Abdominal Imaging

Published online: 17 July 2012

Abdom Imaging (2013) 38:774–777
DOI: 10.1007/s00261-012-9935-x

Eosinophilic abscesses: a new facet of hepatic visceral larva migrans

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Abstract

Hepatic visceral larva migrans (VLM) refers to a condition characterized by granulomatous liver lesions containing eosinophils and inflammatory cells associated with migration of second-stage larvae of certain nematodes such as toxocara canis. The typical imaging findings described in the literature include small, ill-defined, oval or elongated, low-attenuating nodules with fuzzy margins, non-spherical shape, and absent or insignificant rim enhancement on contrast-enhanced CT scan. The present series in contrast depicts a new imaging manifestation of hepatic VLM presenting as confluent and clustered complex cystic liver lesions. Pre-treatment imaging studies including contrast-enhanced CT/MRI of three patients are presented. One of the patients underwent liver resection while post-treatment follow-up scan at 6 months in the remaining two displayed regression of the lesions with antihelminthic treatment.

Key words: Visceral larva migrans—Eosinophilic abscess—CT—MRI

Visceral larva migrans (VLM) refers to the migration of second-stage larvae of certain nematodes such as toxocara canis through the human viscera (human beings being the accidental host). It is acquired via the fecal–oral route mostly due to the ingestion of eggs or encapsulated larvae [1]. From the gut, the nematode enters the portal circulation and reaches the liver. Its slow transit within the liver parenchyma incites an inflammatory response coupled with eosinophilic infiltration leading to eosinophilic granuloma or sometimes eosinophilic abscess formation [2, 3]. Eosinophilic granulomas are characterized by hepatic infiltration of eosinophils and other inflammatory cells along with palisade of giant cells and epithelioid histiocytes with central necrosis, whereas, eosinophilic abscess is a lesion containing copious eosinophils along with destroyed liver parenchyma and associated inflammation [2, 3]. Imaging findings of hepatic VLM has been reported in certain case reports, series, and few studies as well, nevertheless some imaging features are under recognized, or more precisely, not been reported [3–8]. Here, we present three cases of VLM having multiple well-defined cystic lesions in liver which were histopathologically proven to be eosinophilic abscesses.

Case 1

A previously healthy 14-year-old girl presented with vague long standing upper abdominal discomfort predominantly located in the epigastria. Clinical examination revealed non-tender hepatomegaly. An ultrasonogram revealed complex predominantly cystic mass lesion in the left lobe of liver. The lab parameters were unremarkable except for marked eosinophilia (18%). Contrast-enhanced CT of abdomen revealed a multi-cystic coalescent lesion in the left lobe (Fig. 1), which was also confirmed at MR imaging. The diagnostic possibility of VLM was foremost owing to marked eosinophilia, however, other differential including cystic biliary neoplasm was also considered. Fine needle aspiration cytology of the lesion favored eosinophilic abscesses (Fig. 1) and subsequent serological investigations (with ELISA) for toxocara canis (IgG anti-toxocara canis) came positive. The patient was initiated on antihelminthic treatment and showed good response in the

