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# COMPARATIVE STUDY ON GROSS AND HISTOPATHOLOGY IN GOAT TO EXPERIMENTAL INFECTION WITH FIELD ISOLATES OF *MYCOPLASMA*

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**A B S T R A C T**

Contagious Caprine Pleuropneumonia (CCPP) is a complex respiratory syndrome causing high morbidity and mortality in goats resulting in substantial economic losses in the goat farming. A total of 20 healthy goats were randomly divided into three group's i-e, (A, B, and C). Each A and B group included 08 animals, while group C contained 04 animals. The animals in group A of about one year of age (with milk teeth), while group B was about two years of age (two permanent incisors). Group C had two animals from each age group. Groups A and B were infected by the intra-tracheal route at the dose of $3\times10^7$ CFUs of field isolated *Mycoplasma capricolum* subspecies *capripneumoniae* (Mccp) as described by Wesonga et al. (2004) with slight modification. At the same time, the goats in control group C was kept away from the infected animals. Necropsies were performed on animals dying during the course of infection. Among the surviving animals, two goats from group A and B was slaughtered every ten days on days 10, 20, 30, and 40 days post-infection (dpi). Gross lesions in organs like lungs, and trachea were recorded. Tissue samples from the lungs and trachea were collected on days 10, 20, 30 and 40 post-infection (dpi) for histopathological examination. The tissue samples were fixed in 10% formalin, processed, sectioned, and stained with Hematoxylin and Eosin. The tissue slides were examined for any histopathological lesions. Results indicated that gross pathological changes were observed in the trachea and lungs of experimental infected CCPP goats. In trachea, congestion, thin fibrinous mucous plug in lumens, haemorrhage. Purulent exudate, enlarged mesenteric lymph nodes were observed. In lungs, consolidations, enlarged bronchial and medistinal lymph nodes, frothy and fibrinous exudates, thoracic cavities filled with straw-colored fluid, viscous straw colored fluid was found in pleural cavities, multifocal lesions of necrosis and abscess were prominent on lungs surface. Hepatization colours of lungs varying from deep blue to reddish in colour were also observed. Histopathological examination of trachea sections revealed severe CCPP infection. Group A & B plates showed the respiratory ciliated epithelial layer was erupted and erosion of lamina propria in later days of infection. Histopathological examination of lungs sections revealed that in group A, interalveolar capillaries were severely congested, the bronchioles were flooded with fibrinous or serofibrinous exudate, the bronchioles were narrowed by exudate, and the alveolar gaps were filled with fluid. In group B, contagious caprine pleuropneumonia with fibrous tissue mixed with inflammatory infiltration was observed.

**Keywords:** CCPP; Goats; *Mycoplasma;* Gross pathology; Histopathology

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## INTRODUCTION

Goats are valuable to the rural economy since they produce milk, meat, and skin. Given that farmers from lower socioeconomic classes rely on animal income for their livelihood, goats are often referred to as the "poor man's cow." But the goat population suffers several constraints,

including diseases, a lack of feed, inadequate management, and harsh weather conditions (Samiullah, 2013). Contagious caprine pleuropneumonia (CCPP), among other illnesses, poses a serious risk to the goat herd. In Pakistan, CCPP is pervasive and significantly damages goat populations (Awan et al., 2009; Rahman et al., 2003). This disease is widespread worldwide but is most common in nations with a large goat-raising industry (Rurangirwa and Kinyili, 2000). It is widespread throughout Europe, the Middle East, Africa, and the Indian subcontinent (Mondal et al., 2004). The World Organization for Animal Health has designated it as a list B disease (OIE, 2008).

Bacteria of the genus Mycoplasma are the CCPP-causing microorganisms in goats (Laura et al., 2006). *Mycoplasma capricolum* subspecies *capripneumoniae* is the pathogen responsible for the disease's classic form (Mcc). *Mycoplasma mycoides* subspecies capri (Mmc), a prevalent organism on the "subcontinent," is the culprit behind acute septicemic illness and CCPP (Mondal et al., 2004). Mycoplasmas are the tiniest self-replicating microorganisms, measuring between 100 to 250 nanometers. They are unique in that they lack a rigid cell wall. A lipoglycan-containing cell membrane encircles mycoplasma. Their genome is incredibly tiny, ranging from 0.58 to 2.20 Mbp (megabase pair). Due to its fragility, this organism has little chance of surviving in its natural habitat and is parasitic in nature, with stringent host and tissue specialization (Gelagay et al., 2007).

The most common pathological finding in CCPP is extensive lung consolidation, which is typically unilateral (Hussain et al., 2012). Young, immune weak animals frequently exhibit acute pathologic lesions, whereas resistant and healthy animals typically experience chronic lesions (OIE, 2017). The extracellular pathogens, known as mycoplasma, are believed to be on mucous membranes and can adhere to epithelial cells (Nicolet, 1996). Pathogen adhesion to host cells promotes colonization and infection setup. Mycoplasma’s metabolic processes produce free radicals, which can damage cell membranes and cilia. Similar to other mycoplasma species, these occurrences might be related (Tanaka et al., 2014). This characteristic may be significantly influenced by a type of capsule present in various mycoplasmas, notably the Mccp galactan (Nicolet, 1996). Immunity may be inhibited or suppressed by the antigens of Mccp stimulating immune cells. After stimulation, immune cells (macrophages and monocytes) produce pro-inflammatory cytokines like tumor necrosis factor alpha (TNF-a), interleukins, and interferon C are generated, but the exact mechanism for Mccp is still unknown (Totte et al., 2015). Along with this interaction and the production of cytokines are primarily mediated by mycoplasmal structures. It is thought that mycoplasma's biological membranes, particularly the plasma membrane, and their constituent lipids and lipoproteins cause the release of cytokines (Nicolet, 1996).

