Mastitis is inflammation of the parenchyma of the mammary gland

Balemual Abebaw

balemual.abebaw@gmail.com

Abstract: Mastitis is inflammation of the parenchyma of the mammary gland. The predominant pathogens for the disease throughout the world include staphylococcus species, streptococcus species and coliform species. The principal bacterial infection associated with ingestion of milk and milk products are caused by different bacterial genera. The bacteria that are transmitted through milk and cause disease problems in man are bacteria causing mastitis in cattle and transmissible to human when man uses raw milk from infected udder. Example of such type of bacteria includes Mycobacterium, Brucella, and Staphylococcus and Streptococcus species.
[Balemual Abebaw. **Mastitis is inflammation of the parenchyma of the mammary gland.** *Rep Opinion* 2017;9(5):99-106]. ISSN 1553-9873 (print); ISSN 2375-7205 (online). http://www.sciencepub.net/report. 10. doi:10.7537/marsroj090517.10.

Key words: *Mastitis,* Mammary Gland and Bacterial Infections

1. Introduction

Bovine mastitis is characterized by inflammation of the mammary gland. The inflammation severity depends on the causative agent and the host response. Mastitis is caused by a wide spectrum of pathogenic agents that penetrate the teat canal and multiply in the udder cistern. The majority of mastitis cases are produced by a relatively small group of bacteria, including Staphylococcus aureus, Streptococcus uberis, Mycoplasma spp and Escherichia coli (Calvinho & Tirante, 2005). Bovine mastitis is the most prevalent and costly disease, affecting dairy farms worldwide. Economic losses associated with mastitis derive mainly from a decrease in milk production and to a lesser extent, from the culling of chronically infected cows, cost of veterinary treatment, and penalties on milk quality (Seegers *et al*., 2003). The losses caused by clinical mastitis do not take into account those caused by sub-clinical mastitis which is less obvious and may only be detectable by measuring the milk's somatic cell counts (SCC). 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 inconsistency and accompanied by fever, depression and anorexia. 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 quarter's generally contained below 200,000 somatic cells /ml. A value of SCC above 300,000 is abnormal and an indication of inflammation in the udder (Hillerton, 1999). According to Shearer and Harris (2003), subclinical mastitis is important due to the fact that it is 15to 40 times more prevalent than the clinical form (for every clinical case of mastitis there will be 15-40 subclinical cases). Therefore, the objectives of this seminar paper are:

- To address about mastitis.
- To provide highlights on public health significance of mastitis due to milk borne disease.
- To address the economic significance of mastitis specifically subclinical mastitis.

2. Literature Review On Mastitis In Domestic Animals Espesialy In Bovine

2.1. Definition of Mastitis

Mastitis as a result of infectious pathogen in cow (figure 1)

Mastitis is inflammation of the mammary gland. It is caused by microorganisms; usually bacteria, that invade the udder, multiply, and produce toxins that are harmful to the mammary gland. Clinical mastitis has visible signs of the disease like flakes or clots in the milk, may have slight swelling of infected quarter in mild sign and secretion abnormal, hot, swollen quarter or udder; cow may have a fever, rapid pulse, loss of

appetite, dehydration and depression; death may occur in sever sign. Subclinical mastitis has no visible signs of the disease, but somatic cell count (SCC) of the milk will be elevated; bacteriological culturing of milk will detect bacteria in the milk (Abera *et al,* 2012).

2.2. Epidemiology

2.2.1. Agent

Staphylococcus aureus, coagulase negative staphylococci, Streptococcus dysgalactiae, Streptococcus agalactiae and Streptococcus uberis are common causes of both clinical and subclinical mastitis (Pyorala, 1995). Coliform bacteria such as Escherichiacoli and Klebsiella spp. most commonly cause clinical mastitis and seldom give rise to subclinical cases (Hogan & Smith, 2003). Staphylococcus aureus and Str. agalactiae are referred to as contagious udder pathogens as they are bound to the bovine udder or the cow and are mainly transmitted from cow to cow. Good milking hygiene is one important factor in order not to spread these organisms within a herd (Pyorala, 1995). The coliform bacteria are called environmental pathogens as their main source of transmission is from the surroundings of the animal and are best managed by good environmental practices (Hogan & Smith, 2003). Coagulase negative staphylococci, Str. uberis and Str. dysgalactiae are considered to be both contagious and environmental (Taponen & Pyorala, 2006; Todhunterb *et al.*, 1995; Pyorala, 1995).

