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Review Article

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Major Pulmonary Lesions and Their Etiology in Dromedary Camel (*Camelus dromedarius*) with particular emphasis to Ethiopian situation

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Abstract

There are a few investigations about camel lung pathology worldwide. Various pathological lesions including pneumonia, hydatidosis, pulmonary abscesses, pleuritis, bronchitis, pneumonicosis, tubercle nodules, linear scars and primary bronchioalveolar adenocarcinoma have been described in the lungs of camels in literature. In addition to these lesions, pulmonary involvements have been observed in specific diseases such as camelpox, tuberculosis, aspergillosis and respiratory sonbobe disease. This review emphasizes on major
The aim of this review article was to summarise relevant clinical, etio-epidemiological and pathological data available in the current literature regarding **gross** and microscopic pulmonary lesions of dromedary camels and their causative agents. Scientific resources such as Pubmed, Google scholar and Research gate were searched for all published articles in dromedary camels.

Keywords: Dromedary camels, pathology, pulmonary lesions

Introduction

The respiratory system in general constitutes the most extensive surface and gets exposed directly to the environment. Any sudden change in the environment precipitates the infection by interfering with the local defense and rendering the system more susceptible to infections. Various infectious and noninfectious agents can damage lungs and produce significant lesions (Mekuriaw *et al.*, 2016). This implies that the respiratory tract is more prone to injurious agents although major airways and lung parenchyma neutralize or remove the infectious agents that are deposited (Gabinaitiene *et al.*, 2011).

Different infectious agents such as bacteria, viruses and parasites, play a synergistic and interactive role in the etiology of respiratory system diseases observed in cattle. Out of them, bacterial diseases have drawn attention due to variable clinical manifestations, severity of

diseases, and reemergence of strains resistant to a number of chemotherapeutic agents (Chakraborty *et al.,* 2014). Even though the primary factors are infectious agents, environmental and management factors are also important in clinical development and rise in economic losses (Pinar and kadir, 2012). The trend of these animals to huddle and group rearing practices further predispose ruminants to infectious and contagious diseases (Kumar *et al.,* 2016).

Although camels are well adapted to their environment and seem to be spared from devastating epidemic infections which threaten other livestock species in the same region, there are however a number of economically important diseases that affect camels. Pulmonary diseases are among the emerging problems of camels that are causing considerable loss in production and death (Abubakar*et al.,* 2010).

Tuberculosis

Bovine tuberculosis (bTB) is a major disease of cattle that can also affect humans, and many other livestock and wild animal species. Human infection has not been a major public health problem in developed countries since the introduction of milk pasteurization. Advanced cases in cattle experience loss of condition, and this directly affects the economic value of the animal, but in most developed countries detection of infection leads to movement restrictions being placed on the herd, mandatory slaughter and considerable indirect losses for the farmer(Godfray *et al.,* 2013).

Mycobacteria of the Mycobacterium tuberculosis complex cause tuberculosis in various mammalian hosts but exhibit specific host tropisms. The two major pathogenic species in this complex are *M. tuberculosis* and *M. bovis*, the causative agents of tuberculosis in humans and cattle, respectively. However, it is well known that *M. bovis* is zoonotic, while infection with *M. tuberculosis* has been sporadically reported in domestic and wild animal species, most frequently in animals living in prolonged, close contact with humans. Among domestic animals, infection with *M. tuberculosis* has been most frequently identified in cattle (Ameni *et al.,* 2011). According to(Mamo*et al.,* 2011) in Ethiopia they isolate *M. bovis*strain (SB0133) from camel which is similar to cattle strain in pastoral area of East Africa, implies the existence of potential inter-species transmission of the strain among livestock of pastoral area.

Mycobacterium bovis can be shed from virtually any body orifice. The respiratory route is consistently described as the major route of infection although oral infection is also common. This inference is largely based on location of lesions. Once bacteria entered through aerosolized droplets or ingestion it is established in a herd of cattle. The incubation period can range from months to years with the severity depending on the immune system of each individual animal. The bacteria usually enter the respiratory system of a cow and settle in the lungs. Macrophages in lungs are then responsible for phagocytizing the organism. The organism replicates intracellularly after it has been taken up by the macrophages. A granuloma or tubercle forms as the body tries to wall off the infected macrophages with fibrous tissue. The infection can spread hematogenously to lymph nodes and other areas of the body and cause smaller, two to three mm in diameter, tubercles. The formation of these smaller tubercles is known as “miliary tuberculosis” (Jemal, 2016).

