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A paradoxical occurrence of toxocariasis in goat kid

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Abstract

A goat kid was presented for post-mortem examination at Central University Laboratory (CUL) Madhavaram, Chennai, TANUVAS, and a detailed necropsy was conducted to find the exact cause of death. A 2 -month old female kid had diarrhea and was found dead. On post-mortem examination, we accidentally encountered the Ascarid worm in the abomasal region and enlarged mesenteric lymph nodes. Further detailed parasitological investigation confirmed it as Ascarid. Histopathological investigation revealed the enteritis lesion. Morphometric details of the worm was 14 cm in length, 0.7 cm, 1.5 cm, and 0.7 cm in diameter in the anterior, middle and posterior parts respectively. It would be of first of its kind of study on goat kids in India.

Keywords: Paradoxical, Toxocara canis, Toxocara cati, Ascaris, goat, kid

Introduction

Toxocariasis is caused by members of the genus *Toxocara*, nematodes in the family Toxacaridae, and the superfamily Ascaridoisea. Recognized species include *Toxocara canis*, *T. cati*, *T. vitulorum*, which have domesticated animals as their definitive hosts; and *T. tanuki*, *T, pteropodis*, *T. apodemi*, *T. lyncus*, *T. mackerrasae*, *T. paradoxura*, *T. vajrasthirae*, which mature and shed eggs in the wild animals.

Adult *Toxocara* can cause intestinal illness in their definitive hosts, while larvae migrating through the tissues can affect both definitive and paratenic hosts. In paratenic hosts, toxocariasis is often called larva migrants. Visceral larval migrans is a general term used to indicate the presence of larvae in various internal organs. While the larvae in the eye are termed, ocular larval migrans. Larvae in the brain are sometimes called cerebral larval migrans.

These ascarids are very common in man, animals and birds. Younger animals are more susceptible. The adult worms are usually found in the intestines. Sometimes they may migrate to stomach (and from there to oesophagus), the bile duct and pancreatic duct. Infection is by ingestion of eggs in which the 2nd stage larvae are present. The larvae that are liberated in the stomach and intestine burrow through the gastric and intestinal wall and reach the liver via portal vein. Very little work has been done on the causes of morbidity & mortality and the influence of improved management practices to reduce them is being tried in commercial goat farms. Therefore, the present study has been designed.

Materials and Methods

A detailed necropsy was conducted on all the 52 dead kids. Which died during the study period of nine months in a commercial goat farm located in the Kancheepuram district of Tamil Nadu. Samples were collected and subjected to necessary laboratory investigation procedures to confirm the etiological agent. Representative samples were collected and fixed in 10% buffer formalin and subjected to routine histological processes. The tissue section was stained with Haematoxylin and Eosin. The stained tissue sections were examined for histopathological changes.

For bacteriological investigation samples were collected for isolation and identification. The samples were streaked and inoculated in specific media (Nutrient agar, blood agar, MacConkey agar, and Robertson's cooked meat medium). Suspected colonies were subjected

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to Gram staining followed by biochemical tests (Quinn and Carter, 2004) ^[8]. For anaerobic bacteria, the isolate was confirmed by PCR.

Results and Discussion

Necropsy findings and histopathology

The autopsy revealed the presence of a roundworm in the abomasal region (Fig. 1 & 2) and enlarged mesenteric lymph nodes (Fig. 3). Further, parasitological examination confirms it as Toxocara sp (Fig. 5 & 6). This is a paradoxical finding in goat kid. Histopathological examination revealed the enteritis lesion (Fig. 4). Morphometric details of the worm are as follows 14 cm in length, 0.7 cm, 1.5 cm, and 0.7 cm in diameter in the anterior, middle and posterior parts respectively. In this regard, scanty pieces of literature are available. Kantzoura et al. 2013 ^[9] in a study on seroprevalence and risk factors associated with zoonotic parasitic infections in small ruminants reported that 32% of Toxocara seroprevalent cases in sheep and goats and further they concluded occurrence of T. canis infection were related to lower temperature and higher precipitation compared to absence locations of T. canis in Greek and this the first time we are reporting the paradoxical occurrence of Toxocara infestation in goat kid. Calves become infected by ingesting third-stage larvae from an infected dam's milk (Mia et al. 1975, Roberts et al. 1990, Starke et al. 1992) [1, 2, 4], but not from ingesting eggs in the environment (Mia et al. 1975)^[1]. Larvae ingested by the calves develop into adults in 3-4 weeks and then begin shedding eggs in the feces (Kassi1999). Patent Toxocariasis is seen in young calves up to 6 months of age when adult worms are spontaneously eliminated (Roberts *et al.* 1990, Kassai 1999)^[2, 7]. As documented in the present study Srivastava et al. 1963 reported internal toxocariasis is associated with diarrhea and poor performance followed by death in beef calves infected with Toxocara vittulorum. In this present study, all the parent stocks are reared under a slatted floor system with the access to run area. Further, all the animals were routinely dewormed. Hence, the chance of trans-mammary transmission of infective L3 larvae is very minimal. Since this is closed intensive flock so the opportunity for the entry of new stock into the existing for introducing this parasite is not possible. At the farm on which research was carried out; most of kids were bottle-fed by animal attendees. A human can be infected by ingesting embryonated eggs, typically from dirt (eg, unwashed hands) or in contaminated foods or water; or by eating larvae in raw undercooked tissues of other paratenic hosts. or Embryonated T. canis or T. cati eggs on the fur of dogs and cats could also represent a source of infection for people. In some cases, drinking of unpasteurized bovine milk contaminated with Toxocara vittulorum larvae will result in infection but this is not possible in the present study, because kids were fed only goat milk. There were only the following possibilities for this incidence of ascarid, viz ingestion of infective L3 larvae from the cross-contaminated pasture and equipment with fecal material as reported by Davila et al. 2010 [10] in beef calves studies of Toxocara vittulorum infection (as the farm premises had canine and felines) or by unhygienic bottle feeding of kids by the animal handlers or by accidental transfer of embryonated infective stage (L3) to young kids through a nail or by infected human fecal contamination. These infective L3 larvae of T.canis or T. cati usually complete their lifecycle in their respective hosts but in the current study, it is not clear how that life cycle was completed without the definitive host. Need further

detailed study.



Fig 1: PM - Abomasum -Round worm



Fig 2: Round worm



Fig 3: PM- Mesenteric lymph node- enlargement



Fig 6: Contrast MC - Posterior part



Fig 5: Contrast MC – Anterior part



Fig 6: Contrast MC - Posterior part

Conclusion

The present case study reported the aberrant occurrence of *Toxocara* infection in goat kids; this might be due to unhygienic pastures, unhygienic feeding and handling of younger animals, and access to canines and felines on the farm premises. Periodic deworming of parent stocks, younger animals, dogs, and cats. The handler should get dewormed against ascarids regularly is a take-home message of the research. Apart from this proper good nutrition, sanitation and hygiene are the knockdown steps for controlling ascarid infection spread.

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Conflict of Interests

The authors declare that they have no conflict of interests.

Ethical issues: Not applicable.

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