Mastitis as a result of s. aures in goat (figure 2)

2.2.3. Risk factor of mastitis

The large number of predisposing factors that contribute to the emergence of mastitis in dairy cattle may be physiological, genetic, pathological or environmental.

Age of the cow: It has been demonstrated that occurrence of mastitis in infected quarters increases with age in cows, being the highest at 7years of age. This may be due to an increased cellular response to intramammary infection or due to permanent udder tissue damage resulting from the primary infection. Effient innate host defence mechanisms of the younger animals are one possibility that makes them less susceptible to infection.

Inherited features of the bovine: Various genetic traits may also have a considerable impact upon the susceptibility of the animal to mastitis. These genetic traits include the natural resistance, teat shape and conformation, positioning of udders, relative distance between teats, milk yield and fat content of milk. High milk yielders with higher than average fat content are reported to be more susceptible to mastitis. The conformation of the udder and shape of the teat are inherited characteristics that may also affect susceptibility to mastitis. Cows with elongated teats are more vulnerable to mastitis infection than cows with inverted teat ends. Broad udders, lower hind-quarters and teats placed widely help the infectious agent and should be selected against it (Smith and Hagon, 2003).

Stage of lactation: The incidence of mastitis is reported to be higher immediately after parturition, early lactation and during the dry period, especially the first 2-3 weeks due to probably to increased oxidative stress and reduced antioxidant defense mechanisms during early lactation (Biru, 1989).

Mammary regression: There are significant functional changes in the udder during the early and late lactation and dry period, which affect the cow's susceptibility to infections. Lactating cows under stress show premature mammary regression. Such a condition compromises udder's natural defense mechanisms leading to invasion of the teat canals by potential pathogens.

Milking machine: Extraneous factors such as the milking habits of farmers and faulty milking machines favor the pathogens to gain access to mammary land and proliferate, potentially leading to mastitis (Copeland, 2007).

Nutrition: Vitamin E is one of the important supplements in dairy feed to boost the immune response of cows as it has been reported to enhance the neutrophil function as well as the phagocytic properties of neutrophils after parturition. Vitamin E is often combined with selenium, which acts as an anti-oxidant by preventing oxidative stress.

Weather and climate: Heat, humidity, cold and draught are the important predisposing factors. A higher incidence of mastitis has been reported to occur particularly during summer rainy months. As heat and humidity increases, so does the bacterial multiplication as well as the load of pathogens in the environment. Conversely, an alternative study has reported a higher incidence of coliform mastitis during the cold months of the year when the temperature was reported to be less than 21°C (Kivaria et al., 2014).

2.2.4. Host Range

Bovine mastitis due to damage to the teat canal allows organism's access to deeper structures. Staphylococci, streptococci, and the coliforms dominate the picture of bovine mastitis.

Mastitis in the ewe and doe is usually caused by Staphylococcus aureus or Mannheimia hemolytica. The latter can be very necrotizing and affects vasculature resulting in cyanosisand so is sometimes known as blue bag. Maedi-visna virus, caprine-arthritis encephalitis virus cause mastitis in their respective species (sheep and goats). Mastitis in dogs and cats is uncommon and is usually transient and due to staphylococci or streptococci. Mastitis in swine: Mastitis-metritis-agalactia syndrome occurs in postparturient sows and is caused by a number of bacteria, most of whom are coliforms (Kader *et al.*, 2003).

2.2.5. Transmission

Contagious mastitis is caused by bacteria present in teat canal and udder is transmitted from infected cow to uninfected cow while milking. Environmental Mastitis: Some organisms such as Escherichia coli do not normally live on the skin or in the udder but they enter the teat canal when the cow comes in contact with the environment which is highly contaminated. The pathogens are generally found in feces, bedding materials and feed. The pathogens in the contaminated get the earliest opportunity to invade the udder when the teat orifice is open at or soon after milking or after teat damage. These environmental pathogens are, thus, described as opportunistic invaders of the mammary gland. It is noted that environmental mastitis cases are only about10% of the total mastitis cases in the herds.

2.3. 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 infection from outside, a sphincter of smooth muscles surrounds the teat canal which functions to keep the teat canal closed (Murphy *et al*., 1988). 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 (Bramley and Dodd, 1984). 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 electro statically 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 (Habbit *et al.,* 1969). 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 (Capuco *et al.,* 1992). 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 greater than 90% of the total leukocytes (Sordillo *et al.,* 1987). 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 (Sordillo and Nickerson, 1988). 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 (Politis *et al.,* 1992). 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 (Radostitis *et al.,* 2007).

