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Review Article

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Review on the Pneumonic Pasteurellosis of Cattle in Ethiopia

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Abstract

Pneumonic pasteurellosis is a common disease of the respiratory system of cattle as a result of inflammation of pulmonary parenchyma which is usually accompanied by inflammation of bronchioles and often by pleurisy. This review was made to overview pneumonic pasteurellosis which is caused by a gram-negative bacteria pathogen called *M. haemolytica* (*P. haemolytica biotype A) serotype* 1. The disease occurs most commonly in young growing cattle. Stress is an intrinsic condition that was consistently reported to increase the susceptibility to various types of infectious disease. Pasteurellosis with its a worldwide occurrence but it is a particular problem in the tropics especially the hot, humid tropics where environmental stress is an important trigger mechanism of the disease complex. The global economic impact of the disease is very well recognized and more than one billion dollars are annually cost in beef cattle industry. Taking tracheal swabs as microbiology culture from the lower respiratory tract is the most important sample for laboratory diagnosis. Broad spectrum antibiotics are used commonly. Prevention and control of Pneumonic Pasteurollosis should give an emphasis on minimizing the predisposing factors in addition with vaccination and management where herds are at high risk. Chemoprophylactic measures are also mandatory for the prevention of seasonal outbreaks. The disease has a great impact on animals' production. Improvement of the management system and providing prophylactic drugs in case of transportation during loading of animals are use full.

Keywords: Cattle, Pneumonic Pasteurellosis, Predisposing Factors, Vaccination

Introduction

Respiratory tract infections are a common occurrence in various species of domestic and farm animals. However, pneumonic pasteurellosis, also known as the respiratory mannheimiosis, is the most common example with a wide prevalence in ruminant animals. *Mannheimia haemolytica*, *Bibersteinia trehallosi* and *Pasteurella multocida* were involved as etiological agents of the disease, which are commensally resident in the upper respiratory tract of healthy ruminants and are capable of causing infection in animals with compromised pulmonary defense system [1]. Hence the disease is essentially triggered by physical or physiological stress created by adverse environmental and climatic conditions such as

extremely bad weather, poor management, overcrowding, transportation or previous infection with respiratory viruses, mycoplasma or some other pathogenic organisms [2].

The disease, in its typical clinical form, is highly infectious, often fatal, and with a very serious economic impact on the animal industry. It is well established that pneumonic pasteurellosis is responsible for the largest cause of mortality in feedlot animals in which the disease accounts for approximately 30% of the total cattle deaths worldwide [1]. The global economic impact of the disease is very well recognized and more than one billion dollars are annually lost in beef cattle industry in North America alone [3]. The catastrophic effect of the disease was also evident in sheep and goat farming and remarkable economic losses were also attributed to massive fatalities in feedlot animals and acute field outbreaks. In addition, substantial amount of money was further lost, almost every year, in improving farm management, animal husbandry and chemotherapeutic and vaccination programs, due to death and loss of production following illness [3]. *Mannheimia haemolytica* biotype A serotype1 is the most common cause of pneumonia. Eleven serotypes have been demonstrated within *M. haemolytica*, Serotypes 6, 2, 9 and 11 and untypable serotypes have been found in lesions of Pneumonic Pasteurellosis [4].
Several respiratory viruses including PI-3 virus, BHV-1 and BRSV may predispose the bacterial incubation [5]. An acute fatal respiratory disease was also induced in cattle by previous infection BHV-1 challenged with *M. haemolytica* [4]. Clinical signs of respiratory distress usually develop within 10 to 14 days, in adult animals after being exposed to stress [6]. Infected animals appear extremely dull with reduced appetite, depression, bronchopneumonia, abnormal lung sound, coughing, mucoid to mucopurulent nasal discharge, high fever (40-41°C or 104-106°F). Risk factors such as animal risk factor, environmental, managemental risk and the pathogen risk factor are involved in the complexity of the disease, as well as virulence factors including endotoxin, fimbrrae and leukotoxin are involved in the pathogenesis [7].

