

Therapeutic management of *Muellerius capillaris* infection in Himalayan musk deer (*Moschus leucogaster*)

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ABSTRACT

An investigation was conducted to determine the cause of mortality and ill health in a captive musk deer farm at the Musk Deer Research Centre, Bageshwar, Uttarakhand. Six musk deer died within a period of two months, and pathological findings confirmed verminous pneumonia due to nematode infestation. Faecal samples collected from eleven animals and examined using standard flotation and sedimentation techniques revealed infection with *Muellerius capillaris* in all animals, with mild infection in 7 and moderate and severe infection in two animals. All animals were initially treated with ivermectin (0.2 mg/kg body weight, subcutaneously), but follow-up examination on day 10 indicated persistence of infection in seven animals. Subsequently, fenbendazole (5 mg/kg body weight orally for 15 days) was administered to all animals. Post-treatment faecal examinations on days 10 and 30 confirmed complete clearance of lungworm infection and overall improvement in animal health. The poor response to ivermectin may be due to limited drug availability within nodular lung lesions, whereas prolonged fenbendazole treatment proved effective in eliminating persistent *M. capillaris* infection in captive musk deer.

Key words: Anthelmintic, Lung worm, *Muellerius capillaris*, Musk deer

INTRODUCTION

The Himalayan musk deer (*Moschus leucogaster*) is a small deer species found in the Himalayan forests of India, also locally known as the Kasturi Mrig and known for the musk gland present in males¹. Due to illegal hunting for musk and habitat loss, its population has declined significantly. Therefore, it is listed under Endangered on the IUCN Red List and Schedule I of the Wildlife Protection Act, 1972, giving it the highest level of legal protection in India². The Himalayan musk deer is susceptible to several infectious and parasitic diseases that can affect its survival. Parasitic bronchitis is one of the most important diseases affecting domestic and wild ruminants and widely prevalent in Himalayan belt of Jammu and Kashmir, Himachal Pradesh and Garhwal hills of Uttarakhand³. The free ranging wild ruminants, usually share the grazing habitat of domestic ruminants (migratory flocks) very often in these areas and thus there is potential of cross-transmission of lungworm infections, besides gastrointestinal (GI) parasites.

The common etiological agents of the parasitic bronchitis in small ruminants include *Dictyocaulus filaria*, *Protostrongylus rufescens* and *Muellerius capillaris*⁴. Pathogenesis of these nematodes differs greatly because of variation in their predilection sites. *D. filaria* lives in the trachea and bronchi, whereas *P. rufescens* is found in the small bronchioles and *M. capillaris* inhabit the lung parenchyma of the host⁴. Epidemiology of disease depends usually on the pasture contamination by carrier animals and rainfall/precipitation appears to be the stimulant for both larvae and snail intermediate hosts. Clinical course of disease depends on severity of the infection, age and immunological status of the host⁵. The common clinical signs include coughing, unthriftiness, tenacious nasal discharge, respiratory distress and pneumonia⁵. However, pneumonia is rarely observed in *P. rufescens* and *M. capillaris* infected animals and these infections are almost unapparent

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being identified only at necropsy⁶. The present communication describes the impact and successful management of *M. capillaris* infection in musk deer maintained in captivity.

MATERIALS AND METHODS

History

Musk Deer Research Centre, Mahroori in Bageshwar, Uttarakhand, is involved in breeding Himalayan musk deer for musk production, which has high medicinal value. The centre is located at about 7320 ft above mean sea level and is managed

under the Central Council for Research in Ayurvedic Sciences, an autonomous body of the Ministry of AYUSH, Government of India. The farm maintains musk deer in individual captive pens with adequate feed and water. During the monsoon season, mortality of six musk deer occurred within two months, and the cause was investigated. Systematic necropsy was performed and tissue samples from major organs (liver, lungs, kidney, spleen, heart, intestine, and brain) were fixed in 10% neutral buffered formalin for histopathological examination. Sections (4–5 µm) were prepared using a microtome and stained with haematoxylin and eosin following standard procedures⁷. Heart blood and heart blood smears (heat fixed) were collected aseptically for bacteriological examination (culture isolation). Nasopharyngeal and oropharyngeal swabs, together with samples of the spleen, lymph nodes, intestine, and lung, were collected for genome detection of viruses by RT-PCR (PPRV and blue tongue virus). Stomach contents

Ivermectin and Fenbendazole. Blood samples were analysed using Giemsa stain on thin blood smears. Faecal samples were examined using conventional parasitological methods, and lungworm larvae were recovered using the Baermann technique⁸. The number of larvae per gram (LPG) of faeces was calculated and larvae were identified morphologically using standard keys. Based on the findings, infected animals were treated with ivermectin (0.2 mg/kg, subcutaneously), followed by fenbendazole (5 mg/kg orally for 15 days). Treatment efficacy was assessed on days 10 and 30 after the final treatment.