When examined under a microscope, the disease shows features of fibrino-purulent pleuropneumonia and catarrhal broncho-pneumonia, along with a thickening of the spaces between lobes of the lungs (Laura et al., 2006). Alveolar exudates, or fluids that escape into the alveoli, are primarily composed of macrophages, with some presence of neutrophils and occasional pulmonary fibrin deposits. Other notable microscopic alterations include fibrin deposits in alveoli, fibrous tissue formation around bronchi and within lung septa, larger fibrous strands, chronic fibrous inflammation of the pleura, lymphoid structures around the airways, inflammation of alveoli with predominantly mononuclear cells, bronchointerstitial pneumonia, and bronchial lymph nodes showing an increase in lymphoid tissue (Hussain et al., 2012; Sadique et al., 2012; Sheikh et al., 2016). Additional microscopic changes that have been noted include collapse or overinflation of the alveoli (atelectasis or emphysema), thickening of the septa between lobes, granulation tissue, infiltration of inflammatory cells, and deposition of proteinaceous material within alveoli, all of which are indicative of CCPP in affected goats (Parray et al., 2019).

Mycoplasma mycoides subspecies capri (PG3) infects goats and cause CCPP infection. Gross pathological and histopathological findings revealed that the prominent changes were seen in younger animals than older ones. Nevertheless, cELISA revealed that CCPP antibody titers were higher in older animals as compared to young ones (Ayaz et al., 2023). Seroprevalence of CCPP was found higher in older goats of age 2 years (23.7%) than young goats of age 1 and less than years (10 and 8 %) (Solangi et al., 2023).

Much of the existing research on contagious caprine pleuropneumonia (CCPP) has been centered around isolating and identifying the causative agent of the disease. However, there is an apparent necessity for more comprehensive studies. Such studies should observe and document the infection's progression closely and gather detailed information on how the disease develops in experimentally infected animals. This further research would contribute significantly to our understanding of the disease's pathogenesis, enhancing our ability to manage and treat it effectively. The present study was planned to give insight into the accurate and timely identification of CCPP in experimentally infected goats based on gross and

histopathological changes.

## MATERIALS AND METHODS

Twenty goats were purchased from the local market in Hyderabad. The animals were kept in the experimental animals' section of the Vaccine Production Unit, Central Veterinary Diagnostic Laboratory, Tandojam, under hygienic environmental conditions. Animals were kept for one week as an adaptation period and dewormed. The animals were feed green fodder and watered *ad libitum*.

### Experimental design

A total of 20 healthy goats were randomly divided into three groups i-e, (A, B, and C). Each A and B group included 08 animals, while group C contained 04 animals. The animals in group A of about one year of age (with milk teeth), while group B was about two years of age (two permanent incisors). Group C had two animals from each age group. Groups A and B were infected by the intra-tracheal route at the dose of $3\times10^7$ CFUs of field isolated *Mycoplasma capricolum* subspecies *capripneumoniae* (Mccp) as described by Wesonga et al. (2004) with slight modification. At the same time, the goats in control group C was kept away from the infected animals.

### Necropsy

Necropsies were performed on animals dying during the course of infection. Among the surviving animals, two goats from group A and B was slaughtered every ten days on days 10, 20, 30, and 40 days post-infection (dpi). Gross lesions in organs like lungs, and trachea were recorded.

### Histopathological examination

Tissue samples from the lungs and trachea were collected on days 10, 20, 30, and 40 days post-infection (dpi) for histopathological examination. The tissue samples were fixed in 10% formalin, processed, sectioned, and stained with Hematoxylin and Eosin. The tissue slides were examined for any histopathological lesions.

### Tissue processing

The fixed tissue samples of the lungs, trachea, and heart of necropsied goats were used for histopathological examination. The tissues were washed twice in phosphate-buffered saline (PBS). After that, the tissue samples were placed in labeled cassettes and loaded in an automatic tissue processor (Leedo. HS.566) pre-programmed for tissue processing, including dehydration for 60 minutes in each serial dilution of ethanol as 75, 85, 95, 95, 100 and 100 respectively.

After that, the tissues were cleared in pure xylene with two changes for 30 min each. Tissues were then infiltrated with melted paraffin wax at 65°C for 01 hour, 3 times (Table 1).

Table 1. Tissue Processing regents and time.

| S. No. | Process | Reagent | Reagent % Processing time | | | | |
|---|---|---|---|---|---|---|---|
| 1 | Washing | PBS Solution | 05 min | 05 min. | | | |
| 2 | Dehydration | Isopropyl alcohol (IPA) | 75% 1 hour | 85% 1 hour | 95% 1 hour | 100% 1 hour | 100% 1 hour |
| 3 | Clearing | Xylene | 30 min. | 30 min. | | | |
| 4 | Infiltration | Paraffin wax | 1 hour | 1 hour | | | |

### Embedding

Once the tissue processing was completed, each specimen was embedded in paraffin wax, heated to 65°C, utilizing steel embedding molds. This process was facilitated by an automatic embedding system, which ensured that the specimens were properly situated in their areas of interest. To solidify the blocks, these were then placed on a cooling plate.

### Sectioning and mounting

Five-micrometer slices were cut using a rotary microtome. They were then put in a water bath heated to 45°C. The stretched paraffin ribbons were placed onto slides in groups of two to three. These slides were dried in an oven set at 37°C for the whole night before staining.

