German Journal of Veterinary Research

eISSN:2703-1322

Review article

Contagious caprine pleuropneumonia: A review of the global situation with a special reference to Oman

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Article History:
Received: 30-Aug-2023
Accepted: 05-Oct-2023
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Abstract

Contagious caprine pleuropneumonia (CCPP) is a severe infectious disease caused by *Mycoplasma capricolum* subsp. *capripneumoniae* (Mccp) and infects goats, sheep, and wild ruminants. CCPP is characterized by high morbidity and mortality rates reaching up to (100% and 80%), respectively. The disease affects goat farming around the globe in more than 30 countries, particularly in Asia, the Middle East, and Africa. CCPP is manifested in peracute, acute, or chronic forms. The general characteristic clinical signs of the disease are rapid, painful, and labored respiration, dyspnea, nasal discharge, coughing, hyperthermia (41°C), anorexia, emaciation, and abnormal posture. Lesions induced by CCPP are restricted to the pleural cavity in the form of unilateral serofibrinous, pleuropneumonia, accumulation of fluid in the chest cavity, lung congestion, hepatization, formation of adhesion to the pleural coastal, and swollen mediastinal and bronchial lymph nodes. Disease diagnosis encompasses a range of methods, including bacterial culture, isolation, and identification, pathological, serological, and molecular tests. The present review provides an overview of the historical perspective, epidemiological factors, and recommended diagnostic and control strategies for CCPP in Oman.

Keywords: CCPP, Pleuropneumonia, *Mycoplasma*, Seroprevalence, Oman

Citation: Ali, H., Altubi, A., El-Neweshy, M. and Elshafie, E. I. 2023. Contagious caprine pleuropneumonia: A review of the global situation with a special reference to Oman. Ger. J. Vet. Res. 3 (3): 19-26. https://doi.org/10.51585/gjvr.2023.3.0060

Introduction

The goat population in Oman represents 64% of the 3.2 million heads of livestock and is present in four breeds: Jabal Akhdar, Batinah, Dhofar, and Ash Sharqiyah (Al-Araimi et al., 2017). Contagious caprine pleuropneumonia (CCPP) is a devastating infectious disease of goats listed by the World Organization of Animal Health (WOAH) as one of the notifiable diseases. The disease is caused by *Mycoplasma capricolum* subsp. *capripneumoniae* (Mccp), a species of the genus *Mycoplasma* that is among the most important pathogenic bacteria of livestock worldwide (Fischer et al., 2012).

CCPP was detected in Algeria for the first time in 1873. The highly contagious nature of the disease was confirmed following an epidemic in South Africa in 1881, which resulted from the importation of goats infected from Turkey (Fischer et al., 2012; Nicholas and Churchward, 2012). However, the isolation and characterization of the causative agent were performed in 1976, and it was identified as *Mycoplasma* spp. type F38 (MccF38) (Thiaucourt et al., 1996). This study delves into the epidemiological aspects of CCPP, examining seroprevalence, economic impacts, geographical distribution, clinical manifestations, and pathophysiological factors. It places particular emphasis on diagnostic methods, treatment options, and preventive measures within the context of Oman.

Economic losses

CCPP is one of the most prevalent contagious diseases affecting goats and can result in massive economic losses in countries where goats play a significant role in the economy. CCPP has caused huge livestock losses, especially in Asia and Africa, and it is a threat to disease-free nations (Fischer et al., 2012). Nicholas (2002) mentioned that CCPP causes serious losses in goat herds in at least 30 countries in Africa and Asia, including Turkey. Goats play an important role in feeding families, thus contributing to maintaining them in rural areas and reducing migration to ur-

Table 1: Reported seroprevalence of contagious caprine pleuropneumonia (CCPP) based on country, host, and detection method.

Country	Seroprevalence %	Host	Detection method*	Reference
Kenya				
Turkana West	63.9	Goats	cELISA	Kipronoh et al. (2016)
Kajiado Central	48.6			
Pokot East	29.2			
Pakistan	32.5	Goats	CIE	Hussain et al. (2012)
Northern Ethiopia	32.6	Goats	CFT	Hadush et al. (2009)
	18.25	Sheep		
Northwest Ethiopia	8.5	Goats	cELISA	Abrhaley et al. (2019)
Jammu and Kashmir, India	9.93	Goats	cELISA	Parray et al. (2019)
Oman	53.8	Goats	cELISA	Hussain (2021)
	10	Sheep		

*Abbreviations: cELISA; competitive enzyme-linked immunosorbent assay, CIE; counter immunoelectrophoresis technique, CFT; complement fixation test.

ban places (Thiaucourt and Bölske, 1996). In Turkana County, Kenya, goats form a significant source of income for rural households (Kipronoh et al., 2016).

