Review on the current status of bovine mastitis and its risk factors in dairy farms of Ethiopia

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ABSTRACT

Mastitis is an inflammatory disease condition of the udder affecting milk production negatively and having a serious impact on the economy of dairy enterprise. 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. Pathologic changes to milk-secreting epithelial cells from the inflammatory process 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. In Ethiopia, the available information indicated that bovine mastitis is one of the most frequently encountered diseases of dairy cows and continues to be the most economically important disease of dairy cattle, due to the expense of antibiotic treatment, along with the associated costs of decreased milk production and decreased fertility or, in cases where antibiotic treatment is ineffective, culling or death. Field surveys of major livestock diseases in Ethiopia have ranked mastitis as number one disease of dairy animals. Therefore, conducting research on its prevalence and incidence will contribute a lot to designing appropriate preventive measures and treatment regimen in the specific dairy farm.

Key words: Bovine mastitis, dairy farms, Ethiopia, risk factors.

INTRODUCTION

Ethiopia is believed to have the largest livestock population in Africa. This livestock sector has been contributing considerable portion to the economy of the country and still promising to rally round the economic development of the country. The Ethiopian total cattle population is estimated to be about 56.71 million. Out of this, the female cattle constitute about 55.45% and the remaining 44.55% are male cattle. 98.66% of the total cattle in the country are local breeds and the remaining are hybrid and pure exotic breeds that accounted for about 1.19 and 0.14%, respectively (CSA, 2015). However, milk production often does not satisfy the country’s requirements due to a multitude of factors. Mastitis is among the various factors contributing to reduced milk production (Biffa et al., 2005). Bovine mastitis is an infectious inflammation or irritation of the mammary glands that interferes with the
normal flow and quality of milk (Pyorala, 2003). Pathogens invade the mammary glands, develop and multiply, producing some toxic substances that results in inflammation, reduced milk production and altered milk quality, leading to a clinical condition known as mastitis, which is either sub-clinical or clinical (Rall et al., 2013). Mastitis causes reduced milk production, not only at the occurrence of the mastitis but throughout the rest of the lactation (Hagnestam et al., 2007), and increases the risk of new cases of mastitis and the risk of culling (Schneider et al., 2007). Schneider et al. (2007) revealed that the welfare of cow is negatively influenced by mastitis as it can induce pain and even cause death.

Mastitis is characterized by physical, chemical and bacteriological changes in the milk and pathological changes in the glandular tissue of the udder. The most important changes include discoloration, presence of clots and presence of large number of leukocytes (Sharma et al., 2007). It is also defined as inflammation of mammary gland parenchyma, which is caused by bacteria and its toxins (Sharma et al., 2006). The bacterial contamination of milk from affected cows render it unfit for human consumption and provide a mechanism of spread of diseases, such as tuberculosis, sore-throat, Q-fever, Brucellosis, Leptospirosis etc. and has zoonotic importance (Sharif et al., 2009).

Bovine intramammary infections are single, most costly disease on dairy farms. Depending on individual circumstances, each case of clinical mastitis costs approximately two hundred dollar, accounting for treatment costs and lost production (Fetrow et al., 2000). Mastitis continues to be the most costly disease of dairy animals. Field surveys of major livestock diseases have ranked mastitis as number one disease of dairy animals. Mastitis is the most important and expensive disease of dairy industry. In dairy cattle, it results in severe economic losses from reduced milk production, treatment cost, increased labor, milk withheld following treatment and premature culling (Seegers et al., 2003). It is recognized that if this disease is diagnosed in early stages, the greater portion of this loss can be avoided. It is undoubtedly the most important disease with which the dairy industry is encountered.

Mastitis is known to be a complex and costly disease of dairy cows that results from the interaction of the cow and environment including milking machine and microorganism (Azmi et al., 2008). It has been known to cause a great deal of loss or reduction of productivity to influence the quality and quantity of milk yield and to cause culling of animals at an unacceptable age. Moreover, due to its latent form, heavy financial losses and great nutritional and technological impacts can result. This is because valuable components of the milk, such as lactose, fat and casein, are decreased, while undesirable components, such as ions and enzymes are increased, making the milk unfit for processing technology (Girma, 2001).

Subclinical mastitis is a major problem affecting dairy animals all over the world. It causes enormous losses for breeders and consequently influences the national income of the country (McDougall et al., 2009). According to Getahun et al. (2008), economic losses are due to loss in milk production, discarding abnormal milk and milk withheld from cows treated with antibiotics, degrading of milk quality and price due to high bacterial or somatic cell count (SCC), costs of drugs, veterinary services and increased labor costs, increased risk of subsequent mastitis, culling mastitic cows, herd replacement, and problems related to antibiotics residues in milk and its products. Its incidence usually depends on exposure to pathogens, effectiveness of udder defense mechanisms, and presence of environmental risk factors, as well as interactions between these factors (Oviedo-Boyso et al., 2007; Suryasathaporn et al., 2000).

Mastitis, as a disease, has received little attention; especially the sub clinical form of mastitis in Ethiopia (Mekonnen and Tesfaye, 2010; Hunderra et al., 2005). Owing to the heavy economical implications involved and the inevitable existence of latent infection, mastitis is obviously an important factor that limits dairy production. Until now, efforts have only been so far concentrated on the treatment of clinical cases and not in minimizing the risk factors through appropriate methods of prevention and control. Therefore, the objective of this study is to review the current status of bovine mastitis and major risk factors associated with bovine mastitis in Ethiopia.

