Bovine Mastitis - A Disease of Serious Concern for Dairy Farmers

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Abstract

Mastitis is an inflammatory disease condition of udder affecting milk production negatively and having a serious impact on the economy of dairy enterprises. It is considered to be the most costly disease of dairy animals and losses mainly occur through discarded milk, reduction in milk yield, premature culling of animals and replacements. It is usually caused due to the effects of infection by bacterial or mycotic pathogens. Pathological changes to milk-secreting epithelial cells due to the inflammatory processes often bring about a decrease in functional capacity. Depending on the pathogen, functional losses may continue into further lactations, which impair productivity and potential weight gain by offspring. Although most infections result in relatively mild clinical or subclinical local inflammation, more severe cases can lead to agalactia or even profound systemic involvement resulting in death. Mastitis has been reported in almost all domestic mammals and has a worldwide geographic distribution. Climatic conditions, seasonal variation, density and housing of livestock populations, and husbandry practices may affect the incidence and etiology. However, it is of greatest frequency and economic importance in species that primarily are producers of milk, particularly dairy cattle.

Key words: Mastitis, Bovine, Etiology, Losses, Control

Introduction

Mastitis, an inflammatory reaction of mammary gland is the most dreaded disease for dairy farmers because of reduced milk production, increased treatment costs, labour, milk discarding following treatment, death and premature culling (Yang et al., 2011). Annual losses in the dairy industry due to mastitis have been approximately 2 billion dollars in USA and 526 million dollars in India due to subclinical mastitis where as clinical mastitis is responsible for approximately 70% and 30% respectively of dollar losses (Varshney and Naresh, 2004). The decrease in milk production per cow due to the clinical and subclinical prevalence of mastitis is usually recognized as the main pathway in causing economic losses due to this disease (DeGraves and Fetrow, 1993). Other production effects that cause economic loss are mainly reduced longevity and short term lethality, the negative effects on body weight and feed intake, penalties or loss of premiums related to the somatic cell count of bulk tank milk and the milk withdrawn during and after antibiotic treatment, and also the money spent on its treatment. Mastitis has been recently reported to have a detrimental effect on reproductive performance in lactating dairy cows (Hertl et al., 2010 and Nava-Trujillo et al., 2010).

Mastitis is the inflammation of parenchyma of mammary gland which is characterized by physical, chemical and usually bacteriological changes in milk and pathological changes in glandular tissues
(Radostits et al., 2000). Clinical mastitis is characterized by sudden onset, swelling and redness of the udder, pain and reduced and altered milk secretion from the affected quarters. The milk may contain clots or flakes or become watery in consistency accompanied by fever, depression and anorexia. The subclinical mastitis is characterized by having no visible signs either in the udder or in the milk, but the milk production decreases and the somatic cell count (SCC) increases, having greater impact in older lactating animals than in first lactation heifers. A negative relationship generally exists between SCC and the milk yield. Milk from normal uninfected quarters generally contain below 200,000 somatic cells/ml. A value of SCC above 300,000 is abnormal and an indication of inflammation in the udder.

**Epidemiology**

Prevalence of mastitis in dairy herds is about 50% of cows and a quarter infection rate is about 25%. The average annual incidence rate of clinical mastitis in individual herds ranges from 10-12 % in most herds (Miltenburg et al., 1996) but higher values, ranging from 16-65% also occur in some herds (Firat, 1993). The greatest risk of first acquiring mastitis occurs early in lactation, usually in the first 50 days (Sargeant, 1998). The risk of clinical mastitis also increases with increasing parity (Sargeant, 1998). In beef herds, 32-37 % of cows and 18% of quarters may have intramammary infection which has a significant negative effect on new born calf weaning weights (Simpson et al., 1995). Case fatality rates vary widely depending upon the causative agent. There is a definite impact of seasonal variation on incidence of bovine mastitis and the microbes associated with it (Ranjan et al., 2011). The pattern of mastitis occurrence is significantly increasing in both cattle and buffaloes which is a major challenge for field veterinarians and researchers (Awale et al., 2012).