Goats are used for meat and milk production in many countries globally. For example, Samiullah (2013) reported that in Pakistan, goat farming is crucial to society because it provides meat, milk, mohair, skin, and manure on a small scale. Due to the devastation and high mortality rate that CCPP causes, goat rearing was abandoned in some countries in Asia and Africa (Bölske et al., 1996). Economic losses further extended to reduce the number of newborns due to induced fatalities in infected does (Renault et al., 2019). In addition, CCPP causes high mortality rates in wild animal sanctuaries (Arif et al., 2007). Renault et al. (2019) reported that the absence of CCPP vaccination could lead to an average of 1,712.66 € yearly financial losses for a standard flock of 100 heads in Kenya. The estimated CCPP vaccine costs are 309 €/4,000 animals/day (Renault et al., 2019).

Host susceptibility

Goats are the main susceptible animals to Mccp infections, while sheep exhibit a lower susceptibility to pulmonary mycoplasmosis (Bölske et al., 1996). However, limited reports documenting the isolation of Mccp from both healthy and diseased sentinel sheep within a herd of CCPP-infected goats in Kenya (Thiaucourt and Bölske, 1996; Bölske et al., 1996; Arif et al., 2007). The exposure of African buffalo and camels to Mccp was confirmed by the Complement Fixation Test (CFT) (Arif et al., 2007). Arif et al. (2007) reported that an outbreak of CCPP occurred for the first time in Qatar among wild species populations at Al Wabra Wildlife Preservation.

The species found to be affected were wild goat (*Capra aegagrus*), Nubian ibex (*Capra ibex nubiana*), gerenuk (*Litocranius walleri*), and wild sheep; Laristan mouflon (*Ovis orientalis laristanica*). Another outbreak was reported among captive gazelles and other deer populations in the United Arab Emirates, resulting in a 10% mortality rate (Nicholas and Churchward, 2012). In the study by Paling et al. (1988), antibodies against *Mycoplasma* sp. (strain F38) were elucidated in four buffalos and 64 camels on a farm housing mixed domesticated wild herbivores and livestock in Kenya. Furthermore, Chaber et al. (2014) reported the initial occurrence of CCPP in Arabian Oryx, with the isolated Mccp strain closely resembling the one previously identified in a sand gazelle.

Serodiagnosis and seroprevalence

The seroprevalence of CCPP was recorded in various countries in goats and sheep using different detection methods (Table 1). Various techniques have been employed to detect the antibodies against Mccp. Kipronoh et al. (2016) used a monoclonal antibody-based competitive enzyme-linked immunosorbent assay (cELISA) to investigate the seroprevalence of CCPP in goats in Kenya. The study revealed approximately 63.9%, 48.6%, and 29.2% seroprevalence rates in Turkana West, Kajiado Central, and Pokot East. Similarly, Hussain et al. (2012) confirmed a seroprevalence of 32.5% in Pakistani Beetal goats, confirmed by the counter immunoelectrophoresis technique. The CFT was conducted to estimate the seroprevalence of CCPP in Northern Ethiopia. The results showed a seroprevalence of 32.68% in goats and 18.25% in sheep (Hadush et al., 2009). Additionally, in Western Amhara, Northwest Ethiopia, the positive specific antibodies against CCPP in goats were determined to be 8.5% using cELISA (Abrhaley et al., 2019). In the case of Himalayan Pashmina goats, a 9.93% seroprevalence of CCPP was reported (Parray et al., 2019). In Oman, Hussain et al., 2021 utilized cELISA to investigate the seroprevalence of CCPP nationwide. The study revealed that the overall seroprevalence of CCPP in Oman ranged from 10.0% to 53.8% in 147 sheep and goat flocks across various governorates.