ETIOLOGY OF MASTITIS

More than 135 different pathogenic microorganisms have been identified as causative agents of bovine mastitis. Bacteria, fungi and yeasts may all play a role; but of these, bacteria have by far the largest part. Staphylococci, streptococci and members of the Enterobacteriaceae are responsible for the majority of infections (Bradley, 2002). Although about 20 to 35% of clinical mastitis cases are of unknown etiology (Wellenberg et al., 2002), it is widely accepted that bovine mastitis is mainly bacterial in origin. It can be classified as contagious or environmental. Contagious microorganisms are usually found on the udder or teat surface of infected cows and are the primary source of infection between uninfected and infected udder quarters, usually during milking. The organisms that fit into this category include: Staphylococcus aureus (coagulase-positive staphylococci), Streptococcus agalactiae and the less common sources of infection caused by Corynebacterium bovis and Mycoplasma bovis. They all adapt and survive in the udder, causing subclinical infections.

A large number of Gram-positive and Gram-negative species are in a cow’s environment and they cause clinical
or subclinical infections in the udder and fall into a descriptive category known as environmental mastitis pathogens such as Streptococcus uberis, Streptococcus equinus, Enterococcus faecalis and Enterococcus faecium are Gram-positive species. Gram-negative species include Escherichia coli, Klebsiella spp., Enterobacter spp. and Pseudomonas spp. Environmental pathogens are not well adapted to survive within the udder and, instead, they multiply rapidly following invasion, evoke a swift immune response and are eliminated. They require moisture, favorable pH and organic material for survival and they enter the gland through the teat canal. Environmental pathogens reside in soil, bedding materials, manure and other organic matter. Mastitis caused by environmental organisms is essentially opportunistic in nature and becomes established if the immune system of the host is compromised or if sanitation and hygiene is not adequately practiced (Schukken et al., 2005).

**EPIDEMIOLOGY**

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. Mastitis continues to be the most economically important disease of dairy cattle, due to the expense of antibiotic treatment, along with the associated costs of decreased milk production and decreased fertility or, in cases where antibiotic treatment is ineffective, culling or death (Radostitits et al., 2007).

Except for outbreaks of Mycoplasma, clinical mastitis in most dairy herds is caused by environmental pathogens. In addition, many clinical mastitis cases are transient, especially those that are initial episodes for a cow and quarter. Thus, from an epidemiologic perspective, assessment of clinical mastitis is based on incidence and not prevalence. Almost any microbe, that can opportunistically invade tissue and cause infection, can cause mastitis. However, most infections are caused by various species of streptococci, staphylococci, and gram-negative rods, especially lactose-fermenting organisms of enteric origin, commonly termed coliforms. From an epidemiologic standpoint, the primary sources of infection for most pathogens may be regarded as contagious or environmental (Jones, 2010).

All dairy herds have cows with subclinical mastitis; however, the prevalence of infected cows varies from 5–75%, and quarters from 2–40% worldwide. Many different pathogens can establish a chronic infection in which clinical signs of mastitis will manifest only occasionally. The primary focus of most subclinical mastitis programs is to reduce the prevalence of the contagious pathogens, such as Strep. agalactiae and Staphylococcus aureus, as well as other gram-positive cocci, most notably Streptococcus dysgalactiae (which may also be contagious or an environmental pathogen), Streptococcus uberis, enterococci, and numerous other coagulase-negative staphylococci, including Staphylococcus hyicus, Staphylococcus epidermidis, Staphylococcus xylosus, and Staphylococcus intermedius. Herds have been identified that have considerable subclinical mastitis caused by gram-negative rods, such as Klebsiella sp., Serratiamarcenscens, Pseudomonas aeruginosa, and other atypical pathogens, such as mycotic and algal microbes (Jones, 2010).

According to Shearer and Harris (2003), sub-clinical mastitis is important due to the fact that it is 15 to 40 times more prevalent than the clinical form (for every clinical case of mastitis there will be 15-40 sub clinical cases). It usually precedes the clinical form, is of longer duration, difficult to detect, adversely affects milk quality and production and constitutes a reservoir of microorganisms that lead to infection of other animals within the herd. While clinical cases often receives the most direct attention, there is ample evidence which suggests that sub-clinical cases, as detected by elevated milk somatic cell count, can negatively affect production, but the estimated magnitude of this loss varies greatly between studies. The average loss appears to be approximately 0.5 kg/day for every two-fold increase in milk somatic cell count. Sub-clinical infection also acts as reservoirs of pathogen within the herd and may develop into clinical mastitis (Mungube et al., 2004).

The main types of mastitis depending on mode of transmission

**Cow-associated (contagious mastitis)**

For contagious pathogens, adult lactating cattle are most at risk of infection, either while lactating or during the dry period. The primary reservoir of infection is the mammary gland; transmission occurs at milking with either milkers’ hands or milking equipment acting as fomites. Teat-end dermatitis caused by the horn fly, Haematobia irritans, which can harbor Staph. aureus, has been associated with increased risk of infection in heifers, especially in warmer climates. For the contagious pathogens and coagulase-negative staphylococci, there is little or no seasonal variation in incidence of infection. Except for Mycoplasma spp, which may spread from cow to cow through aerosol transmission and invade the udder subsequent to bacteremia, contagious pathogens are spread during milking by milkers’ hands or the liners of the milking unit. Species that use this mode of transmission include Staph. aureus, Strep. agalactiae, and Corynebacterium bovis (Jones, 2010).

