

CASE REPORT

An Interesting Case of Visceral Larva Migrans (VLM)

Paresh A Thakkar, Amit Dahat, Omprakash Shukla, Bakul Javadekar
 Department of Pediatrics, Medical College and S S G Hospital, Vadodara, Gujarat, India
Correspondence to: Paresh A Thakkar (pareshthakkar@yahoo.com)

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ABSTRACT

Human toxocariasis is primarily a soil transmitted zoonosis. We report a case of two and half year old male child who presented with fever, anorexia and hepatosplenomegaly. Hemogram showed anaemia (Hb 5.8), leukocytosis (TLC 26,500) and marked eosinophilia (AEC 14,100). Further investigations revealed hypergammaglobulinemia, CT scan of Abdomen showed low density lesions in liver and liver biopsy showed noncaseating epitheloid cell granulomas and infiltrates of lymphocytes and eosinophils. We decided to get a confirmatory serological diagnostic test for *Toxocara* and the result was positive. Child was treated for it with Oral Albendazole and responded dramatically.

Key Words: Visceral Larva Migrans; Eosinophilia; Noncaseating Liver Granuloma

INTRODUCTION

Toxocariasis is a soil transmitted helminthozoonosis due to the infection by ascarid larvae of genus *Toxocara*.^[1] Poor personal hygiene, consumption of raw vegetables grown in contaminated kitchen gardens, geophagia or soil eating is a specific type of pica that increases the risk of toxocariasis, especially in children living in homes with puppies^[2]. Humans are accidental hosts. Physiological reactions to *Toxocara* infection depend on the host's immune response and the parasitic load.^[3] Majority of patients are asymptomatic. Symptoms occur as result of migration of second stage *Toxocara* larvae through the body.^[3-5] *Toxocara* infection commonly resolves itself within weeks. In VLM, larvae migration incites inflammation of internal organs and sometimes the central nervous system. Symptoms depend on the organ(s) affected.^[3]

CASE REPORT

A 2 and a half year old boy, was admitted on 11th august 2011 with chief complaints of abdominal distension since 1month, fever since 15 days and decreased oral intake. There was no history of cough or worm expulsion. In past he had received two unit blood transfusions with no other significant past illness, was immunised upto age. Growth and development were normal. He was born of non consanguineous marriage and with no significant family history. On general examination the child had moderate pallor and vitals were stable. Systemic examination of the abdomen revealed distension with non tender hepatosplenomegaly (liver firm with smooth surface, rounded margin, 7cm below costal margin in midclavicular line, left lobe enlarged 4cm below xiphisternum; spleen enlarged 3 cm along the splenic axis). Other systemic examinations were normal. Ophthalmological evaluation was normal. We considered Differential diagnostic possibilities like tuberculosis, malaria, sepsis, malignancy, storage disorder and langerhan cell histiocytosis. Upon investigation patient had

anaemia (Hb-5.8), leukocytosis (TLC 26,500), differential leukocyte count showed Eosinophilia (DLC-60% Eosinophils) (Absolute eosinophilic count - 14,100). Other investigations like renal profile, coagulogram, urine and stool examination, malarial antigen test serum Widal were normal and HIV, sickling test, Koch's workup and viral markers for hepatitis were negative. USG and CECT abdomen confirmed the hepatosplenomegaly with prominent periportal cuffing (Figure 2 & 3) and few retroperitoneal lymph node enlargements.

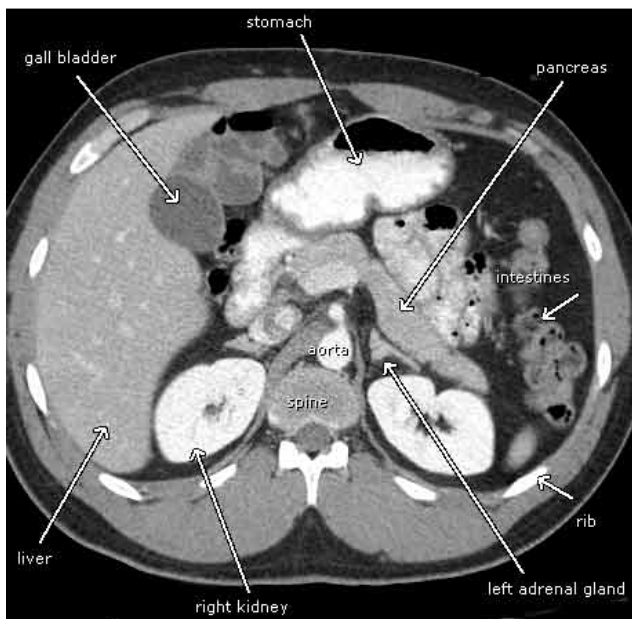


Figure 1: CT slice through the mid-abdomen showing multiple normal-appearing organs