### Staining

Paraffin-embedded thin tissue sections (5 µm) were stained automatically using an automatic tissue strainer, as described in Table 2.

### Mounting

A cover slip was put on top of the slide after a small quantity of DPX mounting media had been poured into it. After that, the slides were left to air dry at ambient temperature. The slides were then scrutinized under a microscope, first at lower magnifications and then progressively progressing to higher ones.

### Microscopic examinations

After mounting, dried sections on slides were observed under a light trinocular microscope attached to a digital camera. The photographs of the lungs, trachea, liver, and heart were captured, and tissue changes such as inflammation, leucocyte infiltration, deposition of exudate or mucus, and other changes were recorded.

Table 1. Hematoxylin and Eosin staining protocol.

| S. No. | Process | Reagent | Reagent % Processing time | | | | | |
|---|---|---|---|---|---|---|---|---|
| 1 | Dewaxing | Xylene | 10 min. | 10 min. | | | | |
| 2 | Rehydration | Isopropyl Alcohol | 100%<br>3 min. | 100%<br>3 min. | 95%<br>3 min. | 100%<br>3 min. | 85%<br>3 min. | 75%<br>3 min. |
| 3 | Washing | Tap water | 10 sec. | | | | | |
| 4 | Staining | Hematoxylin | 12 sec. | | | | | |
| 5 | Washing | Tap water | 05 sec. | | | | | |
| 6 | Differentiation | 0.5% Acid (HCl) Alcohol | 01 sec. | | | | | |
| 7 | Washing | Tap water | 05 sec. | | | | | |
| 8 | Bluing | Ammonia water (0.2%) | 01 min. | | | | | |
| 9 | Washing | Tap water | 05 sec. | | | | | |
| 10 | Dehydration | Isopropyl Alcohol | 75%<br>3 min. | 85%<br>3 min. | | | | |
| 11 | Staining | Eosin Y (0.25%) | 90 sec. | | | | | |
| 12 | Dehydration | Isopropyl Alcohol | 95%<br>3 min. | 9%<br>3 min. | 100%<br>3 min. | 100%<br>3 min. | | |
| 13 | Clearing | Xylene | 10 min. | 10 min. | | | | |

## RESULTS

### Gross pathology

Observations of gross pathology lesions were made to establish a baseline for the confirmed disease diagnosis. Dead animals in the field were subjected to a post-mortem examination to detect pathological alterations in the lungs and trachea.

### Trachea

Noticeable pathological alterations were found in the goats that were slaughtered. On the tenth day (Figure 1, 2, A1 & B2), the trachea showed minor congestion, with a thin fibrinous mucous plug in its lumens. There were slight congestion and fibrinous depositions on the pleural surfaces. The most severe changes, such as hemorrhage, were observed in the trachea's inner lining. A purulent exudate, a hallmark lesion of CCPP, was also identified in the trachea. Hemorrhaging streaks of varying degrees were found in the trachea's lumen. The trachea was hemorrhagic on day 20 (Figure 1, 2, A2 & B2). The animals killed on days 30 (Figure 1, 2, A3 & B3) and 40 (Figure 1, 2, A4 & B4) exhibited similar lesions as noted on days 10 and 20 but with less severity.

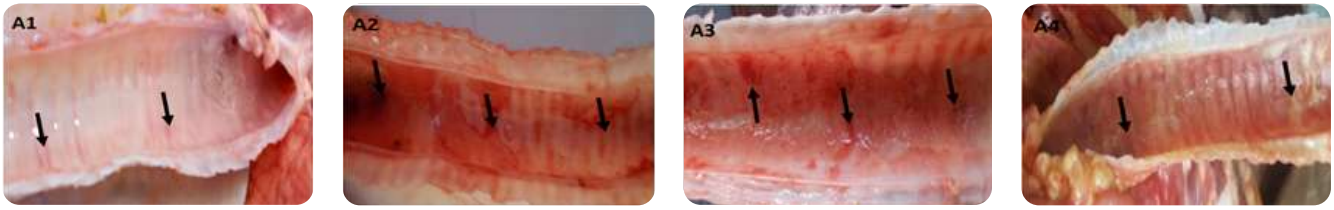


Figure 1. Trachea showed progressive pathological lesion in form of hyperemic changes in response to inflammation; minor in A1, mild to moderate in A2, moderate in A3 and Severe in A4 group of goats due to infection of CCPP.

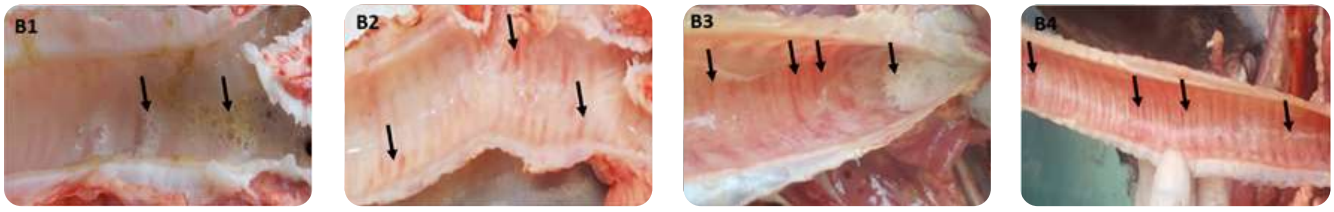


Figure 2. Trachea showed progressive pathological lesions in form of hyperemia and frothy changes in response to inflammation; mild in B1, moderate in B2, moderate to severe in B3 and Severe in B4 group of goats due to infection.