Mastitis is most often transmitted by repetitive contact with the milking machine, and through contaminated hands
or materials. The main bacteria causing contagious mastitis are *Staph. aureus* and *Strep. agalactiae*. They mostly live inside udders or on teat skin and are spread either by splashes of infected milk or sprays during stripping, on milkers’ hands or teatcup liners, and by cross flow of milk between teat cups. *Strep. agalactiae* bacteria tend to locate in duct areas of the udder where antibiotics are effective. *Strep. agalactiae* are very sensitive to penicillin, so there are relatively high cure rates. However, *Staph. aureus* form pockets of infection protected from antibiotics by scar tissue (Bradley and Green, 2010).

**Environmental mastitis**

Soil, manure, bedding, calving pads and water host bacteria cause environmental mastitis. They also occur on parts of the cow other than the mammary gland. Housed cows tend to be more at risk than grazing cows. The main bacteria are *Strep. uberis* which can sometimes persist, and can spread at milking. The other culprit is *E. coli* which does not thrive in the lactating udder and often the infections do not persist. Transition and post-calving cows are very susceptible to these infections because their natural defenses are low. Large infections of environmental mastitis bacteria can contaminate teats, especially if udders are wet and exposed to mud and manure, such as when animals lie down during calving. Most cases of environmental mastitis are clinical but subclinical cases occur too (Jones, 2010).

**PATHOGENESIS**

Mastitis in dairy animals occurs when the udder becomes inflamed 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 by-products. 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 (Hamadani et al., 2013).

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 h after milking and during this period the pathogens may freely enter into the teat canal (Jones, 2006).

Mastitis is induced when pathogenic microorganisms enter the udder through the teat canal, overcome the cow’s defense mechanisms, begin to multiply in the udder, and produce toxins that are harmful to the mammary gland. Mammary tissue is then damaged, which causes increased vascular permeability. As a result of this, milk composition is altered: there is leakage of blood constituents, serum proteins, enzymes, and salts into the milk; decreased synthesis of caseins and lactose; and decreased fat quality (Osteras, 2000). The extent of these changes is determined by the severity of the infection (Pyorala, 2003).

When bacteria enter the udder, the cow sends large numbers of white blood cells to surround and destroy the infection. A small number of udder tissue cells are also shed into milk. Body cells are sometimes called somatic cells (somatic means ‘body’) and their number stays stable after the milk leaves the cow, regardless of filtration or cooling. The concentration of all body cells in milk is called its Individual Cow Cell Count (ICCC) or Somatic Cell Count (SCC). The concentration of cells varies throughout a milking, so the sample tested should be collected throughout the milking. Concentrations also vary between morning and evening milkings, especially with unequal milking intervals. Evenings milking have higher cell counts. The ICCC indicates the likelihood of subclinical mastitis. Uninfected cows generally have ICCC levels of below 150,000 cells/mL. If a cow has had any ICCC above 250,000 during lactation (a peak of 250,000 or more), she is likely to be infected at drying-off and require Dry Cow Treatment. *Strep. agalactiae* infections are easier to treat than *Staph. aureus*; so higher cell counts do not always mean hard-to-treat. High ICCC levels not associated with infection can occur for up to 20 days post-calving. Stress can lift ICCC levels in cows. Elevated ICCC levels can also occur in late lactation when milk volume is low and cells are more concentrated (Hamadani et al., 2013).

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 (Hamadani et al., 2013).

CLINICAL SYMPTOMS OF MASTITIS

Mastitis can be either clinical or subclinical. Clinical cases give rise to visible symptoms. Mild clinical mastitis (CM) causes flakes or clots in the milk, whereas severe cases are associated with heat, swelling and discoloration of the udder, as well as abnormal secretion. Severe CM can also exhibit systemic reactions, such as fever and loss of appetite. Mastitis can exist in the absence of visible signs of infection, and is then referred to as subclinical mastitis (SCM). SCM is the most prevalent form of mastitis (Akers, 2002). The duration of infection further classifies mastitis as acute or chronic manifestations, where a sudden onset defines acute cases and chronic mastitis is characterized by an inflammatory process that lasts for months and results in progressive development of fibrous tissue (Mungube et al., 2005).

Clinical mastitis

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 have clots, flakes or of watery in consistency and accompanied by fever, depression and anorexia. Clinical mastitis is characterized by visual clots or discolorations of the milk, often in combination with tender and swollen udder, sometimes in combination with fever, loss of appetite (Bengtsson et al., 2005).

Sub clinical mastitis

The sub clinical mastitis is characterized by having no visible signs either in the udder or in the milk, but the milk production decreases and the 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. There is a plethora of evidence that the dairy cow milk has a natural level of 100,000-150,000 somatic cells/ml and higher SCC indicates secretory disturbance. Subclinical mastitis is characterized by changes in milk composition e.g. somatic cell count (SCC; leukocytes and epithelial cells), and changes in milk pH and ion concentration, without clinical signs of inflammation (Guidry, 2007).

