against *S. aureus* and improves udder health [86]. Recombinant *Staphylococcal enterotoxin* Type C mutant vaccine (MastaVac) produced measurable antibodies against experimentally induced mastitis in cows 4 weeks after inoculation. Vaccinated cows had lower SCCs. The MastaVac protects cows from S. aureus intramammary infection during lactation and improves udder health [87].

(b). Herd-specific Autovaccines

A herd-specific autovaccine (Best Vac) reduces *S. aureus* mastitis and improves milk output in vaccinated dairy cows. Combination therapy (herd-specific autovaccine alone or with cefuroxime intramammary antibiotic therapy) eliminated S. aureus infection in a herd with high subclinical mastitis. Clinical mastitis was absent in dairy herds after 2 years of immunization. Another study found that a herd-specific autovaccine made from S. aureus JR3 cells and SM capsule of strain 2286 reduced subclinical and clinical mastitis in cows. A trivalent vaccination increased serum antibody titer, CD4+ and CD8+ lymphocyte percentages, and neutrophil phagocytosis activities including S. aureus capsular polysaccharide type 5 (T5), 8 (T8), and 336 (T336). This whole-cell trivalent vaccination induces a protective antibody response against S. aureus' three capsular polysaccharide antigens. Vaccine alone is ineffective and costly on high-mastitis dairy farms. For considerable reduction in intramammary infection incidence and duration, vaccination and other infection control treatments and prophylactic measures are needed. Infection management includes good milking cleanliness, clinical treatment, isolation, and culling affected cows. However, immunization may prevent mastitis [87].

Treatment of Subclinical mastitis

Successful cow mastitis management methods include antibiotics. Alternatively, dry cow treatment is used to manage mastitis in all cattle on 73% of U.S. dairy farms [82]. Rational antimicrobial treatment balances milk quality and yield against veterinarian services, farm labor, pharmaceutical purchases, and rejected milk in subclinical mastitis [88]. Subclinical mastitis is often treated after lactation (dry-cow treatment at the onset of the dry season). Milk bacteriological culture or SCC measurement can detect subclinical mastitis. The culture of milk from postpartum or high SCC cows may be used as a surveillance tool to identify common organisms causing subclinical mastitis during lactation or as part of a mastitis control program to identify cows for treatment, segregation, or culling. In subclinical mastitis, penethamate-treated cows had lower somatic cell counts. Systemic penethamate therapy is beneficial for 2 months and BC of affected quarters improves milk SCC. Acoustic Pulse Technology (APT) is a new therapy for clinical and subclinical mastitis in dairy cows that recovered 65.5% [89].

Treatment of Clinical Mastitis in Dairy Cattle

Restoring milk output and eliminating infection were the main clinical mastitis therapy goals. In acute clinical coliform mastitis, steroidal or nonsteroidal anti-inflammatory medicines are administered, and dairy farmers often milk more. Antibiotics treat clinical mastitis caused by gram-positive cocci such *S. aureus, S. uberis, S. dysgalactiae,* and *S. agalactiae* [82]. β-lactam antibiotics, such penicillin G, cure mastitis caused by streptococci and penicillin-susceptible staphylococci. Systemic therapy is suggested for S. aureus and severe coliform mastitis. Clinical mastitis requires three days of treatment. Systemic and intramammary antibiotics or supportive treatment with nonsteroidal anti-inflammatory medicines work for clinical mastitis [90].

In clinical mastitis during breastfeeding, narrow-spectrum antimicrobials are best. Indian dairy cows' clinical mastitis is treated using homeopathic and allopathic treatments. Good outcomes have been achieved using homeopathy. Phytolacca, Calc. fluor, Silica, Belladonna, Bryonia, Arnica, Conium, and Ipecac (Healwell VT-6) effectively treat fibrotic and non-fibrotic mastitis in lactating dairy cows, outperforming antibiotics [91]. Yi-Xiong-Tang (YXT) is an effective and cost-effective alternative to antibiotics. YXT therapy provides advantages over traditional treatments. Because YXT is a mixture of active chemicals, it has a shorter treatment duration and may reduce drug resistance. Culture-based treatment and severity levels are essential for clinical mastitis management. Antibiotics are recommended for gram-positive clinical mastitis. Light-to-moderate gram-negative clinical mastitis does not require antibiotics. Most severe clinical mastitis requires antibiotics, fluids, and anti-inflammatories. Clinical mastitis caused by yeast, fungal, or no growth isolates does not require antibiotics [92].

