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Short Communication

PULMONARY ADENOMATOSIS IN A NELLORE JODIPI RAM

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ABSTRACT: A three-year-old Nellore Jodipi ram was presented to small ruminant ward, Department of Veterinary Medicine, College of Veterinary Science, Tirupati with inappetence, dullness and profuse nasal discharge for one week. Haemato-biochemical examination revealed moderate anemia and leukocytosis. The animal was treated with parenteral and intranasal antimicrobial therapy for 3 days along with respiratory stimulants. Despite the medical management and supportive therapy animal was succumbed after three days. Upon detailed postmortem investigation of the carcass, lesions of ovine pulmonary adenoma were found in the lungs and were confirmed by histopathological examination and *Oestrus ovis* larvae were found in the lung tissue.

Key words: Jaagsiekte Sheep Retrovirus, Nellore Jodipi, Sheep, Postmortem.

Case history and clinical observations

A three-year-old Nellore Jodipi ram was presented to small ruminant ward, Department of Veterinary Medicine, College of Veterinary Science, Tirupati with a history of profuse nasal discharges and inappetence for one week. On clinical examination the animal was dull and mostly recumbent with pale mucosa (Fig. 2), expiratory dyspnea and tachycardia. Animal had profuse mucopurulent nasal discharges (Fig. 1). Upon laboratory examination, moderate anemia (Hb-6, PCV-18), leukocytosis with severe neutrophilia were noticed. Electrocardiographic findings revealed moderate tachyarrhythmia. Heart rate was 80 bpm. Thoracic radiography performed on left lateral recumbency revealed severe pneumonic changes in lungs. Nasal discharges were collected into a sterile container and cultured, which revealed presence of Gram-negative bacteria.

Treatment and discussion

The animal was treated with Inj. Ceftrioxone @ 20mg/kg b. wt IV, BID; Inj. Chlorphenaramine maleate @ 0.5 mg/kg b. wt IM, SID and Inj. Lasix @ 3 mg / kg b. wt, SID, IM for 3 days. Oral hematinic supplementation was given with Syrup Sharkoferrol- 10 ml, PO, BID for 3 days. The animal showed slight improvement on day 2 post-treatment, but condition worsened on Day 3.

The animal was succumbed on day 4 and Postmortem was performed immediately which revealed enlarged and meaty appearance of lungs with greyish white nodular growths on the surface (Fig. 3). Cut section of the lungs (Fig. 4) showed greyish white nodules. *Oestrus ovis* larvae were also noticed in the lungs (Fig. 5). Histopathology of the Nodular growths revealed hypertrophy and hyperplasia of alveolar cells along with inflammatory cells along with papillary projections of alveoli into lumen and infiltration of leucocytes confirming the diagnosis of Ovine Pulmonary Adenomatosis (Fig 6 and Fig. 7). All other organs were found to be unaffected except lungs.

Ovine Pulmonary Adenomatosis (OPA) is a contagious viral disease of sheep and rarely goats, resulting in pulmonary neoplasia. Following the latest WHO classification for human lung cancers, OPA may be referred to as a mixed adenocarcinoma with associated bronchioloalveolar, papillary and/or acinar subtypes (Beasley *et al.* 2005). Eradication of the disease is difficult since antemortem diagnosis is grim in preclinical stage (Sonawane *et al.* 2016) and death usually results from end-stage respiratory failure (Leroux *et al.* 2007). Postmortem diagnosis of the disease is the most common method.

Ovine pulmonary adenomatosis is a contagious and neoplastic lung disease of sheep caused by Jaagsiekte Sheep Retrovirus (JSRV) and is clinically characterized

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Fig. 1. Sowing pale mucous membrane.

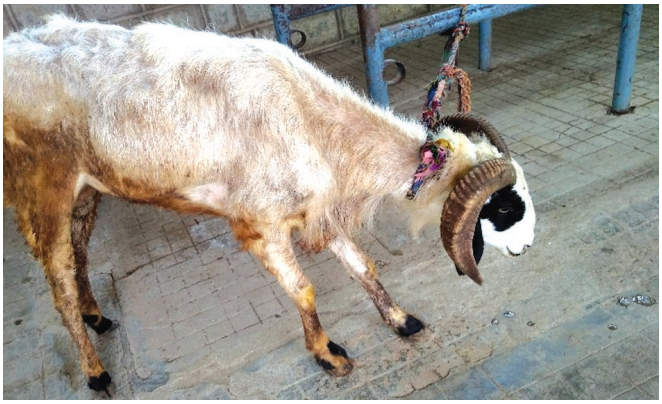

Fig. 2. Nellore Jodipi ram with pulmonary adenomatosis depicting profuse nasal discharges.

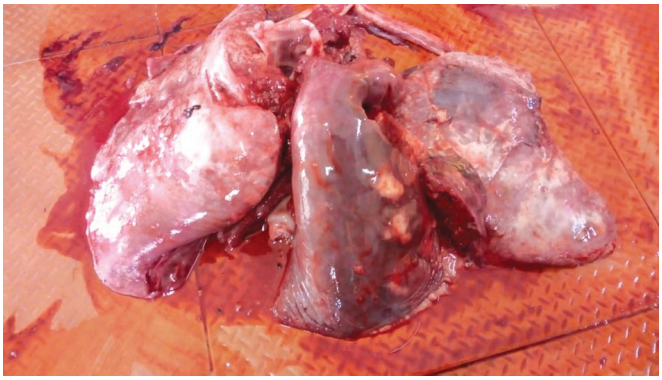

Fig. 3. Lungs showing enlarged and meaty appearance with greyish white nodular growths.

