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Review Article

Mastitis: An Intensive Crisis in Veterinary Science

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Mastitis is an inflammatory disease of mammary gland caused by bacteria and its toxin and it is characterized by physical, chemical and microbiological changes in the milk along with pathological changes in the glandular tissues of udder. Many bacteria, fungus and viruses are responsible to cause mastitis but main causative agent is *Staphylococcus aureus* and its toxin. All breeds of dairy and farm animals are susceptible to mastitis but infection rate is more in successive lactation than the first lactation. Mastitis can cause decline in potassium and lactoferrin which decreases casein, the major protein in milk. The presence of bacteria in the milk collected from infected dairy animal like cow and buffalo renders that milk unsuitable for human consumption and can spread throat infections, scarlet fever, tuberculosis and/or brucellosis. Treatment of mastitis is possible with long acting antibiotics but mastitis may occur after specific period of time even after treatment with long acting antibiotics and so treatment needs effective strategies.

Key words: Mastitis, Mammary gland, *Staphylococcus aureus* infection, Treatment, Strategies

1. INTRODUCTION

Mastitis is defined as inflammation of mammary gland parenchyma, which is caused by bacteria and its toxins. Milk secreting tissue and various ducts throughout the mammary gland are damaged due to toxins by the bacteria. Mastitis can also occur as a result of chemical, mechanical or thermal injury to udder sac. The mammary gland does not produce any milk. The

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udder sac becomes hard, tight and firm. It is the inflammatory condition of the udder irrespective of the cause. It is characterized by physical, chemical and microbiological; changes in the milk and pathological changes in the glandular tissues of the udder. The change in the milk includes change of color, change of consistency (clot) and presence of abnormally large number of leucocytes. Due to improved breeding, the cow's udder has to undergo rapid changes in relation to size, position and adjustment for rapid removal of large volume of milk and such it is prone to injury and infection. The resulted inflammation is referred as mastitis.¹

1.1 Identification

The disease can be identified by abnormalities in the udder sac such as swelling, heat, redness, hardness or pain.



Fig 1: Mastitis infection

Other indications of mastitis may be abnormalities in milk such as watery appearance, flakes, clots or pus.²

1.2 Diagnosis

Majority of cases are subclinical mastitis without any visible change in the milk quality, a somatic cell count helps to detect it. A count of more than 50,000 cells/ml of milk can be taken as positive for mastitis.³

1.2.1 Physical examination of the udder

Shape, size, consistency and contour of the udder should be viewed properly. Signs of Inflammation should be clinically assessed.

1.2.2 Tests for milk abnormalities

a) Strip cup test – This test is easy to conduct and should be practiced every day at the time of milking. This helps to detect the changes in the milk and to

eliminate first few strips since the fore milk contain more bacterial load. By doing this method, the quality of milk improves. A wide mouthed mug made of plastic or aluminum can be taken and a black cloth is placed on it and tied tightly with a thread. The milk can be stripped on it and examine for the presence of streaks of curdled milk. Milk can also be cultured for diagnosis of causative organism.

Diagnosis can also be done by mixing the milk with a coloured reagent to detect the extent of infection. The milk reacts with the reagent and forms a gel if the infection is high. If negative, it remains in the same condition.

b) Bromothymol blue test to determine the pH of the milk- pH of the mastitis milk is alkaline in reaction. The normal pH of milk is 6.6-6.8.

c) White side test- This test depends on the increased leukocyte content of milk. 5 drops of milk is placed on the glass plate underside painted black. Then 2 drops of 4% NaOH is added to it. It is then rapidly stirred with a broomstick for 20-25 seconds. In acute case the mixture becomes thick and viscid. In chronic cases white flakes are noted.

d) Other tests-

- i) California mastitis test (CMT)
- ii) Catalase test
- iii) Chloride test

1.3 Types of Mastitis

1.3.1 Per-acute mastitis

- a) The affected animals show a very high temperature of 106-107°F.
- b) Udder becomes swollen and extremely painful.
- c) Caseation of milk secretion and often blood stained.
- d) This form is caused by *Staphylococcus aureus*, *Streptococcus dysgalactiae*, *Corynebacterium pyogenes*, *E.coli* and *Pseudomonas aeruginosa*.

1.3.2 Acute mastitis

- a) Systemic reactions are not seen.
- b) Udder becomes swollen and changes in the milk.
- c) The milk may be replaced by yellow or brown fluid with flakes or clots.
- d) Infection localized in one quarter or the entire udder.
- e) *Str. agalactiae*, *Staph. aureus* causes this form.