RESULTS

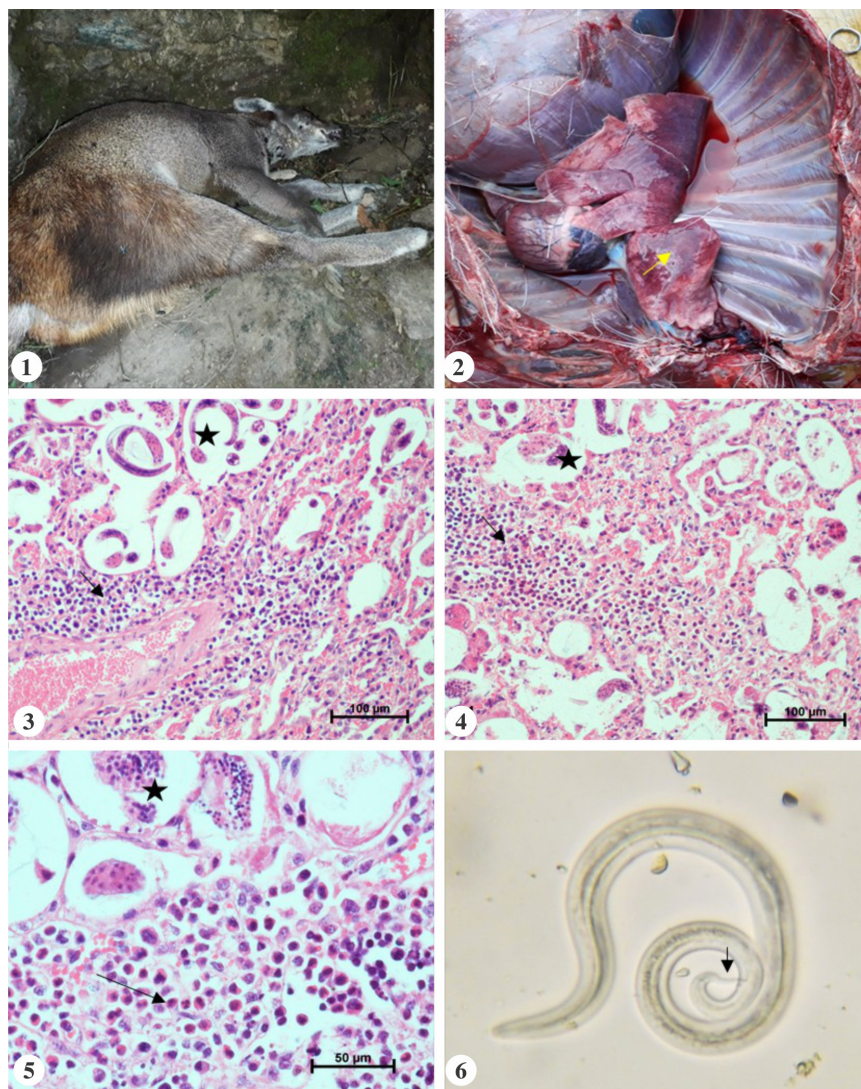
The carcass of musk deer showed emaciation (Fig. 1), pale mucous membranes, and marked reduction in subcutaneous adipose tissue. The lungs exhibited multifocal dark red areas of consolidation involving all lobes of both lungs with serosanguinous fluid in

and tissue samples from the liver and kidney were collected for toxicological analysis.

Health examination of live animals and treatment

During the investigation, individual Himalayan musk deer were carefully captured and examined for general health status. Rectal temperature, respiration rate, and mucous membrane colour were recorded. Blood samples from six animals were collected for haematological and parasitological examination. Faecal samples from eleven animals were collected from the rectum to detect gastrointestinal parasites, and follow-up examinations were conducted after treatment with

Fig. 1. Carcass of musk deer found dead in the enclosure of musk deer farm; **Fig. 2.** Left lung showing multifocal dark red areas of consolidation (arrow) with serosanguinous fluid in the thoracic cavity; **Fig. 3&4.** Lung showing highly engorged pulmonary vessels with numerous parasitic stages in the alveolar lumen (star) surrounded by severe infiltration of inflammatory cells (arrow). HE, x200; **Fig. 5.** Higher magnification of fig.4 showing the parasitic stages in the alveoli (star) surrounded by mixed population of inflammatory cells (arrow). HE, x400; **Fig. 6.** *Muellerius capillaris* larvae with undulating tail tip possessing a small, sharp dorsal spine in fecal examination (arrow).



