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RESEARCH ARTICLE

Clinicopathological studies on respiratory affections in small ruminants with special reference to Mycoplasma species

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ABSTRACT

Pneumonia is a major health concern in small ruminants, significantly reducing productivity and causing substantial economic losses worldwide. Mycoplasma species are key pathogens implicated in respiratory infections of goats and sheep, acting either alone or in combination with other microbes. This study investigated haematological changes and identified major respiratory pathogens in clinically affected small ruminants. It also evaluated the pathological and immunohistochemical changes associated with *Mycoplasma* induced pneumonia in dead animals. A total of 50 animals (42 goats and 8 sheep) showing respiratory signs were examined. Blood and nasal swab samples were collected to assess haematological parameters and identify causative agents. Haematological analysis revealed significantly elevated total leukocyte counts and neutrophilia ($P<0.05$) in affected animals, along with a slight increase in lymphocytes, indicating an active inflammatory process. Bacterial culture of nasal swabs identified *Staphylococcus* spp. (50%) as the most common isolate, followed by *E. coli* (30%) and *Streptococcus* spp. (10%). Postmortem examination of 19 goats with respiratory illness revealed gross lung changes, including consolidation, mucopurulent discharge and tracheal congestion. Histopathological evaluation confirmed interstitial pneumonia in 11 (57.89%) animals, characterized by inflammatory infiltrates, alveolar septal thickening. The presence of mixed inflammatory cell populations suggested both acute and chronic phases of disease. Immunohistochemical analysis confirmed the presence of *Mycoplasma* antigens in all 11 histopathologically positive cases. In conclusion, *Mycoplasma* associated pneumonia, often compounded by secondary bacterial infections, plays a significant role in the respiratory disease complex of small ruminants. Early diagnosis, along with improved management and control measures is essential to reduce economic losses in small ruminant production systems.

Keywords: Mycoplasma species, Pneumonia, small ruminants

INTRODUCTION

Infiltration of inflammatory cells in lung parenchyma, alveoli and bronchioles are hallmarks of pneumonia, an acute, subacute or chronic infection of the lungs[1]. Pneumonia, the main health issue affecting small ruminants, can be brought on by *Mycoplasma* species either by itself or in combination with other microorganisms. In many regions of the world, pneumonia in small ruminants is a major hindrance to the development of this animal group and causes financial losses[2]. *Mycoplasmas* are found in nature in large quantities and can infect humans, animals and plants[3]. The infectious condition known as Mycoplasmosis is brought on by the tiniest, fastidious bacterium called *Mycoplasma*[4]. Extracellular parasites called *Mycoplasmas* frequently live as commensals on the mucous membranes of the upper respiratory tract in mammals. In small ruminants, they can lead to genital disorders, eye infections, respiratory conditions, arthritis and seldom, mastitis. Among other illnesses, respiratory conditions are prevalent in goats. Currently considered an emerging disease, Mycoplasmosis poses significant economic obstacles for farmers and small ruminant-rearing nations[5]. One of the main obstacles to the production of sheep and goats is poor management and husbandry techniques, as well as illnesses with a variety of aetiologies. Since air and blood are the primary means of transmission for respiratory infections, they are the most common among the various diseases that attack sheep and goats. The present study was undertaken to study the pathological changes induced by *Mycoplasma* in small ruminants causing pneumonia in dead animals and to study the haematological parameters along with to harbour main etiological agent causing respiratory affection in live animals.

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MATERIALS AND METHODS

Sample collection from small ruminants suffering from respiratory affections

The goal of the current investigation was to determine the main causative agents of pneumonia in small ruminants. Blood Sample was collected from total of 50 animals (40 clinically sick and 10 healthy animals) to investigate comprehensive

haematology, including TLC, DLC etc., and Nasal swabs were collected from 40 clinically sick and 10 apparently healthy animals to identify the primary etiological agents in clinically unwell small ruminants brought to the hospital for treatment. According to IAEC No. (GADVASU/2024/IAEC73/13), the study was approved.

Blood sample collection

Blood was collected from the animals' jugular vein, which were then placed in tubes with 0.5 ml of ethylenediamine tetra acetic acid. The blood was slowly rotated in order to combine it. For the purpose of determining the differential leukocyte count, a fresh blood smear was produced on a clear, dry and non-oily slide and fixed with methanol for three to five minutes[6].