Prevention and control & Management

Mastitis can be decreased in the research region by using an extensive or semi-intensive rearing approach, cleaning hands and udders with antiseptic before and after milking, and teaching farmers in scientific dairy farming [81,93].

Immunomodulation for mastitis prevention includes dairy cow vaccination. If utilized appropriately, AMS and robotic milking can enhance milking and udder health. Managing clinical mastitis requires culture-based treatment and severity levels [94]. Mastitis may be controlled by teat disinfection, housing, nutrition, breeding mastitis-resistant dairy cows, developing new diagnostic and treatment techniques, employing contemporary vaccinations, and drying animals [93]. Most bovine mastitis management programs use antibiotics. Good cleanliness and management can prevent mastitis. Maintaining milking equipment, disinfecting teats after milking, culling, and dry cow treatment (DCT) minimize infectious diseases (Figure 4) [37].

Challenges for the dairy industry on mastitis

Despite the broad implementation of mastitis control techniques, mastitis remains a significant concern for the global dairy sector. Mastitis poses a persistent threat to the dairy industry worldwide, presenting several challenges (Figure 5):

- Mastitis results in decreased milk production, diminished milk quality, and heightened veterinary expenses, leading to significant economic losses worldwide. The losses are exacerbated by the disposal of milk, premature culling, and the expenses associated with replacements.
- Smallholder farmers frequently experience limited access to cost-effective diagnostic tools, such as the California Mastitis Test (CMT), and insufficient veterinary care, which hinders prompt intervention.
- Inadequate milking hygiene, substandard housing conditions, and insufficient awareness of biosecurity measures contribute to elevated mastitis rates.
- High-yielding crossbred cows are prevalent in the Bangladeshi dairy sector; however, they exhibit increased susceptibility to mastitis due to reduced resistance to infections relative to indigenous breeds.
- The excessive reliance on antibiotics for treatment has intensified the antimicrobial resistance crisis. Resistant pathogens complicate mastitis treatment and present public health risks due to milk residues.
- Subclinical mastitis is a concealed challenge, as it lacks visible symptoms yet significantly affects productivity. Effective detection and management necessitate sophisticated diagnostic tools, which are frequently costly and inaccessible in numerous areas.
- Mastitis negatively impacts animal welfare by causing pain and discomfort related to the condition.
- Many farmers lack knowledge regarding effective strategies for mastitis prevention, including proper milking hygiene, biosecurity measures, and vaccination protocols.

Conclusion and Recommendations

Mastitis in dairy cows is the world's most expensive production disease and causes huge financial losses for the dairy industry [35]. Bangladesh has a high prevalence of SCM, which threatens dairy output. Controlling the disease requires regular screening, cleanliness, management improvements, and farmer awareness [22]. The most severe forms of this disease can kill several high-producing dairy cows every year. Efforts to improve milking and hygiene have reduced the spread of Gram-positive bacteria and reduced *S. aureus* isolates and subclinical mastitis globally [11]. Based on the findings we are recommended following points for prevention and control mastitis in dairy industry-

- Housing management and environmental sanitation require enhancement.
- Consistent assessment of udder health status: Continuous evaluation of the udder, prompt identification of inflammation, and meticulous treatment should be enhanced.
- Regular assessment of the mastitis control program: Systematic evaluation of the mastitis control initiative and monitoring of udder health status.
- Housing places must be consistently maintained to provide cleanliness, dryness, and comfort.
- Mastitis should be systematically examined, particularly for subclinical variants.

