International Journal of Advanced Research in Biological Sciences
ISSN: 2348-8069
www.ijarbs.com
(A Peer Reviewed, Referred, Indexed and Open Access Journal)
DOI: 10.22192/ijarbs Coden: IJARQG (USA) Volume 9, Issue 12 -2022

Review Article

DOI: http://dx.doi.org/10.22192/ijarbs.2022.09.12.006

Review on Epidemiology and Diagnosis of Contagious Caprine Pleuropneumonia

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Abstract

Contagious Caprine Pleuropneumonia (CCPP) is notifiable, trans-boundarydisease characterized by high morbidity and mortality in goats. It is caused by *Mycoplasma capricolum* subspecies *capripneumoniae* (Mccp). The disease mainly affects goats though subclinical cases were reported some wild and domestic animals. It was first described in 1873 in Algeria and spread to the globe. CCPP in Ethiopia had been suspected since 1983 and confirmed in 1990 from an outbreak in Ogaden, Eastern Ethiopia. Occurrence of the disease is influenced environment, production system and host immune status. Livestock mobility and presence of naive populations are the major predisposing factors. CCPP is mainly transmitted by inhalation of infected aerosols. It is initially characterized by high fever after which accelerated dyspnea and productive cough are prominent. CCPP results in heavy economic losses causing estimated annual loss of 507 million US$ in endemic areas. Diagnosis might involve microbiological, biochemical, serological and molecular identification. The pleural fluid and sections of hepatized lung are samples of choice. Currently, molecular tests become novel diagnostic methods because of high specificity and sensitivity, but also because of difficulties in culturing of Mccp. Several serological tests are used on herd basis of which complement fixation (CFT) is sole test prescribed for international trade. Early antibiotic treatment is effectiveandrecovered animals may remain carrier. Prevention and control can be achieved through quarantines, vaccination, movement controls, slaughter policy, and cleaning and disinfection of the premises. The epidemiology of CCPP including the role of reservoirs is unclear and less attention is given to the disease. Therefore more detailed studies with appropriate should be undertaken reveal epidemiology of the disease that help to design and apply effective preventive and control measures. The role of wild and domestic reservoir host in epidemiology of the disease should have to be studied.

Keywords: CCPP, Diagnosis, Epidemiology, Goat, Trans-boundary

1. Introduction

Contagious Caprine Pleuropneumonia (CCPP) is a severe and devastating bacterial respiratory disease characterized with high morbidity and mortality in goats affecting all age and sex groups. The disease is caused by *Mycoplasma capricolum* subspecies *capripneumoniae* (Mccp) bacteria. Contagious caprine plueropneumonia occurs in many countries in Africa, Asia, and Middle East. It is trans-boundary disease included in the list of notifiable diseases by World Organization for Animal Health (Thiaucourt, 2008) as it threatens a significant number of goat populations throughout the world and has a considerable socioeconomic impact in infected territories (Atim et al., 2016). The disease is mainly found in goats. However, subclinical cases were reported in sheep and some wild ruminant species (Yousuf et al., 2012).

Typical cases of CCPP are clinically characterized by extreme fever (41-43 °C), high morbidity and high mortality in susceptible herds affecting all ages. Other associate common clinical signs that can be manifested by the disease include anorexia, weakness, emaciation, dullness, exercise intolerance, and respiratory signs such as dyspnea, polypnea, coughing, and nasal discharges (Soayfane et al., 2018). Abortion have been also reported (Wazir et al., 2016). Contagious caprine plueropneumonia is transmitted by direct contact by inhalation of infective aerosols.After approximately 2 to 3 days of high fever, respiratory signs become apparent. Sheep can be affected occasionally. For diagnosis of CCPP, serological tests such as indirect hemagglutination, complementfixation, competitive enzyme-linked immunoassay (cELISA) and latex agglutination (LAT), and PCR can be used(Teshome et al., 2019).

Treatment of CCPP by antibiotics such as tylosin and oxytetracycline creates carriers but single dose of Di-hydro streptomycin sulphategiven intramuscularly was able to cure goats without creating carriers. In order to prevent global spread of the disease as well as to overcome ineffectiveness of antibiotics for treating CCPP, vaccines are imperative(Yatoo et al., 2019). Prevention and control of the disease can be achieved by quarantines, vaccination, movement controls, and slaughter of infected/exposed animals, cleaning and disinfection of premises. Epidemiology of CCPP is unclear and less attention is given to the disease. Therefore objective of this paper is to review epidemiology and diagnosis of the disease which can help to design and apply effective preventive and control measures.

2. Etiology

Contagious caprine plueropneumonia is caused by bacteria called *Mycoplasma capricolum* subspecies *capripneumoniae(Mccp)* previously, known as *Mycoplasma* biotype F38. These pathogenic bacteria belong to the class *Mollicute*s, which lack cell wall but have galactan and small genomes. They have limited bio-synthetic capability and cause a number of infections in animals (Noah et al., 2011; Yatoo et al., 2019).Five distinct groups of *Mollicutes* have been identified including *Spiroplasma* group. *Spiroplasma* group comprises *Mycoplasma mycoides*, *Spiroplasmaapis*, *Spiroplasmacitri* and *Spiroplasmaixodetis* clusters (Hailu, 2017).

Mccp belongs to the *Mycoplasma mycoides* cluster. This cluster is further divided into *Mycoides* and *Capricolum* sub-groups each of them consisting three species, subspecies or strains causing disease in small and large ruminants. The *Mycoides* subgroup includes *Mycoplasma mycoides* subsp. *mycoides* small colony (MmmSC), *Mycoplasma mycoides* subsp. *mycoides* large colony (MmmLC) and *Mycoplasma mycoides* subsp. *capri* (Mmc) strains. The subspecies within the *Capricolum* subgroup are *Mycoplasma capricolu m* subsp. *capricolum* (Mcc), *Mycoplasma capric-olum* subsp. *capripneumoniae* (Mccp), and *Mycop lasma* subsp. *bovine* 7th group (BG7). Mccp is ver y fastidious slow growing *Mycoplas-ma* with an incubation time ranging from 7-10 days (Rehman et al., 2022). Of the six strains indicated above, some cause similar diseases in sheep and goats in

addition to extra pulmonary involvements they have (Noah et al., 2011).

