

FURTHER OBSERVATIONS ON THE PACKED CELL VOLUME AND HAEMOGLOBIN CONCENTRATION IN CATTLE NATURALLY INFECTED WITH *FASCIOLA GIGANTICA*

M. M. R. Howlader, S. Begum, Kh. N. Islam¹, M. A. Hai² and M. G. Hossain³

Department of Physiology, Biochemistry and Pharmacology, Department of Anatomy and Histology¹, Sylhet Government Veterinary College, Tilagor, Sylhet-3100, Bangladesh

ABSTRACT

The changes on packed cell volume (PCV) and haemoglobin (Hb) in zebu cattle infected with *Fasciola gigantica* were studied in two age and two season groups during the period from November 1999 to June 2000. Eighty-eight selected cattle were divided into two age groups, 2.5 to 5.5 years (n = 44; 22 infected & 22 uninfected control) and 6.0 to 9.0 years (n = 44; 22 + 22 cattle), and similarly two seasons, winter (n = 22 + 22) and summer (n = 22 + 22) were used for this study. Blood samples were collected in glass vials contained EDTA anticoagulant for each age group of all animals in summer and winter seasons. PCV was determined using microhematocrit and Hb concentration by cyanomethemoglobin methods. The PCV of *F. gigantica* infected cattle were significantly (p < 0.01) lower than the non-infected control animals. The average PCV obtained were 24.43% and 32.33% for *F. gigantica* infected and non-infected cattle, respectively. The Hb concentrations of infected cattle were significantly (p < 0.01) lower than the non-infected control animals. The average Hb values found were 7.59 and 10.13 g% for *F. gigantica* infected and non-infected cattle, respectively. There was no significant effect of age and season on the values of PCV and Hb concentrations in infected and non-infected cattle.

Key words: Zebu cattle, *Fasciola gigantica*, packed cell volume, haemoglobin concentration

INTRODUCTION

Fascioliasis of cattle, caused by *F. gigantica* is common and wide spread in Bangladesh (Samad, 2000). The disease causes high economic losses through reduced production and increased mortality in cattle, sheep and goats (Kendall, 1954; Boray, 1969). This loss is due to anemia and poor production performance, condemnation of liver and mortality (Kendall, 1954; Hammond and Sewell, 1974; Fabyi and Adeleye, 1982). Numerous hematological and biochemical changes are associated with liver damage caused by liver flukes (Sinclair, 1962). The causes of anemia in fascioliasis have been the subject of study over many years (Stephenson, 1947; Jennings *et al.*, 1956; Pearson, 1963; Todd and Ross, 1966; Holmes *et al.*, 1968; Berry and Dargie, 1978). It is now considered that the flukes feed on host blood. A marked increase in plasma volume occurs during the first several weeks of infection, which coincides with the rapid drop in PCV (Berry and Dargie, 1978). The adult flukes suck more blood in chronic infections and there is leakage of protein through the bile duct epithelium of the host (Dargie and Berry, 1979). A continuous loss of iron into the intestine is associated with an increased plasma iron turn over rate and a reduction in plasma iron concentration. As a result, iron deficiency anemia develops (Berry and Dargie, 1978). To combat this problem efficiently, an understanding of the effect of this parasite on hematological parameters is essential. Although some reports on the blood pictures of fascioliasis in Black Bengal goats (Nooruddin *et al.*, 1982; Howlader and Huq, 1997) and zebu cattle (Mohsin *et al.*, 1991; Khandaker and Chanda, 1998) are available in inland literatures, however, this paper describes the changes of PCV and Hb of naturally *F. gigantica* infected zebu cattle in Sirajgonj district of Bangladesh.

MATERIALS AND METHODS

Eighty eight zebu cattle of two age groups of 2.5 to 5.5 and 6.0 to 9.0 years old were used in this study during the period from November 1999 to June 2000. Faecal samples were collected from a large number of zebu cattle of the study area to select the *F. gigantica* infected and control animals. A total of 44 zebu cattle having *F. gigantica* infection with faecal egg count above 400 epg (egg per gram of faeces) were included as infected group. While a total of 44 cattle apparently healthy and negative for *F. gigantica* and other parasites on microscopic faecal examination were included as non-infected control group. Faecal samples were collected directly from the rectum of each animal at fortnightly interval for a period of one year and examined using Stoll's ova counting technique. Number of egg per gram of faeces was determined by examining five slides for each sample that were prepared with the emulsified faecal solution. The absence of helminth parasite egg for three subsequent fortnightly faecal examinations was considered as gastro-intestinal parasite free animals.

Present address: ²Veterinary Surgeon, Shahjadpur Government Veterinary Hospital, Shahjadpur, Sirajgonj, Bangladesh, ³Thana Livestock Officer, Shahjadpur, Sirajgonj, Bangladesh.

For each age group 22 blood samples were collected in summer (March–June) and winter (November–February) seasons. About 5 ml blood was collected from the jugular vein in glass vials containing EDTA as anticoagulant. Each blood sample collected from the study animals was analyzed in triplicates for PCV and Hb concentrations. The PCV was determined following microhematocrit and Hb by cyanmethemoglobin methods (Coles, 1980).