**Lungs**

In the goat slaughtered at the end of day 10 (Figure 3, 4, A1 & B1), lung consolidations were observed, particularly in the apical and intermediate lobes. Both bronchial and mediastinal lymph nodes showed slight enlargement. When the lungs were incised, frothy and fibrinous exudates were released. By day 20 (Figure 3, 4, A2 & B2), the thoracic cavities of both slaughtered animals were filled with straw-colored fluid. The lungs exhibited pronounced congestion and consolidation. Both mediastinal and bronchial lymph nodes were enlarged, and fibrinous coats were observed on the lung surface. On the 30th day (Figure 3, 4, A3 & B3), a post-mortem examination of two slaughtered goats revealed extensive lung involvement, showing consolidation and localized abscesses. A thick fibrinous layer was present on the lung surface, along with slight pleural adhesion. A viscous, straw-colored fluid was observed in the pleural cavities. By day 40 (Figure 3, 4, A4 & B4), the lung surfaces showed prominent multifocal necrotic lesions and abscesses. The lungs demonstrated varying shades of hepatization, from deep blue to reddish hues. The primary characteristic lesions of CCPP, such as unilateral and bilateral lung involvement with pulmonary consolidation and a marbled lung appearance, were also observed. It was found that the severity of CCPP infection was more remarkable in group A (Figure 2, A1, A2, A3, & A4) compared to group B (Figure 3, 4, B1, B2, B3 & B4).

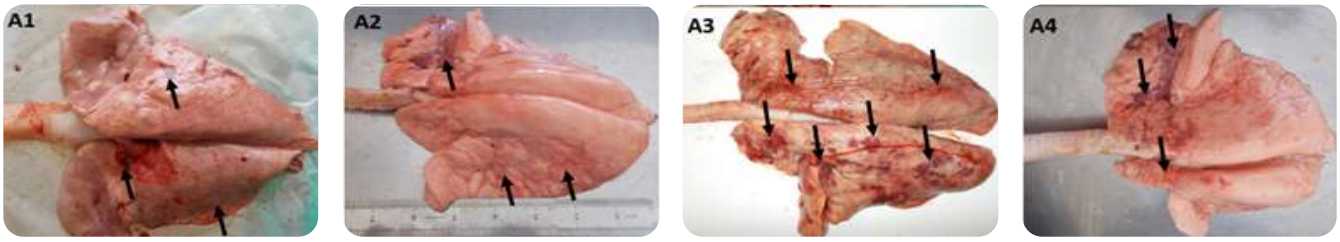


Figure 3. Lungs showed progressive gross pathological lesions in unilateral and then bilateral immersion of lobes of lungs; unilateral hyperemic lesions in group A1, bilateral defuse necrotic lesions in group A2, bilateral multifocal necrotic lesions in group A3, and bilateral multifocal to defuse lesions in group A4.

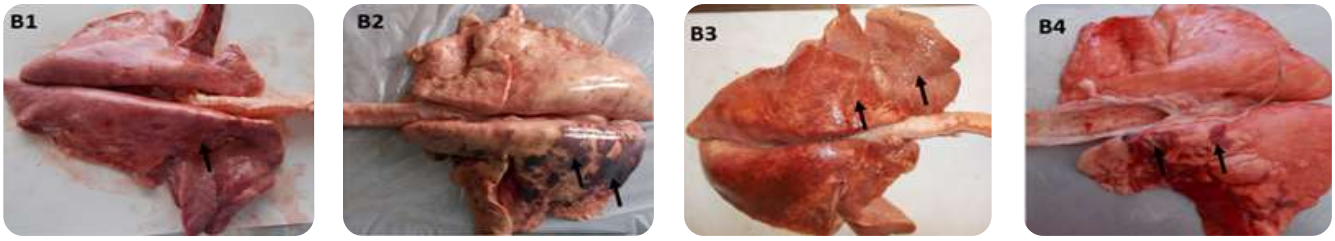


Figure 4. Lungs showed progressive gross pathological lesions in form of bilateral involvement of lungs; overall diffuse inflamed lungs in group B1, discoloration of lung and unilateral extensively defuse necrotic lesions in group B2, bilateral extensively diffused necrotic fibrosis lesions in group B3, and bilateral extensively inflamed with anteriorly multifocal necrotic lesions in group B4.

**Histopathology**

A histopathological examination of the trachea and lungs was performed to investigate histopathological lesions.

**Trachea**

Histopathological examination of the trachea was seriously affected. It revealed variations between the four different timelines of CCPP infection in the form of the respiratory epithelial layer sloughing, erupting, and lamina propria severely affected (Figure 5, 6, A3, A4, B3, and B4) as compared with control (Figure 7).

Histopathologic examination of trachea sections revealed severe CCPP infection. Group A & B plates showed that the gradually increase the respiratory ciliated epithelial layer erupted and erosion of lamina propria in the later days of infection (Figure 5, 6, A2, B2, A3, B3, A3, and B4). Histopathological lesions revealed that the due to CCPP infection trachea were mild to moderate changes in 1 years old goat, but trachea of 2 years old goats were severely infected due to CCPP infection as illustrated in the figure 5 and 6.

**Lungs**

Histopathological examination of the lungs was seriously affected and revealed fluctuations between the four different timelines of CCPP infection in the form of fibrinous broncho- and pleuropneumonia with a control (Figure 8).