Generally, diagnosis relies on bacterial culture and specimen can be collected from the lower respiratory tract by tracheal swabs or bronchoalveolar lavage. Early recognition and treatment with antibiotics are essential for successful therapy [4]. Vaccination regimes for respiratory pathogens should be completed at least 3 weeks before transportation and vaccine *M. haemolytica* which incorporate modified leucotoxin and surface antigen may induce antibody production [8].
Young calves less than two years old age needs more protection and supplement with ration in order to develop immunologically and to satisfy the required growth. But in developing countries including Ethiopia, these practices cannot be learned as much. The farmers in the country simply let their calves to the field, especially in the pastoral areas, since there is lack of education providing calves with potable clear water; feed and disinfected calf house is unexpected. So the young calves lack the ability to gain the required body weight, lack of performance poorly grows and contracted by different disease. This condition might cause high mortality in the susceptible young calves and financial losses to the cattle industry in the country [9]. Therefore, the objectives of this seminar paper are;

Therefore, the objectives of this review paper are:

- To make a review on the epidemiology of pneumonic pasteurellosis in cattle.
- To review on the pathogenesis and virulent mechanism, prevention and control strategies of pneumonic pasteurellosis in cattle.

PNEUMONIC PASTEURELLOSIS

Definition of Pneumonic Pasteurellosis

Pneumonic pasteurellosis is a common disease of respiratory system of cattle as a result of inflammation of pulmonary parenchyma which is usually accompanied by inflammation of bronchioles and often by pleurisy [1].

Etiology of the disease

Pasteurella is a major cause of severe “shipping fever” pneumonia when combined with stress and with and without viral agents. The major bacterial

pathogen involved in pneumonic pasteurellosis of cattle is *M. haemolytica* (*P. haemolytica* biotype A) serotype 1 [1]. Eleven serotypes have been demonstrated within *M. haemolytica*, Serotypes 6, 2, 9 and 11 and untypable serotypes have been found in lesions of Pneumonic Pasteurellosis. *P. trehalosi* strains are commonly isolated causes and *P. multocida* biotype occasionally [10]. However, the pathogenic role of *P. multocida* was more evident in sheep in which it was responsible for many serious outbreaks [1]. The bacteria is Gram-negative, non-motile, non-spore forming, facultative anaerobic, small rods or coccobacilli [11]. *P. multocida* and *M. haemolytica* are similar with each other but unlike *P. multocida*, *M. haemolytica* does not ferment maltose results in hemolysis and *P. multocida* also does not ferment arabinose and cannot result in hemolysis as well as does not grow in Mac Conkay agar [5].

Geographic Distribution and Occurrence

Pasteurellosis occurs worldwide but it is a particular problem in the tropics especially the hot, humid tropics where environmental stress is an important trigger mechanism of the disease complex [12]. It is a common disease of young growing cattle and common in America, the UK and North America. In Canada and the United states the disease occurs most commonly in beef calves after weaning in the fall of the year and is the most important disease in cattle that have been recently introduced into the feedlots [1]. The outbreaks of mannheimiosis are generally noted at the beginning of the rainy season; however, the disease can occur throughout the year in the endemic areas [4].

Factors affecting the disease

1. Animal Risk Factors

The disease occurs most commonly in young growing cattle from 6 months to 2 years of age, but all age groups are susceptible. Calves those are non-immune to *M.haemolytica* than calves that have serum neutralizing antibody to the organism and its cytotoxin. In western Canada auction market calves that originated from many different farms and remixed at the market are at high risk. However the distance that the calves were transported was not associated with fatal fibrinous pneumonia [13]. Although the disease occurs most commonly in young beef cattle soon after their introduction to feedlot, it is not uncommon on dairy herd, especially when recent have been made. Mature beef cows are also susceptible to Pneumonic Pasteurellosis if they are subjected to stress during the summer months or in the fall of the year. Usually associated with the movement of large groups to or from pasture during inclement weather herd outbreak of per acute pleuropneumonia due to *M. haemolytica* have been reported in adult dairy cattle [14].

