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Review on Contagious Caprine Pleuropneumonia and Its Status in Ethiopia.

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Abstract

Contagious caprine pleuropneumonia (CCPP) is caused by *Mycoplasma capricolum* subsp. *caprip-neumoniae* (*Mccp*) which belongs to the *Mycoplasma mycoides* cluster, a group of five closely related *Mycoplasma*s, pathogenic to ruminants. The true lesions of CCPP are restricted to the alveolar tissues of infected goats, which distinguish it from other respiratory diseases of small ruminants caused by members of the *Mycoplasma mycoides* cluster. The typical signs of CCPP are an accumulation of pleural fluid, unilateral hepatisation, adhesions, pleurisy and pleuropneumonia. Transmission of CCPP occurs through aerosol while animals are in nearby contact. There are a number of approaches for the rapid detection and identification of Mccp based on a PCR. In Africa, Asia and the Middle East morbidity and mortality can reach about 100% which causes huge economic loss. The presence of CCPP in Ethiopia reported in 1983 and later confirmed from an outbreak in 1990 in Ogaden, Eastern Ethiopia. Since then CCPP has been considered an endemic disease in Ethiopia. Prevention and control of CCPP is undertaken through vaccination, quarantine, restriction of movement, culling of infected and exposed animals and keeping the hygiene of premises. However, it remains one of the standing problems of goat production. Therefore, more research, awareness creation about CCPP, transmission path way, prevention and control methods in small ruminant rearing areas to reduce the impact of the disease.

Keywords: CCPP, Ethiopia, Goat, Status.

Introduction

Ethiopia is endowed with a huge number of livestock that have not yet been optimally exploited mainly due to technical and institutional constraints [1]. The sector has huge support to Ethiopia's national economy and livelihoods the community and has a promise to the economic development of the country [2].

Based on the Central Statistical Agency of Ethiopia [3], Ethiopia is one of the countries having the largest livestock population. Ethiopia has over 29.11 and 29.33 million heads of goats and sheep, respectively. Nowadays, demand for meat and meat products rising [4], and small ruminants are considered as a main asset for livestock farmers in East Africa and play crucial economic and cultural roles in Ethiopia [5, 6].

Sheep and goats are very important to the household economy of the Borana community in terms of providing a source of a convenient amount of cash on a more frequent basis. This can partially substitute for sales of cattle [7]. At farm level small ruminants serve as investment and insurance due to their short generation interval and high frequency of multiple births [8], as well as small feed requirement and adaptability to harsh environmental conditions [9].

Despite their huge social and economic roles the services and revenue obtained from small ruminants is sub-optimal due to several constraints. The most important constraints to small ruminant production are widespread endemic diseases including parasitic infestation, and viral and bacterial diseases [10, 11]. Among various diseases, Contagious Caprine Pleuropneumonia (CCPP) is a major threat to goats [12], which causes major economic losses [8, 12].

CCPP has become of major concern in some African countries because of high morbidity and mortality. Often uncontrolled movements of animals in search of water and feeds and informal trading systems as well as communal grazing are considered important drivers for the dynamics of CCPP in sub-Saharan Africa [13]. In goats, CCPP is a devastating disease causing high morbidity and mortality occurring in Africa, Asia and the Middle East [12]. It is a classical transboundary animal disease [12, 14]. Moreover, the disease is included in the list of notifiable diseases of the World Organization for Animal Health (OIE) as it threatens a significant number of goat populations throughout the world and causes a significant socioeconomic impact in infected territories [12, 15]. Though the disease is confined to goats, subclinical cases were reported in sheep and some wild ruminant species [8].

Contagious caprinepleuropneumonia is clinically characterized by coughing, respiratory distress and very high morbidity and mortality [16]. The causative agent, *Mycoplasma capricolum* subspecies *capripneumoniae*, is one of the most serious and dramatic mycoplasmas, which is very difficult to isolate and correctly identify [17]. But there are a number of methods for the rapid detection and identification of *Mccp*based on a PCR system by which a segment of the 16S rRNA gene from all members of the *Mycoplasmamycoides* cluster can be amplified. The PCR product is then analyzed by restriction enzyme cleavage for the identification of *Mccp*DNA [18].

In Ethiopia, CCPP occurs in the most extensive goat-rearing areas namely, Afar, Borana, Omo valley, West Gojjam and in the lowlands of Tigray[19]. Reviewing different studies is very important in designing control and prevention methods.

Therefore, the objectives of this paper are;

- ✓ To review epidemiology, diagnosis and control and prevention of CCPP
- ✓ To summarize the status of CCPP in Ethiopia.