Geographic distribution

CCPP is a predominantly fatal goat disease in the Middle East, Western Asia, and Africa (Nicholas, 2002). Mortality rates associated with the CCPP can reach up to 80% (Fischer et al., 2012). The disease was reported in many African countries, including Algeria, Burkina

Faso, Benin, Cameroon, Central African Republic, Djibouti, Egypt, Libya, Mali, Nigeria, Somalia, and Zaire, as highlighted by Nicholas (2002). *Mycoplasma*, the causative agent, has been isolated in Eritrea, Niger (Nicholas, 2002), Sudan, Kenya, Ethiopia, Tunisia, Chad, and Uganda (Bölske et al., 1996; Nicholas, 2002).

In Asian regions, CCPP has been reported in Afghanistan, Bangladesh, India, Iran, Iraq, Jordan, Kuwait, Lebanon, Pakistan, Saudi Arabia, and Syria (Nicholas, 2002). Mccp isolation confirms the disease's presence in Oman, Turkey (Bölske et al., 1996; Nicholas, 2002), the United Arab Emirates, Nepal, and Yemen (Nicholas, 2002). In 2009, Mauritius experienced its first CCPP outbreak, resulting from the introduction of infected goats from the African mainland. This outbreak led to the death of more than 300 goats within a month, as reported by Nicholas and Churchward (2012). Between 2008 and 2009, nearly 600 CCPP outbreaks were reported in Oman, resulting in the deaths of almost 30,000 animals. Similarly, in Iran, between 2006 and 2007, there were 478 reported outbreaks affecting over 16,000 goats. Yemen also recorded several outbreaks during this period, affecting over 800 goats and approximately 200 fatalities (Bölske et al., 1996; Nicholas and Churchward, 2012).

Etiology of CCPP and mode of transmission

Mycoplasma capricolum subsp. *capripneumoniae* is the causative agent of CCPP. The initial organism isolated and characterized during the CCPP outbreak in 1976 was identified as *Mycoplasma* spp. type F38 (MccF38) (Bölske et al., 1996; Thiaucourt et al., 1996). Mccp belongs to the *mycoides* cluster, consisting of subspecies of *Mycoplasma* that share several phenotypic or genomic characteristics. These include *M. mycoides* subsp. *mycoides* biotype SC, *M. mycoides* subsp. *mycoides* biotype LC, *M. mycoides* subsp. *capri*, *M. capricolum* subsp. *capricolum*, and *Mycoplasma* spp. group 7 of Leach (Bölske et al., 1996; Thiaucourt and Bölske, 1996).

Mycoplasma is the smallest detected prokaryotic cell. It is a pleomorphic organism with shapes ranging from spherical to filamentous (Quinn et al., 2011). *Mycoplasma* spp. are characterized by flexible triple-layered membranes that notably lack a cell wall (Quinn et al., 2011; Samiullah, 2013). Phylogenetically, mycoplasmas are related to Gram-positive bacteria with low G and C content (Samiullah, 2013). However, it is important to note that mycoplasmas are not stained by Gram stain (Quinn et al., 2011). Desiccation, heat, disinfectants, and detergents destroy *Mycoplasma* species. They typically grow on the agar, forming micro-colonies with a distinctive appearance resembling fried eggs, characterized by a dense central region. These micro-colonies can be observed using a stereo microscope. Mycoplasmas are known to be host-specific, and the majority of these organisms are facultative anaerobes, growing in 5-10% CO_2 conditions (Quinn et al., 2011).

CCPP is typically attributed to introducing an infectious animal into a vulnerable herd. Direct transmission occurs through the aerogenic route, where the disease spreads via droplets emitted during coughing (Thiaucourt and Bölske, 1996). Notably, a relatively brief period of contact is sufficient for transmitting this disease, although proximity is often required. Only direct transmission is recorded since mycoplasmas are very sensitive and highly fragile in the environment, making them easily inactivated (Thiaucourt and Bölske, 1996; Nicholas and Churchward, 2012). Additionally, it is important to note that the disease can be transmitted through latent chronic carriers, which harbor the infection without displaying any overt symptoms.

Seasonal occurrence

Climate change has been linked to the onset of CCPP outbreaks. In Oman, these outbreaks tend to be more prevalent in January, coinciding with the lowest temperatures and the highest pluviometry, and in July during the peak of high temperatures. The disease was more common in North Africa during the winter season. In addition, recurrent CCPP outbreaks in Pashmina goats have been observed during the winter season in Changthang, Ladakh, India, particularly from November–December to March–April, during a prolonged winter period (Iqbal Yatoo et al., 2019). Disease outbreaks can be triggered by adverse weather conditions (Samiullah, 2013). Despite cold weather being the most common causative factor, any type of stress, such as transportation, dietary, or climatic stress, will expose animals to CCPP.