**Animal risk factors**

Following are the factors that make animals susceptible to mastitis:

1. Age: The prevalence of infection increases with age, peaking at 7 years.
2. Stage of lactation: Infection rate is more in initial and last stage of lactation.
3. Milk yield: High yielders are more commonly affected than low yielders.
4. Breed: Incidence is more in exotic and crossbreds than the zebu cows. In Holstein Friesian cows, incidence has been seen to be more than in Jersey cows.
5. Milking rate and udder morphology: High milking rate and large teat canal diameter have been associated with increased incidence of intramammary infection (Slettbakk et al., 1990). Differences in udder depth, teat length, teat shape and teat orifice morphology are also thought to be associated with differences in mastitis.
6. Herd size: The incidence is more in large sized herd.
7. Nutrition: Heavy protein feeding may be a predisposing factor. Vit. E, vit. A and selenium may be involved in resistance to certain types of mastitis (Erskine, 1993).
8. Hygiene: Bad hygiene and sanitation help in bacterial multiplication.

**Etiology**

138 different pathogens have been identified as a cause of mastitis (Watts, 1988). Variety of pathogenic organisms causing bovine mastitis (Hawari and Fazwi, 2008) are commonly divided into those causing contagious mastitis, which are spread from infected quarters to other quarters and cows; those that are normal teat skin inhabitants and cause opportunistic mastitis; and those causing environmental mastitis, which are usually present in the cow's environment and reach the teat from that source.

1. Contagious mastitis pathogens: There are many contagious mastitis pathogens. The most common are *Staphylococcus aureus* and *Streptococcus agalactiae*. The usual source of contagious pathogens is the infected glands of other cows in the herd; however, the milker’s hands can act as a source of *S. aureus*. *S. aureus* and *S. agalactiae* have been isolated from subclinical bovine mastitis milk samples (Ali et al., 2011). The predominant method of transmission is from cow to cow by contaminated common udder wash cloths, residual milk in teat cups and inadequate milking equipment. *Mycoplasma bovis* is a less common cause of contagious mastitis; it causes outbreaks of clinical mastitis that do not respond to therapy and are difficult to control. Most outbreaks of *M. bovis* are associated with recent introductions of new animals into the herd. Common contagious pathogens have been reported to infect 7 to 40% of all cows (Sharif and Muhammad, 2009).

2. Teat skin opportunistic mastitis pathogens: Teat skin opportunistic pathogens (Bacterial pathogens that normally reside on the teat skin) have the ability to create an intramammary infection via ascending infection through the streak canal. Coagulase - negative *Staphylococci* are the most common teat skin opportunistic mastitis pathogens.

3. Environmental mastitis pathogens: Environmental mastitis is associated with three main groups of pathogens viz. the *Coliforms* (particularly *E. coli* and *Klebsiella* spp.), environmental *Streptococcus* spp. and *Arcanobacterium pyogenes*. The source of these pathogens is environment of the cow. The major method of transmission is from environment to the cow by inadequate management of the environment. Examples include wet bedding, dirty lots, milking wet udders, inadequate pre milking udder and teat preparation, housing systems that allow teat injuries and poor fly control. Coliform organisms are a common cause of clinical mastitis, occasionally in a severe peracute form. The most prevalent species of *Streptococcus* are *Streptococcus uberis* and *Streptococcus dysgalactiae*. *A. pyogenes* is an important seasonal cause of mastitis in dry cows and late pregnant heifers in some parts of the world. Intramammary infections with *A. pyogelies* are severe and the gland is almost always lost to milk production.

Pathogens causing mastitis in cattle can also be divided into major pathogens and minor pathogens:
1. Major pathogens: Major pathogens include those that cause clinical mastitis and are further classified as contagious pathogens and environmental pathogens. Contagious pathogens include *S. agalactiae*, *S. aureus* and *M. bovis* whereas environmental pathogens include environmental *Streptococcus* and Coliforms species. Environmental *Streptococcus* species include *S. uberis* and *S. dysgalactae*, which are the most prevalent; less prevalent is *S. equinus* (formerly referred to as *S. bovis*). The environmental coliforms include the Gram-negative bacteria *E. coli*, *Klebsiella spp.* and *Enterobacter spp.* A. pyogelles mastitis can be an important problem in some herds.

