Prevalence and Haemato-Biochemical Studies of Gastro-intestinal Parasites of Indian Elephants *(Elephas maximus)*

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Abstract

Faecal samples were collected from 40 Indian elephants (*Elephas maximus*) revealed 62.5 percent parasitic prevalence. Amongst the single infection of parasites, high prevalence of *Fasciolias spp*. (15.00 %) was observed followed by percent prevalence of mixed infection. Elephants harbouring parasites were found clinically dull, depressed and lethargic. About 48 percent elephants manifested dehydration and loose faeces grossly along with a habit of soil licking. The haematological studies of elephants harbouring parasites revealed mild anaemia and eosinophilia where as biochemical studies revealed non-significant hypoproteinemia on comparison with elephants that were not harbouring parasites.
Keywords: Elephant, prevalence, haematological, biochemical.

Introduction

Apart from free-living population of Indian elephants *(Elephas maximus)* found in several protected areas, domesticated and trained elephants are being used for drought purpose by forest department, in circus and in temples for religious occasions in India. Several parasitic diseases invariably affect health status of the elephants as like in other domesticated animals. The manifestation of illness of working elephants many a times is ignored on account of lack of awareness. Therefore, an attempt was made in present study to know the prevalence rate of parasites and to evaluate any alteration in haematological and biochemical variation if any.

Material and Methods

Total 40 elephants that were kept in different temples of Gujarat were screened for their health status. The faecal and blood samples were collected and examined in laboratory as per the standard method described by Sloss and Kemp (1978) as well as by Schalm *et al*. (1975). The biochemical estimations were analysed by using the diagnostic kits supplied by Span Diagnostic Pvt. Ltd., Surat-7, India.

Result

In the present study, all together forty elephants were screened for the prevalence of parasitic infection. Faecal samples from the 25 (62.5%) elephants revealed either single or mixed species of parasitic ova where as 15 (37.5%) samples revealed no parasitic ova. The result of the faecal sample examination is presented in table-1. The over all prevalence of parasitic infection was found as 62.5 per cent. The clinical examination of infected elephant revealed dullness, lachrymation, depression and mild dehydration with semi loose faeces in 12 (48 %) elephants along with the vices of soil licking. The haematological and biochemical parameters of parasitic affected elephants were compared with that other healthy elephant is presented in table -2.

Non significant low level of haemoglobin, total erythrocyte count and packed cell volume were found in elephants harbouring the parasites. Where as a significant ($P<0.05$) eosinophilia was recorded in the elephants harbouring the parasites in the present study. No significant biochemical alteration was observed except non-significant low serum protein level in elephants harbouring the parasites.

Discussion

The study was attempted to know the prevalence of parasitic species of the Indian elephants as well as to understand their impact on the body with reference to haematological and biochemical parameters. In the present study, over all prevalence of parasitic infection was recorded as 62.5 per cent. The single parasitic prevalence was found as 60 per cent where as mixed

Table-1: Checklist of parasitic ova observed from the elephants.

Parasitic spp. ova	Number of infected elephants (n=25)(Species specific prevalence %)	Over all prevalence (Percent)
Fasciolia spp.	06 (24.00)	15.00
Paramphistosomum spp.	04 (16.00)	10.00
Strongyloides spp.	02 (08.00)	05.00
Oesophagostomum spp.	02 (08.00)	05.00
Murshidia spp.	01 (04.00)	02.50
Ascaria spp.	01 (04.00)	02.50
Paramphistomum spp. and *Fasciolia spp.*	03 (12.00)	07.50
Ascaria spp. and *Paramphistosomum spp.*	02 (08.00)	05.00
Fasciolia spp. and *Strongyloides spp.*	02 (08.00)	05.00
Fasciolia sp. and *Ascaria spp.*	01 (04.00)	02.50
Fasciolia spp., *Strongyloides spp.* and *Ascaria spp.*	01 (08.00)	02.50
Normal	15	-
Total	**25**	**62.50**

infection was recorded as 40 per cent. The higher prevalence of *Fasciolia spp*. (15.00 %) was recorded in the present study followed by *Paramphistosomum spp.* (10.00 %), *Strongyloides spp*. (05.00 %), *Oesophagostomum spp*. (05.00 %) and *Ascaria spp.* (02.50 %). Dutta and Bordoloi (1989) recorded 23.33 per cent prevalence of *Fasciolia spp*. from elephants of Manas area, Assam. The high prevalence of fasciolia spp. amongst the different parasites in the present study might be due to the preference of the elephants for water bodies and habit of soil licking. The prevalence of various helminthic parasites of elephants has been documented by several workers from the different parts of India (Deka *et al*., 1985, Rao *et al.,* 1990, Tripathi *et al*., 1997). Apart from this the migration of elephants from one place to other for draught purpose and for other ceremony work could have favoured the mixed parasitic infection with clinical signs of dullness and depression.

Haematological findings of affected elephants with non-significant low haemoglobin, packed cell volume and total erythrocyte count suggested anaemic condition on comparison with healthy elephants, which substantiate the findings of anaemia by Sarode *et al. (*1991). The observed significant eosinophilia in affected group attributed to reflection of hypersensitivity to parasites (Coles, 1986). No biochemical alteration was recorded in elephants harbouring the parasites however, non-significant low serum total protein values were recorded in the present study. Hypo proteinaemia due to parasitic infection in different species have been documented. (Ross and Todd, 1965, Soulsby, 1982).

Table-2: Comparison of Haematological and Biochemical Parameters in parasite harbouring and in healthy elephants.