3. Epidemiology

3.1. Host range

For a long time, CCPP has been reported to affect only the domestic goat. Now, it is a threat of wild ungulates exposed to infected goats (Arif et al., 2019). The primary host for CCPP is goat. However, various wild and domestic animals are also found to be susceptible reservoir hosts. Both the wild and domestic animals can get infected from affected goats (OIE, 2009; OIE, 2017; Yatoo et al., 2019; Teshome and Sori, 2021). Clinical disease and seropositivity have been reported in sheep in contact with affected goats, but the role of sheep as reservoirs of infection is unclear (Samiullah, 2015).

CCPP is a serious threat to wild goats (*Capra aegagrus*), Nubian Ibex (*Capra ibex nubiana*), Laristanmouflon (*Ovisorientalis elaristanica*) and Gerenuk (*Litocranius walleri*) causing significant morbidity and mortality. It is also reported to affect endangered Tibetan antelope (*Pantholopshod gsonii*), Arabian oryx (*Oryx leucoryx*), captive*Rhim*, *Dumani Gazelles* and other deer (Hailu, 2017; Teshome and Sori, 2021).

CCPP is isolated from both clinically affected and healthy sheep. There are also findings indicating isolation of Mcpp from sheep (Shiferaw et al., 2006; Hadush et al., 2009; Mbyuzi et al. 2014; Teshome et al., 2018; AbdElrahman et al., 2019) and cattle infected with mastitis (Teshome and Sori, 2021). Furthermore, Mccp antibody was detected from Buffalo (*Syncerus caffer),* Impala (*Aepyceros melampus*) and camel (*Camelus dromedaries*) in Kenya (Hailu, 2017). However, the role of the wild animals and the domestic animals excluding the goat as the reservoir or dead-end host is not clearly studied (Yatoo et al., 2019).

3.2. History and Geographic distribution

CCPP was first described in 1873 in Algeria by Thomas and was called ‘boufrida’ indicating involvement of one lung in the disease. Initially, it was not considered contagious rather climatic conditions were claimed for its occurrence. The 1881 CCPP outbreak in South Africa following the introduction of diseased Angora goats from Turkey lead to exploration of contagiousness of the disease by Duncan Hutcheon. Subsequently the etiology of the disease that remained unclear for long time was investigated and identified to be Mycoplasma F38 in Kenya. Later on, it was officially named *Mycoplasma capricolum* subsp. *capripneumoniae* in 1993 (Yatoo et al., 2019; Teshome and Sori, 2021).

Mccp has been isolated in 20 countries and clinical descriptions have been reported in about 40 countries around the globe. The disease is present in the Arabian Peninsula, North, Central and East Africa and Asia, but its boundaries are still uncertain (Nicholas and Churchward, 2012). After the first isolation of Mccp in Kenya, it has been isolated in a number of African, Asian and Middle East countries. The countries include Sudan, Tunisia, Chad, Niger, Tanzania, United Arab Emirates, Oman, Eritrea, Uganda, Ethiopia and Mauritius, Pakistan, China, Tajikistan, Turkey, Iraq and Kuwait. However, there have been no reports of Mccp isolation in the American continent (Nicholas et al., 2008; AU-IBAR, 2013; Hailu, 2017).

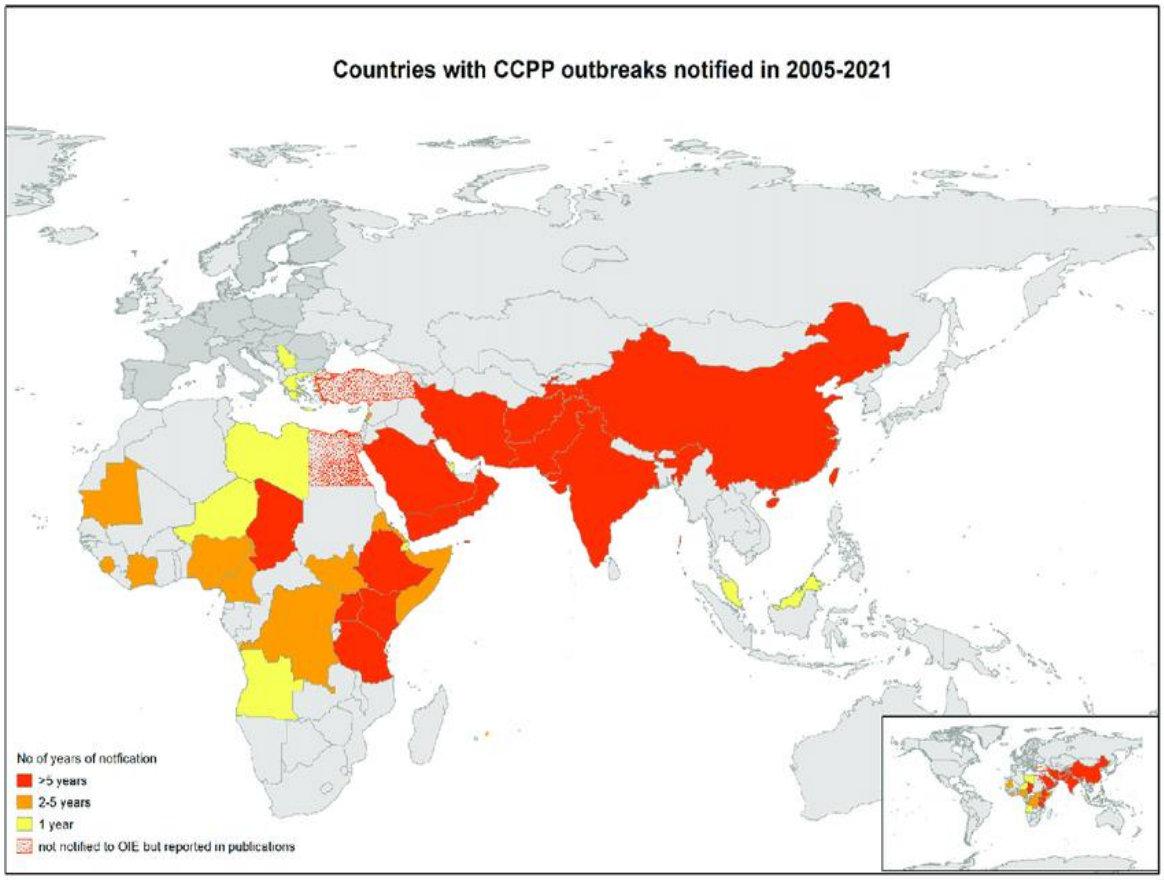

Figure 1: Countries that have notified CCPP outbreaks to the OIE from 2005-2021 by number of years of notifications. Red, orange and yellow colors indicate > 5 years, 2 to 5 years and only 1 year of notification respectively (EFSA Panel on Animal Health and Welfare et al., 2022)