In the healthy lactating mammary gland, the milk SCC is often < 100,000 cells/mL of milk, while the SCC can increase to > 1,000,000 cells/ml of milk during subclinical mastitis. The major factor affecting the SCC at the herd and individual level is the presence of intra mammary infections (IMI) (Radostits, 2007). SCM can be diagnosed by presence of pathogens in bacteriological cultures of milk, but bacteriological sampling is not practically feasible as a routine test. The current standard method of detecting SCM is to measure SCC (Hamann, 2005; Pyorala, 2003).

DIAGNOSIS OF MASTITIS

Qualitative examination of milk

Changes in color of milk can be caused by the presence of blood (red or brownish) or pus (yellow). The consistency may be increased, resulting in thicker, "sticky" milk, or it may be more than usually watery. Flakes and dots are always abnormal. The smell of the secretion may also be altered as a result of mastitis. Mastitis can cause a decline in potassium and lactoferrin. 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 milk. The milk protein continues to undergo further deterioration during processing and storage. Milk from cows with mastitis also has a higher somatic cell count. Generally speaking, the higher the somatic cell count, the lower the milk quality (Jones, 2006).

Mastitis causes considerable changes in milk composition (Table 1). 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 SCC, the concentrations of serum albumin and immunoglobulins 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 impaired coagulation characteristics of mastitis milk. The milk proteins breakdown can occur in milk from animals with clinical or sub clinical 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.
Table 1: Comparison of values (%) of normal milk with that of mastitis milk.

<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 albumin</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>
<tr>
<td>Potassium</td>
<td>0.173</td>
<td>0.157</td>
</tr>
<tr>
<td>Calcium</td>
<td>0.12</td>
<td>0.04</td>
</tr>
</tbody>
</table>


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).

**California mastitis test (CMT)**

Somatic cell counts in milk samples from individual animals can be performed using California Mastitis Test (CMT). The CMT reagent reacts with genetic material of somatic cells present in milk to form a gel. For reliable results, tests should be conducted just before milking after stimulating milk let down and discarding the foremilk. Managers of herds with high SCC may have to cull heavier for mastitis, increase treatments for intramammary infections, increase efforts to avoid antibiotic residues in milk and cull animals, increase cost on facilities or milking equipment, and improve management to reduce the spread of new infections. Thus, emphasis should be on proper milking techniques, improved sanitation, effective use of teat dipping and dry period therapy and maintenance of milking equipment. Lower SCC should result in higher milk yields and better milk quality (Quinn et al., 2002).

**Flow cytometry (FC)**

Flow cytometry (FC) is a method by which physical and chemical characteristics of cells or particles can be measured as they travel in suspension past a sensing point. This method has been developed recently to quantify Somatic Cell Counts in milk, and is particularly good for detecting subclinical mastitis (Tian et al., 2005; Holm et al., 2004).

**Culture method**

The surest way of diagnosing mastitis is by directly isolating and identifying any pathogenic microorganisms which may be present in the milk. This can be achieved by cultural methods and a number of additional determinative tests. To obtain correct results and avoid contamination and hence bias, it is important to work as securely and as accurately as possible under the circumstances. Similarly, the procedure of routine mastitis testing should be standardized and work protocols instituted (Radostits, 2007).

**TREATMENT OF MASTITIS**

Treatment of mastitis should be targeted towards the causative bacteria whenever possible, but in acute situations, treatment is initiated based on herd data and personal experience. Rapid or on-farm bacteriological diagnosis would facilitate the selection of the most appropriate antimicrobial. Treatment protocols and drug selection for each farm should be made by veterinarians familiar with the farm (Sawant et al., 2005; Wagner and Erskine, 2006).

Typically, when clinical mastitis is detected, the cow is milked out and then given an intramammary infusion of antibiotic, that is, infused directly into the infected gland. Prior to intramammary infusion, the teat is cleaned well and the tip of the teat is swabbed with an alcohol swab and allowed to dry for a number of seconds. The antibiotic...
comes in a plastic tube with a plastic infusion cannula at the end. The cannula only goes about half-way up into the streak canal and the antibiotic is expelled from the tube into the teat cistern. After emptying the antibiotic tube, the teat is pinched off and the antibiotic fluid is palpated up into the gland. Clinical mastitis should be treated for at least three days; this recommended treatment duration is longer than label treatments in many countries. All mastitis treatment should be evidence based, that is, the efficacy of each product and treatment length should be demonstrated by scientific studies (Cockcroft and Holmes, 2003).

Antimicrobial agents are the main therapeutic tools for the treatment and control of mastitis. Among main reasons of low efficacy of antibiotic treatment of mastitis cases is the resistance of the bacteria to antimicrobials (De Olivera et al., 2000; Gitau et al., 2003; Haile, 2004). Staph. aureus infections remain the largest mastitis problem of dairy animals. Cure rate with antibiotic therapy during lactation is very low. Many infected animals become chronic cases and have to be culled. Strep. agalactiae respond well to antibiotic therapy and can be eradicated from dairy herds with good mastitis control practices, including teat dipping and dry animal treatment. Strep. dysgalactiae may live almost anywhere: in the udder, rumen, and feces, and in the barn. They can be controlled with proper sanitation and are moderately susceptible to antibiotics (Jones, 2006).