2. Minor pathogens: Minor pathogens are those that normally cause subclinical mastitis and less frequently cause clinical mastitis. They include the coagulase negative *Staphylococcus* spp. such as *Staphylococcus hyicus* and *Staphylococcus chromogenes*, which are commonly isolated from milk samples and the teat canal; *Staphylococcus xylosus* and *Staphylococcus sciu* found free-living in the environment; *Staphylococcus warneri, Staphylococcus simulans* and *Staphylococcus epidermidis* as part of the normal flora of the teat skin. The prevalence of Bovine mastitis coagulase-negative *Staphylococcus* spp. is higher in first-lactation heifers than cows and higher immediately after calving than in the remainder of lactation. *C. bovis* is also a minor pathogen; it is mildly pathogenic and the main reservoir is the infected gland or teat duct. However, in some herds, *C. bovis* appears to be a common cause of mild clinical mastitis. *C. bovis* spreads rapidly from cow to cow in the absence of adequate teat dipping. The prevalence of *C. bovis* is low in herds using an effective germicidal teat dip, good milking hygiene and dry cow therapy. The presence of *C. bovis* in a gland will reduce the likelihood of subsequent infection with *S. aureus*.

Uncommon mastitis pathogens: Many other bacteria can cause severe mastitis, which is usually sporadic and usually affects only one cow or a few cows in the herd. These include *Nocardia asteroides, Nocardia brasiliensis* and *Nocardia jacinina*, *Histophilus somni*, *Pasteurella multocida*, *Pasteurella mannheimia*, *Campylobacter jejuni*, *B. circus* and other Gram-negative bacteria including *Citrobacter* spp., *Enterococcus jaecalis, Enterococcus jaecium*, *Proteus* spp., *P. aeruginosa* and *Serratia* spp. Anaerobic bacteria have been isolated from cases of mastitis, usually in association with other facultative bacteria, e.g. *Peptostreptococcus indolicus*, *Prevotella melaninogenica*, *Eubacterium combesii*, *Clostridium sporogenes* and *Fusobacterium necrophorum*. Fungal infections include *Trichosporon* spp., *Aspergillus jumigatus*, *Aspergillus nidulans* and *Pichia* spp.; yeast infections include *Candida* spp., *Cryptococcus neojormans*, *Saccharomyces* spp. and *Torulopsis* spp. Algal infections include *Prototheca trispora* and *Prototheca zopfii*, *Leptospiras*, including *Leptospira interrogans serovar. pomona*, and especially *Leptospira interrogans hardjo*, cause damage to blood vessels in the mammary gland and gross abnormality of the milk. Some viruses may also cause mastitis in cattle, but they are of little importance.