The disease characterized by a gross pathology named tubercles mostly found in the lungs and lymph nodes. However, it occasionally affects other tissues such as the pleura, lactating glands, gastrointestinal and urogenital tracts(Ahmad *et al.,* 2019). The most common form is pulmonary tuberculosis, characterized pathologically by necrotizing granulomas, associated pneumonia(Gupta *et al.,* 2016), collection of epithelioid and giant cells surrounded by a layer of fibroblasts and lymphocytes, having the center of the granuloma necrotic and calcified(Headley, 2002).

The disease in camel was reported in Ethiopia by Richard, and a spontaneous case of camel tuberculosis in Somaliland has been described by

Pellegrini. It was characterized by progressive debility, coughing and death within six months. Caseous nodules were found in the lungs, liver, spleen and lymph nodes. Granulomatous masses with caseation were present in mediastinal lymph nodes, and grape-like lesions in the pleural cavity (Mustafa, 1997).

Contagious Bovine Pleuropneumonia/CBPP

Contagious bovine pleuropneumonia (CBPP), also called cattle lung disease, is a highly contagious disease of cattle and water buffaloes caused by a bacterium (Elizabeth *et al.*, 2015).CBPP is caused by *Mycoplasma mycoides subsp. mycoides*, a member of the *Mycoplasma mycoides* cluster, comprising four additional closely related mycoplasmas, i.e., *M. mycoides subsp. capri*, *M. capricolum subsp. capricolum*, *M. capricolum subsp. capripneumoniae*, and *M. leachii*, all causing diseases in ruminants (Heller *et al.*, 2016). The bacteria are primarily transmitted through the exchange and inhalation of infectious aerosols when animals are in close contact with each other. The bacteria can be found in nasal discharges, saliva, urine, fetal membranes, and uterine excretions. Transplacental transmission can also occur. Infected CBPP cattle without clinical signs may shed the bacteria when stressed (Admassu et al., 2015; FADPREP, 2017).

Many authors were reported that CBPP is typical example of multi-factorial diseases, where factors such as inter-current infections, crowding, inclement climatic conditions, age, genetic constitution and stress from transportation, handling and experimentation are important determinants of the final outcome of infection. An essential part of the pathogenesis of the disease is thrombosis in the pulmonary vessels, probably prior to the development of pneumonic lesions. Natural infection is by inhalation and results in Bronchitis, alveolitis, bronchiolitis with predominantly neutrophils and mononuclear cellular response constitute the very early inflammation in Mycoplasma pneumonia It is lobar variety of pneumonia in which the inter lobular septa are dilated and prominent due to a great out pouring of plasma and fibrin in to them and it this dilated septa that give the “marbling” effect to the lung in these area (Abera*et al.,* 2016).

The pathological changes, generally characterized by the involvement of only one lung and its gross macroscopic aspects, are well known and may be used for disease surveillance at abattoirs. However, the pathogenetic mechanism of the disease is not yet fully understood. It has been suggested that auto immune and hypersensitive reactions are essential in the development of lesions. CBPP induced lesions vary during the course of the disease (Provvido*et al.,* 2017).

Gross pathological lesions in the acute stage are characterized by fibrinous deposits on the parietal surfaces of lungs and distension of the interlobular spaces with straw colored serofibrinous exudate. Lesions are usually unilateral, localized in the diaphragmatic lobe and present a characteristic marbling appearance. Lesions are detectable on palpation, and upon incision, red and grey areas of hepatization are revealed. In subacute cases, lesions are characterized by necrosis organized within lobules and interlobular septa and early sequestrum formation. Lesions in the chronic stage are characterized by well-defined sequestra surrounded by fibrous capsules. Adhesions, connecting thickened viscera and parietal pleura, are common (Malamsha, 2009).