2.4. Clinical sign

The clinical findings in mastitis include abnormalities of secretion such as discoloration, clots, flakes, pus in milk, abnormalities in the size of the udder which is larger than normal quarter; firm in consistency and temperature of the mammary glands pyrexic, depressed or has decreased appetite or milk production and, frequently, a systemic reaction. In clinical mastitis, the infected quarter often become swollen, sometimes painful to touch, and the milk is visibly altered by the presence of clots, flakes, or discolored serum and sometimes blood. In severe cases (acute mastitis), the cow shows signs of generalized reaction: fever, rapid pulse, loss of appetite and sharp decline in milk production. In contrast, subclinical mastitis is more difficult to detect. The cow appears healthy, the udder does not show any signs of inflammation and the milk seems normal (Radostits *et al*., 2007).

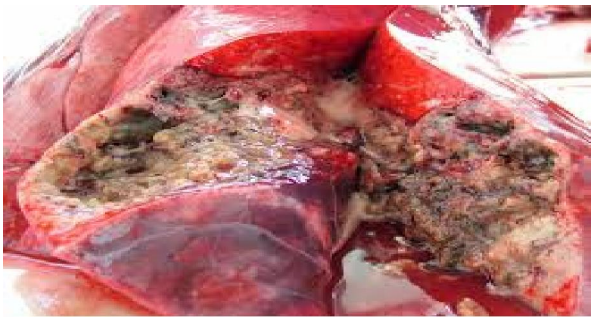
2.5. Pathological lesions

2.5.1. Post mortem findings

- ❖ Pale yellow granular appearance of the udder parenchyma.
- ❖ Light brown edematous udder parenchyma.
- ❖ Enlarged supramammary, iliac and lumbar lymph nodes.

2.5.2. Histopathology

Histopathological evidences revealed that the mammary gland tissue of S. aureus inoculum induced groups produced massive of polymorphonuclear Neutrophil leukocytes (PMN) infiltrations, alveolar damage exhibited irregular epithelial and luminal celllining and unstable secretory products. The presence of functional PMN is crucial to the host defense against bacterial pathogens but neutrophils may promote tissue injury and disturb mammary function, via reactive oxygen metabolite generation. Further, PMN accumulated in the alveolar space damaged secretory products such as milk proteins, lipid droplets and fats, which might be due to the loss of action of phagocytosis of bacteria by the PMN's leading to the tissue damage.



Pathological lesion in of mammary gland as a result of bovine tuberculosis (figure 2)

2.6. Morbidity and mortality

The incidence of clinical mastitis ranges from 5 to more than 10% all mastitis case. Clinical mastitis occurs in all dairy herds. Even well-managed herds, as judged by somatic cell count level and a high level of milk production, may be suffering from a high incidence of clinical mastitis. Clinical mastitis is mostly caused by bacteria. The most important are S. aureus, E. coli, Klebsiella spp. and Streptococci (S. uberis and S. dysgalactia). Subclinical Mastitis account 90 -95% of all mastitis cases in which udder appears normal; milk appears normal but elevated SCC (score 3-5) and Lowered milk output is observed (Jon, 2006).

2.7. Diagnosis

2.7.1. Diagnostic techniques

Methods that have been developed for the diagnosis of mastitis include Visual method, Direct method, Indirect method, CMT method, SCC method, Simplified Resazurin Rennet Test, Stir cup test, Surf filled mastitis test, Bromothymol Blue (BTB) test, Modified Whiteside test, Wisconsin Mastitis test, Electrical Conductivity test and Culture method test (Aielso and Mays, 2005).

2.7.2. Laboratory Diagnosis

Visualization and Palpation of the udder: Regular examination of the udder especially during milking is the primary step in preventing mastitis. With one or two episodes of mastitis, the animal attendant would be able to monitor the physical changes due to mastitis

or any other infection. In clinical mastitis, visually the udder may turn red, hard and hot to touch. Udder may be painful to the cow at the time of palpation. These symptoms show the changes in vascularity and blood flow of the gland when inflamed. On collection of the sample, the presence of pathogen is screened (Capulo *et al.*, 1992.