**Pathogenesis**
Mastitis in dairy animals occurs when the udder becomes inflammed and bacteria invade the teat canal and mammary glands. These bacteria multiply and produce toxins that cause injury to the milk secreting tissue, besides, physical trauma and chemical irritants. These cause increase in the number of leukocytes, or somatic cells in the milk, reducing its quantity and adversely affecting the quality of milk and milk byproducts. The teat end serves as the first line of defense against infection. From outside, a sphincter of smooth muscles surrounds the teat canal which functions to keep the teat canal closed. It also prevents milk from escaping, and bacteria from entering into the teat. From inside, the teat canal is lined with keratin derived from stratified squamous epithelium. Damage to keratin has been reported to cause increased susceptibility of teat canal to bacterial invasion and colonization. The keratin is a waxy material composed of fatty acids and fibrous proteins in the teat. The fatty acids are both esterified and non-esterified, representing myristic acid, palmitoleic acid and linolinic acid which are bacteriostatic. The fibrous proteins of keratin in the teat canal bind electrostatically to mastitis pathogens, which alter the bacterial cell wall, rendering it more susceptible to osmotic pressure. Inability to maintain osmotic pressure causes lysis and death of invading pathogens. The keratin structure thus enables trapping of invading bacteria and prevents their migration into the gland cistern. During milking, bacteria present near the opening of the teat find opportunity to enter the teat canal, causing trauma and damage to the keratin or mucous membranes lining the teat sinus. The canal of a teat may remain partially open for 1-2 hour after milking and during this period the pathogens may freely enter into the teat canal (Jones, 2006). Bacterial pathogens which are able to traverse the opening of teat end by escaping antibacterial activities establish the disease process in the mammary gland which is the second line of defense of the host. In dairy animals, the mammary gland has a simple system consisting of teats and udder, where the bacteria multiply and produce toxins, enzymes and cell-wall components which stimulate the production of inflammatory mediators attracting phagocytes. The severity of inflammatory response, however, is dependent upon both the host and pathogen factors. The pathogen factors include the species, virulence, strain and the size of inoculum of bacteria, whereas the host factors include parity, the stage of lactation, age and immune status of the animal, as well as the somatic cell count. Neutrophils are the predominant cells found in the mammary tissue and mammary secretions during early stage of mastitis and constitute >90% of the total leukocytes. The phagocytes move from the bone marrow toward the invading bacteria in large numbers attracted by chemical messengers or chemotactic agents such as cytokines, complement and prostaglandins released by damaged tissues. The neutrophils exert their bactericidal effect through a respiratory burst and produce hydroxyl and oxygen radicals that kill the bacteria. During phagocytosis, bacteria are also exposed to several oxygen-independent reactants such as peroxidases, lysozymes, hydrolytic enzymes and lactoferrin. In addition to their phagocytic activities, neutrophils are a source of antibacterial peptides called defensins, killing a variety of pathogens that cause mastitis. Masses of
neutrophils pass between the milk producing cells into the lumen of the alveoli, thus increasing the somatic cell counts and also damaging the secretory cells. Increased number of leukocytes in milk causes increase in the number of somatic cells.

Clots are formed by aggregation of leukocytes and blood clotting factors which may block the ducts and prevent complete milk removal, resulting in scar formation with proliferation of connective tissue elements. This results in a permanent loss of function of that portion of the gland. The milk ducts remain clogged, secretory cells revert to non-producing state, alveoli begin to shrink and are replaced by scar tissue. This helps in formation of small pockets making difficult for antibiotics to reach there and also prevents complete removal of milk (Jones, 2006). Macrophages are the predominant cells found in milk and tissue of healthy involuted and lactating mammary glands. Macrophages ingest bacteria, cellular debris and accumulated milk components. The phagocytic activity of macrophages can be increased in the presence of opsonic antibody for specific pathogens. Because of indiscriminate ingestion of fat, casein and milk components, the mammary gland macrophages are less effective at phagocytosis than are blood leukocytes. Macrophages also play a role in antigen processing and presentation. Conditions which contribute to trauma of mammary gland include: incorrect use of udder washes, wet teats and failure to use teat dips, failure to prepare milking animals or pre – milking stimulation for milk ejection, over milking, insertion of mastitis tubes or teat canulae, injury caused by infectious agents and their toxins and physical trauma.

Types of mastitis & clinical signs

Mastitis can environmental or contagious

A. Environmental mastitis: Environmental mastitis is caused by organisms such as Escherichia coli which do not normally live on the skin or in the udder but which enter the teat canal when the cow comes in contact with a contaminated environment. The pathogens are normally found in faeces bedding materials, and feed. Cases of environmental mastitis rarely exceed 10% of the total mastitis cases in the herd.

B. Contagious Mastitis: Caused by bacteria living on the skin of teat and inside the udder. Contagious mastitis can be transmitted from one cow to another during milking. Contagious mastitis can be divided into three groups:

1. Clinical mastitis: Clinical mastitis during early lactation, markedly and negatively influence the reproductive performance of dairy cows (Yang et al., 2012 and Ahmadzadeh et al., 2009). It is characterized by the presence of gross inflammation signs (swelling, heat, redness and pain). Three types of clinical mastitis exist.