Contagious

Contagious caprine pleuropneumonia (CCPP) is one of the most severe diseases of goats. This disease, which affects the respiratory tract, is extremely contagious and frequently fatal; in some naive flocks, the morbidity and mortality rates may reach 100%. CCPP causes major economic losses in Africa, Asia and the Middle East, where it is endemic. Definitive diagnosis can be difficult, as the causative agent is one of the most fastidious mycoplasmas and can be missed during routine bacteriological analysis. CCPP is now known to also affect some species of exotic ungulates. This has raised concerns for zoos and for the conservation of some endangered species exposed togoats [18].

Etiology

Contagious caprinepleuropneumonia is caused by *Mycoplasma capricolum*subsp. *capripneumoniae*(formerly *Mycoplasma* biotype F-38), a member of the family Mycoplasmataceae. Epidemiological studies of

this organism are still limited; however, genetic analyses have grouped *M. capripneumoniae*isolates into two major clusters representing two evolutionary lines of the organism, five lineages which correspond to geographic regions, or six genotypes (A toF) [20].

M. capripneumoniae belongs to a closely related group of mycoplasmas called the *Mycoplasma mycoides*cluster. Another organism in this group, *M. mycoides*sub sp.*capri* (aspeciesnow containing both *M.mycoides*subsp.*capri* and the former*M. mycoides*subsp. *Mycoides* large-colony type) can cause a disease that resembles CCPP but may have extrapulmonary signs and lesions. Some texts consider *M. mycoides*subsp. *capri*to be a minor cause of contagious caprine pleuropneumonia; however, the World Organization for Animal Health (OIE) limits the cause of CCPP to only *M. capricolum subsp. Capripneumoniae* [21].

Species Affected

Goats are the primary hosts for *M. capripneumoniae*, and the only domesticated animals proven to be affected by this organism. At present, the significance of infections in sheep is uncertain; however, at least two papers have reported the occurrence of *M. capripneumoniae* in healthy or sick sheep. There is also a possibility that this organism might have been involved in an outbreak of acute respiratory disease among goats and sheep in Ethiopia in 2002. *M. capripneumoniae* has caused clinical cases in some wild ungulates including wild goats (*Capra aegagrus*), Nubian ibex (*Capra ibex nubiana*), Laristanmouflon (*Ovisorientalislaristanica*), gerenuk (*Litocraniuswalleri*), sand gazelles (*Gazellasubgutturosamarica*), Arabian oryx (*Oryx leucoryx*), and Tibetan antelope (*Pantholopshodgsonii*) [22].

Geographic Distribution

Contagious caprinepleuropneumonia can be found in many countries in Africa, Asia) and the Middle East. *M. capripneumoniae*is difficult to isolate from clinical material, and its presence has not been confirmed in all affected countries. In some cases, reports of its occurrence are based on clinical signs alone [23].

Predisposing risk factors

The occurrence of CCPP is influenced by risk factors related to the environment, production system and immune status of the host population. Livestock mobility and the presence of naive populations in an infected area are major predisposing factors. Contagious caprine plueropneumonia is mostly reported in naive populations and severe in younger animals. The presence of chronically infected animals in close proximity to naive animals, and 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 [24, 25].

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 a distant spread within 50m [26]. 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 [22, 27].

Pathogenesis

The exact mechanism of mycoplasmas pathogenesis is unclear. Mycoplasmas adhere to epithelial cell surfaces by means of 168kD adhesive proteins (P1), one of the major virulence factors. The proteins are found at the tips of the

bacterial cells and bind to sialic acid residues on host epithelial cells [20]. The colonization of the 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 the development of a dry cough. The intimate association of mycoplasma and host cells provides an environment in which toxic metabolic products (such as hydrogen peroxide and superoxide) accumulate and damage host tissues [20].

Furthermore, the mycoplasmas have been shown to inhibit host cell catalase thereby increasing the peroxide concentrations [20, 28].

In general, the pathogenesis of CCPP involves inhalation, attachment, cryostasis, 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 means of different membrane structures followed by colonization and initiation of pathological inflammation characterized by epithelia cryostasis, serofibrinous pleuropneumonia, vasculitis and fibrinocellular exudation [28].

Mycoplasmal antigens activate the immune system and stimulate the inflammatory and oxidative cascade resulting in pathologic changes and hence wide spread serofibrinous inflammatory reaction and fluid exudation in the lungs, pleura, thorax and sometimes heart, liver, and kidneys. These pathological alterations lead to fibrin deposition in the pleural cavity, fluid exudation and hydrothorax [22].

