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Case Report

Hepatic visceral larva migrans: A radiological case report☆,☆☆

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ABSTRACT

Visceral larva migrans (VLM) occurs due to migration of the second stage of larvae of nematodes through human viscera. It is an underdiagnosed entity which commonly affects the liver as eosinophilic abscesses and appears as coalescing, conglomerated cavities on imaging. This case report details the sonographic and CT features of an 8 year old female patient with right upper quadrant pain and peripheral eosinophilia on laboratory reports, diagnosed as a case of VLM on biopsy. Imaging of VLM shows overlap with neoplastic lesions and other infective pathologies hence this case aims to highlight the clinical, laboratory, and radiological features to help narrow the differential diagnosis.

Background

Visceral larva migrans (VLM) denotes the migratory larval phase of nematodes in humans. Primary hosts for disease-causing nematodes, such as Toxocara canis (T. canis) and Toxocara cati (T. cati), are predominantly dogs and cats [1,2]. Additionally, less recognized parasites, including Capillaria hepatica, Ascaris suum, Baylisascaris procyonis, and various Ancylostoma species, can infect humans, leading to a similar disease pattern, particularly affecting the liver [3]. Human transmission of the infection occurs through accidental ingestion of embryonated eggs in soil or the arrested second-stage larvae of nematodes from animal host tissues, often present in consumed meat. This transmission route mirrors parasitic infestations seen in primary animal hosts like dogs and cats. As humans are not the primary hosts, ingested eggs or larvae can only mature into migrating larvae, subsequently released into the small bowel. These larvae then penetrate the portal venous system through the intestinal walls, systematically migrating to infest the liver, lungs, brain, heart, and eyes [4,5]. The migratory process and resulting clinical symptoms collectively characterize this larva as VLM.

Abbreviations: VLM, visceral larva migrans; CT, computed tomography; MRI, magnetic resonance imaging; CBC, complete blood count; LFT, liver function test; SGPT, serum glutamic pyruvate transaminase; SGOT, serum glutamic oxaloacetic transaminase; GGT, gamma-glutamyl transferase; US, ultrasonography.

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VLM presents in the liver as eosinophilic granulomas, representing a lesser-known manifestation of parasitic liver abscesses. These granulomas, characterized by necrotizing lesions, present as indistinct, merging, and occasionally distinct focal lesions with imaging features resembling generic abscesses or resembling cystic/liquefied masses. Over successive imaging sessions, these lesions may dynamically change in shape and position, consistent with the characteristics of migratory larva disease [6]. Sonographically, these lesions typically manifest as multiple rounded or oval hypoechoic lesions, ranging from a singular sub-centimetre focal area to a confluent mass large enough to involve an entire liver lobe.

The imaging characteristics of these lesions on contrast-enhanced computed tomography (CT) or magnetic resonance imaging (MRI) involve poorly defined enhancing walls surrounding conglomerating liquefied lesions, appearing predominantly oval, rounded, or asymmetrical during the venous and equilibrium phases [6]. A distinctive attribute of these abscesses in noncontrast MRI studies is the existence of a hyperintense rim on T1-weighted sequences, coupled with evident diffusion restriction observed in echo planar imaging [7].

The imaging characteristics of VLM on CT and MRI provide suggestive but noncharacteristic information, requiring correlation with laboratory parameters such as antigen serology and eosinophilic counts [1–3]. The gold standard for diagnosis involves cytology or biopsy from the lesions [6,7]. VLM exhibits a global prevalence, with a higher incidence in developing countries [1]. While case series and reports exist on VLM, detailing radiological findings through sonography, CT, and MRI [8–11], the available literature lacks long-term follow-up studies assessing the therapeutic response of hepatic VLM.

Case presentation

Herein, we discuss the case of an 8 year old female child who presented to the outpatient department with complaints of dull aching abdominal pain, and undocumented on and off low grade fever for the last 2 months. Child's parents denied any history of tuberculosis in the child or in the family. Dietary history revealed that she was nonvegetarian.

