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**Summary:**

# Mastitis

## Introduction

- Mastitis is inflammation of the parenchyma of the mammary gland regardless of the cause.
- Mastitis is characterized by physical and chemical changes in the milk, and pathologic changes in the mammary gland, with or without systemic reactions.
- The most important changes in the milk include:
  - Discoloration.
  - Presence of clots.
  - Presence of large numbers of leukocytes (detected by laboratory tests).
    - The increased somatic cell concentration is, in almost all cases, caused by an increased neutrophil concentration, which represents a reaction of glandular tissue to injury and is preceded by changes in the milk that are the direct result of damage to glandular tissue.

Clinical mastitis is detected using the physical examination:
- A useful definition of clinical mastitis is answering the question: “Would you drink this?” Milk from cows with clinical mastitis is not suitable for drinking.
- In many **clinical cases**, the pathologic changes in the mammary gland include: swelling, heat, pain, edema.
- In **subclinical mastitis**, the pathologic changes in the mammary gland are not readily detectable by manual palpation or by visual examination of the milk.
  - The diagnosis depends on indirect tests, e.g., the somatic cell concentration or electrolyte (sodium or chloride) concentration of milk.

# Bovine Mastitis

## Etiology

- **Contagious pathogens**:
  - *Staphylococcus aureus*
  - *Streptococcus agalactiae*
  - *Corynebacterium bovis*
  - *Mycoplasma bovis*

 The source of contagious pathogens is the infected glands of other cows in the herd.

 The infection is transmitted from cow to cow by:

- Hands of milkers.
- Contaminated milking equipment and common udder washcloths.
- Residual milk in teat cups.

**Environmental pathogens:**

- Environmental coliforms including:
  - *Escherichia coli*
  - *Klebsiella spp.*
  - *Enterobacter* spp.,
  - *Trueperella* (formerly *Arcanobacterium*, or *Actinomyces*, or *Corynebacterium*) *pyogenes.*
- Environmental *Streptococcus spp.* Including:
  - *Streptococcus uberis* and *Strept. dysgalactiae* - the most prevalent.
  - *Streptococcus equinus* (formerly referred to as *S. bovis*) - less prevalent.

 The source of these pathogens is the environment of the cow.

 The major cause of transmission from the environment to the cow is the inadequate management of the environment. Examples:

- inadequate premilking udder and teat preparation,
- housing systems that allow teat injuries,
- poor fly control.

 **Teat skin opportunistic pathogens:**

- *Coagulase-negative staphylococci* (CNS)
  - They have the ability to cause an intramammary infection by ascending infection through the streak canal.

**• Uncommon pathogens:**

- *Nocardia spp.*
- *Pasteurella spp.*
- *Mycobacterium bovis*
- *Bacillus cereus*
- *Pseudomonas spp.*
- *Serratia marcescens*
- *Citrobacter spp.*
- anaerobic bacterial species
- fungi
- yeasts

Clinical differentiation of the different causes of mastitis is difficult.

**Major pathogens that cause mastitis are:**

- Environmental: (*E. coli, Klebsiella spp., Enterobacter spp. T. pyogenes, Strept. uberis and Strept. dysgalactiae).*
- Contagious: (*Strept. agalactiae, Staph. aureus,* and *M. bovis*)

## Epidemiology

**Occurrence**

- Incidence of clinical mastitis ranges from 10% - 12% per 100 cows at risk per year.
- Prevalence of intramammary infection is about 50% of cows and 10%-25% of quarters.
- Case–fatality rate depends on cause of mastitis.

**Method of transmission**

- Contagious pathogens are transmitted at time of milking.
- Teat skin opportunistic pathogens take any opportunity to induce mastitis.
- Environmental pathogens are from the environment and induce mastitis between milkings.

**Risk factors**

- Animal risk factors:
  - Prevalence of infection increases with age.
  - Most new infections occur in the dry period and in early lactation.
  - Morphology and physical condition of the teat:
- High milking rate and large teat canal diameter can increase SCC and the infection.
  - Dirty udders are associated with increased SCC and an increased prevalence of intramammary infection caused by contagious and environmental pathogens.
  - Selenium and vitamin E had a beneficial effect on udder health (antioxidants) by decreasing the incidence and duration of clinical mastitis.
  - Cows with a history of mastitis in the previous lactations may be almost twice as susceptible

**Economic importance:**

- Economic losses due to clinical mastitis can be due to
  - Loss of milk production.
  - Discarded milk from cows with clinical mastitis and treated cows.
  - Replacement cost of culled cows.
  - Extra labor required for treatment and monitoring.
  - Veterinary service for treatment and control
  - Cost of abortions due to mastitis
  - Cost of decreased conception rate.
  - Cost of control measures.
- Subclinical mastitis can cause decrease in milk production and increase of somatic cell counts (i.e., both quantity and quality of milk are affected).