- Trained milkers must adhere to adequate sanitary milking techniques.
- Mastitis treatments should be initiated only after identifying the causal agent and assessing the susceptibility profile of the microorganisms involved.
- Effective dry cow treatment during the drying-off time.
- Chronically diseased and elderly cows should be systematically isolated or killed as part of a regular marketing strategy.
- Uphold biosecurity measures for infectious diseases.
- Consistent upkeep of milking apparatus to provide steady teat end vacuum.
- In cases of elevated risk for environmental mastitis during the peripartum period, contemplate the use of internal teat sealants.
- Regulate cross-suckling in calves and juvenile livestock.
- Manage all clinical cases and maintain accurate records.
- Establishing a vaccination program for dry and fresh cows helps reduce mastitis in the herd.
- Ensure the utilization of fully operating milking equipment.
- Implement effective feeding management: a comprehensive nutrition program.
- Disinfection of teats following milking.
- Early and precise identification is crucial for formulating successful mastitis management strategies, as preventive intervention can mitigate the disease's consequences and enhance recovery rates.

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Declaration of Conflict of Interest

The authors declare that there is no conflict of interest.

Ethical of approval

No animals or plats are involved directly in the research.

TABLE 1. Classification of mastitis-causing pathogens.

Source	Mammary gland	Environment	References
Major pathogens	*Str. agalactiae* *(Str. dysgalactiae)* *Mycoplasma bovis* *S. aureus*	• *Streptococci: Str. uberis, Str. equinus, Str. Dysgalactiae, Str. bovis.* • *Enterococcus spp.: E. faecalis, E. durans, E. faecium.* • Coliforms: *E. coli, K. oxytoca, K. pneumoniae, Enterobacter aerogenes*. • Non-coliforms: *Yersinia spp., Proteus spp., Serratia spp.,* • Others: *Pseudomonas aeruginosa, Arcanobacterium pyogenes*	[36]
Minor pathogens	Coagulase-negative *Staphylococci: S. chromogenes, S. haemolyticus,* *S. epidermidis,* *S. simulans, S. sciuri Corynebacterium bovis*	Yeasts, Fungi	[37]

TABLE 2. Pathology of bovine mastitis.

SL	Types of Mastitis	Bacteria involved	Gross Lesions	Microscopic lesions
1	Mixed mastitis		In a pounced lobular mammary pattern, thin white septa separated tiny yellowish nodules (0.2–0.5 cm) in the parenchyma that projected into the duct and gland cistern lumen.	Neutrophils in the alveoli and ducts and neutrophils, lymphocytes, plasma cells, and macrophages in the interstitium form a distinct to moderate inflammatory infiltrate. Epithelial cell hyperplasia, degradation, and distinct fibrosis.
2	Suppurative	*Streptococc*us spp. and CNS C. bovis.	A lobular mammary pattern with thin white septa and little yellowish nodules (0.2–0.5 cm) in the parenchyma that projected into the duct and gland cistern lumen.	Alveoli, ducts, and interstitium had modest to moderate levels of intact and degenerate neutrophils.

SL	Types of Mastitis	Bacteria involved	Gross Lesions	Microscopic lesions
3	Lymphoplasmacytic	*Streptococcus* spp., CNS, and *C. bovis*	Solid mammary quarters with fewer lobulations and thick white septa dissecting the parenchym.	Lymphocytes, plasma cells, and occasional macrophages in a moderately fibrotic interstitial inflammatory infiltrate.
4	Pyogranulumatous	*P. aeruginosa,S. aureus*	Botryomycotic lesion in the mammary parenchyma	Splendore-Hoeppli phenomenon, immunoglobulin aggregates
5	Abscedative mastitis	*T. pyogenes*	Extensive destruction of the mammary parenchyma	Single or multiple abscesses within the mammary parenchyma
6	Necro suppurative	*E. coli* and *Klebsiella*, CNS and *E. coli*.	Vascular damage, edema, hemorrhage, and thrombosis,	Severe parenchyma necrosis

TABLE 3. Risk factors of bovine mastitis.

