

## Hepatic Toxocariasis in a Child: A Case Report from Shiraz, Southern Iran

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**Toxocariasis is a worldwide human helminthiasis. This disease is mostly asymptomatic and caused by *Toxocara canis* and *Toxocara cati*, intestinal nematodes (roundworms) in dogs and cats. These can cause visceral larva migrans syndrome in humans who ingest eggs from contaminated soil or consume of meat of paratenic hosts. A 6-year-old child with fever, chills, pain in right upper quadrant, eosinophilia of 20% and elevated total serum immunoglobulin levels is presented. Ultrasonography demonstrated two hypochoic heterogeneous hepatic lesions measuring 0.7 × 0.7 cm in size located in the right lobe of liver. An enlarged periportal lymph node was noted. The case was diagnosed as hepatic toxocariasis based on sonographic and biopsy findings. The final diagnosis was confirmed by enzyme-linked immunosorbent assay (ELISA) test. It can be concluded that hepatic toxocariasis should be included in the differential diagnosis of multiple liver nodules, particularly in cases with eosinophilia.**

**Keywords:** Hepatic Toxocariasis, Eosinophilic Granuloma, Ultrasonography, Computed Tomography

### Introduction

Toxocariasis is a zoonotic disease caused by the larval stage of *Toxocara* species. Humans are infected by ingestion of embryonated eggs in the soil or through contaminated hands and fomites <sup>(1)</sup>. These clinical entities have been recognized in humans: Visceral Larva Migrans (VLM), Ocular Larva Migrans (OLM) and Covert Toxocariasis (CT).

Most commonly, VLM is a febrile disease of childhood, particularly affecting children between the age of one and five <sup>(2)</sup>. Focal hepatic lesions which are common in VLM and periportal lymph node enlargements have been reported in patients evaluated sonographically <sup>(3)</sup>. We reported serological, pathological and imaging findings of a child with hepatic toxocariasis.

### Case Report

In August 2007, a 6 year-old boy with a history of fever, chill, nausea, fatigue and pain in right upper quadrant was brought to the emergency room. On

complete blood count he showed a Leukocyte count of 9700/  $\mu$ l with marked eosinophilia (20%) (Table 1).

Ultrasonography of the patient's abdomen showed normal liver size and shape but two hypochoic area in right lobe. A computed tomography (CT) revealed a large mass in the posterior segment of the right lobe with extension into the caudate lobe and left lobe with some peripheral early enhancement (Fig. 1). In delayed cuts, enhancement showed a thickened zone then early central parts were not enhanced.

To evaluate the liver findings further, a core liver

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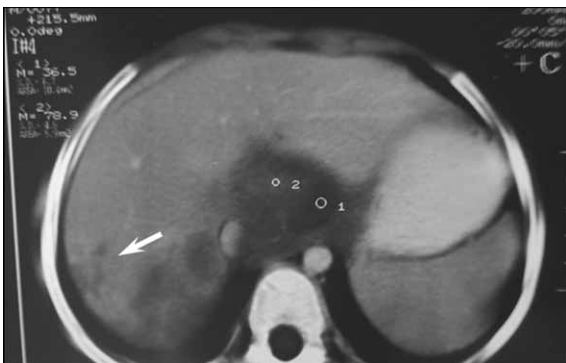
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**Table 1.** The patient's laboratory test results.

Indicator	The patient's values	Normal
White blood Cell count (×1000/μl)	9.7	4-10
Eosinophil	20%	1-10
Hemoglobin concentration (g/dl)	10.8	11.5-18
Platelet count (×1000/μl)	378	150-400
SGOT (IU/L)	24	6-40
SGPT (IU/L)	42	0-35
ALP (IU/L)	310	100-280

SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase, ALP: Alkaline phosphatase.



**Figure 1.** An abdominal CT scan of the patient showing a hypoechoic heterogeneous lesion in the right lobe of the liver (arrow).

biopsy was taken. Histological analysis of the biopsy sample revealed the presence of two round small nodules each measuring: 0.7 × 0.7× 0.7cm and eosinophilic granuloma lesions with central necrosis. This finding was suggestive of visceral larva migrans. A serological test was carried out which detected elevated levels of *Toxocara* in the serum using an enzyme-linked immunosorbent assay (ELISA) that detects antibodies against second stage larvae *Toxocara* excretory- secretory protein. The patient was treated with albendazole 400 mg twice daily for 7 days. After 40 days, the patient's symptoms diminished, laboratory studies revealed reduced leukocytes (7800/ μl) and eosinophilia (2%).

**Discussion**

Toxocariasis is a worldwide distributed helminthzoonosis caused by the infection of humans with second stage larvae of the nematode worms *Toxocara* species. Both *Toxocara canis* and *Toxocara cati* are considered to be causative agents of human toxocariasis (4). The eggs of the worms are excreted in the feces of their definitive hosts, dogs and cats,

respectively. Transmission to humans occurs when infective eggs in soil are ingested by mammalian hosts. These eggs can survive many months in the soil and can infect a wide range of paratenic hosts including humans (5). Children may become affected while playing in sandboxes or playgrounds. Sometimes human beings become infected by eating the meat of paratenic hosts containing encapsulated larvae (6). The disease is more prevalent in children; therefore, the seroprevalence of *Toxocara* infection is estimated to be 4% to 31% in developed countries and may increase to 86% in tropical regions, where environmental conditions favor the transmission of geohelminths (7, 8).

Some clinical features of liver toxocariasis can mimic tumors and may be interpreted histologically as granulomatous hepatitis, eosinophilic infiltrate of the hepatic portal vein, and/or necrotizing eosinophilic abscesses (9, 10). Eosinophilic granulomas have been detected on tissue liver biopsy (11, 12). The symptoms of VLM usually include fever, anorexia, cough, nausea, vomiting and right upper quadrant pain. Hepatic involvement of VLM is common due to portal venous drainage of visceral organs. However, a clinical history with laboratory findings of eosinophilia, a rise in serum total immunoglobulin and a positive serologic test along with histological proof of the affected organ confirm the diagnosis of toxocariasis.

Several studies have evidenced that visceral toxocariasis can be detected by CT and MRI (13-19). The hepatic lesions are seen as low density areas on CT and as high signal areas on T2 weighted images (17). Bhatia and sarin reported that their ultrasonographic findings changed from hypoechoic mass lesions with hyperechoic rims as the disease advanced (13). The hepatic lesions in our patient were homogeneously hypoechoic masses. In the case discussed here, hepatomegaly was not confirmed but two hypoechoic hepatic nodules and enlarged perportal lymph nodes were note. Subsequently, laboratory and histopathologic findings confirmed the diagnosis of toxocariasis.

In conclusion, toxocariasis should be thought of in differential diagnosis of multiple liver nodules, particularly in cases with eosinophilia, and ruled out by imaging technique such as CT and MRI and also by confirmatory laboratory and histopathological findings.

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