1.1- Peracute mastitis Characterized by gross inflammation, disrupted functions (reduction in milk yield, changes in milk composition) and systemic signs (fever, depression, shivering, loss of appetite and loss of weight).
1.2- **Acute mastitis** Similar to peracute mastitis, but with lesser systemic signs (fever and mild depression).

1.3- **Sub-acute mastitis** In this type of mastitis, the mammary gland inflammation signs are minimal and no visible systemic signs.

2. Sub-clinical mastitis: This form of mastitis is characterized by change in milk composition with no signs of gross inflammation or milk abnormalities. Changes in milk composition can be detected by special diagnostic tests. Subclinical mastitis is always related to low milk production, changes to milk consistency (density), reduced possibility of adequate milk processing, low protein and high risk for milk hygiene since it may even contain pathogenic organisms (Sharma *et al.*, 2011).

3. Chronic mastitis: An inflammatory process that exists for months, and may continue from one lactation to another. Chronic mastitis for the most part exist as subclinical but may exhibit periodical flare-ups sub-acute or acute form, which last for a short period of time. Only relatively few udder infections result in “clinical mastitis” in which the udder is noted to be abnormal and the quality of milk secreted is altered. The vast majority of mastitis is “subclinical”. The number of somatic cells in the milk, an indicative of the inflammatory response, may be elevated and bacterial can be cultured from the milk. For every case of clinical mastitis, there are 20-40 times as many cases of subclinical mastitis. Subclinical mastitis may progress and develop into clinical cases, or they may persist for a long time at a subclinical level. Without somatic cell counting or bacterial cultures, subclinical mastitis goes unnoticed.

**Effect of mastitis on milk composition**

Mastitis reduces milk yield and alters milk composition. Casein, the major milk protein of high nutritional quality, declines and lower quality whey proteins increase which adversely affects the quality of dairy products such as cheese. Serum albumin, immunoglobulins, transferrin and other serum proteins pass into milk because of increased vascular permeability. Jones (2006) has reported that with higher somatic cell count (SCC), the concentrations of serum albumin and immunoglobins are increased which reduces heat stability of mastitis milk and pasteurization gives lower grade scores after storage. Also there is a decrease in calcium absorption from blood into milk, resulting in impaired coagulation characteristics of mastitis milk. Haenlein *et al.* (1973) reported a significant decrease in casein content when SCC in milk exceeded 500,000/ml. The milk proteins breakdown can occur in milk from animals with clinical or subclinical mastitis due to the presence of proteolytic activity by more than 2-fold during mastitis. Plasmin and enzymes derived from somatic cells can cause extensive damage to casein in the udder before milk removal. Mastitis increases conductivity of milk and sodium, and chloride concentrations are elevated. Potassium, normally the predominant mineral in milk, declines and because most of the calcium in milk is associated with casein, the disruption of casein contributes to lowered calcium in milk. The reduced lactose concentration is one important factor for impaired acidification properties of milk with
elevated SCC, after adding starter cultures (Schallibaum, 2001). Comparison of mastitic milk with normal milk is given in Table 1 (Jones, 2006).

<table>
<thead>
<tr>
<th>Constituent</th>
<th>Normal Milk</th>
<th>Mastitis milk with high SCC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fat</td>
<td>3.5</td>
<td>3.2</td>
</tr>
<tr>
<td>Lactose</td>
<td>4.9</td>
<td>4.4</td>
</tr>
<tr>
<td>Total Protein</td>
<td>3.61</td>
<td>3.56</td>
</tr>
<tr>
<td>Total Casein</td>
<td>2.8</td>
<td>2.3</td>
</tr>
<tr>
<td>Whey Protein</td>
<td>0.8</td>
<td>1.3 ↑</td>
</tr>
<tr>
<td>Serum Protein</td>
<td>0.02</td>
<td>0.07 ↑</td>
</tr>
<tr>
<td>Lactoferrin</td>
<td>0.02</td>
<td>0.1 ↑</td>
</tr>
<tr>
<td>Immunoglobulin</td>
<td>0.1</td>
<td>0.60 ↑</td>
</tr>
<tr>
<td>Sodium</td>
<td>0.057</td>
<td>0.105 ↑</td>
</tr>
<tr>
<td>Chloride</td>
<td>0.091</td>
<td>0.147 ↑</td>
</tr>
</tbody>
</table>