Risk factor	Categories	Variables	Rate of Mastitis (higher/ lower)	Ref.
Pathogen factors	a) *Staphylococcus aureus*	Infected mammary glands store S. aureus. Toxins and degradative enzymes irreversibly harm milking tissue, lastly lowering milk supply. Because it forms biofilm and adapts to the host environment, treating such infections is tougher.	Increase the infection rate/mastitis	[45]
	b) *Streptococcus agalactiae*	Spread by milking equipment and polluted drinking water. Its biofilm helps it to live in cows' mammary glands, making it resistant to the host factor and food scarcity.	Causes sub-clinical mastitis with high SCC and low milk production	[35]
	c) *Mycoplasma* spp.	Extremely severe, it destroys secretory tissues, produces abscesses, creates biofilm, infiltrates the host cell, and is antibiotic-resistant.	Causes contagious mastitis	[35]
	d) *Escherichia coli*	Through the teat, it multiplies in the udder and inflames dairy cows. *E. coli's* severe clinical mastitis can cause milk loss, mammary gland damage, and even death in dairy cows. *E. coli* can also stay in the mammary gland and produce recurring, difficult-to-treat mastitis. This may be because *E. coli* may produce different biofilms.	Ultimately causes recurrent mastitis infections	[46]
	e) Enterococcus spp (*Enterococcus faecalis, Ent. faecium*)	Pathogenesis of *Ent. faecalis* involves biofilms. Due to biofilm, Ent. faecalis and Ent. faecium are resistant to erythromycin, lincomycin, tetracycline, kanamycin, streptomycin, quinupristin/dalfopristin (Synercid), chloramphenicol, and tylosin. Due to this, chronic and recurring enterococci infections are common and difficult to cure.	Causes Entecoccus infection which leads to mastitis infections.	[47]
	f) Coagulase-negative Staphylococcus (Staph. simulans, Staph. chromogens, Staph. hyicus, and Staph. Epidermis)	New mastitis pathogens are generally subclinical but persistent, increasing SCC and decreasing milk quality. Also contagious and environmental pathogens.	Elicits subclinical mastitis	[48]
	g) *Streptococcus uberis*	Environmental pathogen. Milk's -casein and -casein component promote biofilm development, which helps *Strep. uberis* tolerate environmental stress and antibiotics. In animals, it has been detected in the rectum, lips, tonsils, skin, oral cavity, rumen, respiratory tract, sick udders, feces, and wounds.	causes recurrent mastitis, associated with clinical and sub-clinical infections	[49]
Host factors	a) Breeding and genetics	High-yielding cattle (particularly, Holstein-Friesian	More vulnerable to mastitis	[50]

Risk factor	Categories	Variables	Rate of Mastitis (higher/ lower)	Ref.
		Medium-yielding cattle (Jersey cattle)	Lower rate of mastitis than Holstein-Friesian cattle	[51]
		Lower-yielding Rendena cattle	Higher resistance and resilience to diseases including mastitis	[52]
		Multiparous cows	More vulnerable to IMI than primiparous cows due to immunoincompetence	[50]
	b) Udder structure	large funnel-shaped teats/pendular-shaped udder and blind quarters	Greater risk of sub-clinical mastitis	[53]
		teat size and teat to floor distance	Decrease the in vitro activity of leukocytes in milk hence increasing the occurrence of IMIs	[50]
	c) Age	Young cow	Less vulnerable to mastitis	[35]
		Old cow	Frequent milking widens or permanently partly opens the teat canal, making it more vulnerable to infections. Older cows' mammary epithelium is more permeable due to permanent inflammatory damage.	[35, 54]
		≤ 4 years	Low	
		≥ 7 years	Medium	
		5–6 years	High	
	d) Transition period	Periparturient period (period between 3 weeks before and after parturition)	During immunosuppression, cows are at risk of mastitis due to oxidative stress and inadequate antioxidant defense. Peripartum IMIs occur. Immunosuppression increases oxidative stress and lowers antioxidant defense.	[55, 56]
	e) Host nutritional stress and immune system	Deficits in trace minerals, amino acids, and vitamins (e.g., selenium, iron, copper, zinc, cobalt, chromium) might result from inadequate feed consumption.	Cause cellular and humoral immunosuppression during breastfeeding, increasing infection risk.	[50, 57]
		Proper management during the transition period (diet supplements of vitamin E and zinc)	prevent mastitis infection and promote lactation,	[58]
	Interval of cleaning bedding	Once a week	Low	[59]
		< Once a week	Medium	
		> Once a week	High	
	Udder & leg hygiene score	Slightly dirty	Low	[55]
		Moderately dirty	Medium	
		Very dirty	High	
	Udder position	Normal	Low	
		Pendulous	High	
	Teat end shape	Pointed	Low	
		Round	Medium	
		Flat	High	
	Herd size	≤10 cows	Low	
		>10 cows	Medium	
		muddy	High	
	Production system	Semi-intensive	Low	
		intensive	High	