Clinical signs

Contagious caprine plueropneumonia can be manifested in per-acute, acute or chronic forms. In per-acute form, death is sudden usually within 24-72h [29]. Contagious caprine plueropneumonia 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, and frequent and violent productive cough are prominent. Lethargy, lagging behind the flock, lying down, anorexia and abortions in pregnant goats are also noticed [30, 31].

Continuous nasal discharge that is initially serofibrinous straw-colored exudate followed by thick mucoid or purulent and rust-colored discharge might be observed [30]. 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 drip from their mouth and mucopurulent discharge obstructs their nostrils. The tongue may protrude and they bleat distressingly. Finally, death can occur within 7 to 10 days after the appearance of the signs but can be as fast as 2 days [31].

Pathological lesions

Mccp causes lesions specifically in the thoracic cavity [32] and fibrinous pleuropneumonia is the typical gross pathological lesion observed at necropsy [12, 33]. Macroscopic lesions of pleuropneumonia are often unilateral [34], with rare cases involving both lungs or an entire lobe may become consolidated [35]. Lesions in classical CCPP are confined to the thoracic cavity. Pea- sized yellowish nodules are seen in the lungs in early cases, whereas in more established cases there is marked congestion around the nodules [34]. The pulmonary pleurae become thickened, sometimes covered by a deposit of fibrin and pleural adhesion to the thorax is observed [36- 38]. Lung hepatisation, serofibrinous, pleuritis, accumulation of straw-colored pleural fluid in the pleural cavity and a varying degree of lung consolidation or necrosis with marble appearance is common [39- 41]. In addition, bronchial and mediastinal lymph nodes are swollen, edematous with areas of congestion in acute cases. In some circumstances, pericardial sacs are filled with a sero-haemorragic fluid. The liver and kidneys are enlarged with hemorrhages and diffused necrotic foci [42]. In severe and advanced cases tracheal congestion and in some

cases, hepatisation and abscessation of the lungs [38], as a consequence of secondary bacterial infection are encountered [43].

Histological examination of the lung tissues has shown acute serofibrinous to chronic fibrino-necrotic pleuropneumonia with serofribinous fluids and inflammatory cells (dominated by neutrophils) in the alveoli, bronchioles, interstitial septae and sub-pleural connective tissue [23, 44]. Pulmonary fibrosis and peribronchiolar mononuclear cuffing have also been observed [45]. Intralobular oedema is more prominent, but interlobular oedema has also occasionally been reported. Peribronchial and peribronchiolar lymphoid hyperplasia with mononuclear cell infiltration is also described [23, 46]

Diagnostic tests

Confirmatory diagnosis is based on the isolation of *Mccp* from clinical samples of the lung [23]. The ideal sample for *Mccp*isolation is a pleural fluid obtained from a recently slaughtered or live-infected goat [47]. Unlike the true CCPP caused by *Mccp*, other *Mycoplasma* infections can spread beyond the thoracic cavity [40]. In the laboratory, the major problem in *Mccp*isolation is its slow growth and frequent contamination of the culture by other *Mycoplasma*s [23, 47]. Under an ordinary microscope, the organism has a branching, filamentous morphology in exudates, impression smears or tissue sections, while other caprine *Mycoplasma*s usually appear as short filamentous organisms [27]. *Mccp* and other members of the *Mycoplasma* mycoides cluster cross-react in the serological test and share biochemical and genetic similarities, so biochemical and growth inhibition tests are not reliable and specific [27, 48]. The best and most accurate diagnostic method is molecular typing of *Mccp* [49].

Culturing

A number of media have been used for the general growth and isolation of *Mycoplasma*. *Mycoplasma* agar and broth media (Oxoid; Sigma), are used for the selective isolation of *Mycoplasma* spp. An agar non-selective media under the product code name CC1A (Mycoplasma Experience Ltd. Product), is available that allows the development of *Mccp* as red colonies over seven days of incubation (MEPG **online**). *Mccp* have been successfully grown and isolated from infected lungs through culturing on Hayflick medium broth (H25P) by Noah *et al.* [18], Balikci *et al.* [50] and Cetinkaya *et al.* [51]. Similarly modified Hayflicks media have been used for the growth and isolation of *Mccp* organisms [52]. Other than *Mccp* (five to seven days *invitro* growth), all *Mycoplasma mycoides* cluster members grow within 24–48 h *in vitro*, producing colonies 1–3mm in diameter [47].

