

CLINICO-PATHOLOGICAL STUDY OF CONTAGIOUS CAPRINE PLEUROPNEUMONIA (CCPP) IN SMALL RUMINANTS

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ABSTRACT

This study was conducted to determine clinico-pathological manifestation of contagious Caprine pleuropneumonia (CCPP) in field outbreak in Kyber Pakhtunkhwa. A total of 120 samples were collected from goats exhibiting clinical signs of pneumonia suspected for CCPP during disease out break in field in different regions [northern (NR), central (CR) and southern(SR)] of Khyber Pakhtunkhwa Pakistan. All physical and clinical parameters of animals were examined and detail observations were documented. The predominant clinical findings include pyrexia (NR 80%, CN 83%, SR 69%), cough (NR 78%, CN 70%, SR 72%), excessive lacremation (NR 70%, CN 72%, SR 69%), nasal discharges catarrhal initially turned into mucopurulent in the advance stage, unilateral and bilateral conjunctivitis with corneal opacity, dysponia, abduction of the elbow and diarrhea. The majority (83 %) of animals presented pathological lesions in the form of consolidation (NR 83%, CN 87%, and SR 79%) and marbled appearance of lungs with fibrinopurulent membrane on pleural surface. Straw colored pleural fluid was present in pleural cavity with pleural adhesion (NR 39%, CN 43%, SR 51%), hydro pericardial fluid in pericardial sac, necrotic foci on surface of the liver and pus in the pelvis of kidneys. Microscopic lesions characteristic of CCPP i.e., pulmonary emphysema, atelectasis and thickening of interlobular septa were found in almost all cases. Widespread lesions in different organs of the infected animals and the septicemic nature of the disease suggest *Mycoplasma mycoides Capri (Mmc)* is the causative agent of CCPP.

Key words: Contagious Caprine pleuropneumonia, *Mycoplasma mycoides Capri*, clinico-pathological, Histopathological lesions.

INTRODUCTION

Livestock sector contributes 11.33 percent to national GDP and its share in agriculture is approximately 51.8 percent which is more than the crop sector (Economic survey, 2008). Among livestock sector goat population play a prominent role as it contributed largest number 58.3 million to the total animal populations of 146.6 million (Economic survey, 2008). Goats play important role to fulfill requirements like milk, meat, skin and leather. Livestock holders belong to lower socioeconomic class and their livelihood depends on income generation from animals. Goat is also called poor man cow. However, Goat population faces various challenges in the form of intense harsh climatic condition, poor management, scarcity of fodder and disease. Among various diseases, contagious Caprine pleuropneumonia (CCPP) is major threat to goat population. CCPP is wide spread in Pakistan and causing heavy losses to goat population (Awan, 1985, Rahman *et al.*, 2003 and Awan *et al.*, 2009).

CCPP is characterized by high morbidity and mortality. In field conditions of disease, morbidity may reach to 100% whereas mortality is 60-70% (Kaliner and MacOwan, 1976, Msami *et al.*, 2001). Typical cases of CCPP are characterized by pyrexia (41-43°C) with respiratory sign. After episode of high fever, respiratory signs become more pronounced accompanied by increased nasal discharge and lacremation. Respiration is painful, because of violent and frequent productive coughing. Other signs include lameness, diarrhea, unable to move stand with abducted front legs, stiff neck and lie on ground with lateral recumbancy in advance stage of disease (OIE, 2008).

An accurate and reliable diagnostic technique is essentially required for rapid detection and confirmation of infected animal (Rurangirwa *et al.*, 1987). Confirmation of the disease is difficult for reasons that, from clinical point of view, CCPP can not be differentiated from a number of diseases presenting similar respiratory signs in small ruminants, such as Peste Des Petits Ruminants and Pasteurellosis. Secondly being a fastidious organism it is very difficult to isolate

Mycoplasma on ordinary media in vitro. Consequently, isolation trials are usually failed. It has been observed that a negative result of cultivation of Mycoplasma does not indicate the absence of infection (Thiaucourt and Bölske, 1996). However, once isolated, it is very difficult to identify a particular strain due to common antigenic epitopes. Similarly cross reactions are usually noticed among different species on serological examinations (Thiaucourt *et al.*, 1994 and Rurangirwa *et al.*, 1997). However the gross and histopathological manifestation of the diseased animal provide adequate information for diagnosis of the etiological agents.