Factors exacerbate CCPP

In areas where the disease is already present, the severity of the disease can be determined by various factors (Thiaucourt and Bölske, 1996):

- Viral infections: Pre-existing viral infections such as orf and Peste des Petits Ruminants (PPR), as well as *Mycoplasma* infections like M. ovipneumoniae, can encourage and accelerate the development of CCPP in animals that have previously survived a CCPP infection (Thiaucourt and Bölske, 1996; Nicholas and Churchward, 2012).
- Environmental conditions: Poor environmental conditions, such as significant temperature fluctuations between day and night or a sudden climate change, particularly during the time between the dry and rainy seasons, can exacerbate the severity of CCPP.
- Stress: Stress induced by long-distance movement can increase the severity of CCPP. The duration of the disease varies according to the environmental conditions. Animals kept in good condition can survive for an extended period, exceeding one month or even recovering from CCPP. However, animals in harsh conditions, such as those experiencing underfeeding, parasitism, walking long distances for watering, and adverse climatic conditions, may only survive for a few days (Thiaucourt and Bölske, 1996).

Morbidity and mortality rates

CCPP is undeniably highly contagious, capable of swiftly spreading throughout a herd and causing significant morbidity. In the absence of medication, the mortality rate associated with CCPP is very high, reaching up to 80% (Thiaucourt et al., 1996; Thiaucourt and Bölske, 1996). Nicholas (2002), Arif et al. (2007), and Nicholas and Churchward (2012) mentioned that naïve herds that are exposed to the disease for the first time can suffer losses that reach up to 80% mortality and 100% morbidity.

Clinical signs

CCPP can infect all goats regardless of their age or sex. This disease is recognized by its specific respiratory-related clinical signs aggravated by *Mycoplasma* that affects the lungs, pleural fluid, pleural cavity, heart, and sometimes the upper respiratory tract (Iqbal Yatoo et al., 2019). Generally, CCPP has an incubation period of 10 days, although this duration can vary between 2 and 28 days (Thiaucourt and Bölske, 1996), however, the disease may manifest itself in different forms:

- **Peracute form**: The livestock affected by CCPP in the peracute form are unable to feed or drink, showing rapid shallow respiration and stiff gate followed by sudden death (Arif et al., 2007).

- **Acute form**: The animals become lethargic and anorexic (Arif et al., 2007), exhibit an abnormal posture, and extend their head and neck with foam from the mouth and nostrils. Respiration is painful and labored (Arif et al., 2007), and the affected animals have dyspnea, sometimes with grunting and snoring (Samiullah, 2013). In the Al Wabra Wildlife Preservation outbreak, some animals had a percussive dullness over the affected lung areas and the ventral regions of the lungs; moist rales and harsh friction sounds were heard (Arif et al., 2007).

- **Chronic form**: Animals may exhibit nasal discharge, salivation, and coughing following exercise. Affected animals are hyperthermic, and rectal temperatures range from 40 to 41.2°C. High mortality in kids is because of septicemia (Nicholas and Churchward, 2012). The clinical signs recorded in a CCPP experimental outbreak (MacOwan and Minette, 1976) showed fever up to 41°C, anorexia, coughing, depression, and emaciation. Sometimes, the disease is associated with frequent abortions in pregnant goats (Thiaucourt et al., 1996).

Pathogenesis

In CCPP-affected Pashmina goats, Iqbal Yatoo et al. (2019) observed an increase in proinflammatory cytokine TNF-α and total oxidant status, as well as a related depletion in total antioxidant status that is resulting in the principal pathological lesions of the disease like fibrin deposition in the pleural cavity, fluid exudation, and hydrothorax. Iqbal Yatoo et al. (2019) studied the pathogenesis of Mccp, where it begins with pathogen inhalation via aerosols from infected goats, which bind to layers of superficial cells via different membrane structures and is followed by colonization. This resulted in pathological inflammation characterized by epithelial ciliostasis, sero-fibrinous pleuropneumonia, vasculitis, and fibrinocellular exudation. Mycoplasmal antigens such as polysaccharides, lipoprotein, and galactan stimulate the immune system. The inflammatory and oxidative cascades are also activated. Mccp may affect other organs such as joints, the udder, and the skin.