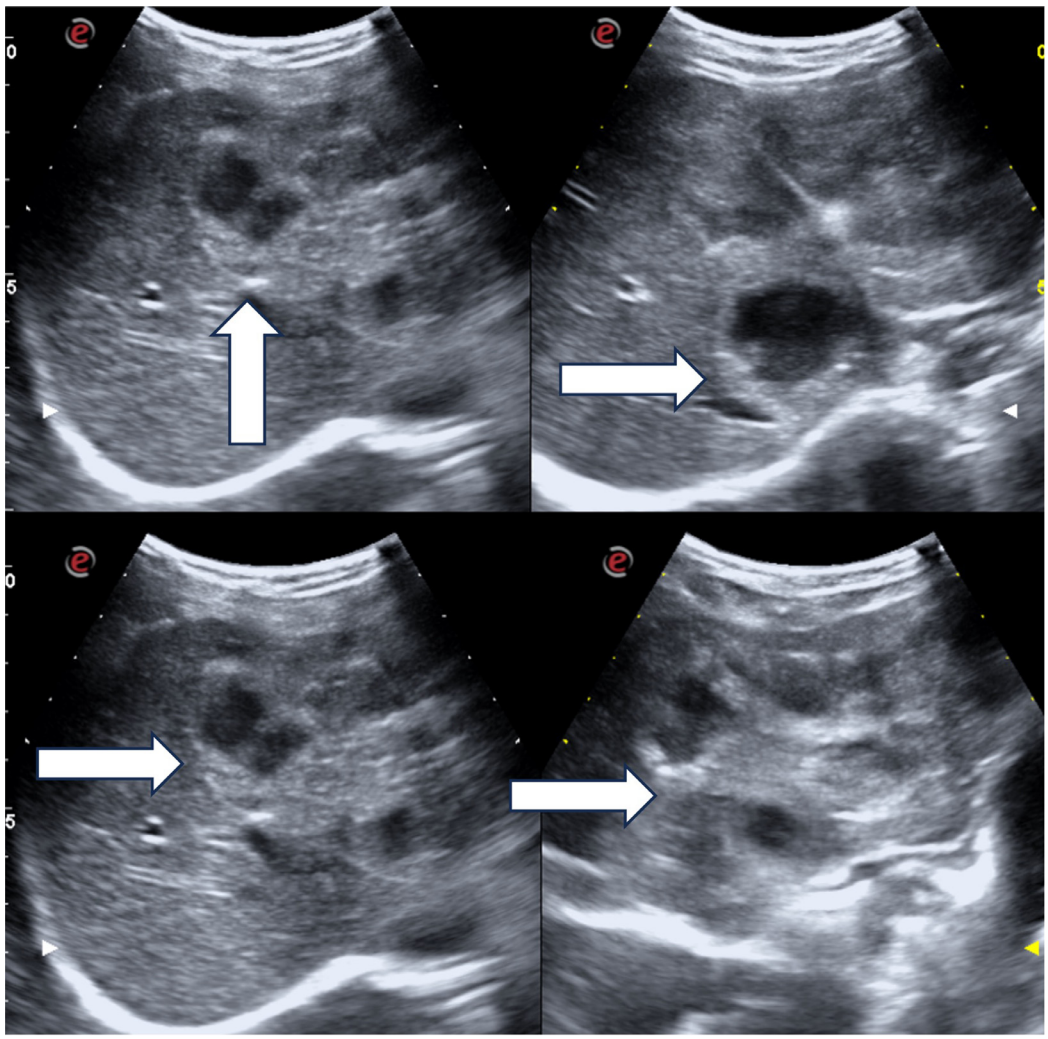

Fig. 1 – Shows ultrasound images of the liver. There was a conglomerated mixed density lesion involving the left lobe showing anechoic areas within (block white arrows).

Upon clinical examination, there was presence of hepatomegaly, while all other examination parameters were within normal limits.