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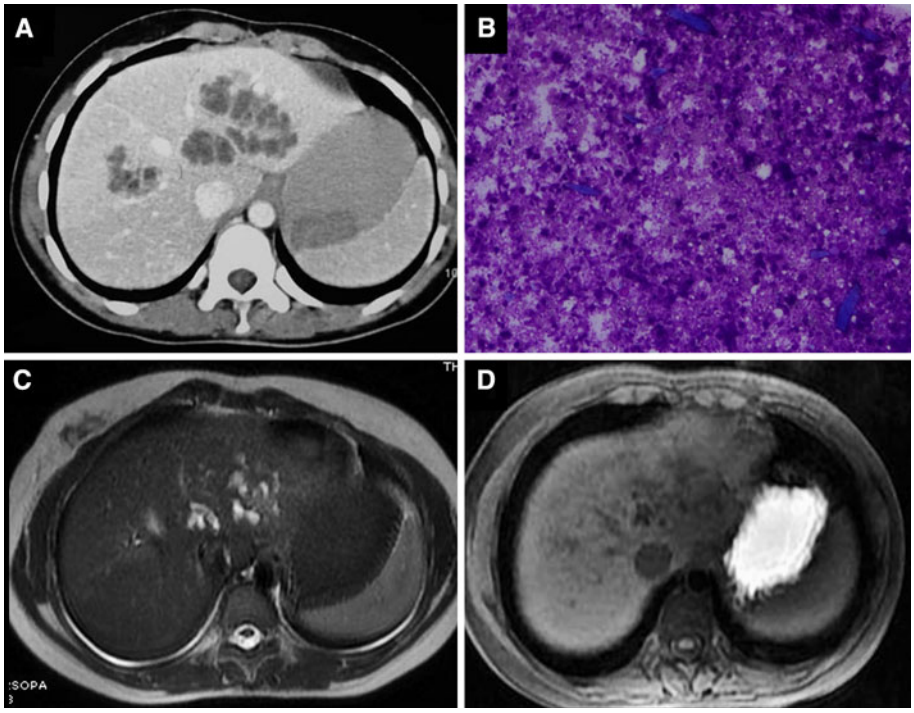

Fig. 1. Axial contrast-enhanced CT scan of a 14-year-old female patient (**A**) (portal venous phase) shows clustered cystic lesions in both lobes of the liver, Geimsa-stained smear (**B**) showing degenerated inflammatory cells, including large number of eosinophils along with Charcot–Leyden crystals (×100). Follow-up MRI scan (6-month), **C** T2- and **D** T1-weighted images showing significant reduction in the size and number of lesions.

form of symptomatic improvement and lesion regression on follow-up scans (Fig. 1).

Case 2

A 30-year-old female presented to the outpatient department with a history of pain abdomen for 3–4 years and accompanied by low-grade intermittent fever. There was no history of jaundice, vomiting, upper or lower gastro-intestinal bleed. Routine blood investigations including complete blood count, liver function tests were unremarkable except for marginally raised eosinophil count (6%). Contrast-enhanced CT abdomen showed mild left lobe atrophy within which there were nodular hypo-attenuating lesions having variable internal density (Fig. 2). Based on CT findings, a likelihood of hepatic Hydatid cyst was given along with differentials of hepatic VLM and biliary cystadenoma. MRI of the upper abdomen and MRCP was also performed before surgery and revealed the larger lesion to be hyperintense on T1-weighted and hypointense on T2-weighted images. Conversely, the smaller lesion was hyperintense on T2-weighted and hypointense on T1-weighted images. Considering all three possibilities, decision of left hepatectomy was taken. Surgery revealed firm lesions in segments 2 and 3 and on resection the lesions were found to contain cheesy material. On histopathology, these were confirmed to be eosinophilic abscesses (Fig. 2). Subsequent serology revealed high titer of toxocara antibodies.

Case 3

A 58-year-old male presented to the emergency department with complaints of severe right upper quadrant pain and fever with chills. Bedside screening, ultrasound revealed a few complex cystic lesions in both lobes of the liver. His complete blood count showed leukocyte count of 9000/μL with severe eosinophilia (38%). He was advised regarding a contrast-enhanced CT scan, but, in lieu of some minor reaction to contrast media during a previous investigation, MRI of the abdomen was performed. MRI showed multiple well-defined complex cystic lesions having lobulated outlines in the right lobe of the liver (Fig. 3). A few of them had irregular wall with internal heterogeneous contents. On T1-weighted images, the lesions had varying intensity ranging from hypo- to hyperintense as compared to the normal liver parenchyma, while on T2-weighted images cystic areas were brightly hyperintense and the wall and solid components were isointense to slightly hyperintense. Based on these imaging findings along with severe eosinophilia, a possibility of VLM was suggested. Subsequently, fine needle aspiration of the lesion confirmed it to be eosinophilic abscess. The diagnosis of VLM was confirmed by serum titer evaluation for toxocara canis antibodies. Follow-up scan performed after 6 months of antihelminthic therapy showed reduction in both the size and number of lesions (Fig. 3C, D).