1.3.3 Sub-acute mastitis

- a) There is variable change in the milk but practically no changes in the udder tissues.
- b) Culture of milk will show presence of pathogenic bacteria.

1.3.4 Chronic mastitis

- a) Udder becomes hard due to fibrosis.
- b) The supramammary lymph node becomes palpable.
- c) The quarters may become thickened, firm, nodular and at times atrophic.
- d) Sometimes milk may look green or yellow-green and foul smelling.
- e) Tuberculous infections often give rise to chronic form of mastitis.⁴

2. ETIOLOGY

A large number of species of microorganisms have been implicated as cause(s) of mastitis. They are bacteria, fungus, mycoplasma and virus.⁵

- a) Bacteria- *Staphylococcus aureus*; *Str. Agalactiae*; *Str. zooepidemicus*; *Str. Faecalis*; *Str. pyogenes*; *Corynebacterium pyogenes*; *Mycobacterium bovis*; *E. coli*; *Brucella abortus*; *Pseudomonas* spp.; *Leptospira Pomona*; *Pasteurella multocida*; *Mycoplasma*: *Mycoplasma bovis*; *Mycoplasma agalactiae*.
- b) Fungus- *Aspergillus* spp.; *Trichosporon* spp.; *Candida* spp.
- c) Virus- Vesicular stomatitis; Infectious rhinotracheitis.

2.1 Susceptible hosts

All breeds of dairy cows, buffaloes, goats, sheep, pig, horses are susceptible. High yielding cows are more commonly affected than low yielder. Infection rate is more in successive lactation than the first lactation⁶.

2.2 Mode of transmission

The avenue of infection is the teat canal. Through teat the infection reaches the mammary gland. There are two sources of infective agent- the udder- where many bacterial like *Streptococcus agalactiae* and *Staphylococcus aureus* may be present as normal inhabitant and the environment- where bacteria like *E. coli* persist. The cutaneous surface of the cow many have many organisms as resident population and from where the organisms may have the chance to invade through contamination by the handlers. The contamination of the milkers hands, clothes, and machine cup by milk from the affected quarter may lead to the spread of the disease to other non- infected teats of cows. Flies and other insects may also spread the infection from one place to the other. Spread of infection is possible through bedding ground by discharges of affected gland.⁷

2.3 Pathogenesis

After entry through the teat canal the bacteria enter into the udder tissue, multiply and produce toxins causing inflammation of the udder or the corresponding teat. Due to inflammation, the body releases leucocytes and the quality of the milk gets affected. The milk becomes watery or curdled; sometimes blood streaks may also be present depending on the severity of infection. Infection of the udder usually takes place directly through teat canal. But, organisms may get settled in the mammary tissues via blood in case of tuberculous mastitis. Broadly, two stages have been described viz. invasive stage and infective stage. In invasive stage the organism gets entry from the exterior to the teat canal and milk. Infective stage denotes the stage of bacterial