CBC revealed a raised total leukocyte count of 14.7 × 10^3/uL (Normal range 5 × 10^3 - 11 × 10^3/uL). The differential count showed increased eosinophil count of 27 % (normal range 1%-4%). LFT revealed Total bilirubin level of 0.23 mg/dL (normal range 0.3-1.3 mg/dL), direct bilirubin level of 0.12 mg/dL (normal range of 0.1-0.4 mg/dL), SGOT-53.8 U/L(normal range <40 U/L), SGPT-52.3 U/L (normal range <40 U/L), Alkaline phosphatase- 625U/L(normal range 35-130 U/L), and GGT- 157U/L (normal range 10-48U/L). Mantoux test was negative.

She was then referred for an ultrasound scan of the whole abdomen. The scan revealed conglomerated solid-cystic hypoechoic lesions in the caudate lobe and left lobe of liver (Fig. 1).These lesions were subjected to colour doppler evaluation and showed no significant vascularity.

For further evaluation, the patient underwent a contrast enhanced CT scan of the abdomen which revealed a conglomerated multiloculated hypodense lesion with peripheral rim enhancement seen in the left lobe and caudate lobe with minimal adjacent biliary radicle dilation likely secondary to mass effect (Figs. 2 and 3).

Few enlarged peri-pancreatic and retro-peritoneal lymph nodes were also seen.

An ultrasound (US) guided biopsy was planned and under local anesthesia 3 cores of tissue were taken from the hepatic lesion and sent for histopatholgical examination.

The tissue sections were prepared and stained with hematoxylin and eosin stain. The micrograph revealed fragmented cores of liver parenchyma showing necrotizing granuloma with numerous Charcot Leyden crystals within dense inflammatory infiltrates comprising lymphocytes, plasma cells, and eosinophils (Fig. 4). These findings were consistent with an Eosinophilic abscess which pointed towards a diagnosis of hepatic visceral larva migrans.

She was then started on Albendazole therapy 400 mg, twice a week for 3 weeks and showed symptomatic improvement.

Discussion

Hepatic visceral larva migrans is a rare parasitic infection caused by the migration of larvae, commonly Toxocara species, into the liver. This case report presents a distinctive radiological perspective on VLM, shedding light on the diagnostic challenges, and imaging findings associated with this condition.

The imaging modalities employed in this case, including abdominal ultrasound and computed tomography (CT), played a pivotal role in establishing the diagnosis. The characteristic features observed on imaging, such as hypoechoic lesions with irregular borders on ultrasound and low-density areas on CT, are consistent with the literature on hepatic VLM. Notably, these findings may mimic other hepatic pathologies, necessitating a comprehensive diagnostic approach. The radiological differential diagnosis included eosinophilic abscess/hepatic visceral larva migrans, alveolar echinococcosis, hepatic mesenchymal hamartoma, and undifferentiated embryonal sarcoma. The latter 2 were excluded based on the clinical background and the cytological and serological findings and echinococcosis was excluded as echinococcal serology was negative in this patient.

The patient's clinical history, including exposure to contaminated soil and close contact with pets, aligns with known