**Somatic cell count (SCC)**

Somatic cells are mainly milk-secreting epithelial cells that have been shed from the lining of the gland and white blood cells (leukocytes) that have entered the mammary gland in response to injury or infection (Anonymous, 2009). Somatic cells are normal constituent of milk and only when they become excessive do they indicate a problem. Somatic cells are composed of leucocytes (75%) and epithelial cells (25%). Leucocytes (white blood cells) increase in milk, in response to infection or injury while increase in epithelial cells is the result of infection or injury. The number of cells reflects the severity of mastitis. Somatic cells are expressed either as cells/ml of milk (Somatic Cell Count, SCC) or as Somatic Cell Score (SCS). In milk obtained from a healthy mammary gland, the SCC is normally lower than 1×10^5 cells/ml, while in bacterial infection it can increase to above 1×10^6 cells/ml (Bytyqi et al., 2010). During winter somatic cell counts are generally lowest and highest during the summer season (Khate and Yadav, 2010). To be used for human consumption, milk must have less than 750,000 SCC. Milk markets rely routinely on SCC to help ensure good quality milk and usually pay a premium for milk with low SCC. This is because high SCC causes a rise in whey protein and a decrease in casein resulting in a considerable lower cheese yields. A shorter shelf life and adverse milk flavors are other consequences of high SCC. Higher counts are also associated with decreased production. When individual cows have a SCC between 2,50,000 – 3,00,000 (SCS = 4) that usually indicates a subclinical mastitis caused by major pathogens. Elevated SCC levels over a long period of time suggests that affected quarters have been in a state of chronic inflammation and could adversely affect development of milk-producing tissues (Nickerson, 2009). Timely detection of mastitis should be done based on SCC values; otherwise subclinical mastitis could develop into a clinical disease (Sandgren et al., 2008).

**Economic losses due to mastitis**
Mastitis results in economic loss for producers by increasing the costs of production and by decreasing productivity. The premature culling of potentially profitable cows because of chronic mastitis is also a significant loss. The component economic losses 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 first trimester abortions due to clinical mastitis (Risco et al., 1999) and cost of control measures. However production losses contributed major part to the economic losses (Huijps et al., 2008 and Nielsen, 2009). There are additional costs such as antimicrobial residues in milk from treated cows, milk quality control, dairy food manufacturing, and nutritional quality of milk, degrading of milk supplies due to high bacteria or SCC, and interference with the genetic potential of some cows from early involuntary culling because of chronic mastitis. The total annual cost of mastitis in the dairy cattle population is estimated to be 10% of the total value of farm milk sales, and about two-thirds of this loss is due to reduced milk production in subclinically affected cows. Losses due to mastitis may even be higher in developing countries because standard mastitis control and prevention practices recommended by national mastitis council of USA are not being carried out promptly (Sharif and Muhammad, 2008). Estimated loss due to mastitis is shown in Table 2 (Anonymous, 1996). The production and economic losses are commonly divided into those associated with subclinical and clinical mastitis.

**Losses due to subclinical mastitis:** Total milk losses from quarters affected with subclinical mastitis have been estimated to range from 10-26 % (DeGraves and Fetrow, 1993). Most estimates indicate that on average an affected quarter results in a 30% reduction in productivity, and an affected cow is estimated to lose 15% of its production for the lactation. In addition to these losses, there is an added loss of about 1 % of total solids by changes in composition (fat, casein, and lactose are reduced and glycogen, whey proteins, pH, and chlorides are increased), which interferes with manufacturing processes, and other losses include increased culling rates and costs of treatment. Approximately 75% of the economic loss from subclinical mastitis is attributable to loss of milk production. Other costs include discarding milk from treated cows, drug costs, veterinary costs, labor and loss of genetic potential of culled cows.