The lesions develop first in the lymphatic system. Thrombi that develop in the lymphatics cause coagulation of lymph, distension of interlobular septa and focal perivascular round cell infiltration. The formation of cuffs of round cells around the arterioles is the only histological pathognomonic characteristic of CBPP. The secondary lesion is characterized by alveolar involvement due to the accumulation of exudate from the foregoing changes. Necrotic foci surrounded by a band of polymorphonuclear granulocytes often develop. These foci may develop into sequestra in chronic cases (Laak, 1992).

Emphysema

Pulmonary emphysema is anatomically defined as the ‘‘abnormal permanent enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of their walls” (Fehrenbach, 2006).Pulmonary emphysema is distension of the lung caused by over distension of alveoli with rupture of alveolar walls with or without escape of air into the interstitial spaces. Over inflation describes the situation in which there is enlargement of airspaces without tissue destruction. Pulmonary emphysema is always secondary to some primary lesion which effectively traps an excessive amount of air in the alveoli. It is a common clinic pathological finding in many diseases of the lungs of all species and is characterized clinically by dyspnea, hyperpnea, poor exercise tolerance and forced expiration (Radostits, *et al.,* 2006).

Pulmonary emphysema is an important lesion only in cattle, although occasional cases occur in pigs. The bovine lung is highly susceptible to the development of emphysema from many different causes, not all of them respiratory in origin. In those of respiratory origin it is common to find pulmonary emphysema when the primary lesion in the lung causes trapping of air in alveoli or terminal bronchioles. Acute interstitial pneumonia, parasitic pneumonia with pulmonary, edema in acute anaphylaxis, Perforation of the lung by foreign body as in traumatic reticuloperitonitis are recorded as causing pulmonary emphysema in cattle. An imbalance between protease and antiprotease activity in the lung is proposed as the major mechanism resulting in emphysema. The imbalance is mostly due to an increase in the numbers of alveolar macrophages and neutrophils. Emphysema can also develop from increased alveolar wall cell death and/or failure in alveolar wall maintenance. Chronic inflammation and increased oxidative stress contribute to increased destruction and/ or impaired lung maintenance and repair (Sharafkhaneh, *et al.,* 2007).

Chronic bronchitis or bronchiolitis can causes obstruction of airways on expiration due to exudates plugging airway passages. This causes airway imbalance where the volume of air entering exceeds the volume of air leaving the lung. “Check valve” lesion in which air is still able to enter alveoli on inspiration but is unable to leave freely”. Pulmonary emphysema can be divided into two broad categories, alveolar and interstitial. In alveolar emphysema the alveoli are distended by excessive amounts of air pressure and often times rupture. In interstitial emphysema the air accumulates in the sub-pleural, interstitial, and intralobular regions of the lungs (Schroeder, 2005). The lungs do not collapse when the thorax is opened and often carry the impression of the ribs. Air pockets are often present and these may be partially filled with coagulated blood. The emphysema may extend to the mediastinal space and reach the musculature and subcutaneous tissue of the dorsal cervical and thoracic regions. Edema is present in the lungs and the air passages contain frothy fluid. The pericardial sac often contains gelatinous transudate. Histopathologically, there is edema and interstitial and alveolar emphysema of the lungs (O'Donoghue, 1960).

Edema

Pulmonary edema is the accumulation of excess fluid in the extravascular space of the lungs (McGavin and Zachary, 2012). Edema of the lung was, in many respects, similar to edema of other tissues, and is governed by the permeability of the vascular wall and by Starling forces-the balance of hydrostatic and osmotic pressures between the intravascular and interstitial compartments. Pulmonary edema is often complication of many diseases and is therefore one of the most commonly encountered pulmonary abnormalities. If severe, pulmonary edema has a catastrophic effect on lung function by reducing pulmonary compliance, blocking ventilation of the alveoli, obstructing gas exchange across the alveolar septa, and reducing the surface area of the air-liquid interface in the alveoli. In addition, proteins present in the edema fluid interfere with surfactant function, further reducing compliance and contributing to pulmonary dysfunction (Jubb*et al.,* 2005).Diffuse pulmonary edema was the predominant change in cattle. Edema is

prominent in the pleura and the pulmonary interstitium, and may form shallow pools in the hilus of the lung or the mediastinum (Muhammad *et al.,* 2010).