3.3. Predisposing risk factors

The occurrence of CCPP is influenced by risk factors related to environment, production system and immune status of the host population. Livestock mobility and presence of naive populations in an infected area are major predisposing factors. Contagious caprineplueropneumonia is mostly reported in naive populations and severe in younger animals. The presence of chronically infected animals in close proximity with naive animals, animal movement, accompanied by porous borders and poor cross-border quarantine systems are also key predisposing factors for CCPP spread. Other stress factors such as overcrowding, cold, long distance transportation, poor nutrition and concurrent parasitic and bacterial infections aggravate the clinical disease (Khan, 2015;Abrhaley et al., 2019).

3.4. Transmission

Inhalation of infected aerosols is the main route of CCPP transmission. The main source of contamination is direct contact with affected animals. Airborne transmission can result in distant spread with in 50m (Chaber et al., 2014). Contagious caprine plueropneumonia always appears after the introduction of an infected animal into a susceptible herd. The transmission of the disease is direct by the aero-genic route, through droplets released during coughing. Very short periods of contact are sufficient to transmit the disease, but intimate contact is needed. Indirect transmission does not seem to occur. Infected objects, vectors, fomites and animal products are yet to be known in transmission (OIE 2009; Yatoo et al., 2019).

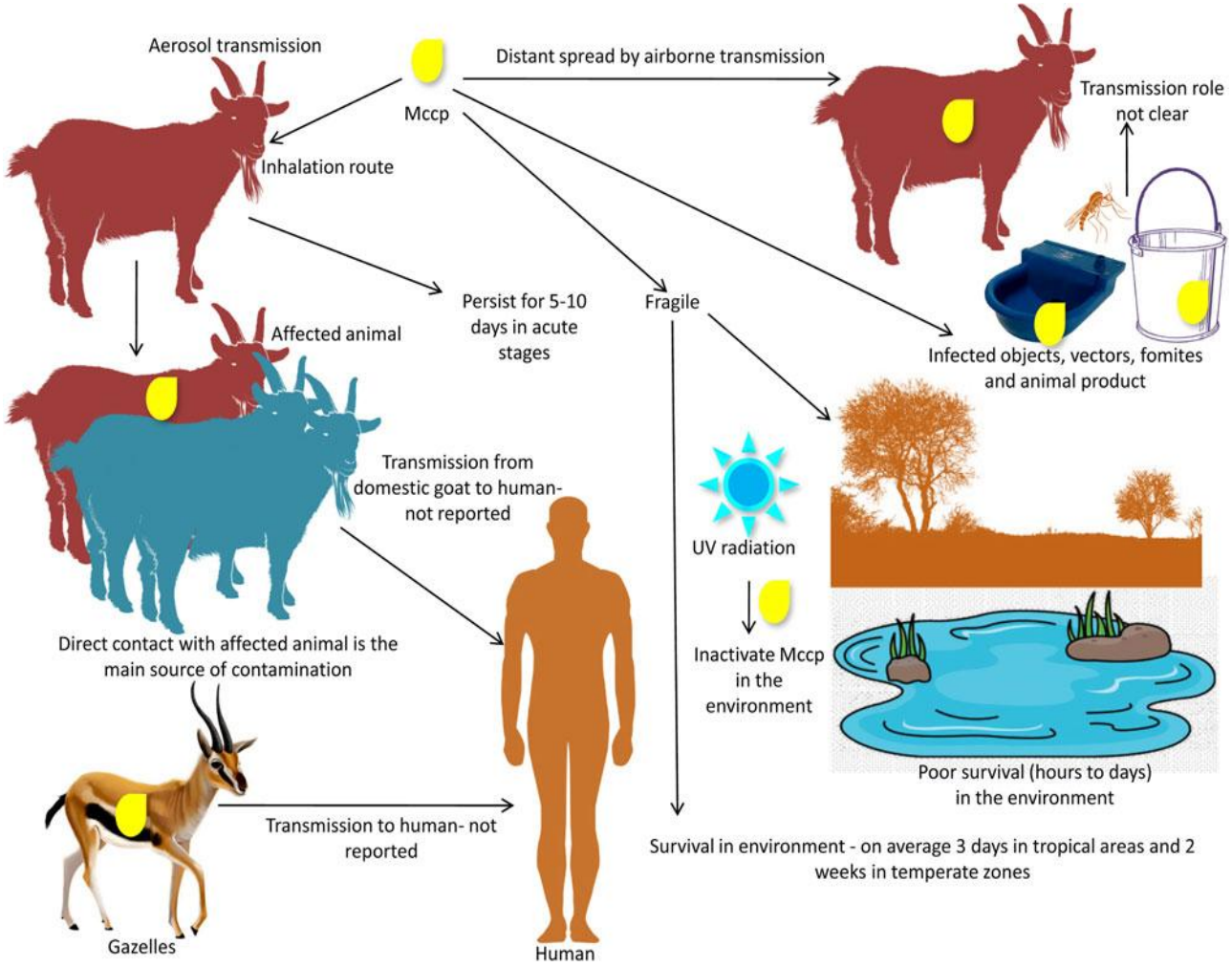

Figure 2 :Transmission of Mccp(Yatoo et al., 2019)

4. Pathogenesis

The exact mechanism of mycoplasmas pathogenesis is unclear. Mycoplasmas adhere to epithelial cell surfaces by a means of 168kD adhesive proteins (P1), one of the major virulence factors. The proteins are found at tips of the bacterial cells and binds to sialic acid residues on host epithelial cells (Hailu, 2017). The colonization of respiratory tract results in the cessation of ciliary movement. Consequently, the clearance mechanisms of the tract do not function, resulting in contamination of the respiratory tract and development of dry cough. The intimate association of mycoplasma and host cells provides environment in which toxic metabolic products (such as hydrogen peroxide and superoxide) accumulate and damage host tissues. Furthermore, the mycoplasmas have been shown to inhibit host cell catalase there by increasing the peroxide concentrations (Tigga et al. 2014; Hailu, 2017).