In general, the use of narrow-spectrum antimicrobials is preferable. Prudent use guidelines have been developed which also include antimicrobial treatment of mastitis (Passantino, 2007). First choice antimicrobials for treating mastitis caused by streptococci and penicillin-susceptible staphylococci are β-lactam antimicrobials, particularly penicillin G. Broad-spectrum antimicrobials, such as third or fourth generation cephalosporins should not be used as first alternatives for mastitis, as they may increase emergence of broad spectrum β-lactam resistance. Systemic treatment is recommended in clinical mastitis due to Staph. aureus and in severe cases of coliform mastitis, preferably in combination with IMM treatment (Barkema et al., 2006). Too short duration of standard treatment is probably an important reason for poor cure rates in mastitis therapy. A longer treatment improves cure rates, and duration of treatment should generally be extended in mastitis caused by Staph. aureus and Strep. uberis (Oliver et al., 2007; Deluyker et al., 2005).

ANTIBIOTIC SENSITIVITY TEST

Antimicrobials have been used to treat mastitis for more than fifty years, but consensus about the most efficient, safe, and economical treatment is still lacking. The concept of evidence-based medicine has been introduced to veterinary medicine (Cockcroft and Holmes, 2003) and should be applied also to treatment of mastitis. The biggest problem is the widespread resistance of staphylococci, particularly Staph. aureus, to penicillin G (Olsen et al., 2006; Hendriksen et al., 2008). Cure rates for mastitis caused by penicillin-resistant strains of Staph. aureus seem to be inferior to those of mastitis due to penicillin-susceptible strains (Sol et al., 2000; Taponen et al., 2003). It is not known if this is due to pharmacologic problems of the drugs used, or virulence factors possibly linked to β-lactamase gene of the resistant isolates (Haveri et al., 2005). Using an in vitro β-lactamase test for determining resistance to penicillin G of staphylococci before treatment is recommended (Olsen et al., 2006). Coagulase negative staphylococci tend to be more resistant than Staph. aureus and easily develop multi resistance (Sawant et al., 2009). Mastitis causing streptococci have remained susceptible to penicillin G, but emerging resistance to macrolides and lincosamides has been detected (Loch et al., 2005).

CONTROL AND PREVENTION OF MASTITIS

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. 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 (Passantino, 2007).

Good management

The control of mastitis has been successfully achieved through the establishment of effective herd health control programs (Erskine et al., 2002). 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 rubber gloves while milking, and machines should be cleaned regularly to decrease the incidence of transmission. A good milking routine is vital. This usually consists of applying a pre-milking teat dip or spray, such as an iodine spray, and wiping teats dry prior to milking. The milking machine is then applied. After milking, the teats can be cleaned again to remove any growth medium for bacteria. A post milking product, such as iodine-propylene glycol dip is used as a disinfectant and a barrier between the open teat and the bacteria in the air. Mastitis can occur after milking because the teat holes close after 15 min if the animal sits in a dirty place with dung and urine (Laven, 2015).
Controlling environmental mastitis can be achieved by reducing the number of bacteria to which the teat end is exposed. The animals’ environment should be as clean and dry as possible. The animal should have no access to manure, mud, or pools of stagnant water and calving area must be clean. Post milking teat dipping with a germicidal dip is recommended. Proper antibiotic therapy is recommended for all quarters of all animals at drying off; it helps to control environmental streptococci during the early dry period (Laven, 2015).

The milking practice is of paramount importance as this is common route of infection. The udder should be prepared before milking by washing the teats, followed by disinfection and drying with clean paper towels. If the teat area is dripping with water from run-off of areas that were heavily soiled it could lead to pathogens gaining access to the teat canal. Milker’s hands should also be disinfected to prevent the transfer of pathogens. Post milking treatment is also important and all cows should be treated with a teat dip disinfectant to reduce the risk of infection. The primary goal would be to reduce the rate of new infections and the duration of current infections within a herd. It would also be essentially important to maintain normal udder health ensuring that the natural immune response in the cow can resist and fight disease while still producing the required level of milk (Laven, 2015).

**Dry cow therapy**

Dry cow therapy (DCT) is the treatment of cows at the end of lactation with a long acting antibiotic preparation with or without a teat sealant. This is to treat for any intramammary infections (IMI) contracted during lactation and provides protection against new infections during the dry period. During a cows lactation, the probability of infection increases from repeated milking and potential exposure in the parlour and environment. The dry period of the lactation cycle is a critical time for the udder health of dairy cows. The mammary gland undergoes marked biochemical, cellular, and immunologic changes. Involution of the mammary parenchyma begins 1–2 days after the end of lactation and continues for 10–14 days. Although many infections will self-cure either during lactation or the dry period, some will persist and cause an elevated somatic cell count (SCC) which, if not treated, may persist into the next lactation. Additionally, using antibiotics at drying off is more successful at eliminating IMI than treatment during lactation since higher concentrations of antibiotic can be used (Hamadani et al., 2013).

**Vaccination for mastitis**

Development of potential vaccines to prevent or control mastitis continues to be an important goal. Excellent progress has been made toward coliform mastitis control with the development of mutant gram negative vaccines. The organisms used (E. coli and Salmonella) have lost the ability to synthesize outer polysaccharide antigens, resulting in exposure of common gram negative LPS (lipopolysaccharide) antigens. Antibodies produced against these antigens are cross-reactive among gram negative pathogens. When used as directed, there is approximately a 70% decrease in clinical coliform mastitis, as well as a decrease in severity of clinical signs. Many attempts have been directed toward development of an effective vaccine for Staph. aureus (Amanda, 2015).