The effects of mastitis on milk yield are highly variable and depend on:

- The severity of the inflammation.
- The causative agents and the lesions produced:
  - *Staph. aureus* results in persistent but moderate infections with high risk of culling.
  - *Trueperella pyogenes* results in suppurative lesions, greatest decrease in milk production, poor response to treatment, and culling.
  - Environmental streptococci is associated with the smallest decrease in milk production.
  - *M. bovis* causes chronic induration and almost complete loss of milk production without recovery.
- The efficiency of treatment.
- The production level.
- The stage of lactation.
  - Mastitis in early lactation causes a larger decrease in milk yield with long-term effects than mastitis in late lactation

**Zoonotic Potential**

- Raw (unpasteurized) milk can be a source of food-borne pathogens.

## Pathogenesis

Entrance of the pathogen via teat canal ⟶ Multiplication and invasion of the mammary tissue

Recruitment of inflammatory cells ⟶ Release of endotoxins (certain bacteria, e.g., *E. coli*)

⟶ Release of inflammatory mediators (e.g., $PGF_{2}\alpha$ and pro-inflammatory cytokines

⟶ Systemic reactions

 The severity and duration of mastitis are related to neutrophil migratory response and their anti-
bactericidal activity at the site of infection:

- If neutrophils move rapidly from the bloodstream and are able to eliminate the bacteria, then recruitment of neutrophils ceases and the SCC returns to normal levels.
- If bacteria are able to survive this immediate host response, then the inflammation continues.
- Prolonged diapedesis of neutrophils damages mammary tissue, resulting in decreased milk production.

## Clinical signs

 Gross abnormalities in the secretion consistency and/or color, and presence of clots, flakes, or pus.

- Wateriness (except the first few streams) is usually indicating chronic mastitis when the quarter is lactating.

- Clots or flakes are usually indicating a severe degree of inflammation, even if they are small and present only in the first few streams.
- Flakes at the end of milking may be indicative of mammary tuberculosis in cattle.
- Notes:
  - Blood clots are of little significance in a mastitis case.
  - Small plugs of wax are often present in the milk during the first few days after calving.
  - During the dry period in normal cows, the secretion changes from normal milk to a clear watery fluid, then to a secretion of color and consistency of honey, and finally to colostrum in the last few days before parturition.

**Physical abnormalities of udder**:

- Acute diffuse inflammatory swelling accompanied by heat and pain.
- In severe cases, gangrene may develop, or abscesses develop in the glandular tissue.
- Local fibrosis vary in size may occur.
- There may be a diffuse increase in connective tissue, giving the quarter a firmer feel, and usually a more nodular surface on light palpation.
- Atrophy in the terminal stage of chronic mastitis.
  - On casual examination, an atrophied quarter may be classed as normal because of its small size, whereas the normal quarter is judged to be hypertrophic. Careful palpation may reveal little functioning mammary tissue remains in atrophic quarter.

 Systemic response: May be normal or mild, moderate, acute, peracute with varying degrees of anorexia, toxemia, dehydration, fever, tachycardia, ruminal stasis, recumbency and death.

- Daily feed intake is decreased.
- Cows eat more slowly and are less competitive at the feed bunk
- Cows spend more time standing and less time lying with affected quarter
- A systemic response is usually associated with severe mastitis associated with *E. coli*, *Klebsiella spp.*, or *T. pyogenes* and occasionally with *Strept. spp.* or *Staph spp.*

 Clinical mastitis episodes are categorized according to their **severity** and **duration**:

- **Severity** is characterized as follows:
  - **Peracute**: severe inflammation with swelling and heat and pain of the quarter with a marked systemic reaction, which may be fatal.
  - **Acute**: severe inflammation without a marked systemic reaction.
  - **Subacute**: mild inflammation with persistent abnormality of the milk.

- **Duration** is characterized as follows:

  - Short-term (as in *E. coli* and *Klebsiella spp.*)
  - Recurrent (as in *Staph. aureus* and *Strept. dysgalactiae*)
  - Persistent (as in *Strept. agalactiae* and *M. bovis*)

**Clinical pathology**

**Detection of Clinical Mastitis**

- The initial diagnosis of clinical mastitis is made during routine physical examination.
- Laboratory culturing of milk samples for bacteria and Mycoplasma spp., and for determining the antimicrobial susceptibility

**Detection of Subclinical Mastitis**

- Subclinical mastitis can only be detected by laboratory examination.
- Detection at the Herd Level:
  - Bulk tank milk somatic cell counts (SCCs).
  - Culture of bulk tank milk
- Detection at the Individual Cow Level:
  - Culture of individual cow milk.
  - Indirect tests for subclinical mastitis:
  - SCCs using automated electronic counters.
  - California Mastitis Test (CMT).
  - Increases in electrical conductivity of milk.
  - Increases in the activity of cell associated enzymes in milk.