Diagnosis

Proper sampling

Proper sampling is essential for accurate disease diagnosis. Samples should be obtained in an aseptic condition following standard protocols. Different types of samples collected from goats exhibited clear clinical signs of CCPP, such as nasal swabs, blood samples, pleural fluid, and lung tissues from necropsied animals. Heamato-biochemical and serological tests require collecting blood or serum samples, secretions, exudates, and tissues for isolation and molecular studies (Iqbal Yatoo et al., 2019). After cleaning the external nares, nasal swabs must be collected and inserted into transport media (El-Deeb et al., 2017; Iqbal Yatoo et al., 2019). For pleural fluid collection, at least 10 mL should be obtained aseptically by sterile syringes from necropsied animals. Thoracentesis is an alternative method to collect pleural fluid from live animals that show signs of CCPP, and it is preferred to minimize contamination (Thiaucourt et al., 1996; Iqbal Yatoo et al., 2019). At necropsy, lung samples are obtained aseptically from the hepatized area, ensuring that the borders of normal tissue are included. These samples can be stored in sterile plastic bags.

Furthermore, a hot spatula is used to sterilize the surface of lung tissue with lesions, and sterilized scissors are used to mince the deep tissue. 9 mL of modified pleuro-pneumonia-like organisms (PPLO) medium is combined with one gram of minced tissue and stored for isolation (El-Deeb et al., 2017). Collected samples can be stored at +4°C for one to two days or at -20°C for a few months. For the longer time of storage exceeding ten months without compromising *Mycoplasma* viability, samples can be preserved at -70°C. It is recommended to add penicillin or ampicillin to the samples to prevent the growth of contaminants (Thiaucourt et al., 1996). Blood or serum samples are particularly important for serological examinations, while discharges, exudates, blood, and tissues are crucial for culture, isolation, and gene/DNA-based studies related to CCPP diagnosis research.

Isolation and identification

Mccp organism is quite difficult to isolate and identify correctly despite the highly improved media formulations due to its fastidious nature (Bölske et al., 1996; Nicholas and Churchward, 2012). Mccp is characterized by slow growth in broth and solid media, forming pinpoint micro-colonies ranging from 0.1-0.6 mm in diameter (Bölske et al., 1996). *Mycoplasma* species unstained microcolonies have a characteristic appearance resembling fried eggs when examined under a microscope at low magnification. The Dienes stain can be useful in identifying these micro-colonies, as it stains the central zone dark blue and the peripheral zone brighter blue (Quinn et al., 2011). Several specialized media are used for Mccp growth. Mccp growth is achieved on a medium enriched with sodium pyruvate (Arif et al., 2007). In addition, enriched media that contain animal protein, a source of DNA or adenine dinucleotide, and sterol components are required (Quinn et al., 2011)).

Mycoplasma requires sterols to develop and grow, expressed in their sensitivity to digitonin inhibition. Generally, mycoplasmas are cultured in agar or broth media (often heart-infusion) supplemented with (20%) horse serum and extract of yeast to provide vitamins and amino acids. Penicillin is added to suppress Gram-positive bacteria, and thallous acetate is used to suppress Gram-negative bacteria and fungi. Samples are incubated in 10% CO2 in a humid atmosphere for 14 days (Quinn et al., 2011). Mccp could be grown successfully and isolated from the clinical materials through culturing on Hayflick medium broth (H25P) (Samiullah, 2013), a modification of the pyruvate-enriched medium.

According to Wesonga et al. (2004), Mccp was isolated from the lungs and tracheal lavage by culturing it in modified Newing's broth with 0.2% sodium and on Medium F with 0.2% sodium pyruvate. El-Deeb et al. (2017) illustrated that the growth in liquid media should be observed daily. The growth is obvious as turbidity, a pH change indicated by color change, and floccular material appearance. The growth is subcultured on PPLO agar. After 10 days of incubation, tubes that displayed no evidence of growth were discarded. Staining with Giemsa stain could help in the identification.