2. Environmental and Managemental Risk Factors

Mixing of cattle from different source is an important risk factor, mixing of recently weaned beef calves from different source at auction market was associated with an increased risk of fatal fibrinopneumonia in calves moved to feedlot in a western Canada, especially in November shortly after action sales had peaked and when feedlot was reaching capacity [13].

Stress is an intrinsic condition that was consistently reported to increase the susceptibility to various types of infectious disease. Stress can be induced artificially by administration of certain drugs and chemical compounds like dexamethasone [15]. Containment in dairy, humid and poorly ventilated barns, exposure to increment weathers, deprivation from feed and water are commonly followed by an outbreak of the disease in cattle. The reason for increased susceptibility to *M. haemolytica* infection in stressed animals was primarily attributed to the breakdown of innate pulmonary immune barriers by stressors [16].

3. Pathogen Risk Factors

The frequency of isolation of Mannheimia species from the nasal passage of normal healthy unstressed calves is low but increased as animals

are moved to auction market and then feedlot. The virulence factors of pasteurella species include fimbriae, polysaccharides, endotoxin and leucotoxins have been identified [7]. Respiratory viruses were reported to impair the phagocytic function of pulmonary alveolar macrophage. An acute fatal respiratory disease was also induced in cattle by previous infection with Bovine herpes virus-1 challenged with *M. haemolytica. M. haemolytica* serotype A1 and A2 can survive for long period at time at least 156 days in bovine and trachea bronchial washings [17].

Morbidity and Mortality

Morbidity and mortality are affected by a number of factors and their interaction. Age, endemicity of the region, previous exposure and immunity are important factors. The high susceptibility of young animals has been established in several studies in Sri Lanka. Morbidity may reach 35%, the case fatality rate may range from 5-10% and population mortality rate may vary from 0.75-1%. However, these morbidity and mortality may not be reliable because of wide variations in the method used to calculate disease incidence and prevalence. More recently in Britain, morbidity rates of 73-100% with mortality of 0-8% of those affected and average mortality of 4% have been reported [16].

Method of Transmission

Transmission of pasteurellas probably occur by inhalation of infected droplet, coughed up or exhaled from infected animals which may be clinical case or recovered carriers in which the infection persist in the upper respiratory tract. *Mannheimia haemolytica* is carried in the nasopharynx and tonsils of apparently health animals where, interestingly, serotyping is most commonly isolated from cattle [18].

Pasteurella multocida and *Mannheimia haemolytica* are highly susceptible to environmental influence and it is unlikely that mediated contagion is an important factor in the spread of the disease. When conditions are optimal, particularly when cattle are closely confined in inadequately ventilated trains or held for long periods in holding pens and feedlots, the disease may spread very quickly and affect a high proportion of the herd within 48 hours. Animals at pasture are able to move freely and the rate of spread may be slower [19].

Pathogenesis and Virulence Factors

Remaining in the URT of unstressed cattle, *M. haemolytica* A1 causes no disease in the animal. Under times of stress in the form of transportation, crowding, irregular feeding or watering, abrupt climate change, exposure to viral agents, or combination of the above, M. haemolytica1: A proliferates in the URT [20]. Stress also disrupts *M. haemolytica*1: A localization and allows the bacterium to colonize other parts of the respiratory tract. Conditions such as stress or cell damage and inflammation are ideal for *M. haemolytica* A1 colonization and proliferation in the upper respiratory tract (URT). Stressed cattle shed large numbers of *M. haemolytica*1: A, which can be isolated during and immediately after shipment. Increased colonization and proliferation of *M. haemolytica* A1 in the URT allows the organism to be inhaled into the lung [20].