## Treatment

**Clinical mastitis in lactating cow:**

- Mild cases of clinical mastitis (abnormal secretion only) may not require treatment.
- Clinical mastitis accompanied by an abnormal gland or systemic signs of illness should be treated with intramammary antimicrobial (all cases) and parenterally (selected cases).
  - Veterinarians should always ask and answer four questions related to antimicrobial therapy in bovine mastitis:

1. Is antimicrobial therapy indicated?
2. Which route of administration (intramammary, parenteral, or both) should be used?
3. Which antimicrobial agent should be administered?
4. What should be the frequency and duration of treatment?

- Identify treated cows and remove them from the milking herd.
- Acute and peracute mastitis cases require supportive therapy (fluid and electrolytes) and nonsteroidal anti-inflammatory agents.

**Treatment of Subclinical Mastitis**

- It is generally considered not advisable to treat subclinical mastitis during lactation, because it is much less rewarding.
- Subclinical infections associated with environmental streptococci, or coliform organisms, can be found in moderate numbers in some herds. Spontaneous cure rates are higher, but the treatment during lactation might be recommended.

**Failure to respond to therapy of the lactating cow may be caused by the following:**

- Presence of micro-abscesses and inaccessibility of the drug to the pathogen.
- Ineffective drug diffusion.
- Inactivation of the antimicrobial by milk and tissue proteins.
- Inefficient killing of the bacteria and intracellular survival of bacteria.
- Increased antimicrobial resistance.
- Development of L-forms of bacteria (bacteria that lack cell wall).

**Dry cow therapy:**

- Intramammary infusion of long-acting antimicrobial agents at drying off provides the best treatment for subclinical mastitis caused by contagious pathogens.
  - Intramammary infusions approved for dry cow therapy contain high levels of antimicrobial agents in a slow-release base that maintains therapeutic levels in the dry udder for long periods of time.
- Dry cow therapy has been efficacious and economically beneficial in reducing the prevalence of intramammary infections.

## Control

**Principles of control:**

- Eliminate existing infections
- Prevent new infections
- Monitor udder health status

**Components of a mastitis control program:**

- Use proper milking management methods.
- Proper installation, function, and maintenance of milking equipment.
- Dry cow management.
- Appropriate therapy of mastitis during lactation.
- Culling chronically infected cows.
- Maintenance of an appropriate environment.
- Good record keeping.
- Monitoring udder health status.
- Periodic review of the udder health management program.
- Setting goals for udder health status.

## Mastitis of Sheep (Ovine Mastitis)

Most cases of clinical mastitis are caused by:

- *Staph. aureus* or *Mannheimia spp.* (predominantly *M. haemolytica* and *M. glucosida*, and also *M. ruminalis*).

- *Staph. aureus* is responsible for around 80% of clinical cases.

○ *Strept. agalactiae* is also important, and associated with persistent subclinical infections and elevated SCCs.

○ Other infectious agents include *E. coli, Histophilus somni* (formerly *H. ovis*), *Clostridium perfringens* type A, *Pseudomonas spp., C. pseudotuberculosis*, or *Enterococcus faecalis.*

 Pathogenesis, clinical mastitis, diagnosis and treatment are similar to those for bovine mastitis.

○ Most clinical cases occur with 2 to 3 weeks of parturition or at weaning, and take the form of gangrenous mastitis, affecting one or both halves.

○ The treatment of ewes with peracute gangrenous mastitis requires systemic treatment, but this is often not successful and the affected half sloughs after several weeks.

## Mastitis of Goats

 *Staph. aureus* and *E. coli* are the most common causes of clinical mastitis.

 Other infectious agents include *Pseudomonas spp., Staph. hyicus, Strept. dysgalactiae, S. pyogenes, S. intermedius, A. pyogenes, and Bacillus spp. and,* more rarely, *K. pneumoniae, C. pseudotuberculosis, M. haemolytica,* and *Actinobacillus equuli*.

 Goats have naturally higher SCCs than cows or sheep, which increase with age and stage of lactation.

 Pathogenesis, clinical mastitis, diagnosis and treatment are similar to those for bovine mastitis.

## Mastitis of Mares

 Mastitis in mares is generally regarded as rare.

 Many mares may affected with typical signs of severe swelling and soreness of the udder, but without abnormal milk.

 Because of the high frequency of gram negative bacteria as causative agents in mares, treatment should include a broad-spectrum antibiotic in an intramammary infusion plus parenteral antibacterial treatment such as with gentamicin-penicillin or trimethoprim sulfonamide combinations.
Hot packs and frequent milking are also recommended

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