Sl. No.	Identification No.	Larval count per gram of faecal pellets (LPG) before treatment	Larval count (LPG) after treatment		
			Ivermectin Treatment after 10 th dpt	Fenbendazole treatment for 15 days	
				10 th dpt	30 th dpt
1.	Musk deer 1	<10	<10	absent	absent
2.	Musk deer 2	<10	absent	absent	absent
3.	Musk deer 3	<10	absent	absent	absent
4.	Musk deer 4	10-50	<10	absent	absent
5.	Musk deer 5	<10	<10	absent	absent
6.	Musk deer 6	>50	10-50	absent	absent
7.	Musk deer 7	10-50	<10	absent	absent
8.	Musk deer 8	<10	absent	absent	absent
9.	Musk deer 9	<10	absent	absent	absent
10.	Musk deer 10	>50	10-50	absent	absent
11.	Musk deer 11	<10	<10	absent	absent

LPG <10- Mild, 10-50-Moderate, >50 Severe

the thoracic cavity. (Fig. 2). Histopathology revealed numerous parasite stages and desquamated epithelial cells in the alveoli, bronchi and bronchioles. These lesions were associated with moderate to severe infiltration of inflammatory cells, predominantly eosinophils, macrophages, and lymphocytes, along with highly engorged pulmonary blood vessels and oedema (Fig 3-5). Other organs did not reveal any significant histopathological changes. Bacteriological culture of heart blood on blood agar did not yield any growth of *Pasteurella* spp. Additionally, microscopic examination of Giemsa-stained heart blood smears did not reveal any bipolar organisms. Virological examination of the lung, spleen, lymph nodes, and intestinal samples by RT-PCR did not detect significant viral pathogens, including PPRV and BTV. Toxicological analyses of the stomach contents, liver, and kidney samples were negative for the presence of heavy metals, nitrate, nitrite, hydrogen cyanide (HCN), and commonly used insecticides.

All the live Himalayan musk deer examined had normal rectal temperature and respiratory rate while pale mucous membranes indicated possible anaemia. Haematological examination showed a mild decrease in haemoglobin, PCV, and RBC counts (9.0 ± 1.0 g/dL, $35.0 \pm 4.2\%$, and $2.2 \pm 0.6 \times 10^6/\mu\text{L}$). In contrast, the total white blood cell (WBC) count remained within the normal reference range. Microscopic examination of Giemsa-stained blood smears did not reveal any haemoparasites. No gastrointestinal parasites were detected by direct faecal examination. However, faecal examination using the Baermann technique revealed first-stage larvae of *Muellerius capillaris* (Fig. 6). Mild infection (<10 LPG) was observed in seven animals, moderate infection (10-50 LPG) in two animals, and heavy infection (>50 LPG) in two animals. After treatment with Ivermectin, lungworm infection persisted in seven animals at day 10

post-treatment. Subsequent treatment with Fenbendazole resulted in complete clearance of the parasite, as no larvae were detected on days 10 and 30 after the final treatment.

DISCUSSION

The Himalayan musk deer belongs to the family *Moschidae* and resembles true deer of the *Cervidae*, but lacks antlers. It is widely distributed across the Himalayan regions. They feed primarily on leaves, flowers, grasses, mosses, and lichens. The declining population of musk deer in India and other parts of the world has been attributed to infectious diseases and anthropogenic factors such as habitat loss, ecological degradation, and poaching². As herbivores sharing grazing areas with domestic and wild ruminants, musk deer are exposed to various parasitic infections through contaminated pastures. Among these, Parasitic bronchitis (lungworm infection) is common in small ruminants of the Himalayan region⁴. The disease is mainly associated with *D. filaria*, while *P. rufescens* and *M. capillaris* are also reported in grazing animals. Lungworm infections have also been documented in wild ruminants such as spotted deer and blue sheep in Himalayan ecosystems^{7,9}.

In the present study, poor health in captive musk deer at the breeding farm was associated mainly with *M. capillaris* infection. Previous records from the same farm indicated mortality due to Fasciolosis and verminous pneumonia⁶. Persistent lungworm infection at the farm may be associated with the use of fodder collected from adjacent forest areas, which could serve as a source of infection. *Muellerius capillaris*, commonly known as the nodular lungworm, generally causes mild or subclinical disease. However, pathological findings frequently reveal diffuse broncho-interstitial pneumonia with minimal or absent characteristic pulmonary nodules,

indicating that significant pathological changes may occur even in the absence of overt clinical signs¹⁰. Control of lungworm infections in ruminants generally involves the use of anthelmintic drugs such as benzimidazoles, pro-benzimidazoles, and Ivermectin¹¹⁻¹³. However, these drugs may not completely eliminate adult parasites and may only temporarily reduce the fecundity of small lungworms¹⁴. In the present study, ivermectin treatment showed limited efficacy against *M. capillaris*, which is consistent with earlier reports indicating incomplete effectiveness of this drug¹³. In contrast, treatment with fenbendazole for 15 days effectively eliminated adult parasites, and no larvae were detected in subsequent faecal examinations up to 30 days post-treatment. Since the parasite forms nodules in lung tissue that may later become fibrotic or calcified, a prolonged treatment regimen is often necessary. Therefore, the present findings support the use of fenbendazole for extended treatment in lungworm infections of domestic as well as wild ruminants.

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