Edematous lungs macroscopically characterized by large, firm, dark red, heavier than normal and incision results in flow of fluid from cut surface (Jubb*et al.*, 2005). Section of the lung with edema histologically characterized by faintly pink stained edematous fluid is acidophilic granular material with in alveoli except for occasional discrete holes that represent trapped air bubbles and fibrosis in chronic cases. Chronic edema is accompanied by a diffuse increase in the number of alveolar macrophages, and in heart failure these may contain phagocytosed erythrocytes or hemosiderin (Rashid *et al.,* 2013).

Hemorrhage

Haemorrhage is escape of the blood from a vessel. It classified as haemorrhage by rhexis when there is rupture of a blood vessel; and haemorrhage by diapedesis when blood leaves through intact blood vessels. Pulmonary hemorrhages are associated with conditions affecting the blood vessels and conditions affecting the blood like severe septicemia or traumatic lesion to the lung (McGavin and Zachary, 2012).

In addition they can cause by lacerations, vasculitis, infarction, ruptured aneurysms, trauma, haemophilia, tumors that have undergone necrosis, or drug reactions. Aspiration of blood is frequent at slaughter, and has a characteristic pattern of multiple, small, bright-red foci with feathery or indistinct borders. Pulmonary hemorrhages vary from petechiation to massive filling of large regions by blood. Affected animals may be found dead with blood flowing from the nares. Agonal hemorrhages resulted from seizures and struggling during slaughter also results in pinpoint hemorrhage over lung surface particularly in the anterior lobes. Histological alterations are resulted from intra alveolar hemorrhage rather than the cause, and can be produced by intrapulmonary injection of auto logos blood and the affected regions characterized by hemosiderin-laden macrophages in airspaces and in the interstitium, alveolar septal fibrosis, and mild bronchiolitis and bronchiolar fibrosis (*Jubb et al.,* 2005). Grossly a haemorrhagic lung appears as variable size red, brown or grey discoloration of the lung or as patchy, blue-brown subpleural foci (McGavin and Zachary, 2012).

Hydatidosis

Hydatidosis (cystic echnococcusis) is one of the most important parasitic diseases of ruminants responsible for huge economic losses due to reduction in carcass weight gain and condemnation of organs. Hydatidosis is a zoonotic parasitic disease caused by larval stages (hydatid cysts) of cestodes belonging to the genus *Echinococcus* and the family *Taeniidae.* Hydatid cyst, which is the larval stage of Echinococcus, is a bladder like cyst formed in various organs and tissues following the growth of the oncospheres of an Echninoccus tape worm in that specific organ or tissue Martinma (Abegaz and Mohammode, 2018).

Certain deep-rooted traditional activities have been described as factors associated with the spread and high prevalence of the disease in some areas of the country. These can include the widespread backyard slaughter of animals, the corresponding absence of rigorous meat inspection procedures, the long standing habit of feeding domesticated dogs with condemned offal and the subsequent contamination of pasture and grazing fields. This can facilitate the maintenances of the life cycle of *E. granulosus* which is the causative agent of cystic hydatidosis and consequently the high rate of infection of susceptible hosts (Getachew*et al.,* 2012).

The life cycle of hydatidosis involves two mammalian hosts. The adult cestode inhibits the small intestine of carnivores (definitive host) and produces eggs containing infective oncosphers. Cestodes segments, proglotids containing eggs (free eggs) released from the intestinal tract of final host in to the environment. After ingestion of eggs by food animals (intermediate host) such as cattle, sheep, goats, swine and camel, the larval

stage (metacestod), develops in the visceral organs typically the matured metacestod produces numerous protoscolices, each having the potential to develop into an adult cestode after being ingested by the carnivore definitive host. Accidentally, ingestion of the eggs infects humans and other aberrant hosts (Abegaz and Mohammode, 2018). Condemnation of edible offal unfit for human consumption is the major economic loss incurred by Hydatidosis (Erbet*et al.,* 2010). Furthermore, Ibrahemet al.(2016) reported that hydatid cyst wall with intact or disrupted laminated membrane moderate to broad fibrous capsule. Protoscolices, congestion, haemorrhage, fibrosis, and heavy cellular infiltration composed of mononuclear cells mainly lymphocytes. Hyperplasia of the bronchial and bronchiolar epithelium, atelectasis and emphysema were seen in distant lung tissues.