Visualization of Milk: At the time of milking, gross changes in the milk such as the presence of flakes, clots or serous, blood and watery secretions may be observed. In clinical mastitis this is the common means of detection. Flakes or clots in the milk are detected by stripping the first few squirts of milk from each quarter into a strip cup at the beginning of milking. The freshly drawn milk is, at first, examined by the naked eye of a dairy man for the visible abnormalities in the milk. Milk turns very much watery during dry period and also changes in milk composition are observed. In advanced cases of chronic mastitis, the appearance of udder secretion is usually abnormal at intervals. The secretion becomes grossly altered in case of acute mastitis.

California Mastitis Test: CMT is not only an easily applicable test at the site but also the only reliable screening test for sub clinical mastitis. The CMT has been developed to test milk from individual quarter and also bulk milk samples. Using the CMT, fresh, unrefrigerated milk can be tested for up to 12 hours and refrigerated milk can be tested for up to 36 hours to get reliable readings. In this test, one teaspoon full milk (2ml) is drawn from each quarter and an equal amount of CMT solution added to each cup in the paddle. The CMT paddle is rotated in circular motion to mix the contents thoroughly. The CMT reaction must be scored within 15 seconds of mixing because exceeding that time weak reactions will disappear. The reagent used in the CMT is a detergent and bromoserol purple as an indicator of pH. The degree of reaction between the detergent and the DNA of cell nuclei determines the number of somatic cells in milk. This CMT helps us to assess the level of infection in each quarter rather than to an overall udder result. The result shows only whether the cell count is high or low. Therefore there is no numerical result (The CMT shows only changes in cell count above 300,000).

Somatic Cell Count: Somatic cells are normal constituent of milk. In mastitis analysis cell and leucocyte counts in milk have been used for over a century. Cells found in normal bovine milk from non-infected glands are inclusive of neutrophils (1 -11 %) macrophages (66-88%), lymphocytes (1 0-27%) and epithelial cells (0-7%) (Paape *et al.,* 2000). Problem arises only when the somatic cells become excessive in milk. When there is infection or injury, leucocytes (up to 75%) and epithelial cells (up to 25%) are increased in milk. In infected glands more than 90% of SCC is composed of neutrophils and a SCC of greater than 2,000,000 cells/ ml is an indicator of mastitis. The severity of mastitis is determined by the number of somatic cells which is expressed as cells/ml of milk (Middleton et al., 2004). Cows with SCC of less than 200,000 are not generally considered to have mastitis. But Cows with SCC above 300,000 are likely to be infected with pathogens.

Simplified Resazurin Rennet Test: This test is simple and useful in practical in the detection of bovine mastitis. In this test, a sterile test tube is placed with a tablet of resazurin and rennet. Milk (10 ml) is directly added from teat into the test tube and incubated for 1 hr. at 37ºC. The change in resazurin colour and the time required for coagulation are observed.

Strip Cup Test: This test is generally used in determining the presence of clinical mastitis by the detection of visible particles of milk. Strip cup method was first used by Moak. Any lay man can make use of this strip cup. In this test an enamel plate divided in four strip cups is used and the bottom of the plate is black in colour so that the milk flakes are easily observed by tilting the cups at an angle.

Surf Field Mastitis Test: The handy readily available household surf (detergent) is used as reagent. This test is easy and cheap besides adequately sensitive to detect all cases of sub clinical mastitis (Muhammad *et al.,* 1995). The surf 3% solution is mixed in equal quantities with milk drawn from quarter in petridishes separately for each quarter. The reaction of somatic cell's DNA with detergent (surf) and the resultant formation of gel in different degree are observed. The formation of gel indicates positive mastitis samples.

Wisconsin Mastitis Test: This is a simple screening cow-side test on producer's milk. When WMT reagent is added to milk, the number of inflammatory cells rises high and results in development of a gel. The aim of WMT test is to maintain a bulk tank reading of less than 300,000 cells/ml. If the WMT score is more than 8, 55,000cells/ml the milk is not considered for human consumption (Ruegg, 2009).

Bromothymol blue test: This test indicates the pH value of milk that has been widely used in the diagnosis of mastitis. When bromothymol blue is added to milk, different colours are developed due to changes in pH of milk. The normal pH of milk is 6.4 to 6.8 and isotonic with blood plasma. The disadvantage is that this BTB test may give false positive reaction, when the cow is in later stages of lactation.