multiplication and their resultant damaging effect on the mammary tissues.⁸

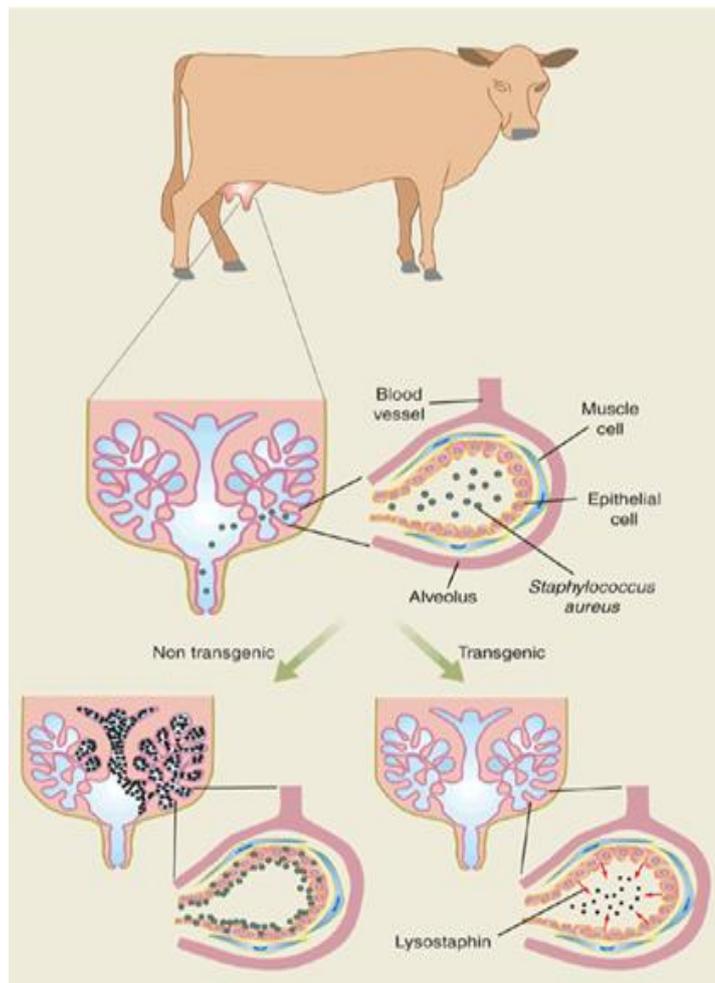


Fig 2: Pathogenesis of Mastitis

2.4 Other common causes

Mastitis is usually caused by poor sanitation in the sheds like unclean floorings, bedding, animal and milking surroundings. Unhygienic conditions favor multiplication of bacteria and infect the animal. Poor milking practice, not washing the animals udder before milking, not practicing the dipping of teats in antiseptic solution and incomplete milking of animals are also responsible for mastitis. Infection also spreads through milking machines if they are not cleaned well before use.

Infection also spreads through milking persons if their hands are contaminated and not cleaned well with disinfectant before milking. Improper milking methods like milking using thumb and pressing or pulling the teat causes a mechanical injury to teat.⁹

2.5 Predisposing factors

- Age- the disease is more prevalent in high age group. More the number of lactation more the possibility of the disease.
- Breed- incidence is more in exotic and crossbred cows than the local cows.
- Complete milking- incomplete removal of milk from the udder is a conducive factor.
- Trauma- any mechanical injury to teat or udder helps the microorganisms to enter the organ.
- Hygiene- bad hygiene and sanitation help bacterial multiplication. Maintenance of adequate hygiene at pre and post milking is important in relation to setting up of infection.¹⁰

2.6 Symptoms

- Swelling of the udder and teat.
- Redness and pain of the udder.
- Milk gets watery, curdled with blood streaks in some cases.
- Fever listlessness and anorexia.
- Reduced milk production.
- In chronic and sub clinical cases the teat canal gets thickened, when the teat is palpated, it appears like a thread inserted in the teat canal.
- In sub clinical cases the milk is not affected and appears normal but the somatic cell count increases and the milk yield is decreased.
- If the sub clinical cases are more in the farm it becomes a problem to control the disease.¹¹

2.7 Effect on Milk Composition

- Mastitis can cause a decline in the potassium and lactoferin. It also results in decreased casein, the major protein in milk.
- As most calcium in milk is associated with casein, the disruption of casein synthesis contributes to lowered calcium in the milk. The milk protein continues to undergo further deterioration during Processing and storage.

- c) Milk from cows with mastitis also has a higher somatic cell count.
- d) Generally, higher the somatic cell count, the lower the milk quality.¹²

2.8 Economic importance

- a) It is one of the most costly disease afflicting dairy cows.
- b) It reduces the milk production.
- c) The milk of the infected cow is inferior in quality and is deficient in butter fat and sugar content.
- d) The milk from the affected cow is unfit for human consumption.
- e) The udder tissue is grossly damaged resulting in loss of one or more quarter and the market value of the animal is reduced.¹³

2.9 Economic losses

- a) Milk discarded due to abnormal characters and presence of antibiotics.
- b) Reduced production due to clinical and sub-clinical infection.
- c) Decreased market value of dairy animal due to damage of quarters.
- d) Cost of veterinary services and drugs to treat acute and chronic cases.
- e) Cost of increased labor to care infected farm animal.¹⁴

2.10 Public health aspect

It is an important disease from public health standpoint. The presence of different bacteria in the milk renders it unsuitable for human consumption and helps in the spread of disease like tuberculosis, brucellosis, staphylococcal toxemia, streptococcal sore throat, scarlet fever and gastroenteritis.¹⁵