**MAJOR RISK FACTORS FOR MASTITIS**

Risk factors associated with clinical mastitis are milking routine, type of housing, feeding, and season, as environmental effects. In addition, older cows, later first calving, first stages of lactation and cows with deep udders, week attachments, and high production are more liable to mastitis. Health problems have negative consequences not only on animal welfare, but also on economics of herds because of additional costs in veterinary medicines, reduction of production, discarded milk, and involuntary culling (Collard et al., 2000).

**Parity**

Demelash et al. (2005), Gizat et al. (2008), Rahman et al. (2009), Matios et al. (2009) and Molalegn et al. (2010) indicated that the higher the parity numbers, the more the prevalence of mastitis. According to Steeneveld et al. (2008), in multiparous cows, the risk of developing clinical mastitis (CM) increases with increasing parity. The first-parity cows have been shown to have as high, or higher incidence of udder disorders in early lactation as older cows (Valde et al., 2004). This can be detrimental to her future life due to reduced milk production (Hagnestam et al., 2007), increased risk of new cases of mastitis and increased risk of culling (Schneider et al., 2007). According to Skrzypek et al. (2004), the level of SCC has been reported to be influenced by parity and SCC increases with advanced parities.

**Udder conformation**

Each quarter is composed of the milk-producing tissue or alveoli that lead into the lactiferous ducts, gland cistern, teat canal and finally the teat opening or duct. The alveoli are lined with epithelial cells that become specialized during the gestation period, before calving, and after
calving. These specialized cells produce colostral and lacteal secretions and finally, milk. Connective tissue and muscle cells support the alveoli glands and contract and squeeze milk from the alveoli during milking. According to Girma (2010) and Sori et al. (2005), animals with pendulous udder showed higher incidence of mastitis than cows with non-pendulous udder and there was an association between the two categories. This is because of more exposure to the environmental pathogens and injurious materials.

**Stages of lactation**

The prevalence of mastitis was significantly higher at 6-10 months after calving than 1-5 months after calving (Rahman et al., 2009). The highest prevalence of sub clinical mastitis occurred during the 4th months of lactation, while the lowest during 5th or more than 5th months of lactation. Gizat et al. (2008) revealed that stage of lactation was found to be significant with the occurrence of mastitis. Risk of new environmental streptococcal infection is influenced by stage of lactation, parity, nutrition, and immunity in addition to factors that increase teat end exposure. The importance of the dry period in control of environmental streptococcal IMI cannot be over emphasized (Green et al., 2002).

**Age of cows**

Studies conducted in different part of Ethiopia by different authors, (Mungube et al., 2004; Demelash et al., 2005; Regassa et al., 2010b) indicated that age is considered as potential risk factor to the prevalence of mastitis. As the age of cow advances, the prevalence rate become higher (older cows were more affected by mastitis than younger cows), with prominent statistical variation (p<0.05).

**Body condition score of cows**

High-yielding dairy cows usually exhibit a negative energy balance after calving, which may influence both the immune system and the metabolic system of the individual. Chronic deficiencies of energy, protein, minerals, or vitamins have repeatedly been associated with increased disease susceptibility as a result of depressed immune function. Because most udder infections occur in the peri partum period, optimal feeding, both in the dry period and during early lactation, may be important in preventing mastitis. It seems difficult for the high-producing dairy cow to ingest enough feed shortly after calving to meet lactation demands for energy and protein. Cows with high BCS at calving lose more condition and achieve positive energy balance later than do cows with lower BCS. These findings suggest that there might be differences in the length and severity of the immunosuppressive period after calving, which may influence the risk for mastitis (Valde et al., 2007). According to Mekonnen and Tesfaye (2010), in the study conducted on dairies of Adama, Ethiopia, a cow with poor body condition score and with previous exposure to mastitis were more liable to mastitis than a cow with good body condition score and non-exposed once, respectively.

**Milk Yield**

A high 305-day previous-lactation milk yield was a significant risk factor for early lactation clinical mastitis and high yields increased the mastitis rate in low bulk milk somatic cell count herds. A high milk protein content at the last milk-test day prior to drying-off was found to be a risk factor for early lactation CM. This may reflect higher energy supplies to the udder and lead to delayed involution of the udder tissue. Cows with a fat to protein ratio of >1.5 at the first test-day after calving had higher risks for clinical mastitis and other production diseases (Schukken et al., 2005).

**Hygiene scoring**

The environment in which dairy cows are kept has a decisive effect on their health and welfare. A clean and comfortable shelter represents the key to maintaining the dairy cows’ health and longevity. The shelter’s hygiene level can be evaluated through several assessment systems based on the quantification of the manure pollution in different body regions of the cows (Chaplin et al., 2000). The hygiene scoring system was elaborated by Cook (2002) in order to quantify the hygiene level in the farm and for the assessment of the improvements which have to be made in hygiene management. Researchers report incidence, prevalence, associations or likelihood ratios but seldom quantify impact. For example, a researcher have reported that cows with dirty udders are 1.5 times more likely to have major pathogens isolated from their milk samples than cows with cleaner udders (Schreiner and Ruegg, 2003). Others report a positive association between dirty udders and hind limbs and individual cow somatic cell counts (Reneau et al., 2005). According to Mollalegn et al. (2010) and Matios et al. (2009), the cow’s hygiene significantly affects the prevalence of mastitis.