In addition, Mccp molecular typing is the most precise and reliable diagnosis technique. Polymerase chain reaction (PCR) was developed to facilitate the recognition and characterization of Mccp due to its difficulties in isolation because of its fastidious nature and confusion of other *M. mycoides* cluster members, especially *M. mycoides* subsp. *mycoides* LC and *M. mycoides* subsp. capri that causes pleuropneumonia in small ruminants that resemble CCPP (Nicholas, 2002). PCR enables the detection of all members of the *M. mycoides* cluster based on the 16S RNA genes, followed by restriction enzyme digestion for specific identification of *M. capricolum* subsp. *capripneumoniae* (Bascuñana et al., 1994; Nicholas, 2002; Arif et al., 2007; Nicholas and Churchward, 2012). PCR/denaturing gradient gel electrophoresis (DGGE) is sensitive, fast, and precise and offers significant advantages in detection. A single pair of mollicute-specific primers will amplify DNA from all animals, which can be classified using DGGE migration patterns. The test also identifies several *Mycoplasma* organisms in a single sample (Nicholas and Churchward, 2012).

Serological tests

Various serological techniques have been used for the diagnosis of CCPP, such as the indirect hemagglutination test (Samiullah, 2013), CFT, cELISA, and the latex agglutination test (LAT) (Nicholas and Churchward, 2012). The LAT is inexpensive, sensitive, easier to perform, and can be used in situ. The principle of LAT work is that LAT utilizes a carbohydrate (capsular polysaccharide [CPS]) harvested from Mccp connected to latex particles (beads) that agglutinate in the presence of particular antibodies in infected goat blood. The LAT for circulating antigen has also been identified, which may allow for earlier identification in infected animals before antibodies appear (March et al., 2000; Nicholas and Churchward, 2012). LAT was used by Nicholas and Churchward (2012) to monitor *Mycoplasma* development *in-vitro*, which can be difficult to detect due to the slight pH changes created by certain strains; the test only needs a few drops of culture fluid. The LAT detects antibodies in the serum samples of goats 22 days after exposure to CCPP, whereas the CFT detects antibodies 24 after exposure (Rurangirwa et al., 1987). The LAT has a higher sensitivity than the CFT. In illustration, both tests were conducted on 763 sera from farms with outbreaks of contagious caprine pleuropneumonia. The LAT detected 63% of positive cases, and the CFT detected only 23%. The LAT was performed in the field using whole blood or undiluted serum, and a result was obtained in less than two minutes (Rurangirwa et al., 1987). The Growth inhibition test (GIT) has low sensitivity and is simple compared to other CCPP diagnostic tests. It relies on the direct inhibition of the growth of *Mycoplasma* on solid media by complex hyperimmune serum and identifies primary surface antigens (Samiullah, 2013).

The Growth inhibition test employed polyclonal rabbit antisera to *M. capricolum*. Wesonga et al. (2004) and Quinn et al. (2011) described growth inhibition tests as filter paper discs containing particular antisera placed on an agar surface cultivated with *Mycoplasma*. Around the disc containing homologous antiserum, a growth inhibition zone extending up to 8 mm wide emerges. Another useful diagnostic method is the indirect enzyme-linked immunosorbent assay (iELISA), which screens goat serum for antibodies to Mccp at a single dilution. The iELISA offers accuracy and specificity and is suitable for large-scale analyses, making it an effective tool for conducting epidemiological analysis related to CCPP (Samiullah, 2013).

Pathological examination

Commonly, Mccp induces severe pathologic lesions in young and immunocompromised animals while caus-