In general, pathogenesis of CCPP involves inhalation, attachment, ciliostasis, alteration and loss of cilia, multiplication and mucosal epithelial cells destruction, dissemination and inflammation and oxidative stress. After entry into respiratory passages, the Mccp may attach to superficial cell layers by a means of different membrane structures followed by colonization and initiation of pathological inflammation characterized by epithelia ciliostasis, serofibrinous pleuropneumonia, vasculitis and fibrinocellular exudation.

Mycoplasmal antigens activate immune system and stimulate the inflammatory and oxidative cascade resulting in pathologic changes and hence wide spread serofibrinous inflammatory reaction and fluid exudation in lungs, pleura, thorax and sometimes heart, liver, and kidneys. These pathological alterations lead to fibrin deposition in the pleural cavity, fluid exudation and hydrothorax (Yatoo et al., 2019).

5. Clinical signs

Contagious caprine plueropneumoniacan be manifested in per-acute, acute or chronic forms. In per-acute form, death is sudden usually within 24-72h (Samiullah, 2013). Contagious caprineplueropneumonia is initially characterized by high fever (41-43^{o}C), high morbidity and mortality rate. After 2-3 days of high fever, accelerated dyspnea with painful grunting, frequent and violent productive cough are prominent. Lethargy, lagging behind the flock, lying down, anorexia and abortions in pregnant goats are also noticed (Ruffin, 2001; Mekuria et al., 2008).

Continuous nasal discharge that is initially serofibrinous straw colored exudate followed by thick mucoid or purulent and rust colored discharge might be observed (Ruffin, 2001). In the terminal stages of the disease, the animals experience mouth breathing, are unable to move and stand with their front legs wide apart, the neck is stiff and extended downward. Stringy saliva can continuously drips from their mouth and mucopurulent discharge obstructs their nostrils. The tongue may protrude and they bleat distressingly. Finally, death can occurs within 7 to 10 days after appearance of the signs, but can be as fast as 2 days(Mekuria et al., 2008).

6. Economic importance

Contagious caprineplueropneumonia results in heavy economic losses to countries involved in goat farming especially in Africa, Asia and the Middle East countries. The total yearly economic loss cost of CCPP is estimated to be about 507 million US$ in endemic areas. The economic losses incur from morbidity, mortality and decline/ loss of production performance in addition to prevention, control and treatment costs. Morbidity and mortality from the disease can be as high as 100% especially in exotic breeds. In naive and native herds, 100% morbidity and 80% mortality rateswas noted(Yatoo et al., 2019). In general, direct economic losses due to the disease result from its high mortality, reduced milk and meat yield, cost of treatment, control, disease diagnosis and surveillance in addition to indirect losses associated with the imposition of trade restriction (Bekele et al., 2011).

7. Diagnosis

Diagnosis of CCPP might involve microbiological, biochemical, serological, and gene-based identification following a clinical tentative diagnosis. Tentative diagnosis of the diseasedepends on clinical signs, postmortem findings and demonstration of Mccp in pleural fluid by dark-field microscopy (Hailu, 2017). The causative agent, Mccpis identified by its branching filamentous structure in exudates, impression smears or tissue sections under the microscope. Other caprine mycoplasmas usually appear as short filamentous organisms or coccobacilli(Spickler, 2015).

Microbiological methods include culture, isolation, and identification. However, the microbiological diagnosis of CCPP is considered difficult due to very poor in vitro growth of Mccp and usual contamination of samples by other easily growing mycoplasmas. In addition, fastidiousness and special requirements of Mccp add to the problem of diagnostics. Hence, other diagnostic methods should be relied on (Yatoo et al., 2019).

7.1. Clinical diagnosis

The CCPP should be suspected when a highly contagious disease occurs in goats with pyrexia of 41^{o}C or greater, severe respiratory distress, high morbidity and mortality, inability to move and standing with front legs wide apart and neck stiff and extended (OIE, 2009).

7.2. Post mortem examination

The lesions of CCPP are limited to the respiratory system. Acute form of the disease is characterized by unilateral or bilateral pneumonia and sero-fibrinouspleuritis with straw colored fluids in the thoracic cavity. The cut surface of the lung is granular in appearance with straw-colored exudates. Yellow nodules surrounded by an area of congestion may also be found in the lungs. There are varying degrees of lung consolidation with enlargement of bronchial lymph nodes. Long term survivors may develop chronic pleuropneumonia or chronic pleuritis, with encapsulation of acute lesions and fibrous adhesions to the chest wall(Spickler, 2015).

7.3. Laboratory diagnosis

Confirmatory diagnosis of CCPP is mainly based on isolation and identification of the causative agent from clinical samples collected from goats showing typical signs of the disease. Samples such as nasal swabs, pleural fluid, lung tissue, blood or serum samples can be collected for diagnosis of CCPP. However, the pleural fluid and sections of hepatized lung are the samples of choice for the diagnosis. Blood or serum samples are essential for serology while discharges, exudates, blood and tissues are for culture or isolation and gene/DNA-based studies. Pleural fluids are collected by sterile syringes. The lung tissue has to be taken from the lesions at the interface between consolidated and unconsolidated area. The preferable method for the tissue sample collection is to sacrifice the goat that did not receive any antibiotic treatment(Hailu, 2017).

The samples are transported to laboratory using an icebox and stored at 4 for days to weeks. If microbiological examination cannot be performed immediately, samples or whole lungs can be stored being frozen at -20^{o}C for long periods (months) with little apparent loss of *mycoplama* viability(Hailu, 2017). Antibiotics such as penicillin or ampicillin has to be added to the sample to be frozen to prevent contamination (Thiaucourt, 1996)**.** Isolation of Mccp is considered as a confirmatory diagnosis. However, it requires a very special growth medium, well-equipped and sophisticated laboratory facility as the pathogen is very fastidious and requires a prolonged initial incubation period at 37 under sterile laboratory environment (OIE, 2009).

