The Bioscan 6(2) : 255-256, 2011
AN INTERNATIONAL QUARTERLY JOURNAL OF LIFE SCIENCES
www.thebioscan.in

THE SPECIFIC ROLE OF LIVER IN EXPELLING *ANCYLOSTOMA CANINUM* LARVAE FROM THE HOST SYSTEM

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KEY WORDS
Liver
Host
Ancylostoma caninum

Received on :
29.12.2010

Accepted on :
17.05.2011

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ABSTRACT
The present study was designed to understand the specific role of liver in the retention and migratory behavior of *Ancylostoma caninum* larvae in male Swiss albino mice. The larvae retention in liver was investigated in male mice infected orally with 500 (group A), 1000 (group B) and 2000 (group C) larvae /mouse. Larval migration in to liver occurred earlier (on day 1 of infection) in all the three groups (A, B and C) and a higher % of larvae migrated in to liver in group A (500 dose) then in groups B and C. interestingly, larvae did not stay in liver from day 9 to 30 in all the three groups.

INTRODUCTION

The role played by liver in expelling and/or destroying the infective *Ancylostoma caninum* larvae and in controlling the severity of ancylostomiasis has received little attention. There have been reports on the migratory pattern of *A. caninum* larvae in oral and sub cutaneous infections in mice (Bhopale and Johri, 1975; Vardhani and Johri, 1981). It was found that about half of the orally administrated larvae migrate through the intestine into liver and lungs and to the musculature. In cutaneous infections, a substantial number of larvae were able to migrate to the musculature directly with out entering the visceral organs. Larval migration and distribution in the different tissues was earlier in mice infected with various multiple doses and more larvae were expelled from the gastrointestinal tract (Bhopale and Johri, 1975). In ancylostomiasis in mice the migratory behavior and survival pattern of larvae was influenced by the passive immunity acquired with the transfer of sensitized peritoneal exudates (Vardhani and Johri, 1983a) and mesenteric lymph node cells (Vardhani and Johri, 1983b). Hookworm infection retards growth and development in millions of children (Holtez and Pritchand, 1995). Enteric infection with *Ancylostoma caninum* is a leading cause of human eosinophilic enteritis in northeastern Australia (Prociv and Croese, 1996). Also, hookworm infection is a major parasitic cause of morbidity in the developing tropical countries (Miller, 1999; Holtez *et al.,* 1999). The risk of atopy was associated with active geohelminth infection among school-age children in rural areas of the tropics (Cooper *et al.,* 2003). Hamsters infected with Ancylostoma *ceylanicum* showed inflamed intestines (Alkazmi and Behnke, 2010).

Studies on the role of liver in hookworm infections are rare. Investigation has been made on the role of liver in expelling *A. caninum* larvae from male Swiss albino mice.

MATERIALS AND METHODS

Procurement of infective larvae, preparation of larval dose and administration of infective larvae were done following the methods reported previously (Vardhani, 1986).Male Swiss albino mice of 6 to 8 weeks old (23-26g wt) were used as experimental animals. Three groups (15 in each group) of each experimental mice were infected orally with 500(group A), 1000 (group B) and 2000 (group C) larvae. Three mice from each infected group were sacrificed on day 1. 4, 9, 16 and 30 after infection for the collection of larvae from the liver. Worm burden from liver was estimated from larvae recovered by Baermann's process.

RESULTS AND DISCUSSION

Larval recoveries from groups A, B and C are summarized in Table 1. No mortality was found from all the groups from day 1 to 30 of experimental period. Total yield of larvae was greater on 1st day in group B (21.0%) than in groups A (16.0%) and C (1.20%) and declined subsequently from day 1 to 4 in groups A(0.8%) and C (0.5%) and from day 1 to 9 in group B (0.4%). Fewer larvae were recovered from liver on day 1 (1.20 %) and 4 (0.50%) in group C which showed complete migration by day 9. No larvae were recovered from liver in group A (500 dose), B (1000dose) and C (2000dose) from day 9 to 30 (except 0.4% of larval recovery on day 9 in group B).

Table 1: Total percentage of *Ancylostoma caninum* larvae recovered at necropsy at different days of infection (values are expressed in mean derived from three observations)

Period of infection	Experimental groups A (500 Dose)	B (1000 Dose)	C (2000 Dose)
1	80 (16.0)	210 (21.0)	24 (1.2)
4	04 (0.8)	10 (1.0)	10 (0.50)
9	-	04(0.4)	-
16	-	-	-
30	-	-	-

The results reveal that the larval retention in the entire experimental group A (500dose), B (1000dose) and C (2000dose) was independent of the dose inoculated. In animals of group A infected with 500 larvae, liver showed maximum retention (16.%) on day 1; similarly animals infected with 1000 larvae (group B) and 2000 larvae (group C) also showed high larval retention on day 1. This is due to the fact that migration of larvae into liver was clearly dependent up on the dose of inoculum. It is worth while to mention that the migratory pattern of larvae into liver was influenced by the degree of immune response stimulated by low (500 larvae) and high (1000 larvae) doses. A substantial number of larval retention (1.2%) in group C (2000 larvae) suggests that animals when inoculated by a high dose, much of the larvae may be expelled from the host system.

According to Alkazmi and Behnke (2010), the heavy doses are unsuitable to stimulate/ stabilize the host's immune response and that may lead to improper migration in the host system. The complete larval migration from liver in groups A and C from day 9 to 30 would suggest that liver acting as an immunological barrier for the retention and/or onward migration of larvae. Velho *et al.,* (2003) also suggested that the heavy burden of hook worms reduced in mice after larval challenging infections.

ACKNOWLEDGEMENT

Thanks are due to UGC, New Delhi (PFship to GS) and for financial support under major research project.

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