CASE REPORT

Hepatic Visceral Larva Migrans Causing Hepatic Venous Thrombosis and Prolonged Fever

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Background: Visceral larva migrans may present with systemic symptoms such as fever, hepatomegaly, pneumonitis or ocular symptoms. **Case characteristics**: A 7-year-old girl with fever, pain abdomen and persistent eosinophilia. Imaging and histopathology were suggestive of visceral larva migrans. **Message**: The diagnosis of visceral larva migrans is often delayed since similar symptoms of fever, hepatomegaly and peripheral eosinophilia occur in more common and identifiable tropical parasitic and non-parasitic diseases.

Keywords: *Budd chiari syndrome, Fever of unknown origin, Liver abscess, Toxocara canis.*

Visceral Larva Migrans (VLM) is a systemic zoonotic parasitic disease due to migration of second stage larva of *Toxocara canis* or *Toxocara catis* through viscera of human beings. Poor hygiene, contact with dogs and geophagia increases the risk of toxocariasis. Young adults and children who are in close contact with animals are at a higher risk [1]. In VLM, the migrating larva incites inflammation of the organs. The majority of patients are asymptomatic and the infection resolves spontaneously. However in some, the inflammation is severe resulting in morbidity if not treated promptly. The clinical manifestations depend on the location of the larvae, intensity of infection, duration of disease and host immune response. VLM is underdiagnosed since there are no specific symptomatology, ova is not identified in the feces and the findings on imaging are very subtle. We present a 7-year-old girl initially treated as pyogenic liver abscess and later diagnosed as hepatic VLM and effectively managed with albendazole.

Case Report

A 7-year-old girl, resident of Jhansi, Uttar Pradesh, presented with to us history of persistent high grade fever with chills and intermittent pain abdomen for past six months. The pain was localized to right hypochondrium with no aggravating or relieving factors. There was no history of jaundice, black stools, bleeding per rectum, passing worms in stool, loss of weight or pica. There were no pets at home but there were many stray dogs in the neighbourhood. Ultrasonography (USG) and Magnetic resonance imaging (MRI) done at referring hospitals were suggestive of liver abscess, and she had received multiple courses of antibiotics and metronidazole. At admission in our health care facility, child was sick looking, febrile with mild pallor. Examination of the abdomen revealed mild distension with non-tender and firm hepatomegaly. There was no free fluid. Growth, development, and rest of the systemic examination were normal. Based on clinical features and available investigations, we considered possibilities of pyogenic/amebic liver abscess, sepsis or enteric fever and treated her with broad spectrum antibiotics. Investigations revealed anemia (Hb 9 g/dL), leucocytosis (TLC: 17.9×10^9/L), eosinophilia (41%), raised absolute eosinophil count (7.4×10^9/L) and normal liver function tests. Blood culture and Widal test were negative. USG showed an irregular non-liquified necrotic area (103 cc) with echogenic wall and multiple other echogenic lesions in right lobe of liver. Middle hepatic vein was thrombosed with a tubular worm like structure seen within it suggestive of VLM with secondary Budd Chiari syndrome. In addition, a small echogenic partial thrombus was seen in the left portal vein. Contrast enhanced computed tomography (CECT) of abdomen showed large well-defined hypodense lesion with multiple internal septations in liver, showing enhancement in the periphery and septa on the portal phase with evidence of middle hepatic vein thrombosis (***Fig.* 1**). A liver biopsy was done which showed presence of microabscesses composed of eosinophils, which were also found in the sinusoidal spaces (***Web Fig.* 1**). No parasites were identified. The biopsy findings corroborated the radiological findings and a provisional diagnosis of VLM was made. Toxocara serology was not available at our centre and could not be sent to referral centers due to financial constraints. Child was treated with

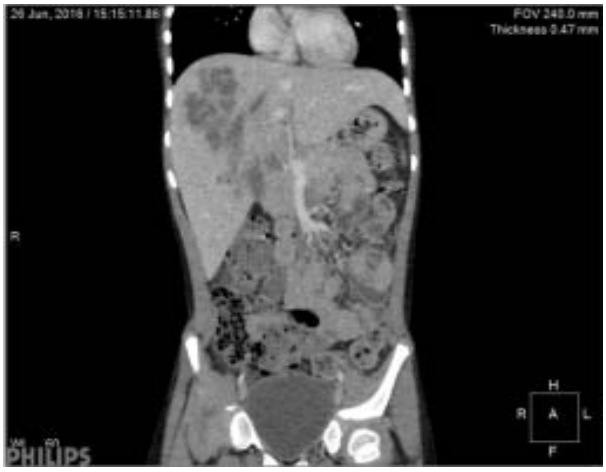

***Fig.* 1** *CECT abdomen showing large well defined clustered hypo-dense lesion with multiple internal septations in liver with enhancement in the periphery and the septa on the portal phase.*

albendazole 400 mg twice daily along with prednisolone 2 mg/kg/day. She improved dramatically and became afebrile within 48 hours of therapy. Prednisolone was stopped and she was discharged after 10 days. Albendazole was continued for 6 weeks as there was recurrence of fever in spite of considerable resolution of hepatic lesions at the 3rd week. After 6 weeks of therapy, USG showed minimal hepatic lesions, and eosinophil count had reduced (AEC 1×10^9/L).