7.4. Culturing

Isolation of Mccp is very difficult because of its highly fastidious nature, slow grows in broth media, and its minute colonies produced on solid media. Furthermore, it is also frequently overgrown by other common *mycoplasmas* (Hailu, 2017). The samples of choice from affected animals are the pleural fluid, which contains high numbers of mycoplasmas and sections of hepatized lung preferably at the interface between normal and diseased tissue(Nicholas and Churchward, 2012).

A number of media have been used for growth and isolation of *Mycoplasma*. *Mycoplasma* agar and broth media are media used for selective isolation of *Mycoplasma* spp. Mccp has been successfully grown and isolated from infected lung tissuesby culturing on Hayflick medium broth (H25P). Modified Hayflicks media have been also used for the growth and isolation of Mccp organisms. Mccp takes longer time usually five to seven days for in vitro growth contrary to all other *Mycoplasma mycoides* cluster members which grow within 24-48 hour producing colonies 1-3mm in diameter. Mccp colonies are known to have red coloration (Samiullah, 2013).

7.5. Biochemical tests

Biochemical tests most commonly used for Mccp isolation are glucose breakdown, arginine hydrolysis, film and spots formation, reduction of

tetrazolium, phosphatase activity, serum digestion and digitonin sensitivity. Glucose breakdown is indicated by acid (yellow color) production and arginine hydrolysis by alkaline (red colour) production in broth media culture (Thiaucourt, 2008).

Film and spots' describes an apparent wrinkling of the agar surface due to the deposition of iridescent film of lipid on the agar, together with the development of black spots within the medium in vicinity of ageing colonies. The test for tetrazolium reduction provides corroborative evidence of mycoplasmal nature of *M. agalactiae* isolates, as this organism is neither glycolytic nor arginine hydrolyzing. Serum digestion distinguishes members of ruminant mycoplasmas and phosphatase production separates Mccp from other members of this cluster. Digitonin sensitivity distinguishes members of the order Mycoplasmatales from those of the order Acholplasmatales(Thiaucourt, 2008).

7.6. Molecular Diagnosis

Molecular tests have become the novel diagnostic interventions for CCPP not only because of high specificity and sensitivity, but also because of difficulties in culturing of Mccp. Globally, various molecular tests are being employed for diagnosis of the disease(Yatoo et al., 2019). The development of PCR has greatly improved the diagnosis as it allow to detect the mycoplasma quickly even in mixed cultures, directly from clinical material such as pleural fluid and lung, and from this material dried on filter paper (Nicholas and Churchward, 2012).

The molecular detection of Mccp directly in clinical samples was found highly sensitive and specific and should be used for diagnosis of CCPP, especially in outbreaks to confirm the disease for rapid control (Teshome et al., 2019)

7.7. Serological diagnosis

Serological tests are used on a herd basis and not for individual diagnosis (Spickler, 2015).

Commonly used serological diagnostic tests used to detect antibody response of goats to Mccp infection are indirect hem-agglutination, complement fixation (CFT), and latex agglutination (LAT). Recently, a competitive enzyme-linked immunoassay (cELISA) for CCPP diagnosis has also been developed and found to be highly specific (Teshome et al., 2019). CFT is prescribed for international trade, demonstrating the potential limitations of antibody detection as a sole diagnostic technique.LAT uses a capsular polysaccharide specific antigen (CPS) coated beads extracted from Mccp to detect early IgM antibodies (Spickler, 2015; Hailu, 2017).

LAT is carried out by mixing a drop of the sensitized beads with a drop of blood or serum from the suspected animal on a glass slide for one minute and the results are read visually and recorded as positive or negative based on presence or absence of agglutination. c-ELISA detects antibodies in sera of naturally infected or artificially immunized animals while it remained negative with hyper immune sera to related strains. It is likely to be more suitable for epidemiological surveillance and sero-prevalence studies. The high cutoff point, maximize the diagnostic specificity but it is expected to decrease sensitivity of the test (Hailu, 2017).
Serological test such as growth inhibition test (GIT), growth precipitation test (GPT) and indirect fluorescent antibody test (IFA) are also used for identification of Mccp. GIT is the least sensitive and simplest of the tests available for CCPP diagnosis. It depends on the direct inhibition of Mycoplasma growth on solid media by specific hyper immune serum, and detects primary surface antigens. The GIT is particularly useful in identifying Mccp because they appear to be serologically homogeneous, and antiserum to the type strain produces wide inhibition zones (Samiullah, 2013).

The GPT detects soluble cytoplasmic and extra membranous antigens released by growing cultures and allowed to diffuse through solid mycoplasma growth medium towards mycoplasma antiserum during growth. IFA test is applied to unfixed colonies on agar. Antiserum

against a single strain is sufficient to identify field isolates of that species, and antisera are diluted before use. Cultures do not have to be cloned, but the test is usually applied only after several passages have indicated whether the culture contains more than one species and the growth characteristics of the organisms present (Thiaucourt, 2012).

7.8. Diagnostic Imaging

Diagnostic imaging techniques such as Ultrasonography (USG) and X-ray are recent advanced tools for diagnosing CCPP associated changes in lungs, pleura, thorax, and associated structures. Irregularity of the visceral pleural surface, which reflects the beginning of consolidation, can be revealed by USG. Accumulation of exudates in lung makes alveoli hypoechoic or even anechoic in pleuropneumonia, which otherwise formhyperechoic zones both in normal or consolidated lungs. However, with disappearance of the consolidation, a reduction of these hyperechoic zones occur and hyperechoic reflective bands may appear(Yatoo et al., 2019).