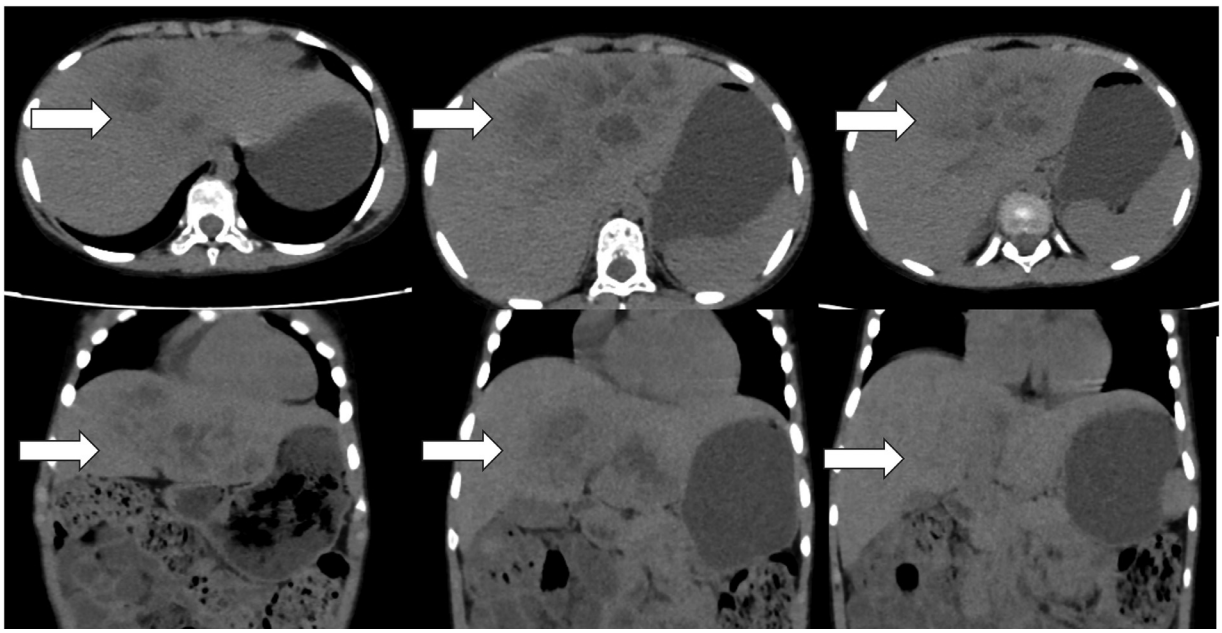

Fig. 2 – Axial (first row) and coronal (second row) non contrast CT images of the abdomen showed an ill-defined hypodense lesion in the left lobe of the liver. No calcifications were seen.

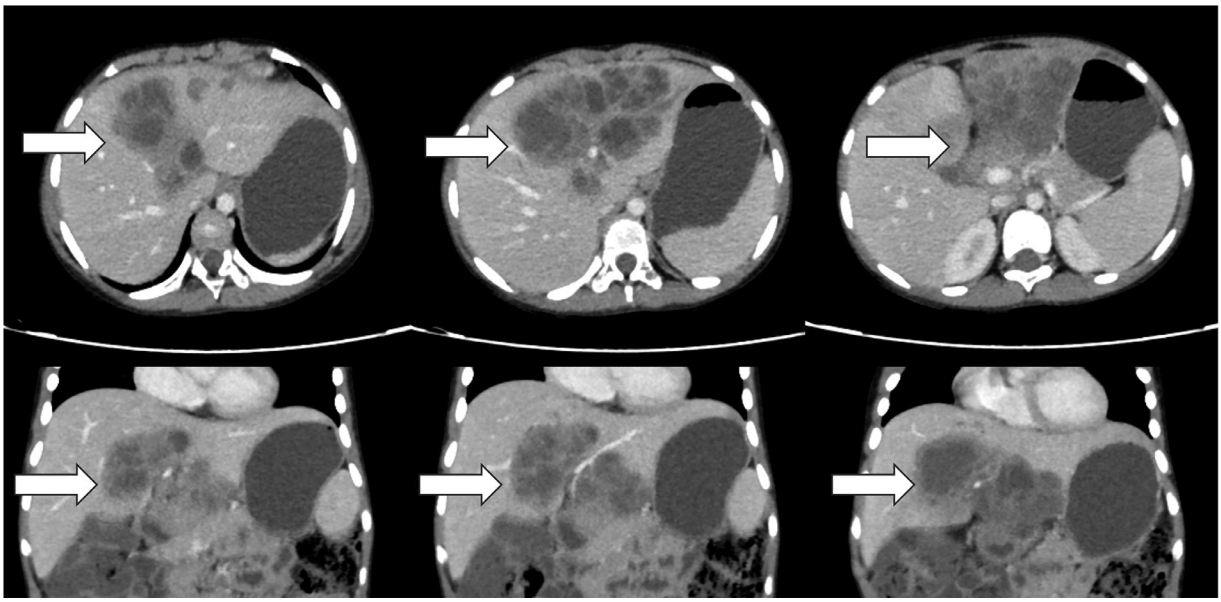

Fig. 3 – Axial and coronal CECT images showed conglomerated multiloculated hypodense lesion with peripheral rim enhancement seen in the left lobe and caudate lobe (block white arrows).