Isolation

Nasal and lung swabs taken from diseased animals during treatment were inoculated on BHI agar plates using the conventional streaking technique in a laminar flow. For a whole day, agar plates were incubated at 37^{O}C. Once more, pure and isolated single colonies of bacteria were obtained by subculturing on respective special media. Thin smears were made from pure isolated bacterial colonies and morphological identification was done by Gram staining.

Sample collection

From January 2024 to March 2025, 19 goats with a history of respiratory disease performed necropsied at the Department of Veterinary Pathology's post-mortem hall at Guru Angad Dev Veterinary and Animal Science University in Ludhiana, India. Following a comprehensive internal and exterior examination, 10% neutral buffered formalin was used to obtain representative tissue samples from the bronchial and mediastinal lymph nodes as well as the afflicted lungs. Additionally, Leishman stain was used to stain cytosmears that were taken from the damaged lung tissues.

Histopathology

Tissue samples were further cut after the first fixation for at least 24 hours, and they were further treated with benzene, water, graded alcohol, xylene, a xylene-benzene combination and melted paraffin. Sections of 3-4 µm were cut after embedding in paraffin blocks. After being moved to glass slides, the thin paraffin slices were stained using standard haematoxylin and eosin (H&E)[7].

Immunohistochemistry

Sections of paraffin tissue (4-5 microns) were placed on slides coated using APES, incubated at 37^{O}C, and then washed three times in 1X PBS (pH 7.2) for five minutes. After 15 minutes of deparaffinization in xylene 1 and 2, the mixture was hydrated with distilled water. After adding citrate buffer (pH 6.0), sections were subjected to antigen retrieval using the EZ Retriever IR System for three minutes at 92^{O}C and seven minutes at 70^{O}C. Sections were treated with 3% H_2O_2 in 100% methanol for 20 minutes after chilling for at least 45 minutes. After 20 minutes of incubation, two drops of 2.5% Normal Horse Serum (NHS) were administered, and any extra NHS was tapped off. Negative controls were given NHS in PBS, while the primary antibody (1:200 dilution in NHS) was incubated for the whole night at 4^{O}C in a humidified room. PBS washing was followed by an hour-long application of HRP conjugated goat anti-mouse IgGs (1:500 dilution). The sections were counter stained with Gill's haematoxylin for one minute after being developed with DAB and washed in double distilled water to stop the reaction. Sections were then cleaned in xylene, dehydrated in increasing alcohol grades and mounted using DPX.

Statistical analysis

The haematological data generated was subjected to paired 't' test using GraphPad Prism (Version 9.3.1.471).

RESULTS

Live animals

Haematological analysis

Haematological analysis was performed on blood samples taken from 50 animals (40 with respiratory diseases and 10 healthy); 42 of the 50 samples were from goats and 8 were from sheep. As previously mentioned, 50 animals' nasal swabs were also gathered in order to identify the primary causative agents of respiratory disorders in small ruminants out of which 40 animals were clinically ill and 10 healthy animals. There was no appreciable difference in the haematological parameters of haemoglobin (HB), packed cell volume (PCV), mean corpuscular haemoglobin concentration (MCHC), MCH and MCV between sick and healthy sheep and goats. Sheep and goats with respiratory disorders have higher White Blood Cell (WBC) counts. All of these pathological states demonstrated a significant rise in relative neutrophil levels in all of the respiratory ill animals. In all of these clinical scenarios, sheep and goats showed no discernible changes in their levels of monocytes, eosinophils or basophils. Leucocytes (neutrophils) were shown to be significantly higher ($P<0.05$) in sheep and goats with pathological diseases such as pneumonia, abscess and haemorrhage. Similarly, Table 1 indicates that the absolute neutrophil count was much higher in clinically unwell small ruminants than in healthy ones.

Isolation of bacteria

Nasal samples were streaked on Brain Heart Infusion agar. Plates were examined for bacterial growth following a 24 hour incubation period. Isolated colonies were stained with the Gram stain. Gram staining revealed that *Streptococci* species were present as purple coloured cocci shaped chains and the *Staphylococcus* species (Table 2) were present as clusters resembling grapes (Fig. 1A).