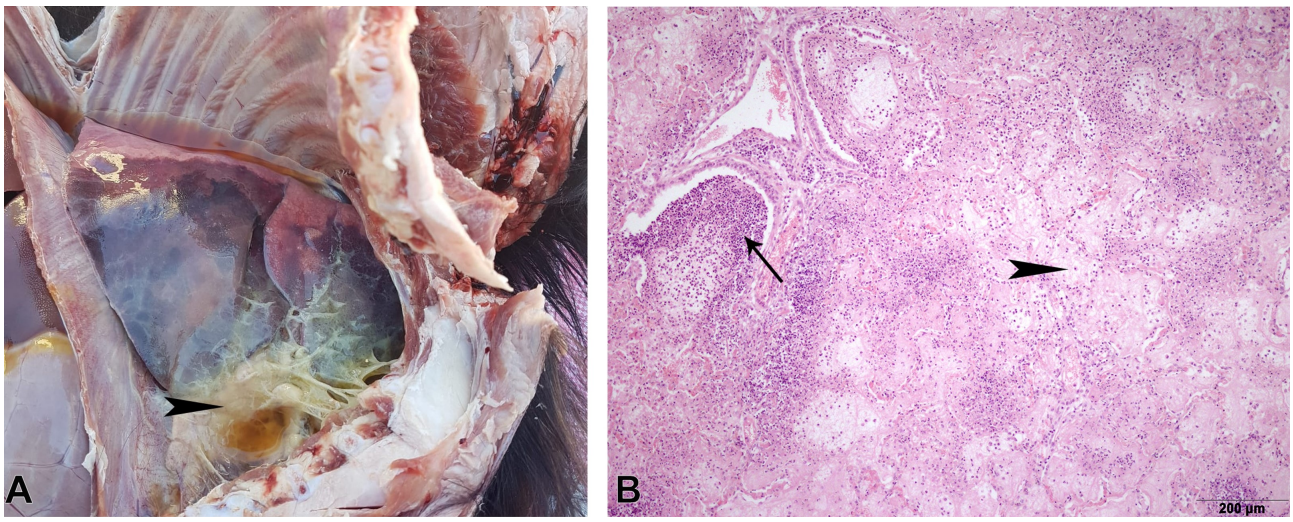

Figure 1: Necropsied CCPP naturally infected goat showing lung hepatization, marbling, hydrothorax, and serofibrinous pleuropneumonia (arrowhead) (A); micrograph of lung tissue showing fibrin threads deposition in alveoli (arrowhead) and obliteration of bronchiole with inflammatory cells (arrow) (B). (Hematoxylin and Eosin, Bar=200 μm).

ing chronic pathological changes in normal resistant animals. Thiaucourt et al. (1996) described the pathological alterations induced by CCPP, restricted in the chest cavity. One of the primary pathological manifestations is the presence of unilateral serofibrinous pleuropneumonia, straw-colored pleural fluid accumulation with fibrin flakes, partial or total lung hepatization with thickening of the interlobular septa, and pleurisy (Figure 1A). Some animals showed yellow fibrin form gelatinous membranes, cover lung lesions, and adhere to the coastal pleura (MacOwan and Minette, 1976; Arif et al., 2007). The influenced lung becomes swollen, hard, oedematous, and varies in color from gray to red. The hepatized lung was characterized by a gray pinpoint necrotization center and hyperemic margins (Thiaucourt et al., 1996). Also, mediastinal and bronchial lymph nodes are swollen (MacOwan and Minette, 1976).

The pathological appearances in gazelles due to CCPP outbreaks in the Middle East were somewhat close to those seen in goats, although the onset of the death was sudden (Nicholas and Churchward, 2012). In an outbreak of Beetal goats, Hussain et al. (2012) stated that 100% of dead animals exhibited lung consolidation, followed by alveolar exudation in 90.9% and pleural adhesion in 72.72% of the cases.

Histological findings in the lung tissues of CCPP-diseased animals revealed the presence of serofibrinous fluid and infiltrates of inflammatory cells, especially neutrophils, in the alveoli, interstitial septa, bronchioles, and subpleural connective tissue (Nicholas and Churchward, 2012) (Figure 1B). Lymphoid hyperplasia of peribronchial and peribronchiolar was notable with mononuclear cell infiltration (Nicholas and Churchward, 2012). The lumen of alveoli is filled out with serous fluid containing neutrophils and lymphocytes, as well as extensive hyperplasia of type II pneumocytes that have destroyed much of their distinctive lamellar ultrastructure. Reducing the production of pulmonary surfactant by Type II pneumocytes and enzyme synthesis, increasing alveolar surface tension and causing collapse, is often found during post-mortem examination (Johnson et al., 2002; Nicholas and Churchward, 2012).

Johnson et al. (2002) reported that type II pneumocytes are responsible for producing surfactants, synthesizing and secreting basement membrane connective tissue components, for instance, fibronectin. Srivastava et al. (2010) found that lung tissue from diseased goats consisted of extensive fibrinopurulent pneumonia, pleurisy, and alveolar and interseptal edema. In the microscopic lesions of dead Beetal goats, (Hussain et al., 2012) found septal peribronchiolar fibrosis in 81.81% of samples, followed by fibrinous pleuritis (63.63%) and peribronchiolar cuffing (54.54%) of mononuclear cells in the lungs.