In group A, interalveolar capillaries were severely congested, the bronchioles were flooded with fibrinous or

serofibrinous exudate, the bronchioles were narrowed by exudate, and the alveolar gaps were filled with fluid. Plate A1: Infectious caprine pleuropneumonia with thick interlobular septa punctuated by inflammatory cells and exudate-filled alveoli. Figure 9, A2 shows a serofibrinous effusion that has enlarged the visceral layer of the pleura and a fibrinous and inflammatory exudate within the bronchiolar lumen (arrowhead). Figure 9, A3 and A4, on the other hand, showed multifocal patches of caseous necrosis (arrowhead) contained in thick fibrous tissue capsules around the bronchioles and surrounded by a zone of inflammatory cells.

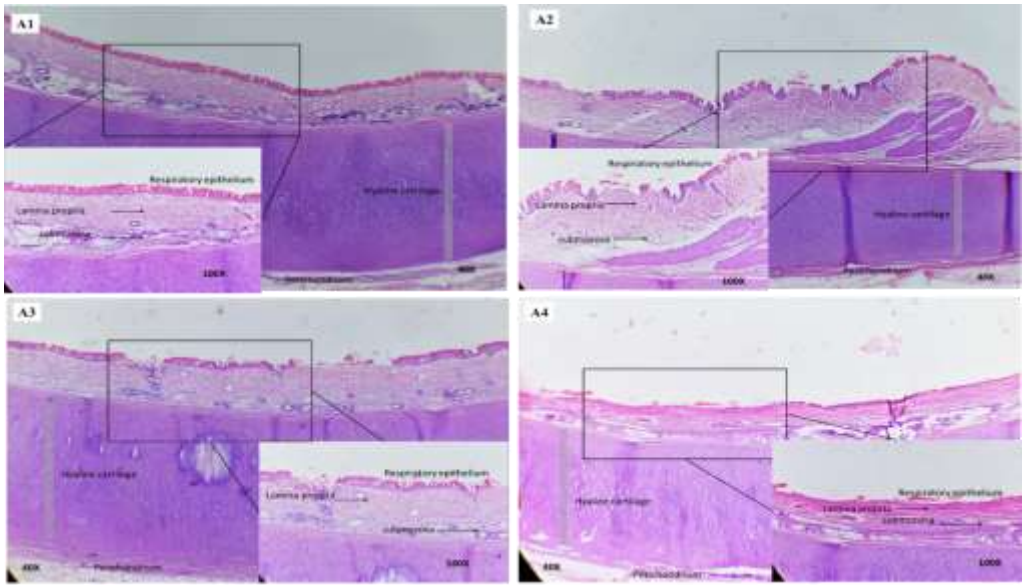


Figure 5. Histopathological finding of trachea infected with CCPP during various days; at 10th day, tracheal (A1) respiratory epithelial layer shown normal, at 20th day, trachea (A2) showed mild eruption of the respiratory epithelial layer, at 30th day, trachea (A3) showed moderate eruption and erosions of respiratory epithelial layer, at 40th day, trachea (A4) showed severe eruption of respiratory epithelial layer and erosion of lamina propria due to CCPP infection. H&E stain. Magnification 40X and 100X.

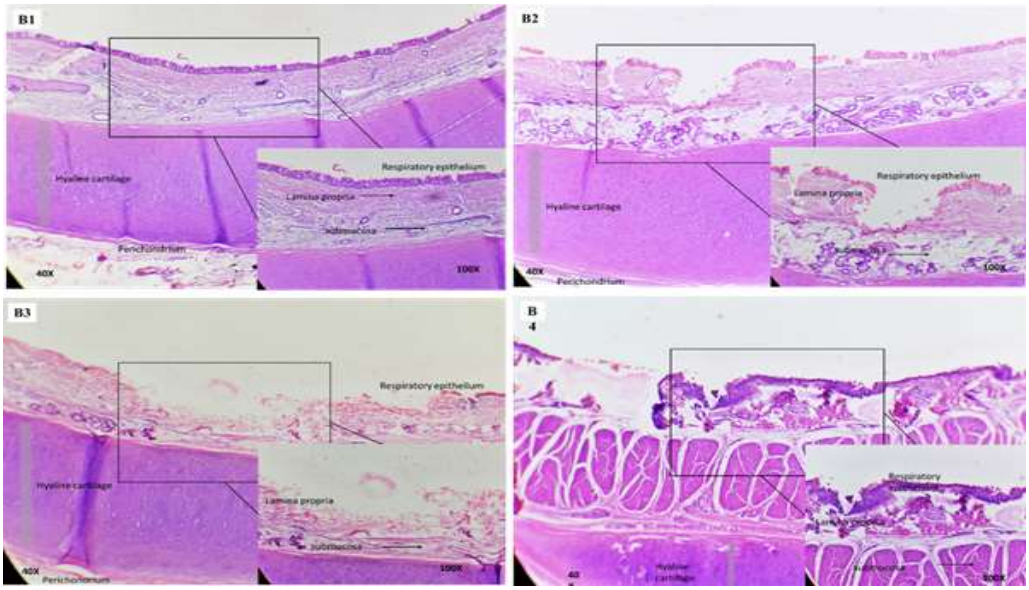


Figure 6. Histopathological finding of trachea infected with CCPP during various days; at 10th day, trachea (B1) showed respiratory epithelial layer was mildly erupted, at 20th day, trachea (B2) showed moderate eruption of the respiratory epithelial layer and lamina propria, at 30th day, trachea (B3) showed severe eruption and erosions of respiratory epithelial layer and lamina propria, at 40th day, trachea (B4) showed severely loss of respiratory epithelial layer and erosion of lamina propria due to CCPP infection.