7.9. Differential Diagnosis

CCPP can be clinically confused with other *Mycoplasma* species or pasteurellosis (Liljander et al., 2015). The classical disease caused by Mccp is restricted respiratory system. However CCPP may be suspected when lesions are restricted to the respiratory tract, affect only one lung and when animals present a conspicuous pleurisy with profuse effusion of pleural fluid (OIE, 2018). Peste des petits ruminants (PPR), pasteurellosis and contagious agalactia syndrome are among the diseases confused with CCPP. PPR can affect sheep in addition to goats.Pasteurellosiscan be differentiated from CCPP on the basis of distribution of gross lung lesions; and what has been called mastitis, arthritis, keratitis, and pneumonia and septicemia syndrome or more often as contagious agalactia syndrome. The disease caused by Mccp was argued to be readily differentiated from contagious agalactia syndrome by Mccp being contagious and fatal to susceptible goats of all ages and sex groups. It is also differentiated from the agalactia syndrome in that Mccp rarely affects sheep and does not affect cattle(OIE, 2009).

8. Treatment

Successful treatment of CCPP varies with affected site, time course of the disease and stage of intervention. Several antibiotic drugs have been used for treatment of CCPP. Antibiotics such as tetracycline, spiramycin, erythromycin, tiamulinsfumarate, tylosin, and streptomycin are recommended to treat the disease their effectiveness depending on early intervention and treatment of the infected herd. However, macrolides especially tylosin is considered to be the drug of choice. Oxytetracycline has been also used for long period of time being proven as effective drug. In early treatment, the prognosis is good. The prognosis for recovery with prompt treatment is estimated to be 8.7% and then animals recovered from clinical diseases may remain carrier(Gladon and Spickler 2011; Teshale 2012; Yatoo et al., 2019).

9. Prevention and control

Prevention and control of CCPP can be done through quarantines, vaccination, movement controls, slaughter of infected and exposed animals, and cleaning and disinfection of the premises. Quarantine measures have to be applied following laboratory confirmation of the disease. There is an effective vaccine; inactivated Mccp vaccine (Formerly F38), for the effective control of CCPP. Vaccination should aim at covering 100% of the population to control the disease. Coordination between neighboring geographical areas and countries in vaccination is very important to control spread of the disease across the region. Regulating animal movement in infected and surrounding area is important. In endemic areas, care should be also taken when introducing new animals into the flock. In addition, antibiotic treatment and reductions in animal density are sometimes employed (Spickler, 2015).

10. Status in Ethiopia

In Ethiopia, the presence of CCPP has been suspected for a long period especially in the remote region of Sudan and Kenya border (Hailu, 2017). It has been suspected since 1983 and was confirmed in 1990 by isolation and identification of Mccp from an outbreak in Ogaden, Eastern Ethiopia by Thiaucourt(Thiaucourt, 2014). Since then, the disease has been known to be endemic in different regions of the country and has been reported from almost all regions of Ethiopia. CCPP is endemic in most extensive goat rearing areas of Ethiopia, mainly in the arid and semi-arid low lands of rift valley, Borana, South Omo, Afar and other pastoral areas of the country (Shiferaw et al., 2006; Teshome and Sori, 2021). Inactivated CCPP vaccine is produced at NVI, Bishoftu from F-38 Kenyan strain/Mccp using saponin as adjuvant. The vaccine has a potential to confer protective immunity for more than one year (Hailu, 2017).

Due to insufficiency of accurate diagnostic services, shortage of vaccination against CCPP, poor management, weather conditions, and concurrent infections, there is a widespread occurrence of the disease in rural and different agro-ecological areas of the country. Environmental stress, particularly hot and humid climate, favors precipitation of this disease. Studies conducted in different localities of Ethiopia reported the prevalence of CCPP to range from 10% to 43%. A meta-analysis of CCPP conducted by Asmare et al., 2016 using more than twelve published articles indicated that the sero-prevalence of the disease to be 25.7% (Abrhaley et al., 2019).

Contagious caprineplueropneumoniasero-prevalence reports of 13.2% (Bekele et al., 2011) from Borena and Guji, 42.8% from Afar (Teshale, 2012), 31.2 % in Borena Zone (Teshome, 2019), 18.1% from Gambella(Aklilu et al., 2015), 8.5% in Northern Amhara(Abrhaley et al., 2019), 31.6% in Borena(Matios et al., 2014), 4.92% in Diredawa(Yousuf et al., 2012), 32.63% in Jijiga zone (Sherif et al., 2012), 18.61 % in Southern Ethiopia (Mekuria and Asmare, 2010), 15.5% in Hammer and Benna-Tsemay districts of Southern Ethiopia(Mekuria et al., 2008) and 14% in Central Ethiopia (Hailu, 2017) have been documented in Ethiopia. Different numbers of CCPP outbreaks have been also reported (Eshetu et al., 2007). The frequently reported outbreaks of CCPP in Ethiopia almost certainly represent an underestimate as this disease is having a major socio-economic impact in the country (Nicholas and Churchward, 2012).

11. Conclusion

CCPP is economically important disease of goats. It can also affect some domestic and wild animals which are reservoir hosts. CCPP is one of the most economically important disease posing losses through production loss, trade restriction, death, treatment and vaccination costs.However, less attention is given to control it compared to other modifiable disease.Early diagnosis and treatment of the disease along with implementation of control/preventive measures is very important to reduce the risk. For diagnosis of the disease pleural fluid and sections of hepatized lung are samples of choice. Currently PCR is novel diagnostic methods because of high specificity and sensitivity, but also because of difficulties in culturing of Mccp. Of several serological tests used complement fixation (CFT) is a sole diagnostic method prescribed for international trade.The epidemiology of CCPP is still unclear and the causative agent (Mccp) is not typed so far. The role of wild animals and other susceptible domestic animals in epidemiology of the disease is also not determined. Therefore, more detailed studies should have to be undertaken to investigate and reveal epidemiology of the disease including the role of the reservoir hosts in the epidemiology to design and apply effective preventive and control measures. Creating awareness to farmers on importance and control/prevention methods of the disease is also important.

12. References

Abd-Elrahman. A.H., Khafaga, A.F., Abas, O.M., 2019. The first identification of contagious caprine pleuropneumonia (CCPP) in sheep and goats in Egypt: molecular and pathological characterization. Trop. Anim. Health Prod. 52 (3): 1179-1186.

Abrhaley, A., Ejo, M., Fentie, T., 2019. Seroprevalence and Risk Factors Associated with Contagious Caprine Pleuropneumonia in Western Amhara, Northwest Ethiopia. J. Vet. Med. 2019: 1-7. https://doi.org/10.1155/2019/9878365.