2.11 Prevention and Control

Practices such as good nutrition, proper milking hygiene and the culling of chronically infected cows can help. Ensuring that cows have clean, dry bedding decreases the risk of infection and transmission. Dairy

workers should wear gloves while milking and machines should be cleaned regularly to decrease the incidence of transmission.¹⁶

General considerations for prevention and control of mastitis-

- a) Proper treatment of teat sores using teat dip and antiseptics.
- b) Disinfection of hand prior to milking.
- c) Udder should be washed and dried prior to milking.
- d) Calves should be prevented from suckling milk of other cows.
- e) Mastitis milk should be properly disposed off. 5% phenol may be added to the infected milk at the time of disposal.
- f) The non-responsive quarter must be permanently dried.
- g) Provide soft bedding to the cows following parturition.
- h) All the equipment and containers should be cleaned and dried properly.
- i) Control of fly population.
- j) Hygienic measures at milking time.¹⁷

3. TREATMENT

Treatment is possible with long acting antibiotics but milk from such dairy animal like cow or buffalo is not marketable until drug residues have left from cows system. Antibiotics may be systemic (injected into the body) or they may be forced upwards into the teat through the teat canal i.e. by intra-mammary infusion.

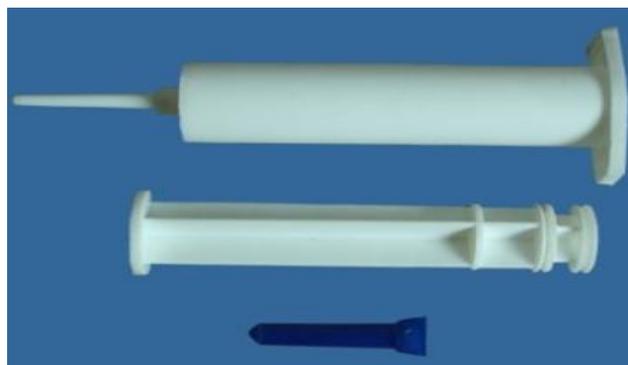


Fig 3: Syringe used for Mastitis

Cows being treated may be marked with tape to alert dairy workers and their milk is siphoned off and discarded. Vaccinations for mastitis do exist but as they only reduce the severity of the condition, and do not prevent new infection, they should be used in conjunction with a mastitis prevention program.¹⁸

Treatment may be enumerated as follows-

- a) Isolate the affected animal from the herd.
- b) Milk the healthy quarters before milking the affected quarter.
- c) Complete removal of milk or secretions as much as possible.
- d) Attempt should be made to culture and antibiotic sensitivity test to provide a rational therapy.
- e) Intra-mammary antibiotic preparations should be used for local treatment.
- f) Systemic antibiotic therapy may be necessary where systemic reactions are evident.
- g) Supportive treatment may be required. It includes parenteral injection of large quantities of isotonic fluid containing glucose, anti-inflammatory drugs, antihistamines and corticosteroids.¹⁹

Drugs used as intra-mammary infusion-

- a) Pendistrin-SH- (Penicillin, Streptomycin, Sulphamerazine and Hydrocortisone).
- b) Tilox- (Ampicillin, Cloxacillin).
- c) Kefelong- (Cephalosporine).
- d) Wokadine- (Povidine Iodine).

3.1 Factors affecting effective treatment

1) Factors related to the cow-

- a) Age
- b) Stage of lactation
- c) Effectiveness of cows immune response
- d) Somatic cell count.²⁰

2) Factors related to the pathogen-

- a) Pathogenicity
- b) Virulence
- c) Response to antimicrobial therapy.²⁰

3) Factors related to the drug-

- a) Spectrum of activity
- b) Route of administration
- c) Concentration of the drug at the site of infection
- d) Duration of treatment.²⁰

3.2 Strategies

Treatment strategies such as use of antimicrobials for various durations, discard of abnormal milk without antimicrobial treatment, culling of cows that are unlikely to respond to therapy, drying off the affected mammary gland or drying off the cow may be recommended depending on the probability of reaching a successful outcome.²¹ Most commonly used antibiotics are-

- a) Penicillin
- b) Ampicillin
- c) Cephalosporin
- d) Tetracyclines

4. CONCLUSION

Mastitis can cause decline in potassium and lactoferrin which decreases casein, the major protein in milk. The presence of bacteria in the milk collected from infected dairy animal like cow and buffalo renders that milk unsuitable for human consumption and can spread infections into humans. Treatment of mastitis is possible with long acting antibiotics but milk from such dairy animal is not marketable until drug residues are not left from milk completely and mastitis may occur after specific period of time even after treatment with long acting antibiotics. Hence, mastitis is very costly and life threatening disease as far as animal survival and public health point is concerned.