Table 1. Haematological parameters of clinically normal and diseased animals (n=50)

Parameters	Control	Diseased animals
Hb(g%)	8.100±0.3861[a]	7.915±0.1818[a]
PCV (%)	41.08±2.081[a]	38.99±1.185[a]
RBC (106/cu.mm)	13.91±0.7899[a]	13.81±0.5211[a]
MCV (fl)	29.73±0.6388[a]	28.68±0.2903[a]
MCH	5.778±0.08784[a]	5.478±0.04613[a]
MCHC	19.35±0.1004[a]	19.28±0.1261[a]
WBC	10.61±0.9849[a]	16.98±1.036[b]
Absolute count of neutrophils	4270±5971[a]	10091±875.3[b]
Absolute count of lymphocyte	6110±703.2[a]	6662±503.8[a]

b-Data represents Mean± S.E.M. differs significantly at p value ≤ 0.05.

Table 2. Table depicting bacterial isolates cultured from nasal swabs of clinically ill animals (n=50)

Serial No.	Bacteria isolated from live animals	Number of animals	Percentage
1	*Staphylococcus* species	25	50%
2	*E. coli* species	15	30%
3	*Streptococci* species	5	10%
5	No growth	10	20%
	Total	50	100%

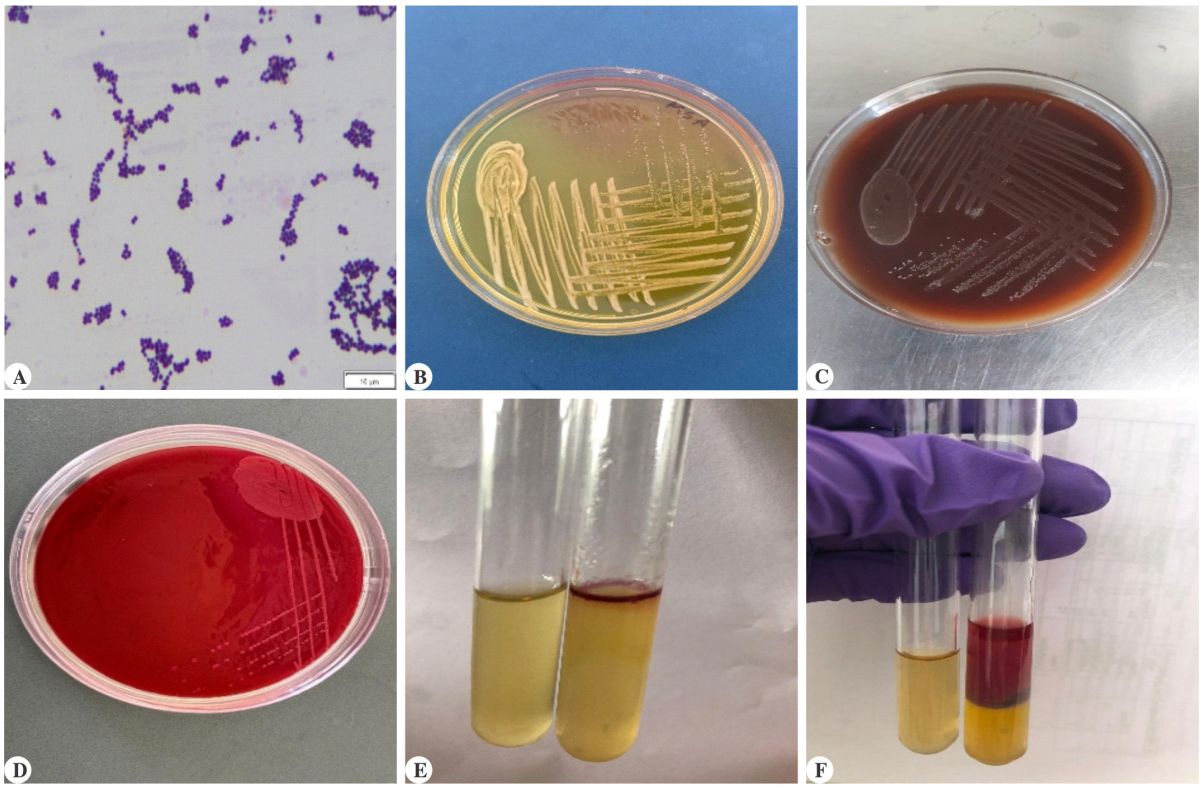

Fig. 1A. Gram stain showing *Staphylococcus aureus*, gram positive in a typical grape shaped (Gram stain x10 µm). **B.** Mannitol salt agar showing typical yellow-coloured colonies of *Staphylococcus aureus*. **C.** Showing colourless colonies of *Streptococci* species on Edward agar. **D.** MacConkey lactose agar showing lactose fermenter colonies of pink colour. **E.** Showing an indole-positive reaction typical for *E. coli*. **F.** Showing methyl red positive reaction typical for *E. coli*.

The catalase test was used to distinguish between *Streptococci* and *Staphylococcus* species, and the coagulase test was used to distinguish between *Staphylococcus aureus* and other *Staphylococcus* species. Of these, *Staphylococcus aureus* exhibited a positive reaction, while other *Staphylococcus* species showed a negative reaction. Coagulase positive *Staphylococci*, such as *Staphylococcus aureus*, produced yellow colonies (Fig. 1B) on Mannitol Salt Agar, whereas *Staphylococcus* species were further streaked on their specific medium, namely Baird Parker media, on which colonies appeared black with clear zones surrounding them. Additionally, *Streptococci* species were streaked on their