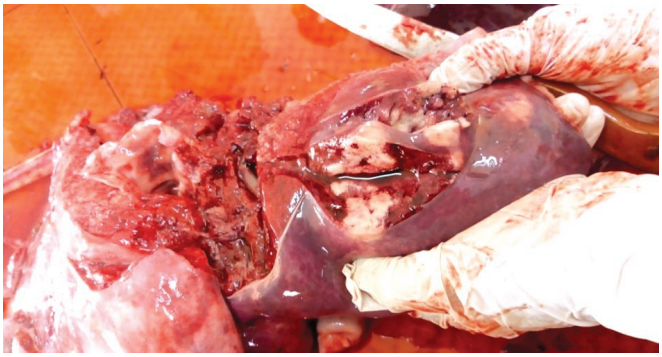

Fig. 4. Cut section of the lungs showing greyish white nodules.

Fig. 5. *Oestrus ovis* larva noticed in cut section of lung.

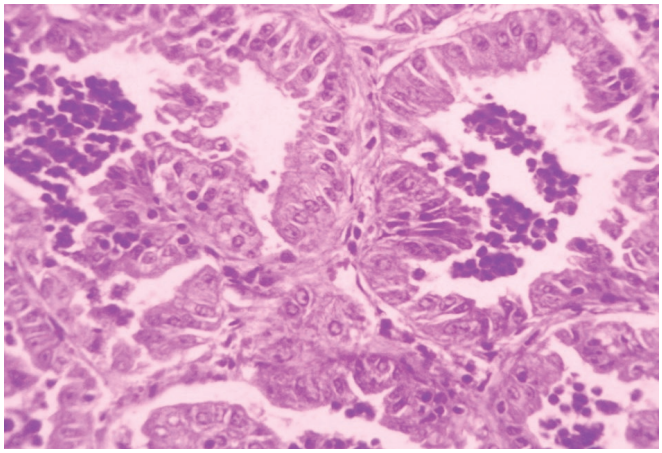

Fig. 6. Lungs showing hypertrophy and hyperplasia of alveolar cells along with inflammatory cells (H&E 40X).

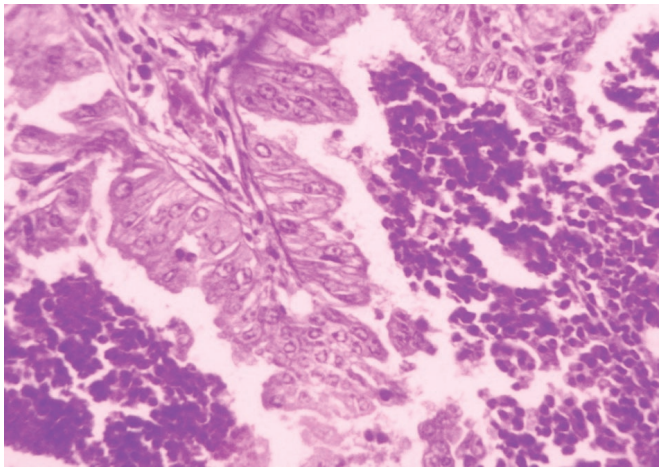

Fig. 7. Lungs showing papillary projections of alveoli into lumen with infiltration of leucocytes (H&E 40X).

by profound respiratory distress, exercise intolerance, copious nasal discharge, cough and deterioration of condition (Griffiths *et al*. 2010). Jaagsiekte Virus being an oncogenic virus, forms invasive broncho-alveolar carcinomas from type II pneumocytes and club cells (Martineau *et al*. 2011). Transmission is possible via

aerosol or through ingestion of milk (Griffiths *et al.* 2010 and Grego *et al.* 2008) and young ones are thus highly susceptible for the disease (Salvatori *et al.* 2004 and Caporale *et al.* 2005). Griffiths *et al.* (2010) reported that clinical disease usually occurs in 2 – 4 years old sheep. JSRV may remain unnoticed for several days since the disease remains in subclinical stage throughout the life span (Caporale *et al.* 2005). In infected animals, the virus does not produce any humoral immunity, thus serological testing is not available for JSRV (Ortín *et al.* 1998). Antemortem diagnosis can be done either by PCR on blood, bronchoalveolar lavage fluid or thoracic ultrasound but with less sensitivity (Lewis *et al.* 2011). Definitive diagnosis of the disease can be done usually by postmortem examination and histopathological examination of lung lesions. Despite antibiotic treatment, sudden death will be noticed due to secondary bacterial pneumonia (Scott *et al.* 2013).

The present case has developed all the characteristic clinical signs of OPA along with presence of *Oestrus ovis* larvae, which might have predisposed to the aggravated pneumonic signs and death despite supportive therapy.

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