Researchers Have Shown That *M. Haemolytica* A1 can be isolated from droplet nuclei in tracheal air [21]. In the lower respiratory tract (LRT), *M. haemolytica*1: A would be easily cleared and no lung damage would occur under normal conditions [22]. However, chronic exposure to the lung allows *M. haemolytica*1: A to overcome clearance mechanisms and to initiate pneumonia. Intranasal and intratracheal inoculation of *M. haemolytica*1: A, as well as transthoracic inoculation of *M. haemolytica*1: A directly into the lung has been shown to produce pneumonic lung lesions [20]. Indeed, *M. haemolytica* is the most common isolated bacterium from shipping fever, but the proportion of fatal cases of respiratory disease in feedlot cattle attributable *to P. multocida* appears to be increasing [23].

Stress and viral infection would eventually impair the local pulmonary defense mechanism by causing the deleterious effects on ciliated cells and mucous coating of the trachea, bronchi and bronchioles. The causative bacteria from the nasopharynx will then reach the central bronchi, bronchioles and alveoli by gravitation at drainage along the tracheal floor and there by become deeply introduced into the lung tissue. Toxin produced by rapid growth and multiplication of the bacteria result in vascular disturbance and inflammatory reaction dominated by fibrinous exudates [24].

Four main virulence factors have been identified in strain of *M. haemolytica* and *P. trehalosi.* Fimbriae are small appendage, present in the surface of many gram-negative bacteria which enhance adherence to and colonization of the target epithelium of the susceptible animals. Two types of fimbriae have been detected in serotype 1 of *M. haemolytica* [25]. Both of them are capable of enhancing mucosal attachment of the organism and colonization of lower respiratory tract epithelium of cattle. Successful colonization will thus enable considerable increase in the number of bacteria needed in the lung tissue beyond the level that normal lung capacity could efficiently resolve [26]. Similar to all other gram-negative bacteria the cell wall of *M. haemolytica* contains Lipid Polysaccharide. This endotoxin is one of the most virulence factors involved in the pathogenesis of Pneumonic Pasteurollosis. It has been shown that serotypes 2 and 8 of *M. haemolytica* possess rough LPS while the other 14 serotypes have characteristic smooth LPS [4].

Endotoxins produced by rapid Growth and multiplication of the bacteria In infected lobules will cause extensive Intravascular thrombosis of pulmonary Veins, capillaries and lymphatics. These Vascular disturbances eventually result in Focal ischaemic necrosis of the pulmonary Parenchyma accompanied by severe inflammatory Reaction dominated by fibrinous Exudate [27]. Experimental evidence indicated that M.haemolytica endotoxin is directly toxic to endothelial cells and capable of altering leukocyte functions and causing lysis of blood platelets [4].

Leukotoxin is heat-labile protein, a pore-forming cytolysin that affects ruminant leukocytes and platelets and also considered as a main virulence factor for *M. haemolytica* [28]. The most susceptible cells are bovine macrophages, neutrophils and lymphocytes. At low concentration, leukotoxin impairs phagocytosis and lymphocyte proliferation while at higher concentration it has cytotoxic effect resulting in cell death due to lysis. The polysaccharide capsule of the organism inhibits complement mediated serum killing as well as phagocytosis and intracellular killing of the organism. The capsules also enhance neutrophil-directed migration and adhesion of the organism to alveolar epithelium. The interactions of these virulence factors contribute to the pathogenesis of the disease [29].

Clinical Signs

An observable clinical signs of respiratory distress usually develop within 10 to 14 days in adult mammals after being exposed to stress. Nevertheless, infected animals in severe cases may die as a result of toxemia even before the development of significant pulmonary lesions. In this case, sudden death may be the first sign of acute out breaks, particularly in young calves [1]. The incubation period of the disease ranges from 3 to5 days, however, acute onset is not uncommon. After the onset of respiratory disturbances, infected animals appear extremely dull with reduced appetite and remarkable depression. They soon develop high fever (40-41°c or 104-106°F). Anorexia and rapid shallow respiration accompanied by mucopurulent nasal discharge. Later on productive cough which is accentuated by physical effort or movement usually develop in most of the infected animals [30]. In acute outbreak, the clinical course of the disease is relatively short (2-3 days) terminating in death or recovery in either treatment or non-treated animals. However, number of sick animals that survive the acute phase may become chronically infected animals. Marked dyspnea with an expiratory grunt may be observed in very advanced stages of disease [31].