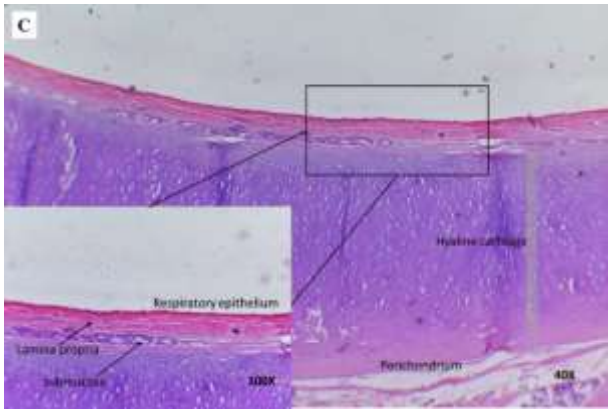


Figure 7. Histology of trachea; shows the epithelial lining, lamina propria, submucosa, and hyaline cartilage of trachea was seen normal in figure C. H&E stain. Magnification 40X and 100X.

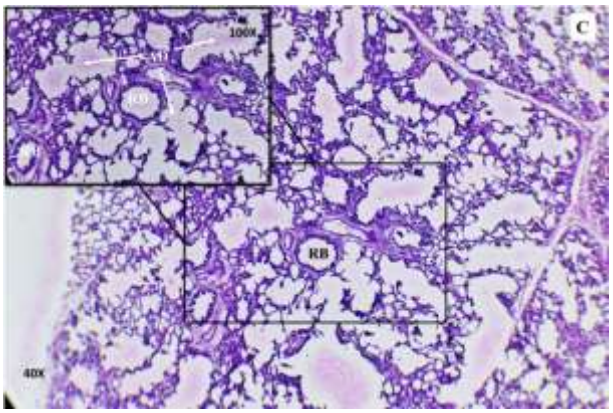


Figure 8. Histopathological photograph of lungs of goat; shows respiratory bronchiole (RB) and alveolar ducts (AD) of normal lungs was seen in figure C. H&E stain, Magnification X40 and X100.

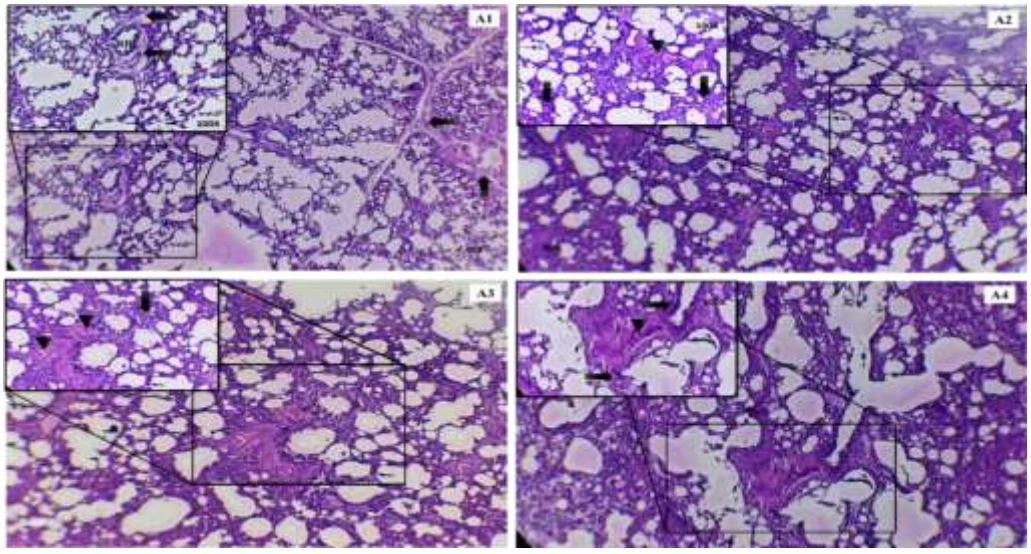


Figure 9. Histopathological photograph of lungs of goat infected with CCPP; shows inflammatory response and exudation (arrow) were seen in group A1, bronchiolar lumen showed serofibrinous (arrowhead) changes and thicken alveolar space (arrow) were seen in group A2, caseous necrosis (arrow) and thicken alveolar space (arrowhead) were seen in group A3, bronchiolar space was thick (arrow) due to caseous necrosis (arrowhead) were seen in group A4. H&E stain, magnification 40X and 100X.

In group B, contagious caprine pleuropneumonia with fibrous tissue mixed with inflammatory infiltration was observed. Serofibrinous exudate, clogged blood vessels, and inflammatory infiltrates caused the interlobular septum to enlarge (Figure 10, B1). Leucocytes have been extensively infiltrated into the bronchial and subepithelial layers (Figure 10, B4), along with intra-alveolar thickening, intra-bronchial fibrin exudation, necrosis of the bronchial epithelium, and the development of intra-vascular thrombi in nearby blood vessels. The necrotic area (arrowhead) is separated from the surrounding area in figure 10, B3 by a zone of inflammatory cells (arrows), a fibrous tissue capsule, and alveoli that are filled with exudate fluid.