Biochemical tests

For preliminary screening, a limited number of biochemical tests are available based on the nutritional capabilities of *Mccp* or specific enzyme activities [18]. Digitonin sensitivity distinguishes *Mycoplasma*s from acholeplasmas, and serum digestion distinguishes members of the *Mycoplasma mycoides* cluster from all other small ruminant *Mycoplasma*s [53]. Phosphatase production separates *Mcc* from other members of the Mycoides cluster, while metabolic differences (such as maltose positive reaction for *Mccp*) allow differentiation between *Mcc* and *Mccp*[54].The inter species variation in some biochemical reactions is often remarkable, rendering their application value less [55, 56]. The lack of arginine catabolism in *Mccp* may help to differentiate it from *Mcc* [18], but in some strains of *Mcc* arginine catabolism is reported to be lacking or very difficult to detect[57, 58].

Serological tests

Quite a few serological tests are available that are used in the field for the confirmatory diagnosis of CCPP. Indirect haemagglutination (IHA) and complement fixation tests (CFT) are used to assay the antibody response of goat to *Mccp*[59]. The CFT used for the detection of CCPP [60, 61], is more specific, though less sensitive than the IHA [59, 62]. The IHA specificity for the *Mycoplasma mycoides* cluster has been evaluated and the results were found to show cross-reactivity between these organisms [59, 63, 64]. The latex agglutination test which detects serum antibodies in CCPP-infected goats is more sensitive than CFT and can be performed in field conditions using whole blood or undiluted serum with a prompt result [65]. An indirect enzyme-linked immunosorbent assay (ELISA) has been developed to screen goat serum at a single dilution of antibody to *Mccp* [66]. The specificity and suitability of ELISA for large-scale testing make it an appropriate tool for epidemiological investigation of CCPP [40]. Direct antigen detection and blocking ELISA detects antibodies in the serum of naturally or artificially CCPP-infected goats [66]. Direct and indirect fluorescent antibody tests are the simple, reliable and rapid serological methods applied to clinical samples for the identification of most *Mycoplasma*s [67].Among many, the indirect fluorescent antibody (IFA) test is the most commonly used and is applied to unfixed *Mycoplasma* colonies on agar [40].

The growth inhibition test (GIT) is the least sensitive and simplest of the tests available for CCPP diagnosis [40]. It depends on the direct inhibition of *Mycoplasma* growth on sol- id media by specific hyper immune serum, and detects primary surface antigens [68, 69]. 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 [40].

Molecular diagnostic tests

Until recently, isolation was the only way to confirm the presence of CCPP. A DNA probe which differentiates *Mccp* from other members of the *Mycoplasma mycoides* cluster was developed [70]. PCR-based diagnostic systems are used for the rapid detection, identification and differentiation of the *Mycoplasma mycoides* cluster members to the serovar and strain level [70]. Sequencing of the gene for 16S ribosomal RNA has also been used to develop a PCR-based test where the final identification of *Mccp* is made depending on the pattern of the products after digestion of the PCR product with the restriction enzyme Pst1 [71,72]. Species identification based on PCR of the 16S rRNA genes and restriction at positions where unique differences occur between the two operons has been demonstrated previously for *Mccp* [71]. An improved resolution method, MLSA (multi-locus sequence analysis) based on the analysis of several genetic markers has also been used for the identification of *Mccp* [52]. Sequence-based genotyping methods for bacterial typing are technically simple, objective oriented and portable [73]; more over they allow direct amplification and sequencing of the organism from clinical material [52].

Treatment and prophylaxis

The duration of the disease varies according to the environmental circumstances [27], however, the infected goat can survive for more than one month or even recover if placed in good rearing conditions coupled with proper treatment [47]. A number of antibiotics and vaccines have been mentioned in the treatment and control of CCPP. In one case report, streptomycin-treated goats suffering from natural and experimental CCPP recovered on the third day of treatment and became completely immune to reinfection with *Mccp*[74]. Anadministration of long acting oxytetracycline stopped morbidity and mortality, and controlled further CCPP spread

immediately [75]. Danofloxacin was found to be highly effective in the treatment of clinical CCPP in goats [76]. Commercially available vaccines such as Pulmovac and *Capri*doll (live) and CCPPV (killed) are produced in Turkey and Ethiopia, respectively. *Capri*vaxis an inactivated CCPP vaccine prepared from an *Mccp* strain by the Kenya Veterinary Vaccine Production Institute, Nairobi. The inactivated *Mycoplasma* strain F38-saponin vaccine in natural CCPP cases showed 100% protection [64].

Treatment

Successful treatment of CCPP varies with the affected site, the time course of the disease and the 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 a long period of time being proven as an 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 [22, 77, 78].