5. REFERENCES

1. Zwald AG, Ruegg PL, Kaneene JB. Management practices and reported antimicrobial usage on conventional and organic dairy farms. *J Dairy Science* 2004; 87: 191-201.

2. Pinzon-Sanchez C, Ruegg PL. Risk factors associated with short-term post-treatment outcomes of clinical mastitis, American Dairy Science Association. *J Dairy Science* 2011; 94: 3397–3410.
3. Wilson CD, Richards MS. A survey of mastitis in the British dairy herd. *Vet Rec* 1980; 106: 431-435.
4. Hutton CT, Fox LK, Hancock DD. Risk factors associated with herd-group milk somatic cell count and prevalence of coagulase-positive staphylococcal intra-mammary infections. *Prev Vet Med* 1991; 11: 25-35.
5. Dodd FH, Westgarth DR, Neave FK, Kingwill RG. Mastitis: the strategy of control. *J Dairy Sci* 1969; 52: 689-695.
6. Miller GY, Bartlett PC. Economic effects of mastitis prevention strategies for dairy producers. *J American Vet Med Assoc* 1991; 198: 227-231.
7. Jarp J, Bugge HP, Larsen S. Clinical trial of three therapeutic regimens for bovine mastitis. *Vet Rec* 1989; 124: 630-634.
8. Sol J, Sampimon OC, Snoep JJ, Schukken YH. Factors associated with bacteriological cure after dry cow treatment of subclinical *Staphylococcus* mastitis. *J Dairy Sci* 2000; 77: 75-79.
9. Pearson JK, Greer DO, Spence BK, McParland PJ, McKinley DL, Dunlop WL, Acheson AW. Factors involved in mastitis control: A comparative study between high and low incidence herds. *Vet Rec* 1972; 91: 615-623.
10. Dargent M, Scarlett J, Pollock RV, Erb HN, Sears P. Herd-level risk factors for *Staphylococcus aureus* and *Streptococcus agalactiae* intramammary infections. *Prev Vet Med* 1988; 6: 127-142.
11. Osteras O. Determinants of success and failure in the elimination of major mastitis pathogens in selective dry cow therapy. *J dairy Sci* 1991; 82: 1221-1231.
12. Bartlett PC, Miller GY, Lance SE, Heider LE. Environmental and managerial determinants of somatic cell counts and clinical mastitis incidence in Ohio dairy herds. *Prev Vet Med* 1992; 14: 195-207.
13. Goodhope RG, Meek AH. Factors associated with mastitis in Ontario dairy herds: a case control study. *Can J Comp Med* 1980; 44: 351-357.
14. Erskine RJ, Eberhart RJ, Hutchinson LJ, Spencer SB. Herd management and prevalence of mastitis in dairy herds with high and low somatic cell counts. *J American Vet Med Assoc* 1987; 190: 1411-1416.
15. Hoare RJ, Critchley DJ, Dettman EB, Sheldrake RF, Fell LR. Mastitis control: a survey of farm practices and their relationship to bulk milk cell counts. *Austr J Dairy Tech* 1979; 34: 91-96.
16. Olsen SJ. A mastitis control system based on intensive use of mastitis laboratories, Proc: Sem. On mastitis control. *Int Dairy Fed Brussels* 1975; 371-372.
17. Ekman T, Astrom G, Funke H. Measures taken by Veterinarians in Sweden in Cases of Bovine Mastitis. *Acta Vet Scand* 1994; 35: 329-335.
18. Robinson TC, Jackson ER, Marr A. Factors involved in the epidemiology and control of *Streptococcus uberis* and coliform mastitis. *Brit Vet J* 1985; 141: 635-642.
19. Hueston WD, Heider LE, Harvey WR, Smith KL. The use of high somatic cell count prevalence in epidemiologic investigations of mastitis control practices. *Prev Vet Med* 1987; 4: 447-461.
20. Howard WH, Gill R, Leslie KE, Lissemore K. Monitoring and controlling mastitis on Ontario dairy farms. *Can J Agric Econ* 1991; 39: 299-318.

21. Sischo WM, Heider LE, Miller GY, Moore DA.
Prevalence of contagious pathogens of bovine mastitis and use of mastitis control practices. J American Vet Med Assoc 1993; 202: 595-600.