A two-factor factorial experiment in a randomized complete block design was done. Season and age group were two factors, each with two levels. Comparisons based on the least significant difference at 5% were done between the means of infected and control group in each season and age group.

RESULTS AND DISCUSSION

Faecal examination showed 400 to 1100 *F. gigantica* eggs per gram of faeces (epg) with a mean of 594 epg which indicated a moderate and patent chronic infection. The average PCV values of zebu cattle of two age groups in two seasons infected with *F. gigantica* are presented in Table 1. The PCV values of two age groups of naturally infected animals did not differ significantly ($p > 0.05$) in summer and winter seasons. The infected group showed significantly ($p < 0.01$) lower PCV values than the non-infected control group irrespective of age and season. This difference could be attributed to hemodilution demonstrated by the increase in plasma and blood sucking activity of the adult flukes for a considerably longer period of time. The results of this study were consistent with the observation of Berry and Dargie (1978) who found a marked drop in PCV of sheep infected with *F. hepatica*. Holmes *et al.* (1968) demonstrated that the red blood cell loss per fluke was approximately 0.5 ml per day in *F. hepatica* infection. In the present study, the PCV ranged from 23.80 to 33.30%. This indicates that the infected animals are able to maintain a low level of PCV despite having patent chronic infections.

Table 1. Changes on PCV and Hb in cattle naturally infected with *Fasciola gigantica*

S / N	Seasons	Type of animal	No. of cattle	Age of animals (years)				Overall	
				2.5 to 5.5		6.0 to 9.0		PCV (%)	Hb (g%)
				PCV (%)	Hb (g%)	PCV (%)	Hb (g%)		
1.	Winter	Infected	22	24.20 ^{uA}	7.01 ^{uA}	24.10 ^{uA}	7.85 ^{uA}	24.15	7.43
		Uninfected	22	31.70 ^{bA}	9.75 ^{bA}	32.20 ^{bA}	10.20 ^{bA}	31.95	9.98
2.	Summer	Infected	22	23.80 ^{uA}	7.56 ^{uA}	25.60 ^{uA}	7.95 ^{uA}	24.70	7.76
		Uninfected	22	32.10 ^{bA}	10.05 ^{bA}	33.30 ^{bA}	10.50 ^{bA}	32.70	10.28
Total		Infected	44	24.00 ^{uA}	7.29 ^{uA}	24.85 ^{uA}	7.90 ^{uA}	**24.43	**7.60
		Uninfected	44	31.90 ^{bA}	9.90 ^{bA}	32.75 ^{bA}	10.35 ^{bA}	32.33	10.13

^uAverage of 22 replications. Means in the same column with a common capital letter, and in a row with similar small letters are not significantly ($p > 0.01$) different, **Significant at $p < 0.01$.

The results of this study are in agreement with the findings of Sinclair (1962) who reported that a small number of *F. hepatica* in sheep did not produce clinical signs of adverse effect on the rate of live weight gain in the early stage of infection. The anemia that developed progressed and depressed the productivity of infected animals in the longer period when there are chronic infections. In an earlier study, Sinclair (1962) found that young flukes did not produce anemia in sheep as they live on liver tissues during their early development and migration. Cameron (1951) observed that the parasite produced a toxin which caused damage to the red blood cells, but Urquhart *et al.* (1996) claimed that a sheep weighing 20 kg which infected with 200 flukes would loss 40 ml of blood per day which would develop a progressive type of anemia in the infected animals. In the present study, however, the PCV values showed a moderately lower degree of anemia and this could be attributed to a loss of smaller amount of blood.

The average Hb values of zebu cattle of two age groups infected with *F. gigantica* in two seasons are presented in Table 1. Statistical analysis showed that naturally infected animals registered significantly ($p < 0.01$) lower Hb values than the animals in non-infected control group. There was no significant ($p > 0.05$) difference in Hb concentrations between two seasons within the same group of infected and non-infected animals of two age groups. On the other hand, animals of infected group registered significantly ($p < 0.01$) lower Hb concentrations than the non-infected

control zebu cattle of two age groups in the two seasons. This results indicate that anemia had developed in the zebu cattle of infected group. In the present study, the PCV and Hb concentrations might have brought about by interference with the production of erythrocytes and or by shortening of the life span of erythrocytes that were in agreement with the findings of Sinclair (1962). The continuous drainage of iron stores and reduction in the total number of erythrocytes were thought to be responsible for reduction in haemoglobin level. A continuous production of erythrocyte depended on the presence of raw materials in the blood that circulated through the bone marrow. Adequate supplies of iron, copper, cobalt, vitamins and protein were essential for erythropoiesis and it was possible that the anemia in fascioliosis was caused by a deficiency of one more of these components. Sinclair (1962) found that a change in the plasma protein occurred in ovine fascioliosis, particularly it was marked in the chronic stage of the disease. In this study, the lower Hb concentrations indicated the presence of a lower plasma protein that might be inadequate for normal erythropoiesis and this might have helped to develop anemia.