Diagnosis of CCPP is practiced in Pakistan by common conventional serological and biochemical test and very less data is available on histopathological diagnosis. To have an insight on accurate and rapid identification, present work was planned, to document details clinical signs, gross and histopathological lesions.

MATERIALS & METHODS

Sample collection: A total of 120 samples were collected from goats exhibiting clinical signs of pneumonia suspected for CCPP during disease out break in field in different regions (northern, central and southern) of Khyber Pakhtunkhwa Pakistan. All physical and clinical parameters of animals were examined and detail observations were documented. Clinical parameters including temperature, coughing, nasal discharge, lacrimation, breathing, diarrhea and posture of animals were recorded. Postmortem was performed on dead animals during disease out break and lesions in different visceral organs were noted. Samples for histopathological study were taken from trachea, lungs, heart, liver, kidney, spleen and intestine and preserved in 10% buffered formalin.

Tissue samples collected were packed, labeled, and transported to laboratory in departments of Pathology and Microbiology, University of Veterinary and Animal Sciences, Lahore, Pakistan.

Histopathological examination: On necropsy, of animal samples were collected from the organs including lungs, trachea, heart, liver, spleen, kidneys and intestine and fixed in 10% buffered formalin. The samples were processed for histopathological examination according to the standard procedure as described by Jalees *et al.*, 2010.

RESULTS

During field study of natural infection of CCPP majority of the animals exhibited common signs with elevated temperature ranging from 105-107°F acute form. There was nasal discharge catarrhal initially and turned to mucopurulent in later stage of disease. Excessive lacrimation, unilateral / bilateral conjunctivitis with

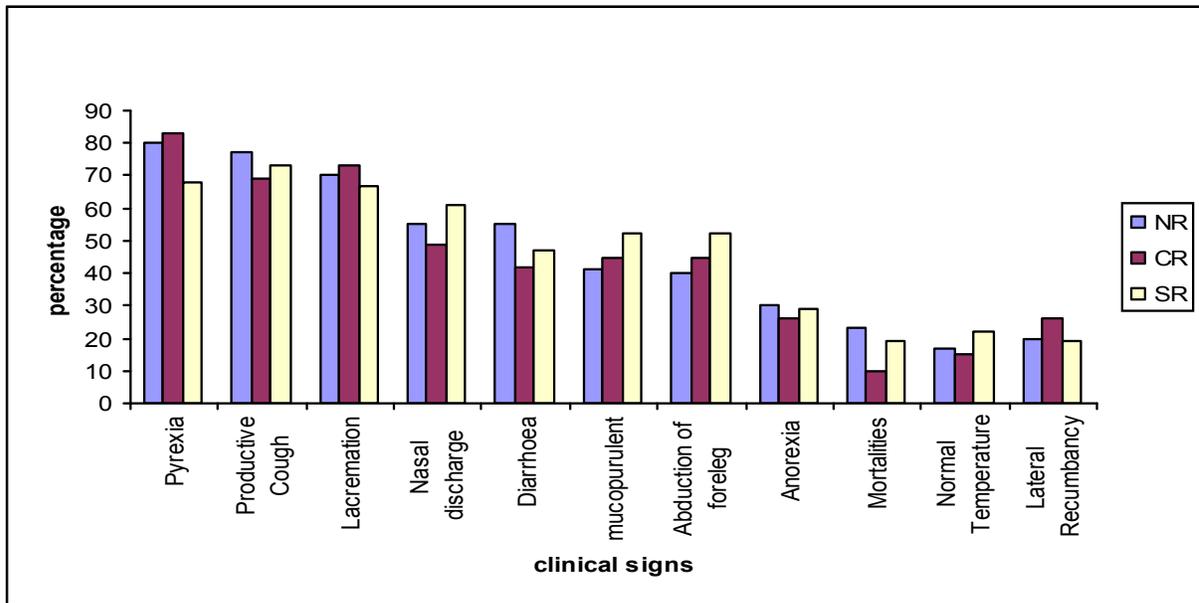
corneal opacity, painful cough, dyspnea, weakness, reluctant movement, extended neck and abduction of elbow. No joint inflammation was recorded in any animal through out the study (Fig. 1). A typical pneumonic sound was observed on auscultation of the thoracic cavity. Animals were slightly anorexic and diarrhea was frequently found. In advance stage of disease the temperature was almost normal or sub normal and the animals were found lie on the lateral recumbancy. On clinical examination the mucous membranes of most of animals were pale in color. Nervous sign were also recorded. Mortality was high in young than old animals.