particular medium, such as blood agar, which displayed white colonies with a haemolysis zone and Edward's agar likewise displayed colourless colonies with a visible haemolysis zone (Fig. 1C). Gram staining made gram negative bacteria look blue. Eosin methylene blue agar (EMB) and MacConkey lactose agar (MLA) were streaked with additional gram negative bacteria. Colonies in MLA had a pink colour, indicating that the bacteria were lactose fermenters (Fig. 1D). These lactose fermenting colonies had a distinctive green metallic sheen that is typical of *Escherichia coli* when streaked further on EMB agar. Additionally, the Indole, Methyl Red, Voges Proskauer, and Citrate test (ImViC) were used to validate it (Fig. 1E & F).

Postmortem examination

11 (57.89%) of the 19 lung samples collected from necropsied animals were tested positive for Mycoplasma species. Grossly, mucopurulent discharge was observed inside the nasal cavity. Six out of eleven small ruminants (54.54%) had mild hyperaemia (Fig. 2A), four out of eleven instances (36.36%) had moderate hyperaemia and one out of eleven (9.09%) had severe hyperaemia. There was also mild to severe tracheal mucosal congestion. Mononuclear cells, primarily lymphocytes and a small number of macrophages were seen in the impression smear upon cytological analysis (Fig. 2B). Histopathological analysis identified the primary features of interstitial pneumonia linked to Mycoplasma infection. The affected lungs were heavy, expanded, light in colour (with a more noticeable pale colour) and rubbery in texture rather than collapsing (Fig. 2C). The lung's pleural surface showed distinct rib imprints (Fig. 2D). In many cases, the rapid drainage of frothy and/or bloody exudate from the lung's cut surface was a sign of oedema and congestion. Patchy, dark red, discoloured patches that were more widely dispersed across the lungs showed pulmonary consolidation. There were times when the caudo dorsal area of the lungs was reddish brown, meaty and sometimes browned lobular (Fig. 2E). Upon opening the lungs, the interlobular septa (marbling) expanded and held a yellowish jelly like