Discussion

Humans are accidental hosts of *Toxocara*, the parasitic larvae of canine or feline roundworms. The parasite's eggs are passed from animal feces into the soil where the larvae hatch and remain viable and infectious for many years owing to their extreme resistance to damage and desiccation [1, 2]. Humans get infected on ingesting food material contaminated by eggs of Toxocara and the

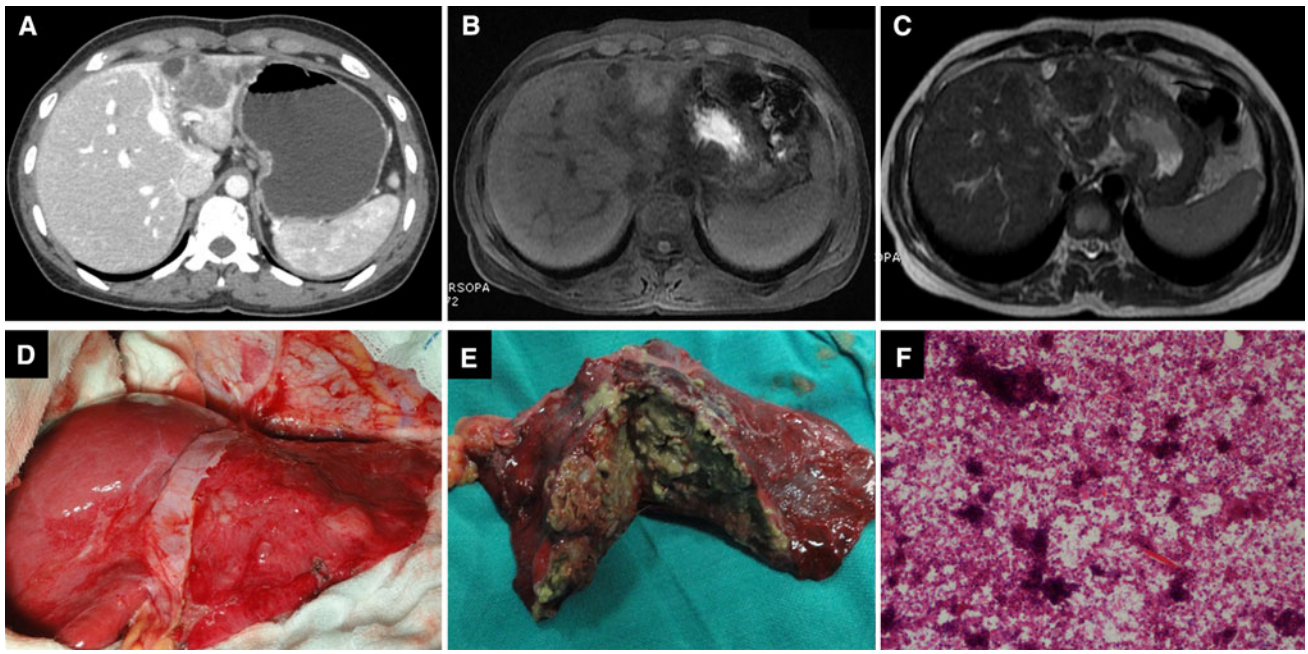

Fig. 2. Contrast-enhanced CT (portal venous phase) of 30-year-old female patient (**A**) shows small left lobe with nodular hypodense lesions. Corresponding MR scan displaying varying signal intensities on **B** T1- and **C** T2-weighted images. Intraoperative photograph (**D**) showing lesion in the left lobe of the liver and cut section of specimen (**E**) showing intralesional pultaceous material. H&E-stained smear (**F**) showing degenerated inflammatory cells, including large number of eosinophils along with Charcot–Leyden crystals (×100).