<table>
<thead>
<tr>
<th>Source of Loss</th>
<th>Loss per Cow ($)</th>
<th>Percent of Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reduced Production</td>
<td>121.00</td>
<td>66.0</td>
</tr>
<tr>
<td>Discarded Milk</td>
<td>10.45</td>
<td>5.7</td>
</tr>
<tr>
<td>Replacement Cost</td>
<td>41.73</td>
<td>22.6</td>
</tr>
<tr>
<td>Extra Labour</td>
<td>1.14</td>
<td>0.1</td>
</tr>
<tr>
<td>Treatment</td>
<td>7.36</td>
<td>4.1</td>
</tr>
<tr>
<td>Veterinary Services</td>
<td>2.72</td>
<td>1.5</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>184.40</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>
Assumptions: One-third of cows infected in average of 1.5 quarters; milk loss 856 pounds per infected quarter; milk price $12.07 per hundred weights.

Losses due to clinical mastitis: Clinical mastitis results in marked decreases in milk production, which are much larger in early than late lactation. Milk production losses are also greater in cows with multiple lactations than first-lactation cows, and clinical mastitis also decreases the duration of lactation and increases the likelihood of culling. Clinical cases of brief duration that occur after the peak of lactation affect milk production very little but can induce abortion during the first 45 days of gestation (Risco et al., 1999). In the National Animal Health Monitoring System of dairy herds in the US, clinical mastitis alone was the most costly disease identified, at a loss to the producer of $27-50 per cow per year (DeGrave and Fetrow, 1993). The costs of clinical mastitis and mastitis prevention in dairy herds have been estimated, based on monitoring 50 dairy herds over 1 year (Miller et al., 1993). Mean incidence of clinical mastitis was 39 cases/100 cow-years; each clinical case cost $38/cow-year, with a mean cost per clinical episode of US$107. Prevention of mastitis cost $14.50/cow-year (Hoblet et al., 1991). Lost milk production was estimated at $14.85/cow year, which does include the losses associated with subclinical mastitis. Economic loss to mastitis in the United States is estimated to be approximately $185/cow annually. Heikkilä (2012) has suggested giving more emphasis to the hidden costs, especially the high cost of premature culling should be underlined, in order to decrease losses due to clinical mastitis.

Diagnosis

Diagnosis of clinical mastitis is based on the appearance of abnormally appearing milk. Milk may be off color, watery, bloody or have the appearance of serum. Abnormal milk may also contain varying amounts of puss and clots. The amount of swelling, severity of pain and the overall appearance of the cow will indicate the severity of infection and serve as a guide for the course of treatment. Diagnosis of subclinical infection is more problematic since the milk appears normal but usually has an elevated somatic cell count. Diagnosis of subclinical mastitis can be made in a variety of ways including direct measurement of the somatic cell count (SCC) level or indirectly by performing a California Mastitis Test (CMT) on suspected quarters. Milk culture of suspected quarters or cows (composite samples) will identify the presence of mastitis pathogens but will not provide a measure of the degree of inflammation associated with the infection. Individual cow SCC will provide a determination of the level of infection within the herd. Bulk tank somatic cell counts (BTSCC) are performed routinely as a indication of milk quality but the BTSCC can be used to monitor the level of udder health when the bulk tank scores are monitored over time. For diagnosing mastitis physical examination of the udder should be done to observe any deviation from normal shape, size, colour consistency of udder. Signs of inflammation viz. hot, swelling, pain, redness and loss of function should be clinically assessed. Diagnostic tests of milk include Strip Cup Test, Bromothymol Blue Test, Bromocresol Purple Test, Chloride Test, Catalase Test, Hotist Test, California
Mastitis Test, White Side Test, Modified California Mastitis Test, CAMP Test, Draminski Mastitis Detector and Electrical Conductivity Test. Direct tests like cultural examination, biochemical tests, animal inoculation test, serological test, etc. Molecular diagnostic technologies have also been as a routine tool in diagnosing mastitis pathogens (Gurjar, 2012).