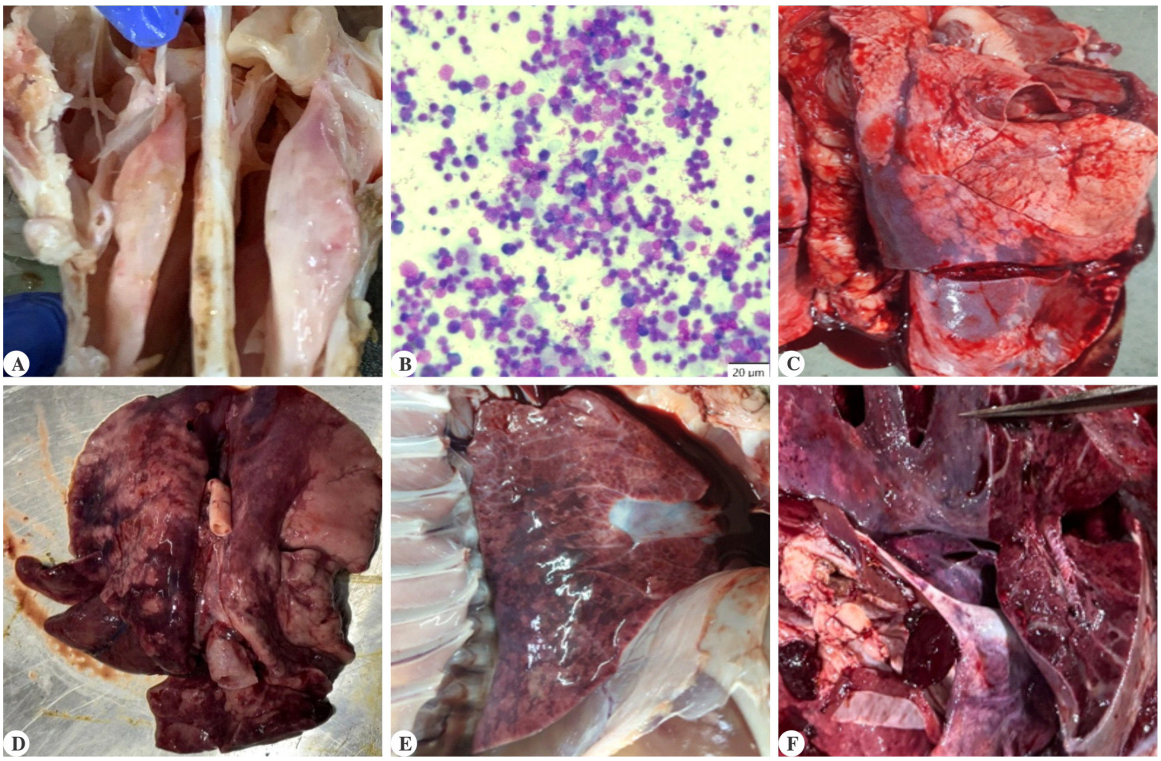

Fig. 2A. Nasal turbinate showing very mild congestion. **B.** Impression smear showing the presence of mononuclear cells (MNCs). Note the presence of some binucleate cells and conjoined cells giving the appearance of syncytia (Leishman x20 µm). **C.** Lungs showing pale and enlarged lungs. **D.** The lung showing irregular areas of consolidation with rib impression. **E.** Lung showing reddish brown colour in caudo dorsal lobe. **F.** Cut surface of lung showing typical marble pattern of Mycoplasmosis.

substance with different levels of lobule consolidation (Fig. 2F).

Microscopic examination

Inflammatory cell infiltration under a microscope mostly consists of a mixed cell population with hyperplastic alterations in the nasal mucosa lining epithelium (Fig. 3A). The lymph node showed the development of syncytia (Fig. 3B) and the presence of binucleate cells. During necropsy, interstitial pneumonia was discovered in 11 (57.89%) of the 19 cases. Four out of seven cases (57.14%) had mononuclear cells (primarily lymphocytes and few macrophages) that suggested chronic interstitial pneumonia, one out of seven cases (14.28%) had neutrophils (Fig. 3C), which suggested acute interstitial pneumonia and two (28.57%) out of seven cases had both mononuclear (MNCs) and neutrophils/polymorphonuclear (PMNCs) cells (Fig. 3D), which suggested a mixed infection. In these cases, the histopathological results showed dilated and enlarged alveolar capillaries and vasculature (Fig. 3E), along with varied degrees of thickened interlobular septa caused by oedema, fibrin deposition, syncytia formation and a significant infiltration of inflammatory cells, primarily MNCs. The interalveolar septa thickened as a result of the infiltration of inflammatory cells and the hyperplasia and hypertrophy of type II pneumocytes. Desquamation of necrotic epithelial cells of the bronchioles inside the lumen, peribronchiolar lymphoid cell infiltration, multifocal nodules of mononuclear cell infiltration and mononuclear cell aggregation in the bloodstream were all observed in the lung tissues exhibiting the chronic stage of pneumonia in *Mycoplasma* positive samples.