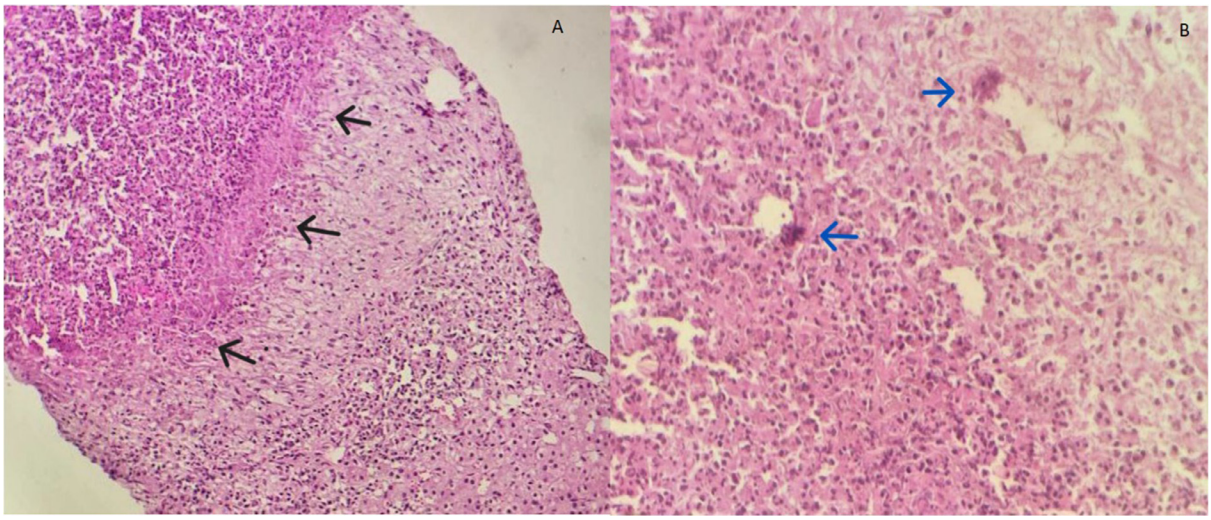

Fig. 4 – (A) Micrograph (Haematoxylin-eosin stain, original magnification, 100X) and (B) Micrograph (Haematoxylin-eosin stain, original magnification, 400X) revealed fragmented cores of liver parenchyma showing necrotizing granuloma with numerous Charcot Leyden crystals (blue arrows) within dense inflammatory infiltrates comprising lymphocytes, plasma cells and eosinophils (black arrows). These findings are consistent with an Eosinophilic abscess.

risk factors for Toxocara infection. This emphasizes the importance of considering parasitic etiologies in patients with compatible symptoms and imaging findings, particularly in regions where such infections are prevalent.

Furthermore, the radiological evaluation in this case underscores the need for a multidisciplinary approach involving radiologists, clinicians, and parasitologists. The correlation between imaging findings and serological tests can enhance diagnostic accuracy and guide appropriate treatment strategies. Serological testing for specific antibodies, in conjunction with imaging, contributes to a more comprehensive understanding of the disease.

Conclusion

In conclusion, this radiological case report highlights the significance of considering VLM in the differential diagnosis of hepatic lesions, especially in patients with relevant exposure history. The integration of imaging, clinical data, and serological tests is crucial for accurate diagnosis and timely initiation of appropriate therapeutic interventions. Continued research and collaboration between disciplines are essential for enhancing our understanding of VLM and improving patient outcomes.

Patient consent

I state that written and informed consent was taken from the patient's father for publication of this case.

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