In this study, all the infected animals were found suffering from chronic infections. The lower PCV and Hb values could be attributed to an abnormal loss of red blood cells due to the feeding habits of the flukes or to an excessive destruction of the red blood cells caused by some hemolyzing factors produced by the flukes. Some researchers reported that blood loss caused by the feeding activities of the flukes as the main factor causing anemia in fascioliosis (Stephenson, 1947; Jennings *et al.*, 1956). However, Sinclair (1962) found that anemia in fascioliosis was due to some other factors. A few studies showed that the flukes caeca contained host's blood (Pearson, 1963; Todd and Ross, 1966). The Hb concentrations of zebu cattle of infected groups showed significantly ($p < 0.05$) lower values than the animals in control group. This could be attributed to a loss of whole blood due to fascioliosis. Dargie *et al.* (1967) reported that a loss of whole blood took place into the gastro-intestinal tract of fluke infected animals. From this study, it may be concluded that chronic *F. gigantica* infection significantly lower the PCV and Hb concentrations in zebu cattle.

ACKNOWLEDGEMENTS

We thank the Director General of the Bangladesh Livestock Research Institute, Savar, Dhaka, for providing the laboratory facilities at the Regional Station, Baghabari, Sirajgonj, to conduct the research in their laboratory.

REFERENCES

- Berry CI and Dargie JD (1978). Pathophysiology of ovine fascioliosis: the influence of dietary protein and iron on the erythrokinetics of sheep experimentally infected with *Fasciola hepatica*. *Veterinary Parasitology* 4: 327-339.
- Boray JC (1969). Experimental fascioliosis in Australia. *Advances in Parasitology* 7: 95-210.
- Cameron TMW (1951). The internal parasites of parasitic animals. 2nd edn., A. and C. Black, London.
- Coles EH (1980). *Veterinary Clinical Pathology*. 3rd edn., W. R. Saunders Company, London.
- Dargie JD and Berry CI (1979). The hypoalbuminaemia of ovine fascioliosis: the albumin metabolism of infected and of paired control sheep. *International Journal of Parasitology* 9: 17-25.
- Dargie JD, Holmes PH, Maclean JM and Mulligan W (1967). Pathophysiology of fascioliosis in the rabbit: studies on albumin turnover. *Journal of Comparative Pathology* 78: 101-105.
- Fabiyyi JP and Adeleye GA (1982). Bovine fascioliosis on the Jos Plateau, Northern Nigeria with particular reference to economic importance. *Bulletin of Animal Health and Production in Africa* 30: 41-43.
- Hammond JA and Sewell MMH (1974). The pathogenic effect experimental infection with *Fasciola gigantica* in cattle. *British Veterinary Journal* 130: 453.
- Holmes PH, Dargie JD, Maclean JM and Mulligan W (1968). The anemia of fascioliosis studies with ^{51}Cr - labeled red cells. *Journal of Comparative Pathology* 78: 415-420.
- Howlader MMR and Huq MM (1997). Haemoglobin concentration and hematocrit value of Black Bengal goats infected with *Fasciola gigantica*. *Asian-Australasian Journal of Animal Science* 10: 118-121.
- Jennings FW, Mulligan W and Urquhart GM (1956). Radioisotope studies on the anemia produced by infection with *Fasciola hepatica*. *Experimental Parasitology* 5: 458-468.
- Kendall SB (1954). Fascioliosis in Pakistan. *Annals of Tropical Medicine and Parasitology* 48: 307-313.
- Khandaker MMU and Chanda PK (1998). Haemato-biochemical changes in *Fasciola gigantica* infected cattle treated with fasciolicide. *Bangladesh Veterinary Journal* 32:136-141.
- Mohsin M, Rahman M, Das PM and Haque AKMF (1991). Haematological observations in cattle naturally infected with *Fasciola gigantica*. *The Bangladesh Veterinarian* 8: 31-34.
- Nooruddin M, Samad MA and Rahman A (1982). A note on certain haematological and biochemical changes of Black Bengal goats infected with *Fasciola gigantica*. *Haryana Veterinarian* 21: 133-136.
- Pearson IG (1963). Use of the chromium radioisotope ^{51}Cr to estimate blood loss through ingestion by *Fasciola hepatica*. *Experimental Parasitology* 13: 186-193.
- Samad MA (2000). An overview of livestock research reports published during the twentieth century in Bangladesh. *Bangladesh Veterinary Journal* 34: 53-149.
- Sinclair KB (1962). Observations on the clinical pathology of ovine fascioliosis. *British Veterinary Journal* 120: 212-222.
- Stephenson W (1947). Physiological and histological observations on the adult liver fluke, *Fasciola hepatica* L. II. Feeding. *Parasitology* 38: 123-127.
- Todd JR and Ross JG (1966). Origin of hemoglobin in the caecal contents *Fasciola hepatica*. *Experimental Parasitology* 19: 151-154.
- Urquhart GM, Armour J, Duncan JL, Dunn AM and Gennings FW (1996). *Veterinary Parasitology*. 2nd edn., Blackwell Science Ltd., UK.