**PREVALENCE OF BOVINE MASTITIS IN ETHIOPIA**

Different studies conducted in different parts of Ethiopia showed variable prevalence of mastitis depending on the type of farm and managements systems. Mandefrot and Zeleke (2016) revealed that the overall prevalence of
mastitis was found to be 44.44% with clinical and subclinical mastitis accounting for 16.11 and 36.67%, respectively. The overall prevalence of mastitis at cow level was 62.06%, with 3.54 and 58.52% for clinical and subclinical mastitis prevalence, respectively (Alemayehu, 2015). Shimels (2014) reported that the overall prevalence of mastitis at cow and quarter levels were 128 (83.1%) and 403 (65.42%), respectively. In another study carried out by Yien (2014), CMT was performed on 121 lactating dairy cows, of these; about 60.33% (73) showed mastitis cases. The prevalence of clinical and subclinical mastitis was found to be 11.57% (14) and 48.76% (59), respectively.

Abinet (2015) examined a total of 216 lactating cows clinically by California mastitis test (CMT) to detect clinical and sub clinical mastitis. Of the total dairy cattle examined, 92 (42.59%) were found to be positively reacted to CMT. From the total count, the clinical and subclinical mastitis forms were 6.48% (14/216) and 36.11% (78/216), respectively. Biniam (2014) also reported that the prevalence of mastitis was 40.9%. Out of this, 4.66 and 36.18% were clinical and subclinical, respectively.

Abera et al. (2013) reported 66.6% in Adama, 68.1% by Zerihun et al. (2013) in Addis Ababa, 71.0% by Mekibib et al. (2010) in Holeta, and Bitew et al. (2010) of 28.2% in Bahir Dar, Ethiopia. This variability in prevalence of mastitis between different reports could be attributed to differences in farms management practice or to differences in study methods agro-climatic condition. Mungube et al. (2005) revealed overall prevalence of sub clinical mastitis in cross breed dairy cows in Ethiopia as 52.3 and 32.4% at cow and quarter levels, respectively.

Biffa et al. (2005) conducted a study on mastitis of 974 lactating dairy cows in Southern Ethiopia as 34.9% (340) had mastitis; 11.9% (116) clinical, and 23.0% (224) subclinical mastitis, respectively. Mastitis prevalence in dairy farms of Holeta town, Central Ethiopia at cow level was 71.0% (76/107), out of which 22.4% (24/107) and 48.6% (52/107) were clinical and subclinical, respectively. The Holeta study also revealed the quarter level prevalence of mastitis as 44.9% (192/428); from this the clinical form was 10.0% (43/428) and the subclinical was 34.8% (149/428). Out of the 43 quarters clinical cases, 31 had blind teats, while 12 of them revealed active cases of mastitis showing visible sign of inflammation on the udder and changes were also observed on milk. Getahun et al. (2008) indicated that in Selalle smallholder dairy farms, central Ethiopia, a total of 14 (12.8%) herds, 16 (3.2%) cows and 17 (0.9%) quarters had clinical mastitis, while 77 (70.6%) herds, 147 (30.4%) cows and 264 (13.6%) quarters had sub-clinical mastitis. The prevalence of clinical mastitis at cow level is 3.9 and 4.8% respectively in Bahir Dar as reported by Almaw (2004) and Bitew et al. (2010), and 3.9% by Abera et al. (2013) in Adama, Ethiopia. Kerro and Tareke (2003) in southern and Hundera et al. (2005) in central Ethiopia reported a rate of 10 and 16.11%, respectively. Matios et al. (2009) reported clinical mastitis prevalence level as high as 14.9%. Abera et al. (2013) and Mekibib et al. (2010) reported the quarter level mastitis as 42 and 44.8% in Adama and Holeta town, respectively which is higher than Biffa et al. (2005) who reported 28.2% and Kerro and Tareke (2003) who found 19% in different area of Ethiopia. This variation might be due to the complex effect of mastitis in the management system of the farm, breeds of cattle and geographical location of the study area.

According to Melesse (2012), the overall prevalence of mastitis at cow and quarter level was found to be 73 and...
37%, respectively which is in line with the report of Regasa et al. (2010b) who found the prevalence rate of 71 and 69.8% in dairy farms of Holeta town and Addis Ababa and its vicinity, Ethiopia, respectively. This is slightly lower than the report of Matios et al. (2009) who reported the prevalence rate of 64.5% in dairy farms of Asella, Ethiopia. In contrast, Matios et al. (2009) also reported a sub clinical mastitis of 30.4% in Asella area. Getahun et al. (2008) and Mekonnen and Tesfaye (2010) however, recorded lower level of sub clinical mastitis prevalence in Selalle (13.6%) and Adama area dairies (22.7%). Regasa et al. (2010a) on the other hand, reported 34.8% sub clinical quarter wise prevalence which is higher than others. Variations in husbandry practices between different areas might, at least, partly explain the difference in prevalence reported by different authors.