Cryptococcosis

Cryptococcosis is a chronic, subacute to acute pulmonary, systemic or meningitic disease, initiated by the inhalation of basidiospores and/or desiccated yeast cells of *Cryptococcus neoformans*. Primary pulmonary infections have no diagnostic symptoms and are usually subclinical. On dissemination, the fungus usually shows a predilection for the central nervous system, however skin, bones and other visceral organs may also become involved. *C. neoformans* and *C. gattii* are regarded as the two principle pathogenic species. Outside the host, *C. neoformans* is believed to exist as a poorly or moderately encapsulated spherical to oval structure with a diameter ranging from 2 to 10 µm. The pathogenesis *of C. neoformans* infection is mediated by four main virulence factors that allow it to survive within the host environment; these include: The ability to grow at 37^0C, synthesis of an extracellular capsule, production of melanin and secretion of extracellular proteases (Refai*et al.,* 2014).

Histologically the cryptococcosis classified in to two categories there reactive pattern and minimally reactive pattern. Reactive pattern is characterized by a granulomatous inflammatory response composed of histiocytes, multinucleated giant cells and lymphocytic infiltration. Regions of necrosis are occasionally associated with neutrophilic infiltrates. Fibrotic nodules cryptococcomas, considered to be a variant of the reactive pattern-are also found. The minimally reactive pattern is characterized by minimal or no inflammatory response. Numerous spherical microorganisms or oval microorganisms (or a combination of the two) of 2-20 µm in diameter, surrounded by a light halo and arranged extracellularly, are seen. In some cases, complete destruction of tissue architecture is observed. According to some authors, the minimally reactive inflammatory pattern can be suggestive of poor prognosis (Severo*et al.,* 2009).

Atelectasis

Development of atelectasis is associated with decreased lung compliance, impairment of oxygenation, increased pulmonary vascular resistance, and development of lung injury (Duggan and Kavanagh, 2005).

Lung atelectasis is the failure of alveoli to open or the alveoli are collapsed and thus do not have air.Atelectasis is the collapse of certain portion of pulmonary tissue in absence of air content in alveoli. These lesions are classically localized in the apical and cardiac lobes, and more rarely in the diaphragmatic one. Seem to come with infections with Mycoplasma and Pasteurellaspecies.Macroscopicallyatelectatic lung characterized by depressed relative to aerated lung, homogeneously dark-red and the texture is fleshy or more firm and non-spongy than normal lung. Section of atelectic lung appears as slightly congested alveolar walls lying in close apposition with cleft-like residual lumina having sharp angular ends. Atelectatic alveoli often contain scant edema fluid and excess alveolar macrophages. The edema may result from hypoxic damage, hypoxic vasoconstriction, or reduced surfactant activity (Jubb*et al.,* 2005).

Pneumonia

Pneumonia is an inflammation of the tissues of the lungs that results from the response of the animal to an infectious agent, either a virus or bacteria, or in most cases both. Common viruses that can initiate pneumonia in cattle include: infectious bovine rhinotracheitis virus, bovine respiratory syncytial virus, parainfluenza 3 viruses, bovine virus diarrhea virus, certain rhino viruses, and a host of uncommon viruses that can affect cattle. Often the virus infection will cause damage to the lung tissue and then bacteria will invade the compromised tissues. The bacteria most often involved include Mannheimiahemolytica (formerly Pasteurellahemolyticum), Pasteurellamultocida, and Histophilussomni (formerly Hemophilussomnus). These bacteria are never far from cattle and are particularly adept at invading lung tissue damaged by viruses. Other bacteria commonly involved in pneumonia include Mycoplasma bovis and Arcanobacteriumpyogenes (formerly Actinomycespyogenes). These are more Latin names than anyone really wants to consider; however, the principal organism involved can influence (Maas, 2008).