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 the spread of the disease across the region. Regulating animal movement in the infected and surrounding areas 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 [79].

Economic importance of CCPP

Goats are important commodities to a large segment of the world's population as a source of meat, milk, and skin. CCPP is a disease of major economic importance in Africa and Asia, posing a major constraint to goat production. The direct losses of the disease result from its high mortality, reduced milk and meat yield, cost of treatment, control, disease diagnosis and surveillance. Moreover, there are indirect losses due to the imposition of trade restrictions [80].

Status of CCPP 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 [20]. 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 [81]. 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 [82, 83]. 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 [20].

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*.[8] using more than twelve published articles indicated that the sero-prevalence of the disease to be 25.7% [25].

Contagious caprineplueropneumonia sero-prevalence reports of 13.2% [84]from Borena and Guji, 42.8% from Afar [78],31.2%inBorenaZone [85],18.1% from Gambella [86], 8.5% in Northern Amhara [25], 31.6% in Borena [87], 4.92% in Diredawa [88], 32.63% in Jijiga zone [17], 18.61 % in Southern Ethiopia [89], 15.5%inHammer and Benna-Tsemay districts of Southern Ethiopia [31] and 14% in Central Ethiopia [20] have been documented in Ethiopia. Different numbers of CCPP outbreaks have been also reported [90]. 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 [23].

Different sero- prevalence has been reported in goats and sheep by various authors as shown in (Table 1). In addition to the different sero-prevalence studies conducted, there have been monthly reports of disease outbreaks to the Ministry of Livestock and Fisheries (MoLF), and the annual outbreak reports from 2011-2015 obtained from Ministry of Livestock and Fisheries (MoLF) are shown in Table 2.

Table 1. Results of sero-prevalence studies done on CCPP in different parts of Ethiopia.

Study area by region	Study animal	Sample size	Prevalence	Author
Afar	Goats	64	29.7	[90]
Afar	Goats	329	22.5	[91]
Afar	Goats	352	38.6	[92]
Afar	Goats	224	47.3	[93]
Afar	Goats	1183	29.1	[94]
Afar	Sheep	21	47.6	[92]
Amhara	Goats	400	8.5	[25]
Dire Dawa	Goats	200	44.5	[93]
Dire Dawa	Goats	244	4.9	[88]
Dire Dawa	Goats	319	6	[95]
Gambella	Goats	1152	18.1	[96]
Oromia	Goats	789	31.2	[97]
Oromia	Goats	900	13.2	[98]
Oromia	Goats	82	26.8	[90]
Oromia	Goats	81	34.6	[90]
Oromia	Goats	169	20.1	[42]
Oromia	Goats	280	51.8	[93]
Oromia	Goats	510	31.6	[99]
Oromia	Goats	200	51.5	[100]
Oromia	Goats	273	24	[101]
Oromia	Sheep	14	7.1	[42]
Oromia	Sheep	28	13	[101]
SNNPR	Goats	73	32.9	[90]
SNNPR	Goats	234	27.8	[89]
SNNPR	Goats	679	15.5	[89]
SNNPR	Goats	679	15.5	[31]
Somali	Goats	334	32.6	[17]
Tigray	Goats	280	43.9	[92]
Tigray	Goats	231	10	[92]
Tigray	Sheep	27	40.7	[92]
Tigray	Sheep	89	4.5	[92]

Table 2. The annual CCPP outbreak reports from 2011-2015 from different parts of Ethiopia.

Year	No. of outbreak	No of case	Death	Slaughtered	No. of animal at risk
2011	23	2327	876	74	243129
2012	28	17924	1659	416	342196
2013	21	3024	538	82	617608
2014	10	575	91	53	126863
2015	1	100	10	0	30000
Total	83	23950	3174	625	1359796

Source: [102]

Conclusion and Recommendations

CCPP is an 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 controlling 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 the hepatized lung are samples of choice. Currently, PCR is a novel diagnostic method because of high specificity and sensitivity, but also because of difficulties in culturing of Mccp. Of several serological tests used complement fixation (CFT) is the 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 the epidemiology of the disease is also not determined.

Therefore, based on the above review the following recommendations are forwarded;

- Small ruminant-rearing countries should promote further research focusing on the understanding of its geospatial epidemiological status to design a feasible control strategy.
- Improvement in the production of vaccine and vaccination coverage
- Development of new diagnostic tools
- Awareness creation about CCPP, transmission pathway, prevention and control methods, for small ruminant farmers
- Promotion of regional and international collaborations for the development of efficient control strategies.

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