According to Mekonnen and Tesfaye (2010) in smallholder dairy farms of Adama, Ethiopia 62, 48 and 24.6% at herds, cows and quarter levels, respectively were affected by clinical and/or sub-clinical mastitis. Out of the total number of 195 quarters affected by clinical and sub-clinical mastitis, 48 (24.6%) were right fore, 50 (25.6%) right hind, 53 (27.2%) left fore and 44 (22.6%) left hind quarters. The prevalence of sub-clinical mastitis was higher than clinical mastitis at herd, cow and quarter levels, respectively by 4.5, 6.6 and 9.3 times. The higher mastitis infection was observed in hindquarters than front quarters (Kerro and Tareke, 2003). This might be due to the fact that hindquarters are highly prone to contamination with dirty, and large amount of milk is produced from hindquarters and as a result, the pressure on teat canal forces the canals to be opened widely, which allows entrance of microbes.

According to Alemayehu (2015), the prevalence of mastitis shows a significant difference (P< 0.000) between crossbreed and local breeds. From the cases, 162 (78.64%) were Crossbred, while 31 (29.52%) were local Fogera breed. Bitew et al. (2010) reported in Bahir Dar, on Cross and Fogera breed, Lakew et al. (2009) on cross and local Arsi breed and Biffa et al. (2005) found significant difference between local Zebu, Holstein-Frisian and Jersey breeds in Ethiopia. Increased milk yield from genetic selection may be accompanied by genetic susceptibility to mastitis. The lower prevalence in local breeds could be associated with difference in genetically controlled physical barrier, such as streak canal sphincter muscles, keratin in the teat canal or shape of teat end where pointed teat ends are prone to lesion. In addition to physical barriers, the difference in occurrence of mastitis in these breeds could arise from differences in cellular immunity (Erskine, 2001; Workineh et al., 2002; Kerro and Tareke, 2003).

Kerro and Tareke (2003) found that the risk of clinical and subclinical mastitis infection increases with the advancing age of the cow. This might be due to the increased opportunity of infection with the time and the prolonged duration of infection, especially in herd without mastitis control program (Radostitis et al., 2007). The higher the parity number, the higher prevalence was observed in a study by Biffa et al. (2005). According to Erskine (2001), primiparous cows have more effective defense mechanism than multiparous cows. This variation might be due to the influence of breed and management of the farm.

Mekonnen and Tesfaye (2010), and Nibret et al. (2011) revealed that contagious bacteria, such as Coagulase negative staphylococci (CNS), Staph. aureus, Strep. agalactiae, Strep. dysgalactiae and environmental microorganisms, such as coliforms (E. coli, E. faecalis, and Strep. uberis) were found to be the major etiology of mastitis in market oriented smallholder dairy farms in Adama and Gondar, Ethiopia.

According to Alemayehu (2015), the 5.2% of the mammary glands examined was being blind. Matios et al. (2009) also found 4.5% of blind quarters. Getahun et al. (2008) and Mekonnen and Tesfaye (2010) reported 2.3 and 3.6% blind quarters, respectively in their study on herds. Kerro and Tareke (2003) reported higher prevalence of mastitis in cows at mid and late stages of lactation. However, Mungube et al. (2004) and Biffa et al. (2005) reported higher prevalence of mastitis in early stage of lactation. The variations in the stages of lactation among different studies might be related to age variation, parity and breed of the sampled animals as indicated by Getahun et al. (2008). The main sources of infection are udder of infected cows transferred via milker’s hand, towels and environment.

**CONCLUSION AND RECOMMENDATIONS**

In this study, mastitis was found to be one of the major constraints to dairy production in dairy farms of Ethiopia. The prevalence rate of sub clinical mastitis was found to be higher than clinical form. High prevalence rate of mastitis implied that it is the most serious health problem of the dairy cow. The higher prevalence level of sub clinical mastitis as compared with clinical form indicates the magnitude of subclinical mastitis problem and low level of attention that is given to it in terms of diagnosis and treatment. The presence of considerable proportion of Enterobacteriaceae suggested contamination of mammary gland and its environment with animal dung. Moreover, dominant number of contagious microbial agents indicated improper milking procedures experienced in the farms. Among the assessed potential risk factors to the prevalence of mastitis, higher infection rates were observed in cows with advanced age groups, pendulous udder conformation, and multiple parity, poor body condition score, bad hygiene score, high milk producers, early lactation stage, previous exposure to mastitis and blind teats. Information relating to mastitis magnitude, distribution, negative effect on milk...
quality and risk factors associated with consumers are limited. Such information are important to envisage when optimizing prevention and control options in different dairy farms of the country that would help reduce its prevalence and negative effects on dairy products.

Based on the above conclusion, the following points are forwarded:

1. Mechanisms to control the risk factors associated with the disease should be implemented;  
2. Veterinarians should reduce repeated use of drugs to minimize drug resistance development;  
3. Awareness should be created among veterinarians, dairy farm owners and dairy workers on the effect of Mastitis;  
4. Mastitis treatments should be preceded with identification of the causative agent and susceptibility test profile of pathogens to select effective antibiotics;  
5. Regular investigation of mastitis especially sub clinical form should be practiced;  
6. Culling of old aged and repeatedly infected cows should be done on regular planned basis;  
7. External parasites especially tick prevention program should be applied;  
8. Further investigation and molecular diagnosis on mastitis causative agents should be done in order to apply proper prevention and treatment scheme.

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