Pneumonias in domestic animals can be classified based on texture, distribution, appearance and exudation into four morphologically distinct types: bronchopneumonia, interstitial pneumonia, embolic pneumonia, and granulomatous pneumonia. By using this classification, it is likely to predict with some degree of certainty the likely causes (virus, bacteria, fungi, parasites) and routes of entry (aerogenous versus hematogenous). On the other hand, overlapping of these four types of pneumonias is possible, and sometimes two morphologic types may be present in the same lung (McGavin and Zachary, 2010).

Pneumoconiosis was a common environmental health hazard for camels. This condition was probably associated with the increasingly dusty environment where camels are reared. It can also predispose camels to secondary infections, as there had been associated bronchopneumonia, by interfering with the defense mechanisms of the lungs (El-mahdy *et al.*, 2013).

Aspiration pneumonia

Pulmonary aspiration in bovines is the inhalation of secretions, forestomach contents or foreign material into the larynx and the lower respiratory tract. The presence of abnormal substances in the airways and alveoli as a result of inhalation is usually referred to as aspiration pneumonia. Injury to the lung will depend, however, on the amount and nature of the aspirate, the frequency of aspiration, the distribution within the respiratory tract and the host's response to the aspirated material (Marik, 2001).

In mature bovines, because of the size of the rumen and the magnitude of contents that can be regurgitated, overwhelming aspiration of regurgitated contents will cause instant death as a result of mechanical asphyxiation. The pH of these contents, even with a severe ruminal acidosis, is more alkaline than that seen in monogastrics, and the chemical burn as seen in the latter is unlikely. However, contamination of the pulmonary tree with pathogenic bacteria is a distinct possibility and even small amounts aspirated can cause aspiration pneumonia after a day or two (Shakespeare, 2012).

Microscopic finding during aspiration pneumonia could be acute inflammatory cells &eosinophilic amorphous exudate in the bronchioles &alveolar space, foamy cells (lipid-laden macrophages) or foreign body giant cell and thickening alveolar septum due to edema and inflammation (Robbins and Cotran, 2005).

Interstitial pneumonias

Interstitial pneumonias are inflammatory conditions in which the predominant exudative and proliferative responses involve alveolar walls. Grossly, the lesions are distributed widely throughout the lungs. Interstitial pneumonias comprise a significant proportion of cattle respiratory diseases known by different names, such as acute bovine pulmonary emphysema and

edema, fog fever, atypical interstitial pneumonia and cow asthma, the condition seems to occur predominantly in late summer or fall (Kerr, 1969).

It is often caused by a blood-borne insult, but can also be aerogenous. Instead, the whole lung seems just bigger and firmer than normal, sometimes even rubbery. The lesions are really easy to see histologically though. The focus of damage is on and within the alveolar walls. There is a wide variety of causes of interstitial pneumonias. Inhalation of high concentrations of toxic gases or fumes will cause interstitial pneumonia. Many of the viruses that arrive at the lung, either from the blood stream, or from the air, settle at the broncho-alveolar junction, and from there quickly move to the alveoli, creating an interstitial pattern (Sorden*et al.*, 2000).

Granulomatous pneumonia

This is a particular type of pneumonia where a pathogen, either inhaled or arriving at the lung via the bloodstream, settles out in the parenchyma to incite a typical chronic granulomatous inflammation. Fungal diseases are most commonly the reason, but also some of the higher bacteria, such as mycobacteria or Rhodococcusequi, will do this as well. Distribution is multifocal, without regard for cranioventral or caudodorsal they can settle out anywhere (Elsiddig and Elsiddig, 2003).

Gross lesions: Granulomas in the lung and sometimes in other organs too. Be aware that granulomatous pneumonia can resemble lung cancer and may require histopathological confirmation. Histopathology: Variable size nodules with a necrotic center infiltrated by macrophages and giant cells and surrounded by connective tissue mixed with lymphocytes and plasma cells (López, 2012).