1. Post Mortem Examinations

There is marked pulmonary consolidation, usually involving at least the antero-ventral part of the lungs. The lung is firm and the cut surface usually reveals an irregular, variegated pattern of red, white and gray tissue due to hemorrhage and necrosis. Occasionally sequestrate of necrotic lung tissue are found. *P. multicoda* cause fibrino-purulent bronchopneumonia without the multifocal coagulation hemolytic necrosis that characteristics of fibrinous lobar pneumonia associated with *M. haemolytic*a. The post mortem findings of lung consolidation and pleurisy are present. The basic post mortem lesions are acute fibrin hemorrhagic pneumonia with pleurisy adhesion [31].

Diagnosis

Diagnosis is depending on the history of age, recent movement, weaning or housing, isolation and identification of the causative agent is important [21]. In addition isolate and identify the *Pasteurella* species from apparently healthy and suspected cases of Pasteurellosis in cattle and comparative cultural, morphological and biochemical characteristics of the isolated *Pasteurella* species as well as comparative pathogenicity of cattle *Pasteurella* species in mice model and its antibiogram should be done [32].

1. Clinical Findings

The spectrum of clinical finding depends in part on whether the disease is occurring in groups of young cattle in large commercial feedlot, to in small farm feedlot or in individual animals such as lactating dairy cows in which illness is more early recognized by drop in milk production and feed intake. In the feedlot situation, affected animals must be identified primarily by visual observation followed by closer physical examination. However, close physical examination such as auscultation of the lungs have not been routinely used in feedlot, because of the time required to examine individual animals and the perceived accuracy of the examination in making clinical diagnosis [1].

The respiratory rate increased from 30 per minute up to 70 per minute as the percentage of lung consolidation increased from 10% to 50%. The typical case of pneumonic pasteurollosis reveals a fever (40-41°C or 104-106°F). In early stages there are loud breathing sounds audible over the anterior and ventral part of the lung. Clinically when viewed from a distance, affected cattle are depressed and up on auscultation, rapid shallow respiration with loudness of breath sounds, nasal and ocular discharge are present [17].

2. Laboratory Diagnosis

Microbiology cultures from the lower respiratory tract by tracheal swabs, transtracheal wash, or bronchoalveolar lavage are the most important laboratory diagnosis. Impression smears show a bipolar staining organism with methylene blue. Examination of nasal swab samples from the clinical cases before treatment often yields bacteriological samples for pasteurella in which *M. haemolytica* biotype A serotype 1 is most common isolate obtained from cattle with Pneumonic Pasteurellosis. *M. haemolytica* or *P. multocida* may be isolated from nasal swabs in live animals [33] Serology and mere isolation of *P. multocida* from nasal swabs is of little value without being able to predict pathogenicity. The application of the polymerase chain reaction (PCR) to detect and differentiate toxin producing and nontoxin producing *P. multocida* may prove to be a useful technique for control of both pneumonic pasteurellosis and atrophic rhinitis [33].

3. Differential Diagnosis

The combined infection with certain respiratory viruses is commonly found to increase the susceptibility of farm animals to secondary bacterial pneumonias [34]. Conditions that may be mistaken for pneumonic pasteurellosis include; Contagious Bovine pleuropneumonia, Infectious Bovine Rhinotrachitis Verminous pneumonia caused by *Dictyocaulus viviparous* and viral interstitial pneumonia and sporadic conditions, such as lung abscesses and aspiration pneumonia. The finding of a fibrinous or fibrino-

bronchopneumonia on necropsy is highly suggestive of pneumonic pasteurellosis, although *Haemophilus somnus*, also a member of the family pasteurelleae, can cause fibrinous lesions [4].