Gross pathological lesions on postmortem examination revealed reddening, consolidation and marbled appearance of lungs. Fibrinopurulent membrane was present on pleural surface. Straw color pleural fluid with pleural adhesion was present. Mediastinal and bronchial lymph node were enlarged. Lesions in lungs were either unilateral or bilateral. Lesions in most of examined animals were limited to apical and middle lobe. However, in few animals proximal portion of caudal lobe was also involved. On cut frothy and purulent exudates was found with in the air ways of the lungs. Trachea showed mild hemorrhages with purulent exudates accumulated in its lumen. Heart was found of normal in size and yellow tinged fibrinous fluid was accumulated in the pericardial sac of most of animals. Kidneys were normal in size but slightly congested and necrotic foci's were present in few cases. On giving incision pus was present in the pelvis region. Liver was pale in color with hemorrhages and multi focal necrotic foci. Mild hemorrhages were found on the mucosal surface of the intestine of animals suffering from diarrhea with enlarged mesenteric lymph nodes. Distribution of gross lesions in different organs of surveyed animals are presented in (Table 1)

Lung sections of animals showed emphysema and atelectasis with interstitial and bronchopneumonia. The lining epithelium of bronchi and alveoli were disrupted and the interlobular septa were thickened with extensive infiltration of polymorph nucleated cells. Hemorrhages, congestions and necrotic area in the form of necrotic pneumonia surrounded by pyogenic band or by granulation tissue with scattered inflammatory cells were also found. Fibrin of different concentration was present in alveoli. Micro thrombi were observed in the small blood vessel within perivascular cuffing of phagocytic cells. The lining ciliated epithelium of trachea was eroded with leukocytic infiltration. Marked edema and hemorrhages was present in muscular layer with hypertrophy of secretory glands. There was glomerulonephritis, discrete hemorrhages and leukocytic infiltrations. Cast of varying degree was present in the urinary tubules. Section of liver showed areas of congestion and necrosis around the central veins. The necrotic focal area was surrounded by polymorph

nucleated cells. Spleen represents multi focal purulent area uniformly distributed through out the parenchyma mainly on the peripheral area. A marked area of congestion and hemorrhages with extensive leukocytic

infiltration was also found. Lining epithelium of intestine was sloughed off with hemorrhages and infiltrated with leukocytic cells. Lesions were more prominent in diarrhetic animals.



NR: Northern Region, SR: Southren Region, CR: Central Region

Fig. 1. Distributions of clinical signs in goats (in percent) in natural out-break of CCPP across different regions of NWFP, Pakistan.

Table 1. Distribution of gross lesions (in percent) in goats suffering from CCPP in natural outbreak across different regions of NWFP

Organ	Lesions	Northern Region %	Central Region %	Southern Region %
Lungs	Bilateral infection	65	69	73
	Unilateral infection	35	31	37
	Consolidation	83	87	79
	Pleural Adhesion	39	43	51
	Abscess	32	39	41
	Hemorrhages	24	21	17
	Pleural fluid	41	33	29
	Trachea	Congestion	40	47
	Pus	39	41	37
Lymph nodes	Enlarged medistinal Lymph Node	80	83	81
	Mesenteric Lymph Node	57	47	51
Heart	Pericardial fluid	49	45	51
Liver	Hemorrhages	33	29	31
	Necrotic Foci	31	25	27
Kidney	Congestion	25	31	29
	Pus	55	59	47
Intestine	Congestion	50	46	47

Table 2. Distribution of Microscopic lesions (in percent) in goat infected with CCPP in field outbreak across different regions of NWFP

