Toxocara elephantis Infection in a Juvenile Asian Elephant and Its Management

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Introduction

Parasites can adversely affect host health, productivity and foraging, and may also modify host behaviour to facilitate parasite transmission (Moore 1984). Parasitism has been shown to directly affect both the evolution and ecology of hosts through processes such as sexual selection (Hamilton & Zuk 1982) or parasite-mediated competition, which can lead to a reduction in population size, or the extinction of one host (Price *et al*. 1986). Parasitic infections can cause diseases and death in wild animals and can become a source of infection for domestic animals and vice-versa.

Globally, the Asian elephant (*Elephas maximus*) is listed as 'Endangered' in the IUCN Red List of Threatened Species (IUCN 2021) and is protected under CITES. Anthropogenic influence on habitats may result in increased parasite prevalence in elephants (Hing *et al*. 2013). Elephants in the wild are susceptible to many gastrointestinal parasites (Watve 1995; Dharmarajan 1999; Vidya & Sukumar 2002). The occurrence of gastrointestinal parasites is higher in wild elephants as compared to captive and semi-captive elephants (Abeysekara *et al*. 2018). Gastrointestinal nematode infections can result in diarrhoea and disruption of gastrointestinal motility (Bueno *et al*. 1982).

Toxocara sp. commonly known as roundworms, infect many species of animals. *Toxocara* infection is mostly seen in young animals, which get infected through faeco-oral or galactogenic transmission and the larvae develop into adults in the intestine, which produce eggs by three weeks (Sarma *et al*. 2012). Infection may cause diarrhoea, poor performance, intestinal and biliary obstruction, and sometimes death (Srivastava 1963). Previously *Toxocara elephantis* infection has been reported in an Asian elephant from a zoo in Switzerland (Fowler & Mikota 2008). This paper describes *T. elephantis* infection in a wild juvenile Asian elephant and its management.

Case study

A 5-year-old female Asian elephant at the Centre for Wildlife Rehabilitation and Conservation (CWRC), Borjuri, Panbari Reserve Forest near to Kaziranga National Park, Assam was showing mild diarrhoea, loss of appetite, weight loss and lethargy (Fig. 1). The animal was rescued during a flood and was at the rehabilitation centre for one week. Clinical examination revealed normal body temperature (36.4°C), congested conjunctival mucous mem-

Figure 1. Juvenile affected with diarrhoea.

Manuscript Editor: Prithiviraj Fernando

branes, increased pulse rate (38 bpm) and eupnoea (14 breaths/min). Dehydration was diagnosed based on weight loss, decreased water intake, diarrhoea and dryness of the tip of the trunk and skin. A diarrhoeic sample was collected in a sterile container for the detection of parasites/parasitic eggs/protozoans. A blood sample (approximately 5.0 ml) was collected by venipuncture of the auricular vein into vials with and without dipotassium ethylenediaminetetraacetic acid (K2-EDTA). The blood with anticoagulant was used for haematology and the blood without anticoagulant was allowed to clot and centrifuged at 2000 rpm for 10 min at 4ºC to separate serum. The separated serum was stored at -20°C till analysis.

The collected faecal sample was processed with a standard protocol (Heinrich 2016) and examined under the microscope for identification of parasites/parasitic eggs/protozoans (Soulsby 1982).

Haematological analysis was conducted with an automated blood analyzer (Autoread, IDEXX, USA). Biochemical analysis was carried out with an automated biochemical analyzer (Catalyst one chemistry analyzer, IDEXX, USA) using commercially available test kits following the manufacturer's instructions. Zinc concentration in the serum was estimated by atomic absorption spectrophotometer (AA 7000, Shimadzu, USA). Oxidant-antioxidant status in the serum was evaluated by measuring lipid hydroperoxide (LPO), superoxide dismutase (SOD) and total antioxidant capacity (TAC), with the help of commercially available test kits as per manufacturer's instructions (Cayman Chemical, USA).

Results and Discussion

Macroscopic faecal examination revealed the presence of adult ascarid worms in the stool (Fig. 2). Microscopic examination of the faecal sample showed the presence of *T. elephantis* ova, which were round-shaped with a thick outer shell (Fig. 3). Based on clinical and laboratory findings, the case was diagnosed as a *Toxocara elephantis* infection.

Figure 2. The presence of adult ascarid worms in the stool.

The haematological analysis revealed a slight elevation in haemoglobin (Hb), total erythrocyte count (TEC) and packed cell volume (PCV) levels (Table 1), which could be attributable to dehydration causing haemoconcentration. Total leukocyte count (TLC) was within the normal range, and eosinophilia was the major finding in the differential leukocyte count (DLC) (Table 1). Eosinophilia is generally caused when parasites migrate through tissues, therefore it is prominent only in early infection of ascariasis (Leder & Weller 2000). In serum biochemistry, there was an increase in total pro-

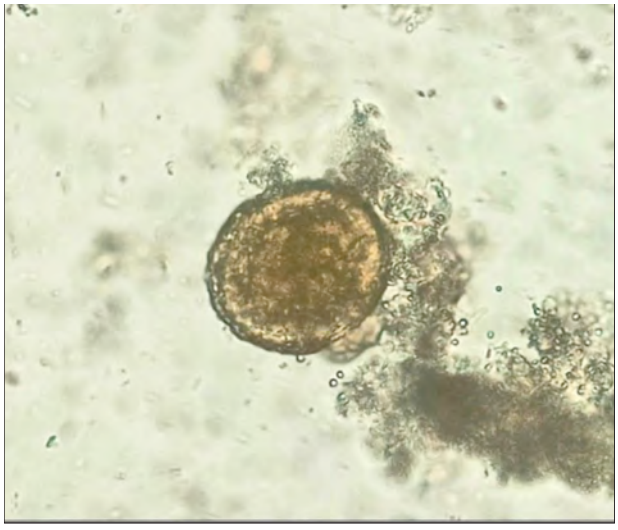

Figure 3. Microscopic examination of the faecal sample showing the presence of *T. elephantis* ova (X40).