Parameters	Affected group (n=25)	Control group (n=15)
Haemoglobin (g/dl)	8.96± 0.43*	11.25± 0.78
Total erythrocyte counts (TEC)(X10^6/cu.mm.)	2.42 ± 0.25*	2.64 ± 0.06
Total Leucocyte count (TLC) (X10^3/cu.mm.)	6.66 ± 0.46	6.90 ± 0.18
Packed Cell Volume (%)	24.41± 1.32*	37.67± 0.80
Neutrophil(%)	65.00± 8.02	32.00± 1.38
Lymphocyte(%)	27.62 ± 8.05	61.17 ± 1.58
Eosinophil (%)	16.16 ± 2.15*	03.50 ±0.37
Monocyte(%)	02.02 ± 0.21	03.24 ± 0.86
Basophil (%)	-	-
Total Protein(g/dl)	08.13± 1.12*	09.05 ± 0.31
Cholesterol (mg/dl)	39.16± 1.87	42.50± 1.65
Blood urea Nitrogen (mg/%)	12.10 ± 0.34	13.65 ± 0.49
Blood Glucose(mg%)	59.28 ± 17.76	61.64± 10.25

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References

1. Coles, E. H. (1986): Veterinary Clinical Pathology. 4th Edn. W.B.Saundrs Company, Philadelphia.
2. Deka, D.C., Baruah, D.K., Barkakoti, M.K. and Lahkar, B.C.(1985):Proceeding of the workshop on wildlife health for veterinarian. Wildlife Institute of India, Dehradun.
3. Dutta, G.C. and Bordoloi, G.C. (1989): *Zoo Print* 4: 10: 23.
4. Rao, K.N.P., Bandhopadhyay, A.C. and Gopal, R.(1990): *Zoo Print*. 5: 10: 18.
5. Ross, J.G. and Todd, J.R.(1965): *Br.Vet. J.* 121:55-64.
6. Sarode, D.B., Hatwar, A.K. and Hiwase, S.D. (1991): *Zoo Print* 6: 11: 10-11.
7. Schalm, O.W., Jain, N.C. and Carroll, E.J. (1975): Veterinary Haematology.3rd Edn. Lea and Febiger, Philadelphia, USA.
8. Sloss, M.W. and Kemp, R.L. (1978): Veterinary Clinical Parasitology. 5th Edn. Iowa State University Press, Ames, Iowa, USA.
9. Soulsby, E.J.L.(1982): Helminths, Arthropods and Protozoa of Domesticated Animals. 7th Edn. Baillere and Tindall, London.
10. Tripathi, S.B., Acharjyo, L.N. and Parhi, N.K. (1997): *Indian J.Vet.Med.*17, 1: 48-49.

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Test tube experiments may help identify the most hazardous prion proteins

Mixing up normal and infectious prions in test tubes can generate entirely new forms of infectious prion. Infectious prion proteins from hamsters can change normal proteins from mice into new, infectious forms of prion - simply by mixing the proteins together in a test tube. Researchers at the University of Texas Medical Branch in Galveston suggest their discovery could be turned into a useful test for whether a given prion strain is transmissible from one species to another. Prion proteins are responsible for Creutzfeld-Jakob disease and "mad cow" disease. But they also found that when a prion jumps species, it produces a new kind of prion. "This is very worrisome," says Claudio Soto, who led the research, published in Cell. "The universe of possible prions could be much larger than we thought." Normal prion protein, or PrP, is found throughout the body but is concentrated in the brain. Its exact role is not known, although it has been linked to cell signalling, metal-ion transport, and blood-cell manufacture. The protein can adopt malformed shapes that cause disease. Those proteins, which are resistant to degradation, bind and convert normal protein to their troublesome conformation. Over time, the diseased protein builds up and forms fibrils in the brain, causing neurodegeneration and ultimately death.

Generally, prions are limited to a specific host and a few related species. But prions sometimes cross the species barrier to infect new hosts. Notably, prions from cows have hopped to humans, causing disease in 208 people, mostly in the UK. Now, scientists wonder if the prion-induced chronic wasting disease (CWD), which afflicts elk and deer in the US, could jump to humans. Since prion diseases have long dormant periods, the fact that there are no human cases of CWD doesn't necessarily indicate that people won't develop symptoms in the future. "At this point, we cannot predict the species barrier just by looking at the sequence" of the prion protein, Soto says. But his laboratory has developed a test-tube method, analogous to the polymerase chain reaction for DNA, to amplify prions. Their protein misfolding cyclic amplification (PMCA) protocol starts with a minute amount of prion protein and an excess of normal PrP from healthy brain extract. Over repeated cycles of incubation (allowing the proteins to interact) and sonication (to break those interactions and allow the malformed prions to access other normal protein), the process makes more and more infectious protein.

In the current study, Soto and his colleagues show that the technique allows hamster prions to convert mouse proteins, and vice versa. Although prion infections can pass between hamsters and mice in vivo, the process takes years and only some animals develop disease. "Here, we crossed the barrier between hamsters and mice in a couple of weeks in vitro," Soto says. "In our technology, it's actually more efficient than real life." "It is exciting and interesting that a well-characterized, naturally occurring species barrier to prion infection can be breached without mutation of the PrP sequence," says biochemist Surachai Supattapone, who researches prions at Dartmouth Medical School in Hanover, New Hampshire, and was not involved in the study. "It is also interesting that the newly produced prions display novel strain properties, because this observation is consistent with the idea that naturally occurring prion strains might arise as a result of cross-species transmission." Whereas PrP has one healthy conformation, there are multiple possible shapes that cause health problems. In the study, the new prions caused disease within different time frames, affected different areas of the brain, and showed different resistance to protein-digesting enzymes compared with the original strains. This suggests that new kinds of prion, with potentially differing characteristics, can be born every time a misfolded prion protein lands in a new species.