Risk factor	**Categories**	**Variables**	**Rate of Mastitis (higher/ lower)**	**Ref.**
	Stage of lactation	Early (1-2 months	Low	
		Mid (3–6 months)	Medium	
		late > 7 months	High	
	Number of parities	≤ 2 calves	Low	[59, 60]
		3 to 5 calves	Medium	
		≥ 6 calves	High	
	Udder washing	Yes	Low	[55, 59]
		no	High	
		Whole udder	Low	
		Teats only	High	
	Breed	Local	Low	[55]
		Cross	High	
	Using towel	No	High	[59]
		yes	Low	
	Udder drying after washing	yes	Low	[55, 61]
		no	High	
	Proper milking technique	yes	Low	
		no	High	
	Use of clean udder drying towel	yes	High	[55, 62]
		no	Low	
	Washing hands between milking	yes	Low	
		no	High	
	Use of teat dips	yes	Low	
		no	High	
Environmental factors	a) Environmental conditions and management practices	Herd uncleanliness and uncomfortable condition	Induces the incidence and severity of mastitis	[63]
		High stocking density	Promote growth of mastitis pathogens and increased exposure of cows, resulting in higher occurrence of mastitis	[55, 62,64]
		contaminated floor		
		Wet bedding		
		Poor ventilation		
		Humid climate		
	Floor type/Type of floor	Concrete	Low	
		Muddy	High	
	Type of Housing	Separate pen	Low	
		Shared barn	High	
	Bedding material	yes	Low	
		No	High	
	Tick infestation	Yes	High	
		No	Low	
		Flat	High	
	Housed with a roof	Yes	High	[55, 59,65]
		No	Low	
	Cleaning of floor	Daily	High	
		weekly	Low	
	Tick infestation	Yes	High	
		No	Low	
		Flat	High	

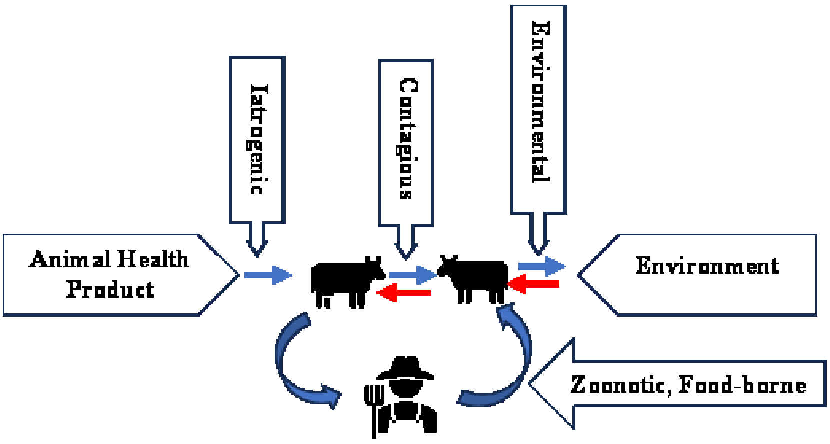

Fig. 1. Possible routes and transmission routes for mastitis pathogen.

Fig. 2. Mastitis affected cow udder, teat (inflammation in udder and teat) and milk (blood-stained milk).