Electronic Somatic Cell Count: This is automated electronic cell count equipment used by Dairy Herd

Improvement Associations. Periodical report of the udder health of individual cows as well as the entire herd can be obtained by this test. The limitation is that the Electronic Somatic Cell Count equipment is more expensive and constant monitoring is needed (Brown, 2006).

2.7.3 Differential diagnosis

- Mammary abscesses.
- Mammary tumor (cancer).
- Mammary traumatic wound.
- Mammary edema.
- Mammary cellulitis.

2.8. Economic Significance

2.8.1 Public Health significant

With mastitis there is a danger that the bacterial contamination of milk from affected cows may render it unsuitable for human consumption by causing food poisoning and provides a mechanism of spread of disease to humans through consumption of raw milk. Many farm families simply consume raw milk because it is a traditional practice and it is less expensive to take milk from the bulk tank than buying pasteurized retail milk. Some believe that raw milk has a higher nutritional value than pasteurized milk. The bacteria that are transmitted through milk and cause disease problems in man are bacteria causing mastitis in cattle and transmissible to man when man uses raw milk from infected udder. Example of such type of bacteria includes Mycobacterium, Brucella, Staphylococcus, streptococcus, Campylobacter and Listeria species (Heeschen, 1996). The presence of residue in milk following treatment of mastitis is a major public health concerns that adversely affects the industry, the practicing veterinarian and the safety of milk for human consumption (Hagsted and Hubbert, 1986). Milk borne infection raw (unpasteurized) milk has been found to participate in spreading out of illnesses caused by Mycobacterium bovis, Brucellaabortus, Staphylococcus aureus, Listeria Monocytogenes, Campylobacter jejuni, Salmonella, Staphylococci species, and E. coli. With severe clinical mastitis, abnormalities of milk are easily observed and milk is discarded by the producer. Such milk normally would not enter the food chain. But when milk of cows with subclinical mastitis, which is with no visible changes, is accidentally mixed into bulk milk, it enters into food chain and can be dangerous to humans. Although pasteurization is likely to destroy most of human pathogens, there is concern when raw milk is consumed or when pasteurization is incomplete or faulty (Jayarao *et al*., 2006).

The impact of mastitis is mainly economical and the most evident costs are reduced milk yield, veterinary costs and the disposal of milk. Examples of more indirect costs are reduced fertility, increased work load for the farmer and reduced quality of milk that aggravate the making of cheese and yogurt (Lavon et al., 2012). Mastitis may also cause severe illness and suffering and may lead to increased costs if the animal is culled or dies from the disease (Nielsen, 2009).

2.9 Treatment

Treating subclinical mastitis is generally not economical during lactation because of high treatment costs and poor efficacy. However, treatment of subclinical mastitis may be considered to reduce the risk of becoming clinically infected and a long term detrimental effect on milk quality. Antimicrobial treatment is one of the most common treatment plans for therapy of bovine mastitis. A standard recommendation for most clinical mastitis is a 3-day intramammary treatment of an antimicrobial agent. However, several antimicrobial treatment regimens are also available, differing in antimicrobial compound, route of application, duration, probability of cure, and costs. Other therapeutic approaches such as asymptomatic therapy using anti-inflammatory drugs, immunotherapies using vaccines or cytokines and even homeopathy have also been described. Cure rates are highly depended on the causal pathogens and other cow factors (Radiostitis *et al*., 2007).

2.10. Prevention and control

While mastitis cannot be totally eliminated from aherd, 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; 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 to approach 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. Controlling contagious mastitis: Staphylococcus 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 eases and have to be culled. Streptococcus 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. Streptococcus 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. Controlling environmental mastitis: This can be achieved by reducing the number of bacteria to which the teat end is exposed. The animal's 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. Attempts to control environmental mastitis during dry period, using either germicidal or barrier dips, have been unsuccessful. 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 (Tanos and Bahas, 2004).

3. Epidemiology Of Mastitis Inethiopia

Mastitis is distributed throughout the world, but it is highly distributed in developing country due to poor management. Mastitis considered being a typical example of a complex disease, which requires the interaction of the host, agent, and the environment (Radostits *et al.,* 1994). Host factors include breed, physiological state of mammary gland, and anatomy of teat canal, sphincter tone and presence of teat lesion. Agent factor includes the ability to survive in the immediate environment of the animal, the ability to colonize the teat duct, the ability to adhere to the mammary epithelium and not to be flushed out with milk flow Environmental factor includes milking practice, housing system and bedding (Quinn *et al.,* 1994).