Aklilu, F., Asfaw, Y., Baumann, M., Abie, G., 2015. Epidemiological study of contagious caprine pleuropneumonia (CCPP) in selected districts of Gambella Region, Western Ethiopia. African J. Agric. Res. 10: 2470-2479. https://doi.org/10.5897/ajar2014.9264.

Arif, A.A., Schulz, J., Thiaucourt, F., Taha, A., Hammer, S., Arif, A., Sc, B. V, Sc, M., Schulz, J., 2019. Contagious caprine pleuropneumonia outbreak in captive wild ungulates at Al Wabra wildlife preservation, State of Qatar captive wild ungulates at Al Wabra wildlife. J. Zoo Wildl. Med. 38: 93-96.

Asmare, K., Abayneh, T., Mekuria, S., Ayelet, G., Sibhat, B., Skjerve, E., Szonyi, B., Wieland, B., 2016. A meta-analysis of contagious caprine pleuropneumonia (CCPP) in Ethiopia. Acta Trop. 158: 231-239. https://doi.org/10.1016/j.actatropica.2016.02.023.

Atim, S.A., Ayebazibwe, C., Mwiine, F.N., Erume, J., Tweyongyere, R., 2016. A Survey for Contagious Caprine Pleuropneumonia in Agago and Otuke Districts in Northern Uganda. Open J. Vet. Med. 06: 9-14. https://doi.org/10.4236/ojvm.2016.61002.

AU-IBAR., 2013. Contagious caprine pleuropneumonia. African union Inter African bureau for animal resources. Www/AU-IBAR Org.

Bekele, T., Asfaw, Y., Gebre-Egziabeher, B., Abebe, G., 2011. Seroprevalence of contagious caprine pleuropneumonia in Borana and Guji lowlands, Southern Ethiopia. Ethiop. Vet. J. 15: 69-76. https://doi.org/10.4314/evj.v15i2.67695

Chaber, A.L., Lignereux, L., Qassimi, M. Al, Saegerman, C., Manso-Silván, L., Dupuy, V., Thiaucourt, F., 2014. Fatal transmission of contagious caprine pleuropneumonia to an Arabian oryx (Oryx leucoryx). Vet. Micro biol. 173: 156-159. https://doi.org/10.1016/j.vetmic.2014.07.003.

EFSA AHAW Panel (EFSA Panel on Animal Health and Welfare), Nielsen, S.S., Alvarez, J., Bicout, D.J, Calistri, P., Canali, E., Drewe, J.A., Garin-Bastuji, B., Gonzales Rojas, J.L., Gortazar, C., Herskin, M., Michel, V., Miranda Chueca, M.A., Padalino, B., Pasquali, P., Spoolder, H., Stahl, K., Velarde, A., Viltrop, A., Winckler, C., Gubbins, S., Stegeman, J.A., Thiaucourt, F., Antoniou, S-E., Aznar, I., Papanikolaou, A., Zancanaro, G. and Roberts, H.C., 2022. Scientific Opinion on the assessment of the control measures for category A diseases of Animal Health Law: Contagious Caprine Pleuropneumonia. EFSA J. 20 (1): 7068-7088 pp. https://doi.org/10.2903/j.efsa.2022.7068.

Eshetu, L., Yigezu, L., Asfaw, Y., 2007. A study on Contagious Caprine Pleuropneumonia (CCPP) in goats at an export oriented abattoir, Debrezeit, Ethiopia. Trop. Anim. Health Prod. 39: 427-432. https://doi.org/10.1007/s11250-007-9041-1.

Hadush, B., Eshetu, L., Mengistu, W., Hailesilassie, M., 2009. Sero-prevalence of contagious caprine pleuropneumonia in Kefta Humera, Alamata (Tigray) and Abaala (Afar), Northern Ethiopia. Trop. Anim. Health Prod. 41: 803- 806.

Hailu, D.M., 2017. Sero-prevalence investigation and associated risk factors of contagious caprine pleuropneumonia in selected districts of Central Ethiopia. MSc Thesis, Addis Ababa University, College of Veterinary Medicine and Agriculture, Bishoftu, Ethiopia.

Liljander, A., Yu, M.O., Brien, E., Heller, M., Nepper, J.F., Weibel, D.B., Gluecks, I., Younan, M., Frey, J., Falquet, L., Jores, J., 2015. Field applicable recombinase polymerase amplification assay for rapid detection of *Mycoplasma capricolum* subspecies *capripneumoniae*. J. Clin. Microbiol. 53(9): 2810-2815.

Mbyuzi, A., Erick, V., Komba, E., Kimera, S., Kambarage, D., 2014. Seroprevalence and associated risk factors of peste des petitsruminants and contagious caprine pleuropneumonia in goats and sheep in the Southern Zone of Tanzania. Prev. Vet. Med., 116 (1): 138-144.

Matios, L., Tesfaye, S., Gelagay, A., Eyob, E., Gebremikael, D., Tadele, T., 2014. Seroprevalence of contagious caprine pleuropneumonia and field performance of inactivated vaccine in Borana pastoral area, southern Ethiopia. African J. Microbiol. Res. 8: 2344-2351. https://doi.org/10.5897/ajmr2014.6806.

Mekuria, S., Asmare, K., 2010. Cross-sectional study on Contagious Caprine Pleuro Pneumonia in selected districts of sedentary and pastoral production systems in Southern Ethiopia. Trop. Anim. Health Prod. 42: 65-72. https://doi.org/10.1007/s11250-009-9386-8.

Mekuria, S., Zerihun, A., Gebre-Egziabher, B., Tibbo, M., 2008. Participatory investigation of Contagious Caprine Pleuropneumonia (CCPP) in goats in the Hammer and Benna-Tsemay districts of Southern Ethiopia. Trop. Anim . Health Prod. 40: 571-582. https://doi.org/ 10.1007/s11250-008-9136-3.

Nicholas, R., Ayling, R., and McAuliffe, L., 2008. Contagious caprine pleuropneumonia. In: Mycoplasma diseases of ruminants. Wallingford, UK: CAB International pp. 114-131.