Organ	Lesions	Northern Region	Central Region	Southern Region
		%	%	%
Lungs	Emphysema	86	79	77
	Atelactasis	83	81	79
	Interstitial Pneumonia	60	67	63
	Thickening of Alveolar septa	80	70	67
	Leukocytic infiltration			
	granuloma	85	81	83
	Fibrosis	39	45	47
Trachea	Micro thrombi	23	27	26
		17	13	9
	Sloughing of epithelium	59	54	49
Heart	Leukocytic infiltration	62	58	56
	Hemorrhages	49	47	43
Liver	Hemorrhages	39	35	41
	Leukocytic infiltration	55	49	51
Kidney	Hemorrhages	36	45	33
	Focal Necrosis	37	31	29
	Leukocytic infiltration	69	55	39
Intestine	Congestion	33	29	30
	Cost in urinary tubules	25	26	19
Spleen	Sloughing of epithelium	49	39	42
	Hemorrhages	22	26	31
	Leukocytic infiltration	55	49	46
Spleen	HemorrhagesLeukocytic infiltration	20	22	10
		22	26	15

DISCUSSION

The clinical signs and symptoms exhibited by the animal during CCPP field outbreak varied differently in different part of the world (Nicholas *et al.*, 2008). This variation in signs and symptoms is due to the fact that CCPP is caused by Mycoplasma clusters having different species and sub species which ultimately contributing in the development of different lesions. The clinical picture of disease is further complicated by involvement of secondary infection like virus and bacteria particularly the pasteurilla (Nicholas *et al.*, 2008). The classical signs of CCPP are characterized by high fever, nasal discharged, lacremation, difficult breathing, forceful cough and ultimately death (OIE 2008, Nayak and Bhowmik, 1991 and Real *et al.*, 1994). In the present study similar findings were observed.

The signs exhibited by diseased animals during field survey were characterized by high fever, nasal discharge, lacremation, painful cough, dyspnea and diarrhea. A high proportion of disease was observed in the northern region of KPK which might be due to intense climatic condition and nomadic nature of the goat's owner. The nomads plays vital role in spreading of the disease to the area from neighboring country

Afghanistan through trans-boundary transmission. The intense climatic condition accompanied by long traveling causes stress and decreases the immune status of the animal. This make the animal more vulnerable to disease, similar observation was made by Gelagy *et al.*, (2007) who demonstrated that pastoralism and season are the efficient means of spreading of CCPP disease. In the present study the disease was not confined to the respiratory tract but along with the respiratory signs the diarrhea was also present. This difference in observation with previous study is due to the difference in species of mycoplasma that causes CCPP. In the former study the cause was Mycoplasma capriculum capripneumoniae (Mccp) while in the present study the species isolated from natural outbreak was confirmed as Mycoplasma mycoides Capri (Mmc). The Mycoplasma mycoides capri tends to cause a more generalized infection as compare to other species of Mycoplasma cluster. This statement is justified by the findings of Laura *et al.*, 2006: Mondal *et al.*, 2004 and Nicholas *et al.*, 2008.

Although the Mmc infection causes systemic form of disease but in the present study no involvement of joints and mammary glands were observed in any animals. Similar observations were also made by Laura *et al.*, (2006). In India it was reported by Mondal *et al.*, (2004) that joints inflammations were frequently

observed in CCPP field outbreak particularly the young kids were severely affected. The involvement of joint would be probably due to MmLC infection. The statement is justified by the observation of (Cottew *et al.*, 1987 and DeMassa *et al.*, 1992). According to their observation the MmLC causes arthritis and mastitis in goats. The systemic involvement of different organs accompanied by high fever suggested the septicemic nature of disease as supported by findings of (Rodriguez *et al.*, 1996).