Treatment

Treatment should begin early. Most cattle will show some improvement within one to three days of initiating treatment. Broad spectrum antibiotics are used commonly. Antibiotics most commonly used are Oxytetracycline at rate 20mg/kg BW, IM, long acting and 10mg/kg daily for 3 days short acting; Tilmicosin at rate 10mg/kg BW, SC and repeat 72hr later if necessary; florfenicol (Analog of thiamphenicol) 20mg/kg BW, IM repeat 48 hr and mass medication with sultomethazine 100mg/kg BW in drinking water for 5-7 days. Tilmicosin is effective in reducing the population of *M. haemolytica* that colonizing the nasal cavities of calves with respiratory disease [35]. In veterinary medicine, particularly food animal production, diagnostic testing is used for prevention and control and monitoring as described which then include vaccination, antimicrobial treatment for infected animals and implementation of a biosecurity plan are mandatory [36]. Florfenical given on arrival reduce the incidence of respiratory diseases and reduce the colonization of nasopharynx by *M. haemolytica* [37]. If pulmonary abscessation has occurred, it is difficult to achieve resolution with antimicrobials and culling of animal should be considered. NSAIDS have been shown to be a beneficial ancillary therapy in treating bacterial pneumonia [4].

Prevention and Control

Prevention and control of Pneumonic Pasteurollosis has centered on the predisposing factors in combination with vaccination and management where herds are at high risk [4]. These bacteria are part of the normal microbiota in the upper respiratory tract making the disease difficult to prevent [38]. On the other hand a study reported that there is a various possibilities to prevent and control infections due to *Pasteurella* and *Mannheimia* species in animals [39].

1. Management Strategies

Because of common occurrence of the disease at the time of shipment from the range to the feedlot, much attention has been given to reduce the incidence of disease at this time. The calves should be transported from the farm of origin directly to the fattening unit. The transport distance should be as short as possible and the animal should be handled in calm and considerate manners at all stages of transport. The calves could weaned and introduced to fattening diet at least two weeks before leaving farm [20].

2. Vaccination

Pasteurella vaccines and respiratory viral vaccines have been used extensively in an attempt to control Pneumonic Pasteurellosis in cattle. However, their efficacy appeared to be low and literature review suggests that at present there is little evidence to show efficacy of such vaccines under feedlot conditions. Vaccination regimes for respiratory pathogens should be completed at least 3 weeks before transportation and vaccine for *M. haemolytica* incorporate modified leukotoxin and surface antigen induce production [8]. The experimental lung challenge of calves with formalin killed *P. multocida* does not provide subsequent protection to challenge with live *P. multicida* [4].

Single vaccination of *M. haemolytica* bacterin toxoid given to calves on arrival in the feedlot reduced overall mortality. Vaccination of calves after arrival in the feedlot with genetically attenuated leucotoxin *M. haemolytica* combined with its extracts reduced morbidity due to bovine respiratory disease. Several outer membrane protein of *P. multocida* type A3, which occasionally causes severe bronchopneumonia in cattle, may be important for immunity for organism [40]. Vaccination of colostrum-deprived calves at 2 and 4 weeks of age with *M. haemolytica*, a culture supernatant vaccine

resulted in high titer of IgA antibody to capsular polysaccharides within one week of vaccination [41].

3. Chemoprophylaxis

Chemoprophylactic measures for preventing Pneumonic Pasteurellosis are useful for preventing the outbreak of the disease, especially when disease provoking stress is consciously put up with. Application of long acting Oxtetracycline before shipping animals over a long distance will protect the animals effectively against shipping fever. The antibiotic chemoprophylaxis of pasteurellosis is the only way to stop the infection immediately during a sudden outbreak and prevents its spreading to other animals or herd. In such cases, the chemoprophylaxis replaces the application of hyperimmune serum which used to be applied [12].

Economic Importance

It is well established that Pneumonic Pasteurellosis is responsible for the largest cause of mortality in feedlot animals in which the disease accounts for appropriately 30% of the total cattle death worldwide. The global economic impact of the disease is very well recognized and more than one billion dollars are annually cost in beef cattle industry. In addition to the death losses, the cost of treatment is considerable. It is also a disease of great importance in North America and Britain were it has caused great loss since before 1990s [3].