Nicholas, R., Churchward, C., 2012. Contagious Caprine Pleuropneumonia: New Aspects of an Old Disease. Transbound. Emerg. Dis. 59: 189-196. https://doi.org/10.1111/j.1865 1 682.2011.01262.x.

Noah, E.Y., Kusiluka, L.J.M., Wambura, P., Kimera, S.I., 2011. Field Isolation of Mycoplasma capripneumoniae in Central Zone of Tanzania. Int. J. Anim. Vet. Adv. 3: 434-442.

OIE, 2017. Contagious caprine pleuropneumonia. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2017.

OIE, 2018. OIE Terresterial manual. Chapter 3.7.4. Contagious caprine pleuropneumonia.Pp 1141-1455.

OIE, 2009. Contagious Caprine Pleuropneumonia Aetiology Epidemiology Diagnosis Prevention and Control References, OIE Terrestrial Manual.

Rehman, F.U., Khan, F.A., Saeed, M., Ali, M., Khan, H., Ahmad, F. and Ullah, Q., 2022. Clinicopathological and Sero-Molecular Detection of *Mycoplasma capricolum* subsp. *capripneumoniae* in Goats in Southern Areas of Pakistan. Vet. Med. Int., 2022: 9508810. https://doi.org/10.1155/2022/9508810.

Ruffin, D.C., 2001. Mycoplasma infections in small ruminants. Vet. Clin. North Am. Food Anim. Pract. 17: 315-332. https://doi.org/10.1016/S0749-0720(15)30031-1.

Samiullah, S., 2013. Contagious caprine pleuropneumonia and its current picture in Pakistan: A review. Vet. Med. 58: 389-398. https://doi.org/10.17221/6977-VETMED.

Sherif, M., Addis, M., Tefera, M., 2012. Contagious caprine pleuropneumonia: Serological survey in selected districts of Jijiga zone, Ethiopia. Asian J. Anim. Sci. 6: 309-315. https://doi.org/10.3923/ajas.2012.309.315.

Shiferaw, G., Tariku, S., Ayelet, G., Abebe, Z., 2006. Contagious caprine pleuropneumonia and Mannheimia haemolytica-associated acute respiratory disease of goats and sheep in Afar Region, Ethiopia. OIE Rev. Sci. Tech. 25: 1153-1163. https://doi.org/10.20506/rst.25.3.1723

Soayfane, Z., Houshaymi, B., Nicholas, R.A.J., 2018. Mycoplasma capricolum subsp. capripneumoniae, the cause of contagious caprine pleuropneumonia, comprises two distinct biochemical groups. Open Vet. J. 8: 393-400. https://doi.org/10.4314/ovj.v8i4.7.

Spickler, A. R., 2015. Contagious Caprine Pleuropneumonia. http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php.

Teshale, A.G., 2012. Seroprevalence and participatory study of Contagious Caprine Plueropneumonia In Gulina Wereda, Afar National Regional State, Ethiopia. Addis Ababa University.

Teshome, D., and Sori, T., 2021. Contagious caprine pleuropneumonia: A review. J. Vet. Med. Anim. Health, 13 (3): 132-143. DOI: 10.5897/JVMAH2020.0906.

Teshome, D., Sori, T., Sacchini, F., Wieland, B., 2019. Epidemiological investigations of contagious caprine pleuropneumonia in selected districts of Borana zone, Southern Oromia, Ethiopia. Trop. Anim. Health Prod. 51: 703-711. https://doi.org/10.1007/s11250-018-1744-y

Thiaucourt, F., 2014. Contagious caprine pleuropneumonia in Ethiopia. https://doi.org/10.1136/vr.131.25-26.585.

Thiaucourt, F., 2012. Chapter 2.7.6 Contagious Caprine Pleuropneumonia., in: Manual of Diagnostic Tests and Vaccines for Terrestrial Animals (Mammals, Birds and Bees). OIE Terrestrial Manual. World Organisation for Animal health (OIE), Paris, France, pp. 995–1007.

Thiaucourt, F., 2008. Contagious Bovene Pleuropneumonia, in: manual of diagnostic tests and vaccines for Terrestrial animals (Mammals, Birds and Bees). OIE Terrestrial Manual, p. 146. https://doi.org/10.5455/ijlr.20121014122526.

Thiaucourt, F., 1996. Contagious caprine pleuropneumonia and other pulmonary mycoplasmoses of sheep and goats. Rev. sci. tech. Off. int. Epiz., 15, 1397-1414.

Tigga, M., Choudhary, B., Ghosh, R., MaliK, P., 2014. Mycoplasmosis: An emerging threat to developing livestock industry. Int. J. Adv. Res. 2(1): 558-564.

Wazir, I., Hussain, I., Khan, M.A, Ali, M.I, Ur Rahman, H., Ashraf, F., Khan S., Ullah, S., Ullah, Q., Khyber, Khan, A., 2016. Seroepidemiological Analysis of Contagious Caprine Pleuropneumonia through cELISA in Selected Districts of Khyber Pakhtunkhwa-Pakistan.Am. Sci. Res. J. Eng. Technol. Sci. 26 (3): 274-281.

Yatoo, M.I., Parray, O.R., Bashir, S.T., Muheet, Bhat, R.A., Gopalakrishnan, A., Karthik, K., Kuldeep, D., Singh, S.V., 2019. Contagious caprine pleuropneumonia–a comprehensive review. Vet. Q. 39: 1-25. https://doi.org/10.1080/01652176.2019.1580826.

Yousuf, E., Melaku, A., Bogale, B., 2012. Seroprevalence of contagious caprine pleuropneumonia in Dire Dawa provisional administrative council, Eastern Ethiopia. J. Vet. Med. Anim. Heal. 4: 93-96. https://doi.org/10.5897/JVMAH12.053.

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DOI:10.22192/ijarbs.2022.09.12.006	

How to cite this article:
Yalew Abiyu and Nebiyu Kassa. (2022). Review on Epidemiology and Diagnosis of Contagious Caprine Pleuropneumonia. Int. J. Adv. Res. Biol. Sci. 9(12): 60-73.
DOI: http://dx.doi.org/10.22192/ijarbs.2022.09.12.006