Furthermore, direct and indirect fluorescent antibody tests are simple and accurate serological methods to identify most mycoplasmas in clinical samples (Samiullah, 2013). Immunofluorescence using polyclonal rabbit antisera (polyclonal antibody) to *M. capricolum* subsp. *capripneumoniae* (Wesonga et al., 2004) and the peroxidase-anti peroxidase method on paraffine-embedded tissues were employed for diagnosis (Quinn et al., 2011). Lung tissues were embedded with the paraffin to investigate Mccp antigen by immunohistochemistry through employing polyclonal anti-*M. capripneumoniae* antibodies (Wesonga et al., 2004). Wesonga et al. (2004) and Arif et al. (2007) detected the Mccp antigen in alveolar macrophages, and positively stained cells looked multifocally and in neutrophils of the affected lung tissue. The existence of antigens in alveolar macrophages shows the importance of these cells in the goat lungs' defense against *M. capricolum* subsp. *capripneumoniae*.

Treatment and control strategies

Thiaucourt et al. (1996) and Nicholas (2002) mentioned that antibiotics belonging to a family of penicillin or aminoxide must be completely prohibited for two reasons. Firstly, these antibiotics are ineffective against *Mycoplasma*. Secondly, they support the

growth of resistant mycoplasmas strains that may take a turn for the worse conditions in herds. The most effective antibiotics against Mccp belong to the tetracycline group and macrolide family, like spiramycin, lincosamine, erythromycin (Thiaucourt et al., 1996; Nicholas, 2002; Nicholas and Churchward, 2012) and tylosin (Arif et al., 2007)), as well as fluoroquinolones, such as enrofloxacin (Thiaucourt et al., 1996; Nicholas, 2002; Nicholas and Churchward, 2012).

Anti-inflammatory drugs can also play a crucial role in improving the effectiveness of antibiotic treatments and protecting animals from succumbing to shocks (Arif et al., 2007). *In-vitro* antibiotic sensitivity test, according to Srivastava et al. (2010), revealed that the isolated Mccp were susceptible to the majority of antibiotics, with minimum inhibitory concentration (MICs) of 0.12 g/mL for oxytetracycline, tilmicosin, tylosin, marbofloxacin, danofloxacin and, and mild sensitivity (1 to 2 g/mL) for chloramphenicol and tulathromycin. (Nicholas and Churchward, 2012) recommended the movement restrictions and slaughtering of infected and contact animals for newly infected regions.

Generally, the inactivated vaccine has proved beneficial, but immunity is short-lived (Nicholas, 2002). In Kenya, an inactivated vaccine with saponin, acting as an adjuvant and inactivating *Mycoplasma*, was developed (Rurangirwa et al., 1987; Thiaucourt et al., 1996). This vaccine has been shown to protect goats for up to one year, as reported by (Thiaucourt et al., 1996). The Kenya Veterinary Vaccine Production Institute in Nairobi prepared an inactivated CCPP vaccine from a Mccp strain, Caprivax, which showed 100% protection. The study stated that there an available live vaccines like (Pulmovac and Capridoll) and killed vaccines like (CCPPV) commercially, and are manufactured in Turkey and Ethiopia, respectively (Samiullah, 2013).

Conclusion

Contagious caprine pleuropneumonia is a contagious and highly fatal caprine disease caused by *Mycoplasma capricolum* subsp. *capripneumoniae*. The disease poses a significant risk to goat farming in more than 30 countries, primarily in regions such as Africa, Asia, the Middle East, and some parts of Europe. Mccp has a fastidious nature and requires special media for culture and isolation. The rise in the number of countries infected by CCPP is primarily due to better diagnostic testing for fastidious *Mycoplasma* when the condition was frequently suspected based on clinical data.

Exporting goats from infected countries should be accompanied by a certificate that provides all details about a group of criteria, including no clinical signs of CCPP in animals, and it has to be tested negative for CCPP on two occasions in the previous six weeks. Antibiotic administration before exportation can mask the clinical signs, making accurate diagnosis challenging. Serological monitoring, such as CFT, is an essential tool for diagnosing CCPP, but not all strains are detectable. Alternative methods, like the LAT, have shown similarity to CFT with less strain-specific, can be more suitable for export testing, and are easier to implement than cELISA. For instance, tetracycline, macrolide, and fluoroquinolones still show effectiveness against Mccp. Annual vaccination is very important to minimize CCPP dissemination and animal loss.

Article Information

Funding. This work was funded by Sultan Qaboos University "grant number: RF/AGR/ANVS/20/01".

Conflict of Interest. The authors declare no conflict of interest.

Authors Contributions. Data collection; HA, AA, ME, and EIE, writing—original draft preparation; HA, ME, writing—review, editing, and approval of the final manuscript; all authors.

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