STATUS OF PNEUMONIC PASTEURELLOSIS OF CATTLE IN ETHIOPIA

Few studies are conducted in Ethiopia to determine the extent of the problem and the relative distribution of different biotypes and serotypes of Mannheimia species. In those studies, there are indications to the prominence of *M. Haemolytica* especially serotype A1 and A2 are the most common in the country obtained from nasal and transtracheal swab with morphology, phenotypical and conventional characterization [42]. However, molecular advances need to know the prevalence and the tangible organisms elaborate bovine and ovine pneumonic pasteurellosis concerning serotype distribution and the etiological diversity of the agent in the country [42]. Losses due to death were also noticed in ruminants confined in quarantine stations for export and on the farm [43].

In a study undertaken in calves with clinical signs of respiratory disease in the same area, *M. Haemolytica* and *P. Multocida* isolates were obtained from nasal and transtracheal swabs [44]. *M. Haemolytica* serotype A and A2 are the most common in the country. However, no study has been done to know the prevalence and the actual organisms involved in Pneumonic Pasteurellosis of cattle but few studies have been done concerning Ovine Pneumonic Pasteurellosis in Central, North, Eastern and South Eastern high lands of the country [45, 46].

In Ethiopia among few studies the prevalence in ruminants is found to be high and eleven of the 17 known serotypes of *M. Haemolytica , M. Glucosida* and *B. Trehalosi* has so far been isolated and identified in ovine in central, northeastern and southeastern high lands of the country [43]. In Milae Districts of Afar region from an outbreak in 2000 in sheep and goat *M. Haemolytica* biotype T was isolated from nasal swabs and lung and pleural fluid. In addition calves with clinical signs of respiratory disease in the same area *M.haemolytica* and *P.Multocida* isolates were obtained from nasal and transtracheal swabs [43]. The prevalence of pneumonia caused by these bacteria has been considered to be important constraints in Ethiopia by Tibbo *et al.* [47],Weldemeskel *et al.* [48] incurring huge economic loss. Similarly, in Ethiopia, respiratory problems due to *M. haemolytica, B. trehalosi* and *P. multocida* cause significant mortality and morbidity and are responsible for huge treatment cost [49].

Recent studies indicated that most cases of ruminant pasteurellosis are caused by *M. haemolytica* and vaccine produced by the NVI against the disease is from *P. multocida* serotype A and B which does not correspond to the real causative agent [42]. This may be one possible explanation for high mortality observed from respiratory distress in North Showa (Ethiopia) despite the annual vaccination using monovalent vaccine. The presence of multiple serotypes of *M. haemolytica* as well as *B. trehalosi* without cross protection becomes a challenge for the development of vaccine that is effective worldwide [43].

Conclusion and Recommendation

Pneumonic Pasteurellosis is a highly complex multifactorial disease of a worldwide prevalence and distribution in cattle. The disease primarily results from interaction of stress, immunity and the causative bacteria (*M. haemolytica*) which is commensally resident in the respiratory tract of susceptible animals. The major factors leading to stress and compromised immunity are naturally created by adverse environmental and climatic conditions and also by previous or co-infection with certain respiratory viruses, mycoplasma or some other types of bacteria. The disease is mostly transmitted by inhalation from infected droplets. Also the disease has typical clinical form, highly infectious, often fatal and economically important. It is a major cause of morbidity globally. Diagnosis is depending on the history of age, recent movement, weaning or housing, isolation and identification of the causative agent is important. Effective control is based on management, vaccination and chemoprophylaxis. Based on the above conclusion, the following recommendations are forwarded:

- Infected animals should be isolated and treated as early as possible.
- Prevent and avoid stress factors that predispose animals to pneumonic pasteurellosis;
- Emphasis should be given on management system improvement practices.
- Providing prophylactic drugs during loading of animals in case of transportation.
- Vaccination of cattle should be given at least 3 weeks before transportation.
- Further investigation has to be conducted on the status of pneumonic pasteurellosis in cattle in Ethiopia in terms of prevention and control of the disease.

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