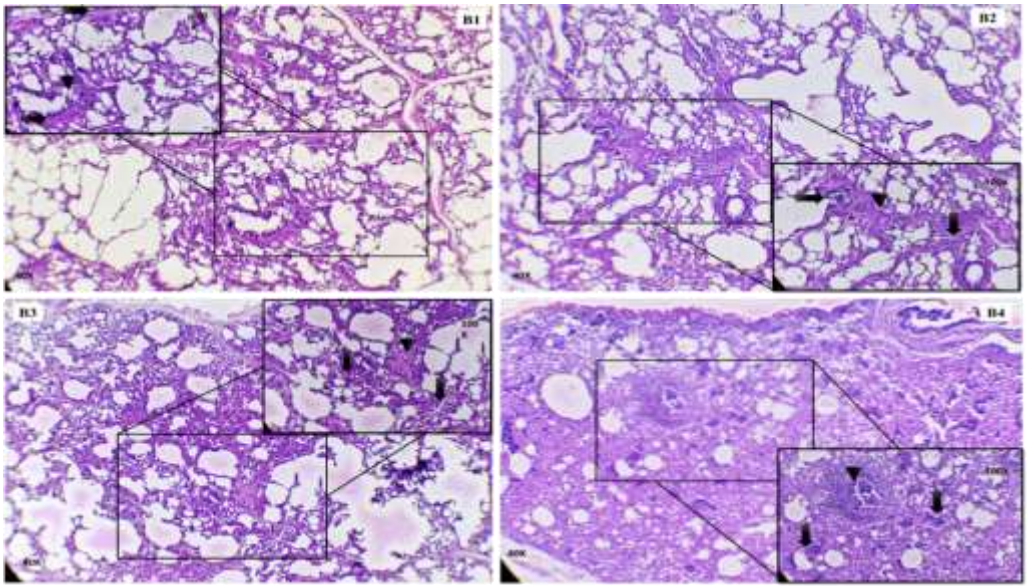


Figure 10. Histopathological photograph of lungs of goat infected with CCPP; shows inflammatory infiltration and exudation (arrow) were seen in group B1, alveolar space was disrupted (arrow) and thick by serofibrinous (arrowhead) changes were seen in group B2, thicken alveolar space and caseous necrosis (arrow) filled in interstitial alveolar space (arrowhead) in group B3, progressively bronchiolar space was thick and filled with caseous necrosis (arrowhead) in group B4. H&E stain, magnification 40X and 100X.

**DISCUSSION**

Our study observed gross pathological changes in the trachea and lungs of experimentally infected goats with CCPP. These changes included congestion, thin fibrinous mucous plugs in the tracheal lumens, hemorrhages, purulent exudate, and enlarged mesenteric lymph nodes in the trachea. The lungs exhibited consolidations, enlarged bronchial and mediastinal lymph nodes, frothy and fibrinous exudates, straw-colored fluid filling the thoracic cavities, multifocal necrotic lesions and abscesses on the lung surface, and varying hepatization colors ranging from deep blue to reddish. These findings are consistent with the gross lesions described by Nicholas and Churchward (2012) and OIE (2008) for CCPP-infected animals, characterized by inflamed lungs with a marbled appearance, fibrinous pleura with consolidation and hepatization, and accumulation of straw-colored fluid with pleural adhesion. Wesonga et al. (2004) also reported lesions characteristic of classical CCPP caused by *Mycoplasma capricolum capripneumoniae*, limited to the thoracic cavity. Similar findings were reported by Mondal et al. (2004), Laura et al. (2006), and Rodriguez et al. (1996) regarding fibrous pleuropneumonia, fibrosis, pleural adhesion, mucopurulent exudates in the trachea, and typical lesions of CCPP in lung sections, including emphysema, atelectasis, broncho- and interstitial pneumonia. However, some variations in findings may be attributed to the different etiological species of Mycoplasma causing CCPP.

Our study's histopathological examination of tracheal sections revealed severe CCPP infection, with the erosion of the respiratory ciliated epithelial layer and lamina propria. Examination of lung sections in group A showed severe congestion of interalveolar capillaries, flooding of bronchioles with fibrinous or serofibrinous exudate, narrowing of bronchioles due to exudate, and filling of alveolar gaps with fluid. In group B, contagious caprine

pleuropneumonia with fibrous tissue mixed with inflammatory infiltration was observed. These findings are consistent with the reports of Rodriguez et al. (1996), Mondal et al. (2004), Laura et al. (2006), and Gelagay et al. (2007), who have reported similar kinds of changes in lung and tracheal sections, including broncho and interstitial pneumonia, atelectasis, emphysema, leukocytic infiltration, hemorrhages in the sub-mucosal layer, hyperactive cells (mucus-secreting), lining of epithelial layer (erosive inflammation). According to Wesonga et al. (2004) and OIE (2008), CCPP lesions are limited to the thoracic cavity. The results may be discrepancies due to variances in the Mycoplasma species producing CCPP. After isolating *Mycoplasma mycoides* subspecies *capri* from diseased goats, Mondal et al. (2004), and Nicholas et al. (2008) made comparable findings.

## CONCLUSIONS

The moderate gross pathological changes observed in the trachea and lungs of experimentally infected goats with CCPP were congestion, thin fibrinous mucous plugs in the tracheal lumens, hemorrhages, purulent exudate, and enlarged mesenteric lymph nodes in the trachea. The lungs exhibited consolidations, enlarged bronchial and mediastinal lymph nodes, frothy and fibrinous exudates, straw-colored fluid filling the thoracic cavities, multifocal necrotic lesions and abscesses on the lung surface, and varying hepatization colors ranging from deep blue to reddish. Histopathological examination of trachea sections revealed severe CCPP infection. Group A & B plates showed the respiratory ciliated epithelial layer erupted and erosion of lamina propria in the later days of infection. Histopathological examination of lungs sections revealed that in group A, interalveolar capillaries were severely congested, the bronchioles were flooded with fibrinous or serofibrinous exudate, the bronchioles were narrowed by exudate, and the alveolar gaps were filled with fluid.