Immunohistochemistry

Immunohistochemical staining revealed positive immunoreactions in 11 (57.89%) of the 19 histopathologically indicated *Mycoplasma* cases. Both the lumen of the lung's blood vessels and the cytoplasm of pulmonary intravascular macrophages in the interalveolar septa displayed positivity. *Mycoplasma* antigen was detected in the pleura, interlobular septa, extracellular space and the cytoplasm of macrophages (Fig. 3F). The necro suppurative exudate that obstructed the bronchiolar lumina and was occasionally observed along the surface of the bronchiolar epithelial cells and the cytoplasm of inflammatory cells implicated in the lesion was related to the immunoreactivity.

DISCUSSION

Leucocytes (neutrophils) were shown to be significantly higher (P<0.05) in sheep and goats with

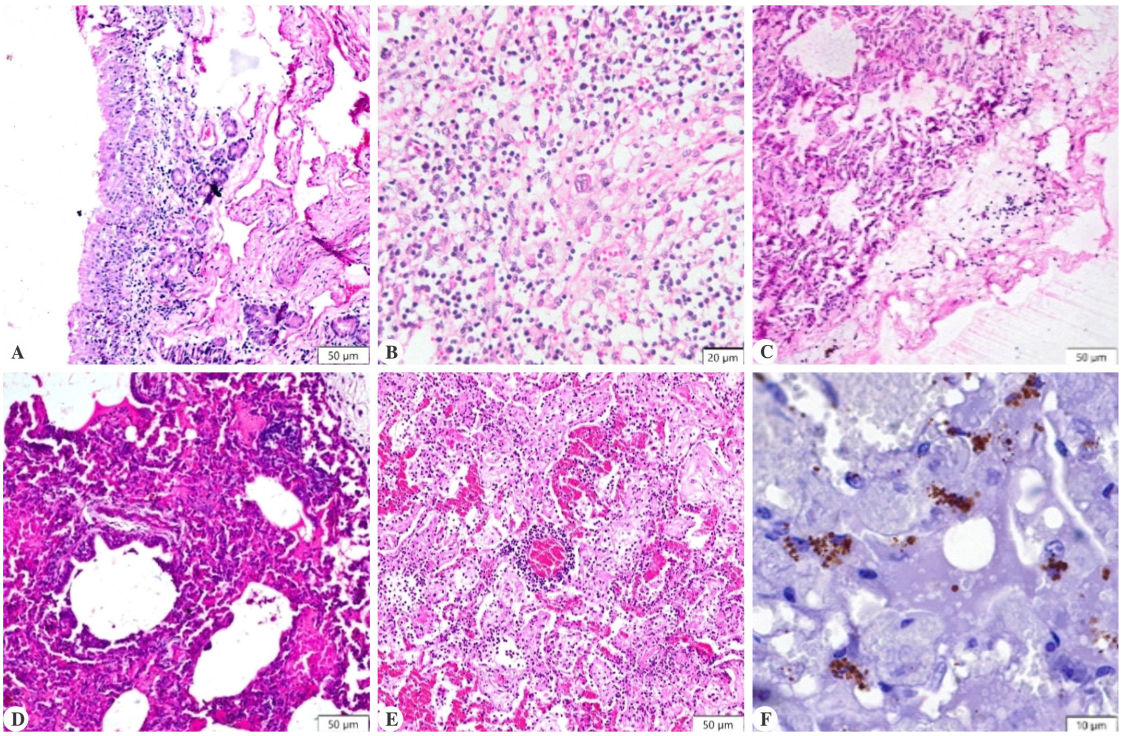

Fig. 3A. Revealed hyperplasia of epithelium along with mixed cell population infiltration (H&E x50 µm). **B.** Lymph node showing typical syncytia formation and lymphoid depletion (H&E x20 µm). **C.** Lungs showing infiltration of cells in the interstitium, giving a thickened appearance along with infiltration (H&E x20 µm). **D.** Showing infiltration of mixed cell population of inflammatory cells in the interstitium, suggestive of interstitial pneumonia along with syncytia (H&E x50 µm). **E.** Showing serous fluid infiltration in alveoli and diffuse infiltration of mixed cell population and the presence of perivascular infiltration (H&E x50 µm). **F.** Immunolocalization of *Mycoplasma* in the interstitium of the tissue and in inflammatory cells (IHC x10 µm).