Embolic pneumonia

This term can be used to include pneumonias caused by any circulating particulates. Lungs are a biologic filter for circulating particulate matter. Causes of embolic pneumonia include those bacteria that tend to travel as septic aggregates Histophilussomni and Actinobacillusequuli. Right-sided vegetative valvular endocarditis often causes an embolic pneumonia, as septic thrombi travel to and lodges in the lung. The granulomas feel fairly firm and you can palpate them in lots of locations. The offending agent could have been inhaled or could have arrived at the lung from the circulation (Millar *et al.*, 2017).

Embolic pneumonia is the result of a showering of septic thrombi into the pulmonary arterial system from major veins such as the caudal vena cava, mammary, uterine and jugular veins. Septic thrombi result from bacterial infections in tissues, resulting in erosion into veins and release of thrombi into the circulatory system. Animals surviving the thromboembolic showering event may later develop pulmonary arterial aneurysms, which may rupture into a bronchus causing rapid blood loss and death. At necropsy, there is severe widespread discoloration, abscessation, and haemorrhage affecting the lungs. Further dissection may find an abscess at the hilus of the liver that has eroded into the vena cava, or an infection of other organs and tissues that has entered other large veins (Jubb*et al.*, 1980).

Bronchopneumonia

The hallmark feature of bronchopneumonia is that the inflammation originates in the bronchial tree. As would be expected, the origin of bronchopneumonia is aerogenous something nasty comes down the conducting tree. Bronchopneumonia frequently comes from something being brought down the trachea into the lungs. In this type of pneumonia injury and the inflammatory process take place mainly in the bronchial, bronchiolar, and alveolar lumens (Fulton, 2009).

Within 2-3 days of bacteria becoming established at the bronchioloalveolar junction, there is red consolidation evident. Leukocytes migrating in in large numbers will change the color of the exudates to more of a gray appearance in 5-7 days. It may begin to resolve by 7-10 days, with slow turnover of type II alveolar cells back to the

more efficient type I variety. The lung can return to normal by 3-4 weeks. Or, on the other hand, the whole lung can go to heck in a hand basket and the end result will be available for full viewing in the necropsy room within days, with all shades of red and gray, consolidation, and fibrin exudation (Britton and Zabek, 2012).

Bronchopneumonia grossly characterized by is of irregular consolidation in cranioventral regions. The cranial and middle lobes are most often affected in those species having well defined lobation (Ertan, 2006). Consolidated lungs vary from dark red, through gray pink, to more gray, depending on the age and nature of the process. Consolidation of the tissue is the single most important gross criterion of pneumonia (Goodwin, 2005). The cut surface of infected lungs shows the variability of involvement seen on the pleural surface. In catarrhal or suppurative bronchopneumonia, the section of consolidated lobules is moist and mucopurulent or purulent material can be expressed from small airways. The cut surface of fibrinous inflammation has a dull and dry appearance (Sorden*et al.,* 2000; Ertan, 2006).

Situation in Ethiopia

Camels are important animals for pastoralists in the northeastern, eastern, southeastern and southern parts of Ethiopia. According to the report by Samuel (2008) in abattoir study of respiratory lesions in 104 adult camels at the Dire Dawa abattoir (88 male and 16 female), 98% of the examined lungs had one or more lesions. The most common lesions were pulmonary fibrosis (50.00%), pneumoconiosis (34.62%), hydatid cyst (30.80%), pulmonary abscess (3.85%) and parasitic bronchopneumonia (0.96%). The distribution of pneumoconiosis and hydatid cyst varied significantly ($p<0.05$) among different lobes, the highest being seen in the caudal lobe. For the different lesions there was no significant ($p>0.05$) difference in distribution among male and female camels. Possible explanations for the occurrence of the lesions are discussed. And recommendations forecasted are made. Southern Ethiopia (Bekele 1999). Many authors also emphasized that camel respiratory problem has received little consideration, even though it is an emerging disease in Ethiopia causing considerable loss of production and deaths (Rufael 1996; Bekele 1999).

In conclusion, camels are important animals for pastoralists in in many Asian, Latin American and African countries. However, there are a few investigations about camel lung pathology worldwide. Camel respiratory problem has received little consideration, even though it is an emerging and re-emerging disease in different part of the globe causing considerable loss of production and deaths.

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