4. Conclusion And Recommendation

Bovine mastitis is an economically important disease due to its involvement in the quantity and quality of milk production. The dairy industry all over the world suffers from significant economic losses incurred due to mastitis. Application of hygienic measures during milk collection, using milking machines, lactation and dry cow therapy, teat sealers, dietary supplements and culling are likely to reduce but not control the incidence of both clinical and subclinical mastitis. Research aimed at developing an effective broad-spectrum universal vaccine capable of providing protection against the predominant environmental and contagious pathogens causing bovine mastitis is highly warranted for reduction of the incidence of bovine mastitis worldwide. Generally, mastitis is usually considered as the most important disease in dairy cattle and human beings and hence remained a concern of most dairy farmers and veterinarians. The bacteria that are transmitted through milk and cause disease problems in man are bacteria causing mastitis in cattle. Based on the above facts the following points are recommended:

- Mastitis control measures should be largely under taken to reduce bacterial contamination of milk from the udder.
- Education of public at large about hazard of raw milk consumption and the possible control and preventive measures through heat treatment should always be encouraged before milk is consumed.
- An appropriate treatment protocol should be selected based on the concept of evidence-based medicine.
- Implement correct and gentle milking technique.
- Divide herd into groups according to udder status in order to establish a milking order.

Reference

1. Abera, M., Habte, T., Aragaw, K., Asmare, K. & Sheferaw, D. (2012) Major causes of mastitis and associated risk factors in smallholder dairy farms in and around Hawassa, Southern Ethiopia. Tropical Animal Health and Production 44, 1175-1179
2. Aielso, S.E. and Mays, A. (2005). Mastitis in Large Animals. The Merck Veterinary Mannual.9th ed., Merck and CO. INC. USA. Pp. 1121-1130.
3. Bramley, A. J. and F. H. Dodd, 1984. Mastitis control: progress and prospects. J. Dairy Sci., 51:481
4. Brown, J. L. (2006). A Survey of Food borne Pathogens in Bulk Tank Milk and Raw Milk Consumption Among Farm Families in Pennsylvania. J. Dairy Sci., 89: 2451-2458.
5. Biru G: Major bacteria causing bovine mastitis and their sensitivity to common antibiotics. Ethiop J Agric Sci 1989; 11:43 −49.
6. Capuco, A. V., S. A. Bright, J. W. Pankey, D. L. Wood, R. H. Miller and J. Bitman, 1992. Increased susceptibility to intramammary infection following removal of teat canal keratin. J. Dairy Sci., 75:2126.
7. Copeland, S. (2007). Livestock animal health. Bovine Tuberculosis facts in Manitoba livestock producers. J. Vet. Res., 40: 50-75.
8. Calvinho, L.F., & Tirante, L. (2005). Prevalenciade microorganismospatógenos de mastitis bovina y evolucióndelestado de salud de la glándulamamaria