## CONFLICT OF INTEREST

The authors declare that there is no conflict in the publication of this article.

## AUTHOR'S CONTRIBUTION

All the authors contributed equally in the manuscript.

## REFERENCES

Awan, M., Abbas, F., Yasinzai, M., Nicholas, R., Babar, S., Ayling, R., Attique, M., Ahmed, Z., 2009. Prevalence of *Mycoplasma capricolum* subsp. *capricolum* and *Mycoplasma putrefaciens* in goats in Pishin district of Balochistan, Pakistan. Pakistan Veterinary Journal 29, 179-185.

Ayaz, H.P., Mansoor, T., Amjad, A.C., Zaheer, A.N., Ayaz, H.M., Riaz, A.L., Ghulam, M.S., Shahrood, A.S., 2023. Pathological study on experimental infection with *Mycoplasma mycoides* subspecies *capri* in different age groups of goats. Biosight 4, 41-50.

Gelagay, A., Ayelet, G., Gelaye, E., 2007. Contagious caprine pleuropneumonia-A review. Bulletin of Animal Health and Production in Africa 55, 143-151.

Hussain, M., Khan, A., Mahmood, M.S., Mushtaq, M.H., 2012. Seroprevalence of contagious caprine pleuropneumonia in Beetal goats in Pakistan. Tropical Animal Health and Production 44, 1669-1672.

Laura, P.M., Vasquez, R.A., Martínez, J.A., López, H.A., 2006. Lesions and clinical findings in goats with contagious pleuropneumonia. Veterinary Record 159, 214-217.

Mondal, B., Sen, A., Mahajan, V., Pathak, A., Ranjan, R., 2004. Epidemiological, clinico-pathological and molecular studies of contagious caprine pleuropneumonia (CCPP) in goats of Bihar. Indian Veterinary Journal 81, 312-315.

Nicholas, R., Ayling, R., McAuliffe, L., 2008. Mycoplasma diseases of ruminants. CAB International.

Nicholas, R., Churchward, C., 2012. Contagious caprine pleuropneumonia: new aspects of an old disease. Transboundary and Emerging Diseases 59, 189-196.

Nicolet, J., 1996. Contagious caprine pleuropneumonia: current status and prospects for the future. Veterinary research communications 27, 203-220.

OIE, 2008. Contagious caprine pleuropneumonia, Manual of Diagnostic Tests and Vaccines for Terrestrial Animals. World Organisation for Animal Health, pp. 1063-1072.

OIE, 2017. Contagious caprine pleuropneumonia, Manual of Diagnostic Tests and Vaccines for Terrestrial Animals. World Organisation for Animal Health, pp. 1163-1172.

Parray, O.R., Bhat, R.A., Mir, M.S., Bhat, J.A., 2019. Contagious caprine pleuropneumonia: An emerging respiratory disease of goats. Indian Journal of Animal Research 53, 1-7.

Rahman, A., Kausar, R., Akhtar, P., 2003. Studies on the occurrence of caprine mycoplasmosis in Pakistan. Pakistan Journal of Agricultural Sciences 40, 117-120.

Rodriguez, J.L., Tola, S., Gómez-Martín, A., Perez de la Lastra, J.M., Poveda, J.B., 1996. Tracheal lesions in goats naturally infected with *Mycoplasma capricolum* subsp. *capripneumoniae*. Veterinary Record 138, 415-417.

Rurangirwa, F.R., Kinyili, G.K., 2000. Contagious caprine pleuropneumonia in Kenya. Journal of Veterinary Medicine 47, 39-46.

Sadique, U., Hameed, S., Qayyum, M., Ahmad, M., 2012. Contagious caprine pleuropneumonia: A comprehensive review. The Journal of Animal & Plant Sciences 22, 1031-1041.

Samiullah, M., 2013. Contagious caprine pleuropneumonia (CCPP): A major constraint to goats' productivity. Journal of Animal Health and Production 1, 12-16.

Sheikh, A.A., Selim, A., Hossain, M.A., Chowdhury, S.D., Islam, M.N., 2016. Contagious caprine pleuropneumonia (CCPP) outbreak in a herd of black Bengal goats at Cox's Bazar in Bangladesh: A case report. Journal of Advanced Veterinary and Animal Research 3, 101-106.

Solangi, G., Nizamani, Z., Tariq, M., Leghari, Z., Kamboh, A., Talpur, B., 2023. Seroprevalence of contagious caprine pleuropneumonia in goats from selected endemic areas of Sindh. Journal of Animal Health and Production 11, 56-61.

Tanaka, Y., Kato, Y., Kubo, M., Nakamura, M., Izawa, H., Suzuki, Y., 2014. *Mycoplasma capricolum* subsp. *capripneumoniae* induces NF-κB-dependent mechanism to upregulate apoptotic cell death in bovine lung epithelial cells. Veterinary research communications 45, 1-9.

Totte, P., Rodrigues, M.X., Micheloud, J.F., Bustamante, A., 2015. Immune response in goats experimentally infected with *Mycoplasma capricolum* subsp. *capripneumoniae*. Veterinary Microbiology 177, 398-404.

Wesonga, H.O., Bolske, G., Thiaucourt, F., Wanjohi, C., Lindberg, R., 2004. Experimental contagious caprine pleuropneumonia: A long term study on the course of infection and pathology in a flock of goats infected with *Mycoplasma capricolum* subsp. *capripneumoniae*. Acta Veterinaria Scandinavica 45, 167.

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