The presence of gross lesion in different organs of CCPP infected animals dependent on the species of Mycoplasma clusters. In general the gross lesions on postmortem are characterized by inflamed lungs with marble appearance, fibrinous pleura with consolidation and hepatization. Straw colored fluid is accumulated in the pleura with pleural adhesion in the chronic cases (Nicholas *et al.*, 2004 and OIE, 2008). The lesions of classical CCPP caused by Mycoplasma capricolum capripneumoniae are limited to thoracic cavity (Wesonga *et al.*, 2004 and OIE, 2008). In the present study wide spread lesions were observed in majority of examined goats suffering from CCPP in field outbreak. The systemic infection of various organs suggests the cause of disease might be Mmc. As the Mycoplasma mycoides capri causes systemic infection. The statement is supported and justified by findings of (Mondal *et al.*, 2004 and Laura *et al.*, 2006 and Rodriguez *et al.*, 1996). There was fibrous pleuropneumonia combined with fibrosis, pleural adhesion, and mucopurulent exudates in trachea. These lesions presented the chronic nature of the disease. Chronic natures of lesions are compatible with poor resolution of pneumonia from Mycoplasma mycoides Capri infection. Similar finding were also reported by Wesonga *et al.*, (2004). Who reported that CCPP infection usually undergoes chronic course of disease with fetal results. However the recovered animals become carrier and serve constant source of spreading of disease. Both bilateral and unilateral involvements of lungs were founded in the present study. However, bilateral occurrence was more 69% as compared to unilateral 38 %. Findings of these lesions were contrast to the findings of (Rodriguez *et al.*, 1996 and Wesonga *et al.*, 2004). Who reported that in Mycoplasma capricolum capripneumoniae infection unilateral involvement of lungs were more as compared to bilateral. This might be due to the difference in etiological species of Mycoplasma. Straw color pleural fluid was present in almost 50% of the examined animals. The pleural fluid was yellow tinged in appearance with watery in nature. Such observations were also made by Laura *et al.*, (2006). Who reported outbreak of CCPP in Mexico caused by Mycoplasma mycoides capri. The lesion in pleura and pleuritis suggested the tropism of mycoplasma infection to these tissues.

The lesions in the lungs were limited to apical and middle lobes. However, in some animals the caudal lobes were also get infected. These observations are matched with the findings of (OIE 2008 and Nicholas *et al.*, 2004). Different species of Mycoplasma clusters have tissue tropism and developed lesions in different organs (Nicholas *et al.*, 2008). In CCPP infection caused by Mccp the histopathological lesions are confined to respiratory tract only (Wesonga *et al.*, 2004). However, in the present study the lesions were present in almost all visceral organs. This wide spread lesion in different organ is due to Mmc infection. The statement is supported by findings of Rodriguez *et al.*, 1996, Mondal *et al.*, 2004 and Laura *et al.*, 2006. Tracheal lesions were characterized by erosive inflammation of lining epithelial layer with hyperactive mucous secreting cell and hemorrhages in submucosal layer with leukocytic infiltration. It has been proved in vitro that growth of Mycoplasma is closely associated with tracheal tissues. The bacteria lodged in the respiratory epithelium, persisted for some time and than ultimately get multiplied. It was also established that peroxide free radicals are produced by Mmc within the tracheal tissue which is an important factor in pathogenesis of CCPP (Cherry and Robinson, 1974).

Marked vascular response was recorded in the form of congestion, zones of hemorrhages with perivascular cuffing of leukocytes. Microthrombosis was present in 13% of sections in small capillaries and blood vessels. Similar observations were made by Gutierrez *et al.*, (1999) who reported that Mmc infection causes diffuse intravascular thrombosis and significant losses of thrombocyte. Secondly it also causes endothelial injury resulting in thrombosis. The histopathological lesions were not confined to respiratory tract only. Diffuse hemorrhages and lesions were also observed in liver, spleen, kidneys and intestine. The wide spread lesion in different organs represents systemic nature of disease. These findings are an agreement with finding of (Mondal *et al.*, 2004 and Nicholas *et al.*, 2008) who reported that Mmc causes more generalized infection. In a study, it was observed that Mmc causes acute form of disease with diffuse interstitial bronchopneumonia and purulent nephritis (Gutierrez *et al.*, 1999). However, these observations are contrary to findings of Wesonga *et al.*, (2004) and OIE (2008) who reported that lesions of CCPP are confined to the thoracic cavity only. This variation in findings is due to differences of species of Mycoplasma clusters that cause CCPP.

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