en Argentina en losúltimos 25 años. Revista FAVE 4(1-2):29–40.
9. Habbit, K. G., C. B. Cole, and B. Reiter, 1969. Antimicrobial proteins isolated from the teat canal of the cow. J. Gen. Microbiol., 56: 365.
10. Heeschen, W.H. (1994). Introduction In: Monograph on the Significance of Microorganism in Raw Milk. Internationaldairy federation. Wolf passing, Austria. Pp.19-26.
11. Hillerton, J. E., 1999. Balancing mastitis and quality. Proc. British Mastitis Conference Stonelergh, UK. Pp: 31-36
12. Janosi, S., & Baltay, Z. (2004). Correlations among the somatic cell count of individual bulk milk, result of the California Mastitis Test and bacteriological status of the udder in dairy cows. Acta Veterinaria Hungarica. 52(2):173-83. Jayarao, B. M., S. C. Donaldson, B. A., Straley, A. A., Sawant, N. V., Hegde and Brown, J. L. (2006). A Survey of Food borne Pathogens in Bulk Tank Milk and Raw Milk Consumption Among Farm Families in Pennsylvania. J. Dairy Sci., 89: 2451-2458.
13. Jones, G. M., 2006. Understanding the basics ofmastitis. Virginia Cooperative Extension. Publication No. 404-233. Virginia State University, USA, pp: 1-7
14. Kader MA, Samad MA & Saha S (2003), Influence of host level factors on prevalence and economics of sub-clinical mastitis in dairy cows in Bangladesh. Indian Journal of Dairy Science 56, 235-240.
15. Lavon, Y., Leitner, G., Voet, H. & Wolfen son, D. (2012) Naturally occurring mastitis effects on timing of ovulation, steroid and gonadotrophic hormone concentrations, and follicular and lutealgrowth in cows. Journal of Dairy Science 93(3), 911-921.
16. Kivaria FM, Noordhuizen JPTM, Kapaga AM 2004: Risk factors associated with subclinical mastitisinsmall holder dairy cows in Tanzania. Tropical Animal Health and Production 36581 - 592.
17. Murphy, S. C., K. Cranker, G. F. Senyk, D. M. Barbano, A. I. Saeman and D. M. Galton, 1988. Influence of bovine mastitis on lipolysis andproteolysis in milk. J. Dairy Sci., 71: 65-69
18. Nielsen, C. (2009). Economic impact of mastitis on dairy cows. Doctoral thesis no 2009:29. Faculty of medicine and animal science, Swedish university of Agriculture
19. Paape MJ, Bannerman DD, Zhao X, Lee JW (2008) The bovine neutrophil: Structure and function in blood and milk. Vet Res 34: 597-627
20. Politis, I., X. Zhao, B. W. Mc Bride and J. H. Burton, 1992. Function of bovine mammary macrophagesas antigen presenting cells. Vet. Immunol. Immunopathol., 30: 399
21. Pyorala, S. H. & Pyorala, E. O. (1995). Efficacy of parenteral administration of three antimicrobial agents in treatment of clinical mastitis in lactating cows: 487 cases (1989-1995). Journal of the American Veterinary Medical Association, 212(3), pp 407–412.
22. Quinn, P.J. Carter, M.E. Markey, B. K. and Carter, G. R (1994): Veterinary Microbiology Microbial Diseases, Bacterial Causes of Bovine Mastitis, 8th Edition, Mosby International Limited, London, pp 465 -475.
23. Radostitis, O. R., blood, D.C. and Henderson, J.A. (1994). Mastitis. In: Veterinary, A Text book of the disease of Cattle, Sheep, Goats and Horse, 8th ed., Bailler Tindal, London, Pp. 563-614. Radostits, O.M., Gay, C.C., Hichcliff, K.W., and Constable, P. D. (2007). Veterinary Medicine. A Textbook of the Diseases of Cattle, Sheep, Pigs, Goats and Horses. 10th ed., Bailler Tindal, London, Pp. 673-762.
24. Reinemann, D.J., Wolters, M.V.H.G., Billon, P., Lind, O., & Rasmussen, M.D. (2003).Review of practices for cleaning and sanitation of milking machines. In: Bull. 381. International Dairy Federation, Brussels, Belgium. pp.1-23
25. Sandholm, M. & Pyorala, S. (1995) Coliform mastitis, In: (University of Helsinki, Faculty of Veterinary Medicine) The bovine udder and mastitis. 149-160. Helsinki.
26. Seegers H, Fourichon C, Beaudeau F. Production effects related to mastitis and mastitis economics in dairy cattle herds. Vet Res. 2003; 34(5): 475-91.
27. Smith, K. L. & Hogan, J. S. (2003). Environmental mastitis. The Veterinary clinics of North America. Food animal practice, 9(3), pp 489–498.
28. Sordillo, L. M. and S. C. Nickerson, 1988. Morphometric changes in the bovine mammary glandduring involution and lactogenesis. Amer. J. Vet. Res., 49: 1112. Sordillo, L. M., S. C. Nickerson, R. M. Akers and S. P. Oliver, 1987. Secretion composition during bovinemammary involution and the relationship with mastitis. Intl. J. Biochem., 19: 1165.
29. Taponen, S., Jantunen, A., Pyorala, E. & Pyorala, S. (2006). Efficacy of targeted 5-day combined parenteral and intramammary treatment of clinical mastitis caused by penicillinsusceptible or penicillin-resistant Staphylococcus aureus. Actaveterinaria Scandinavica,44(1-2), pp 53–62.
30. Todhunter, D. A., Smith, K. L. & Hogan, J. S. (1995). Environmental streptococcal intramammary infections of the bovine mammary gland. Journal of dairy science,78 (11), pp2366–2374.

5/25/2017