pathological diseases such as pneumonia, abscess and haemorrhage. These results are in accordance with research from studies[8,9] which revealed several lung degenerative diseases. In studies[10,11], an increase in leukocytes was found and this was also evident in the current investigation. Additionally, [12]observed a noteworthy reduction in lymphocytes in sheep and goats with respiratory ailments. As a defense mechanism against the etiological agents that cause the body's inflammatory processes, particularly in the spleen, liver, kidneys and lung, where the inflammation prompts the bone marrow to produce a lot of white blood cells (WBC), they did observe a notable increase in neutrophils and a decrease in lymphocytes[13].

The most frequent bacterium responsible for pneumonia in sheep and goats was *Staphylococcus aureus*[14]. However, the most frequent bacterium that caused respiratory problems in small ruminants was *E. coli*, according to a study done by[15]. Other bacteria, including *Klebsiella* and *Streptococci* species, were also reported by him. *Pasteurella multocida* and *E. coli* species were the most often detected bacteria in the respiratory tracts of ill small ruminants, ranking second and third, respectively[16]. However, the geographic variation of the place from where the samples were taken, the mixed bacterial population in animals and the immunological condition of the animal[14] could all be responsible for a variation in the isolation percentage of various etiological agents.

Tracheal mucosal oedema and congestion were indicated by lesions that resembled froth in the trachea and other related organs[17]. The stroma, or supporting tissue, grows as inflammatory cells reach the lungs and in certain cases, cranioventral congestion[18] causes the lungs to appear mottled. The interlobular septa (marbling) grew larger and held a yellowish jelly like fluid with different levels of lobule consolidation[19] when the lungs were cut open. In the pulmonary lobules, compensatory emphysema from alveolar wall rupture and atelectasis from significant thickening of the interalveolar septa were frequently seen. Additionally, some of the animals exhibited many hemorrhagic regions. Pale lungs, especially when the alveolar walls are fibrosed, are caused by severe alveolar capillary obliteration (a reduced blood tissue ratio).

Type II pneumonocytes, which are essentially progenitor cells that differentiate, take the place of necrotic type I pneumonocytes. This causes the alveolar walls to thicken. The reason the lungs don't collapse regularly when the thorax is opened, why they feel rubbery to the touch and why the lung's sliced surface seems "meaty"[20] can all be explained by this mechanism. The complicated aetiology of interstitial pneumonia may be caused by hematogenous damage to the alveolar capillary endothelium or alveolar basement membrane. Alveolar and interlobar septal deposits of antigen antibody complexes (type III hypersensitivity) are created when inhaled antigens and circulating antibodies mix. This results in thickening (marbling), a series of inflammatory responses and damage[20] of the animals had interstitial pneumonia, fibrous pleuritis and excess serosanguinous pleural fluid. The animals with more chronic illnesses showed lung adhesion to the rib cage because to *Mycoplasma* species, which is similar to the findings of[21]. Hydropic degeneration, necrosis and in certain cases, severe hemorrhagic pneumonia[22] were observed in the lining of the bronchi. Acute lesions with a bad prognosis are frequently the outcome of septicemia in these illnesses, which is caused by *Mycoplasma* growth and bloodstream dispersion[21]. The majority of *Mycoplasma* species proliferate and disperse via blood vessels, leading to the formation of microthrombi[23]. The lungs' stoma or supporting tissue receives blood from their blood vessels, so when etiological agents cause microthrombi to form in the tissue, inflammatory cells are drawn to the area. This leads to inflammation of the stroma or supporting tissue, which in turn causes interstitial pneumonia. The thickening of the interlobar and interalveolar septa, which results in interstitial pneumonia characteristic of *Mycoplasma*, is caused by the increased attraction of inflammatory cells to the site, i.e., the interstitium as stated in[21,23,24].

CONCLUSION

In conclusion, *Mycoplasma* associated pneumonia, often compounded by secondary bacterial infections, plays a significant role in the respiratory disease complex of small ruminants. Early diagnosis, along with improved management and control measures, is essential to reduce economic losses in small ruminant production systems.

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