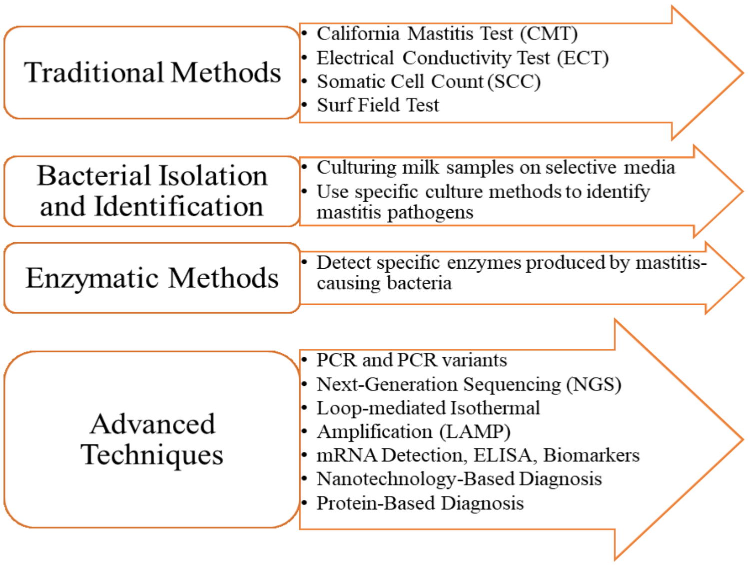

Fig. 3. Diagnostic methods of mastitis in cow.

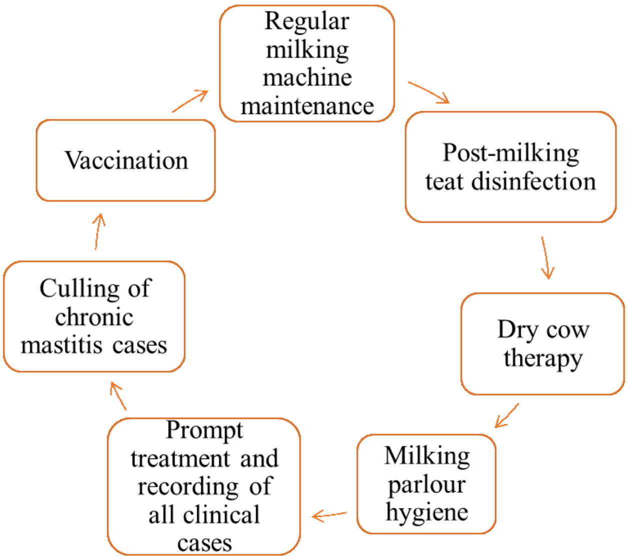

Fig. 4. A plan for prevention and control measures for bovine mastitis.

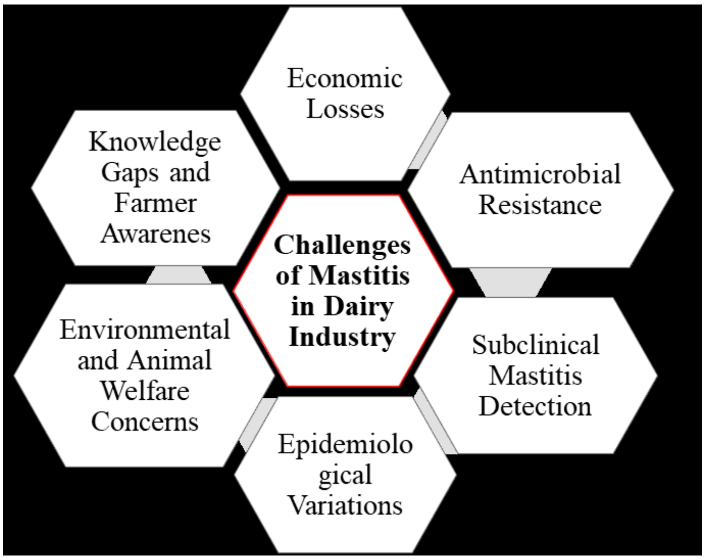

Fig. 5. Challenges of mastitis in dairy industry.

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