Figure 2: CT scan image of patient showing grossly enlarged liver

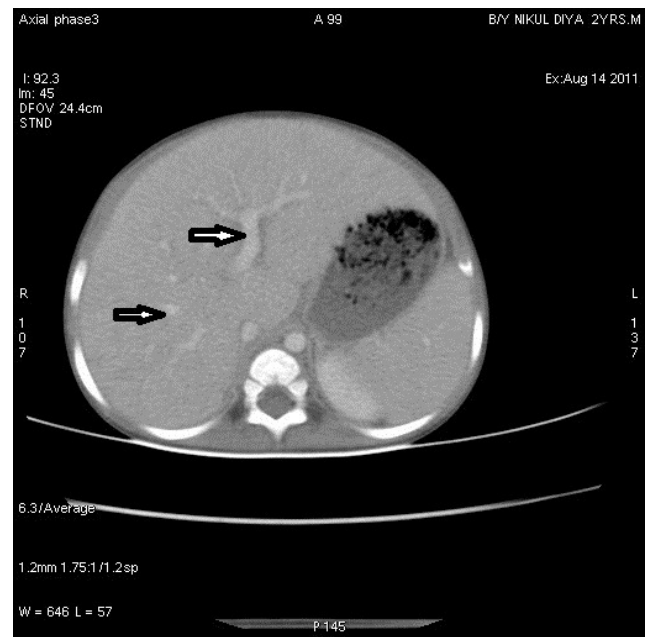


Figure 3: CT scan image of patient showing prominent periportal cuffing

No abnormality was noted in his skeletal survey. A liver biopsy was done and showed non caseating, epitheloid cell granulomas with infiltrates of lymphocytes and eosinophils (Figure 4). Immunohistochemistry of the biopsy was normal. Immunoglobulin assay revealed hypergammaglobinemia (IgG: 2090) after evaluating these investigations, we decided to send *Toxocara* serology which came positive. Thus a final diagnosis of VLM was made. Ophthalmic examination was normal. The child was treated with Albendazole for 2 weeks to which he responded dramatically. His hepatosplenomegaly regressed and eosinophilia gradually subsided.

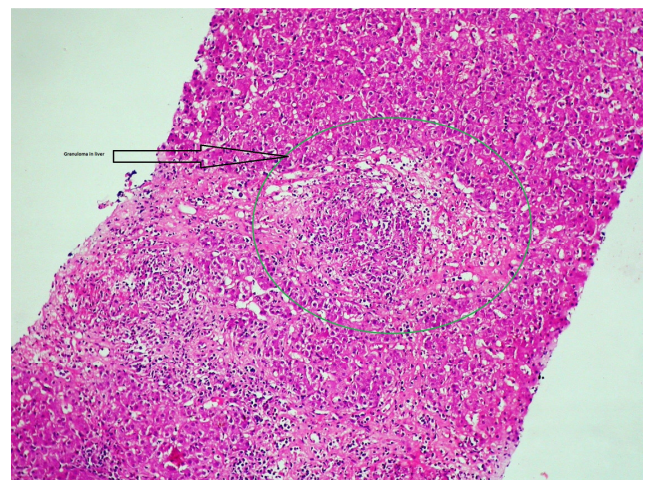


Figure 4: Histology image showing hepatic granuloma

DISCUSSION

VLM is a parasitic infestation caused by larvae of dog roundworm (*Toxocara canis*) or the cat roundworm (*Toxocara cati*).^[1] Other terms used are Weingarten's disease, Frimodt-Moller's syndrome, and eosinophilic pseudoleukemia.^[6] Human *T. canis* infections have been reported in nearly all parts of the world, seroprevalence is higher in developing countries (>50%), but highly underreported due to numerous reasons^[7]. The first description was made in the early 1950's by H.C. Wilder. Two years later, Beaver et al. published the presence of *Toxocara* larvae in granulomas removed from patients^[7,8], since then VLM has been reported from all over the world, although 80% of the dogs in India affected by *Toxocara canis*, to our knowledge there are very few pediatric case reports of VLM from India. *Toxocara* antigens induce immune responses that lead to eosinophilia. The characteristic histopathological lesions are granulomas containing eosinophils, multinucleated giant cells (histiocytes), and collagen. Granulomas are typically found in the liver but may also involve other organs.^[1] Commonly seen in age of 1 - 7 years. The clinical manifestations of *T. canis* infections depend on the number of infective eggs, duration of infection, anatomic location of the larvae and the host immune response.^[1] Most cases are asymptomatic. Symptoms are due to migration of second stage *Toxocara* larvae through the body. There are 3 major clinical syndromes associated with human toxocariasis: VLM, ocular larva migrans (OLM), and covert toxocariasis. The classic presentation of VLM includes Eosinophilia, fever, and hepatomegaly. Other findings include cough, wheezing, bronchopneumonia, anaemia, leukocytosis, and positive *Toxocara* serology. Cutaneous manifestations can also occur. Serologic testing for *Toxocara* had improved the detection rate of VLM in individuals with less obvious or covert symptoms of infection. These children may have nonspecific complaints that do not constitute a recognizable syndrome. Eosinophilia may be present in 50-75% (AEC of >500/ μ L) of cases. A correlation between positive *Toxocara* serology and allergic asthma and epilepsy has also been

described. Diagnosis Supportive laboratory findings include hypergammaglobulinemia. Biopsy confirms the diagnosis. ELISA is the standard serologic test used to confirm toxocariasis. Treatment Albendazole (400 mg bd X 5 days) has efficacy in both children and adults. Mebendazole (100-200 mg bd x5 days) is also useful. Prevention-Transmission can be minimized by public health measures. Children should be discouraged from putting dirty fingers in their mouth and eating dirt.^[1]

CONCLUSION

VLM is not diagnosed commonly in developing countries due to difficulties in defining the parasites in the tissues and the non-availability and/or under-utilization of the available diagnostic methods, combined with a low index of suspicion are probably important underlying factors. Besides this, symptomatology of VLM is similar to the more common and more easily definable tropical parasitic and nonparasitic diseases so that the milder symptoms of VLM are probably ignored by the physicians and the patients alike. We present a case in which quite by serendipity, we made a diagnosis of VLM. To our knowledge this is one of the very few if any, pediatric cases reported of this kind from India.

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