**Treatment**

While mastitis cannot be totally eliminated from a herd, the incidence can be held to a minimum. The key elements in the control of mastitis include: sound husbandry practices and sanitation, post-milking teat dipping, treatment of mastitis during non-lactating period, and culling of chronically infected animals. The efficacy of therapy during the non-lactating period has proved to be superior to that which can be achieved during lactation. Monitoring of somatic cell counts and prompt identification and treatment of mastitis in dairy animals help in the reduction of mastitis. Dry animal therapy can eliminate 70% of environmental streptococcal infections. The fundamental principle of mastitis control is that the disease is controlled by either decreasing the exposure of the teat to potential pathogens or by increasing resistance of dairy animals to infection. Jones (2006) has suggested approaching the treatment in the same way a surgeon approaches surgery. Wash hands with soap and water, wash teats and udder in sanitizing solution, thoroughly dry teats and udder with individual towels, dip teats in an effective germicidal teat dip. Allow 30 seconds of contact time before wiping off teat dip with an individual towel; thoroughly scrub the teat end with a cotton swab soaked in alcohol. If all four quarters are being treated, start by cleaning the teat farthest from you and work toward the closest teat, use commercial antibiotic products in single dose containers formulated for intramammary infusion. Treat teats nearest to you first, then those farthest away to prevent contaminating clean teat ends. Dip teats in an effective germicidal teat dip after treatment. Awandkar et al. (2009) have suggested the necessity of microbiological and antibiogram studies for treatment and control of bacterial mastitis.

**Control of mastitis**

Prevention is the key in mastitis control. A control program should emphasize factors that reduce the rate of new infections. New infections are controlled by adopting measures like proper milking procedures, improved milking hygiene and housing management (Arnold, 2011). A combination of preventive measures and therapeutic use of antibiotics will markedly reduce the incidence of mastitis. The prevention of mastitis can be achieved by:

1. Proper milking hygiene: Bacteria transmit to the uninfected from the contaminated hands of the milker. Thus the milker's hands should be washed thoroughly with disinfected soaps before milking and clinically infected cows should be milked last. Teats should be cleaned and dried before milking. Poor hygienic conditions can lead to *E.coli* mastitis as the udder is infected through teat canal (Sumathi et al., 2008).
2. Milking machine: Should function and operate properly. Vacuum level in the milking unit should be between 275 and 300 mm of mercury with little fluctuation. The vacuum regulator should be kept clean and checked regularly.

3. Dipping the teats after milking: Teat dipping does not reduce existing infection. However, the rate of new infection can be reduced by up to 50% when suitable disinfectant is used to immerse or spry the teats.

4. Dry treatments: Incidence of mastitis during the dry period can be considerably reduced by effective use of antibiotic infused in each quarter of the udder at the last milking of lactation. Dry cow therapy is the best way to cure chronic and subclinical mastitis that are difficult to treat successfully during lactation.

5. Culling of chronically infected cows: This is an effective method because in most herds only 6-8% of all cows account for 40-50% of all clinical mastitis.

6. Nutrition: Deficiencies of selenium and vitamin E in the diet have been associated with an increased rate of new mammary infections.

The udder should be washed thoroughly in a sanitizing solution with individual paper towels and after milking, the teats should be immersed in appropriate teat-dip solution. The teat cup assembly, milk pipes and other utensils should be cleaned and sanitized between each milking. After milking, the sphincter muscle surrounding the teat canal remains dilated for a varying period of time facilitating invasion of the teat canal by bacteria. Thus teat dips are most effective when applied immediately after milking machine is removed. Cows are also exposed to mastitis organisms via the milking machine when milked after a cow affected with clinical or sub clinical mastitis.

References