Table 1. Haemato-biochemical and oxidative stress indices of *T. elephantis* infected elephant calf.

Parameters	Before treatment (Day 0)	After treatment (Day 10)	Normal reference range (Janyamethakul *et al*. 2017)
Hb (g/dl)	17.7	12.8	10.1–15.6
PCV (%)	47.9	34.3	27.8–43.0
TEC (x10^6 cells/μl)	4.93	3.02	1.9–3.1
TLC (x10^3 cells/μl)	18.15	15.40	7.20–23.22
DLC (x10^3 cells/μl)			
Neutrophils	10.83	9.25	0.83–13.51
Lymphocytes	4.90	4.64	1.06–12.03
Monocytes	1.11	1.25	0–3.29
Eosinophils	1.32	0.27	0–1.17
Basophils	0.00	0.00	0–0.036
Platelets (x10^5 cells/μl)	1.28	1.67	1.05–5.98
AST (U/l)	58.19	31.45	10.1–39.6
Creatinine (mg/dl)	1.97	1.02	0.9–1.8
BUN (mg/dl)	23.4	17.20	4.2–19.7
Total protein (g/dl)	9.79	8.32	6.6–9.3
Sodium (mmol/l)	153.0	123.0	105.0–131.0*
Potassium (mmol/l)	8.5	5.1	3.8–5.2*
Chloride (mmol/l)	121.0	97.0	91.0–102.0*
Zinc (ppm)	7.5	12.90	12.5–15.78*
LPO (μM)	7.05	4.67	4.16–4.64*
SOD (U/ml)	0.83	2.13	2.26–2.9*
TAC (mM Trolox equivalents)	1.02	2.67	2.9–3.6*

*Unpublished data

tein, sodium, potassium, chloride, aspartate aminotransferase (AST), blood urea nitrogen (BUN) and creatinine levels (Table 1). Increase in total protein and electrolyte levels may have been due to dehydration, whereas the increase in AST may have been due to cell necrosis caused by parasitic infestation (Benjamin 2013). Reduced renal function secondary to dehydration may have resulted in elevated levels of BUN and creatinine. Oxidative stress may play an important role in parasitic diseases (Deger *et al*. 2008). In the present case, the serum level of lipid hydroperoxide (LPO) was increased whereas the levels of superoxide dismutase (SOD), total antioxidant capacity (TAC) and zinc (Zn) were decreased (Table 1), which again may be due to *T. elephantis* induced diarrhoea. Elevation of oxidative parameters, and depletion of antioxidants and micro minerals were documented in *T. vitulorum* infection in buffalo calves (Sarma *et al*. 2012). The decreased level of Zn noticed in the present case may be due to its excessive utilization to neutralize the overproduction of reactive oxygen species (ROS) (Sarma *et al*. 2012).

The animal was treated with albendazole at 10 mg/kg, orally, once daily for 3 days, Dextrose normal saline at 3 l/day, IV, once daily for 5 days, Ringer's lactate at 2 l/day, IV, once daily for 5 days, zinc glycinate (ProFoodsTM) at 20 mg/kg, orally, once daily for 7 days and probiotic [*Lactobacillus bulgaricus* (Kaypeeyes Biotech Private Limited)] at 1x10^{10} colony forming units/50 kg, orally, once daily for 7 days. On the third day of treatment, there was improvement in appetite and faecal consistency. On the seventh day, the animal voided normal faeces without adult ascarid worms. A faecal sample was examined on the tenth day and found to be

Figure 4. Clinical recovery of the animal in response to treatment.

negative for *T. elephantis* ova. After completion of treatment, there was an improvement in haemato-biochemical and oxidative stress indices (Table 1), and the animal showed an uneventful recovery (Fig. 4).

The case study presented here illustrates the effective management of *T. elephantis* infection. As young animals can get toxocariasis through faeco-oral or galactogenic transmission, deworming should be done regularly to avoid infection.

Acknowledgements

The authors wish to thank the Dean, College of Veterinary Sciences and Animal Husbandry (CVSc & AH), Central Agricultural University (CAU), Selesih, Aizawl for providing facilities to carry out this study and also wish to thank director, Centre for Wildlife Rehabilitation and Conservation (CWRC), Borjuri, Panbari Reserve Forest, Assam, for providing facilities and giving permission to carry out this study. This work was funded under academic activities of the University. The study was approved by the Institutional Research Ethics Committee of the CVSc & AH, CAU, Selesih, Aizawl (CVSC/CAU/IAEC/19-20/P-25).

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