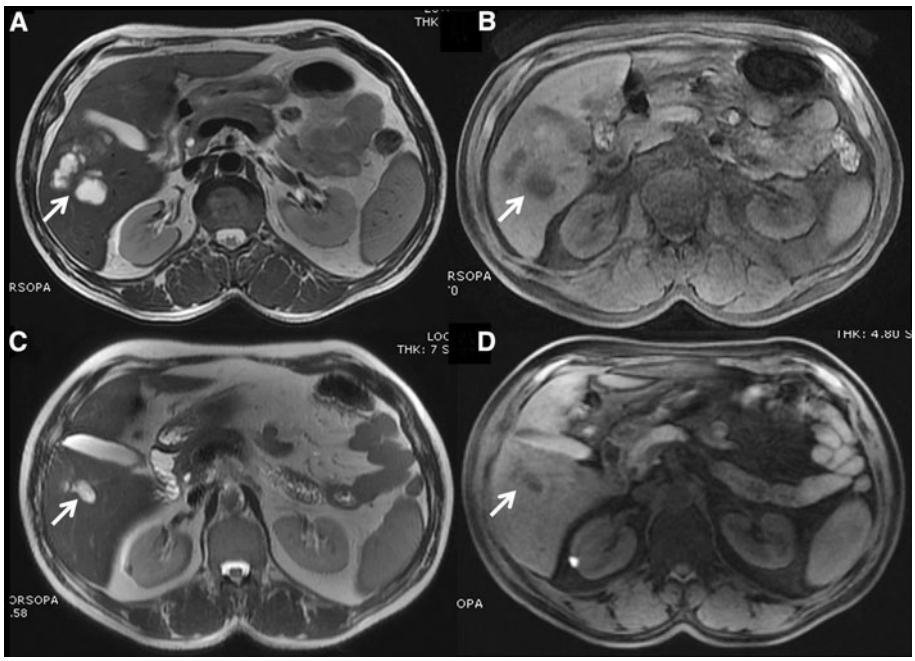

Fig. 3. Axial MR scan of 58-year-old male patient, **A** T2- and **B** T1-weighted images exhibiting variable signal intensities within the lesions. Follow-up (6 months) MR images (**C** T2 and **D** T1) of the same patient showing near total resolution of the lesions.

infection is particularly common in young children with a history of pica. The infection can also be acquired from playgrounds and sandboxes [3, 4]. Once ingested, the eggs hatch and larvae penetrate the gut wall and via the blood stream migrate around the body hence the term VLM. The larvae, which migrate around the body, cause infiltration and involvement of various tissues such as the liver, heart (causing myocarditis), skin, and the CNS (causing dysfunction, seizures, and coma) [5–7]. Since the larvae are unable to develop further and complete their life cycle in humans, they die within few weeks of invasion.

Hepatic VLM refers to a condition characterized by granulomatous liver lesions containing eosinophils and inflammatory cells associated with severe eosinophilia. Mostly this entity is caused by larva of genus toxocara; however, it is also reported to be caused by few species of ascaris and ancyclostoma [1]. The described imaging findings of this particular condition as per the present literature include small, ill-defined, oval or elongated, low-attenuating nodules on contrast-enhanced CT scan which are best appreciated on the portal venous phase [8–13]. The VLM nodules are described to have fuzzy margins and non-spherical shape with no or insignificant rim enhancement; these features help to differentiate them from other nodular hepatic lesions like metastatic deposits [11, 12].