DISCUSSION

In the present case, there were no pets at home but there were plenty of stray dogs in the neighbourhood. The prevalence rates of Toxocara eggs in soil samples in India is reported as 12 % [2]. The larva hatch, penetrate the intestinal wall and travel via the portal vein to reach various organs. Liver is the most common organ to be involved due to portal venous drainage and other involved sites are lungs, heart, eyes and brain. During migration, larva excrete large amount of glycosylated proteins which induce strong immune response leading to eosinophilia and granulomatous inflammation [3]. Most cases are asymptomatic and clinical manifestations depend upon the localization, intensity and chronicity of the infection. As in the present case, the child had been symptomatic for 6 months and there was extensive liver involvement with venous thrombosis. There were no clinical features of Budd Chiari syndrome as only single hepatic vein and a branch of portal vein were blocked. The classic presentation of VLM includes fever, hepatomegaly and eosinophilia as was seen in the present case. Pulmonary involvement may lead to cough, wheeze or pneumonia and neurological involvement may present with headache, seizures or loss of consciousness [4]. The usual laboratory findings include leucocytosis, marked eosinophilia (20% to 70%), raised absolute eosinophil count raised IgE and hypergammaglobulinemia [5]. Imaging forms an important role in diagnosis. As in our patient, eosinophilia pointed towards parasitic infestation but VLM was thought of due to suggestive USG and CT findings. CECT in hepatic toxocariasis usually shows multiple, small, ill-defined, coalescing, low-attenuation nodules, which are best appreciated on the portal venous phase. MRI findings include hypointense lesions on T1 weighted sequence and hyperintense on T2W with peripheral wall enhancement on contrast enhanced T1W images associated with restriction of these lesions on Apparent Diffusion Coefficient (ADC) maps corresponding to diffusion images [6]. The resultant lesion on pathology is marked eosinophilic infilterates also called eosinophilic abscess or granuloma as was found in our patient. Toxocara larvae are rarely found on biopsy [7]. ELISA is the standard serologic test to diagnose toxocariasis [5]. Though these tests are available in India, the cost was a limiting factor in this child.

Anti-helminthic drugs form the mainstay of treatment of VLM. Albendazole is given in a dose of 400 mg twice daily and duration of the treatment depends upon the intensity of the infection [8]. In our patient as there was extensive involvement, she received albendazole for a total of six weeks. Corticosteroids and antihistamines are often used to reduce the inflammation and prevent hypersensitivity. In some cases, there may be no response to the treatment and surgical excision may be needed [9]. VLM is often underdiagnosed due to low index of suspicion and non-availability of the diagnostic methods. VLM may be a cause of prolonged febrile illness and should be suspected in every febrile patient with hepatic involvement and persistent eosinophilia.

Contributors: JK: collected the clinical details and reviewed the literature; AG: managed the patient; NW: supervised the management. All authors were involved in drafting the manuscript.
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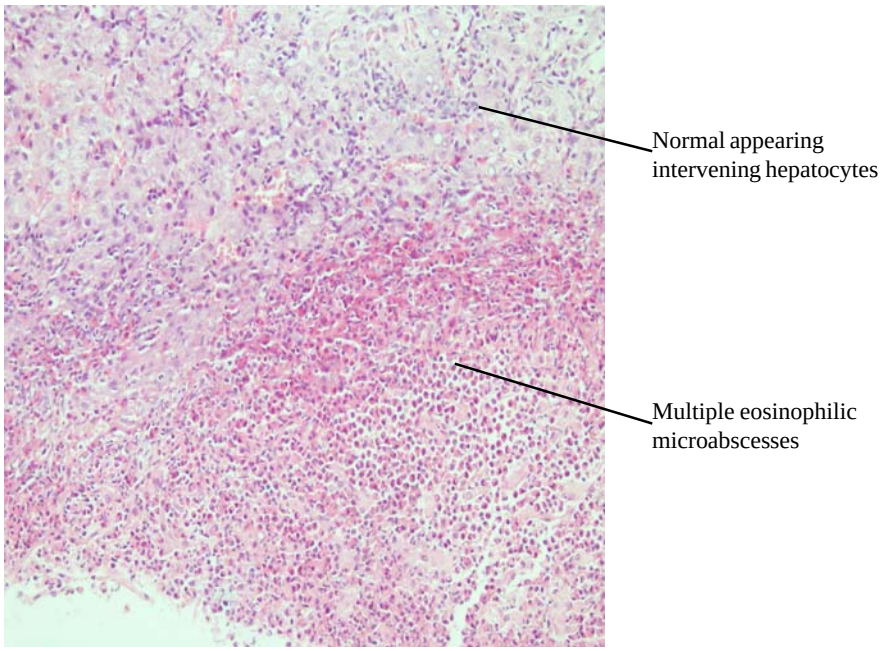

Web Fig. **1** *Liver biopsy showing multiple microabscesses composed of eosinophils and normal intervening hepatocytes.*