In contrast to the above-described imaging features of VLM, we encountered three cases in which lesions were relatively well marginated and had a definite cystic component. In one of the case, only MRI was done while the other two patients underwent both CT as well as MR studies. All patients had multiple well-defined but conglomerate complex cystic lesions of varying sizes in liver. These lesions exhibited variable signal intensities on T1- and T2-weighted images. Some of the lesions were hyperintense on T1-weighted and hypointense on T2-weighted images, whereas others were hypointense on T1-weighted and hyperintense on T2-weighted images with respect to the normal liver parenchyma. The lesions displayed variable diffusion restriction and showed faint enhancement of the walls on administration of contrast (Gd-BOPTA, Multihance, Bracco Diagnostics, Inc.). CT study revealed well-defined clustered cystic lesions and the lesions were best visualized on portal venous phase of contrast-enhanced study. In all our cases, liver lesions were sharply defined and demonstrated a definite cystic component. These findings are contrary to previous reports describing VLM lesions to be subtle, poorly discernable and having ill-defined fuzzy margins [8–13]. Although none of the features seen in our cases are typical of hepatic VLM, they do illustrate a distinctive picture which has not been previously reported. The possible explanation for this imaging finding could be severe inflammatory response along with accumulation of eosinophils in the liver secondary to the passage of larva further leading to formation of sterile cystic areas. The variable MR appearance is possibly due to different age of the lesions along with varying protein content within the abscesses. These were subsequently established to be eosinophilic abscesses on fine needle aspiration cytology/post-surgery histopathology.

Conclusion

The present report demonstrates an atypical imaging manifestation of hepatic VLM presenting as confluent and clustered complex cystic liver lesions; rather, it might seem more apt to label them as hepatic eosinophilic abscesses secondary to toxocara canis infestation.

Conflict of interest None.

References

1. Beaver PC, Jung RC, Cupp EW (1984) *Clinical parasitology*, 9th edn. Philadelphia: Lea & Febiger, pp 320–329
2. Rubinsky-Elefant G, Hirata CE, Yamamoto JH, Ferreira MU (2010) Human toxocariasis: diagnosis, worldwide seroprevalences and clinical expression of the systemic and ocular forms. Ann Trop Med Parasitol 104(1):3–23
3. Carvalho EA, Rocha RL (2011) Toxocariasis: visceral larva migrans in children. J Pediatr (Rio J) 87(2):100–110
4. Akdemir C (2010) Visceral larva migrans among children in Kütahya (Turkey) and an evaluation of playgrounds for *T. canis* eggs. Turk J Pediatr 52(2):158–162
5. Rubinsky-Elefant G, Hirata CE, Yamamoto JH, Ferreira MU (2010) Human toxocariasis: diagnosis, worldwide seroprevalences and clinical expression of the systemic and ocular forms. Ann Trop Med Parasitol 104(1):3–23
6. Enko K, Tada T, Ohgo KO, et al. (2009) Fulminant eosinophilic myocarditis associated with visceral larva migrans caused by Toxocara canis infection. Circ J 73(7):1344–1348
7. Kaplan KJ, Goodman ZD, Ishak KG (2001) Eosinophilic granuloma of the liver: a characteristic lesion with relationship to visceral larva migrans. Am J Surg Pathol 25:1316–1321
8. Kim GB, Kwon JH, Kang DS (1993) Hypereosinophilic syndrome: imaging findings in patients with hepatic involvement. AJR 161: 577–580
9. Chang S, Lim JH, Choi D, et al. (2006) Hepatic visceral larva migrans of Toxocara canis: CT and sonographic findings. AJR Am J Roentgenol 187(6):W622–W629
10. Leone N, Baronio M, Todros L, et al. (2006) Hepatic involvement in larva migrans of Toxocara canis: report of a case with pathological and radiological findings. Dig Liver Dis 38(7):511–514
11. Azuma K, Yashiro N, Kinoshita T, Yoshigi J, Ihara N (2002) Hepatic involvement of visceral larva migrans due to Toxocara canis: a case report—CT and MR findings. Radiat Med 20(2): 89–92
12. Lim JH (2008) Toxocariasis of the liver: visceral larva migrans. Abdom Imaging 33(2):151–156
13. Kakihara D, Yoshimitsu K, Ishigami K, et al. (2004) Liver lesions of visceral larva migrans due to *Ascaris suum* infection: CT findings. Abdom Imaging 29(5):598–602