



Hepatic Visceral Larva Migrans Treated by Hepatic Lobectomy: A Case Report

Fadi Rayya^{1*}, Reem Shamoo², Raiean Al Habba³, Sami Alashi³ and Assad LW²

¹Department of General Surgery, Al-Assad University Hospital, Syria

²Damascus University, Syria

³Damascus Hospital, Syria

Abstract

Visceral Larva Migrans (VLM) is a systemic manifestation of migration of second stage larvae of nematodes through the tissue of human viscera. It is not uncommon but is under diagnosed in developing countries. The liver is the most common organ to be involved due to its portal venous blood supply. The imaging findings are subtle and differentiation from Hepatocellular Carcinoma (HCC), metastases, cystic mesenchymal hamartoma and granulomatous diseases is difficult. This case report highlights the imaging features along with pathological findings of hepatic lesion of VLM along with clinical and laboratory data which help in reaching the final diagnosis.

Keywords: Visceral larva migrans; Hepatic lobectomy; HCC

Introduction

There are three parasites that can cause VLM syndrome, the most common is *Toxocara canis* followed by *toxocara catii*, and less commonly caused by *Ascaris lumbricoides*, *Capillaria hepatica* and *Ascaris suum* [1,2].

Several studies have shown that factors such as dog ownership, rural residence and low sociological status carry an increased risk of *T. canis* infection [3,4].

Definitive hosts, which include humans, are infected with VLM by ingesting the embryonated ova which cause the disease. The ova reach their digestive system directly from the soil, or by eating uncooked meat of an infected animal. Cases of parasite transmission were reported through transplacental or transmembrary transmission of embryonated ova. When the embryonated ova hatch, the larvae are released into the small intestine, where they can penetrate the intestinal wall and reach the bloodstream. After reaching the bloodstream, it is obvious that they can reach different parts of the body. In humans, these second-stage larvae are unable to fully mature and reproduce, and they are destined to be excreted with the stool mass [5]. VLM caused by *T. canis*, a syndrome occurring mainly in young children, is characterized by chronic eosinophilia and hyperglobulinemia with the involvement of lungs, liver, eyes, the central nervous system, skin and the heart as a consequence of local larval migrations [6].

The symptoms of VLM usually include fever, hepatomegaly, abdominal pain, vomiting, and diarrhea, coughing/wheezing, asthma, in appetite, weight loss, fatigue and headache [7].

We report a case of a woman with hepatic involvement in VLM, diagnosed after undergoing a Hepatectomy by the pathological examination.

Case Presentation

A 30-year-old married female with one kid from a rural area, presented to the hospital with right liver mass along, fever since two and half months, weight loss (seven kilograms during three months) and intermittent pain at the right hypochondrium. The pain was not associated with food intake. There was no vomiting, black stool or bleeding per rectum.

Physical examination and past medical history

The patient was slightly pale and had hepatomegaly, but no significant lymphadenopathy.

Physical examination shows no discoloration of the skin or jaundice. Cardiovascular, pulmonary, and extremities examination was normal.

OPEN ACCESS

*Correspondence:

Fadi Rayya, Department of General Surgery, Al-Assad University Hospital, Syria,

E-mail: fadirayya@yahoo.com

Received Date: 22 Apr 2019

Accepted Date: 05 Jun 2019

Published Date: 10 Jun 2019

Citation:

Rayya F, Shamoot R, Al Habbal R, Alashi S, Assad LW. Hepatic Visceral Larva Migrans Treated by Hepatic Lobectomy: A Case Report. *Clin Surg*. 2019; 4: 2461.

Copyright © 2019 Fadi Rayya. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Past medical history was significant for treated Brucellosis 10 years ago and a cesarean section a year and half ago. Patient mother has familial Mediterranean fever and suffer from chronic kidney disease.

Prior to admission, the patient visited gynecologist and during the examination ultrasonography shows a hypoechoic ill-defined mass measuring 6 cm at the right liver lobe.

Abdominal Computed Tomography (CT) at an outside institution showed a low-density liver lesion extending from the fourth segment (LSg IVA) to the seventh segment (LSg VII) in a posterior anterior diameter of 6 cm without calcifications or increased blood supply, associated with and slight expansion of the intrahepatic bile ducts in the right lobe. The rest of the liver tissue was normal.

Investigations

Hematological examination was within normal levels except an elevation in white blood cell due blood eosinophilia (blood eosinophil count being 8.8%). All liver function parameters being normal. Renal functions and coagulation profile were normal. HBs Ag, Anti HCV, Wright and Widal were all negative. Serum α -fetoprotein was within normal limits (5 ng/mL) other tumor markers (CEA, CA19-9) were normal.

On admission to our institution a repeated abdominal computed tomography (CT); revealed a mass which is located in the right hepatic lobe. It occupies the eighth hepatic segment (LSg VIII) and the upper medial sections of the LSg IV, measuring [10 cm] and a diameter [3.5 cm \times 4 cm], and was associated with the expansion of the bile ducts in the right lobe and press a little the Inferior Vena Cava (IVC), In addition to some lymphadenopathy in the hilum of the liver and behind the head of the pancreas, measuring [2.2 cm]. The radiological differential diagnoses included hepatic malignancies such as hepatocellular carcinoma, cholangiocarcinoma, hepatoblastoma and metastases along with cystic mesenchymal hamartoma, sarcoidosis and tuberculosis.

Surgery

Because of the patient weight loss and 10 cm liver mass with high suspicious of malignancy; the patient underwent right hepatectomy. The postoperative period was uneventful. The patient left the hospital on the 7th postoperative day in good condition.

Pathological findings

Microscopic examination of the sample showed granulomatous inflammation with central necrosis and surrounding palisading fibroblasts and inflammatory cells. The central necrosis containing degenerating inflammatory cells and eosinophils associated with charcot laden crystals, the stroma surrounding the granuloma shows abundant numbers of eosinophils. The granulomas coalesce and appear as large multifocal mass lesion which explains the imaging result before surgery.

The differential diagnosis of the granulomatous inflammation in liver includes tuberculosis, sarcoidosis, cat scarch disease, fungal infection and helminthes disease.

Special stains and testing exclude most of the above cases. However, the presence of peripheral eosinophilia, eosinophils at the periphery of the granuloma, necrosis with Charcot-Leyden spindle-shaped crystals that develop from degranulated eosinophils support our diagnosis of hepatic visceral larva migrans.

After conveying the diagnosis to the physician on the case; the patient confirmed that she has close contact with stray dogs and cats, which are a prominent risk factor for the above diagnosis [8].

Treatment

The patient was given 400 mg of albendazole twice a day for 3 weeks.

Outcome and follow-up

The patient was totally relieved of her symptoms after surgical and medical treatment. The patient is symptom free two years after treatment.

Discussion

Visceral Larva Migrans (VLM)–is defined as inflammatory lesions in human tissues produced by the migratory stages of larvae of certain nematode parasites. VLM is caused by larva stages of *Toxocara canis* and *Toxocara cati*, both of which are roundworms found in the intestines of dogs and cats. The other causative organisms implicated are *Ancylostoma caninum* of dogs, and *Ascaris suum* of pigs [9]. Dog hair and soil also contain infective stage eggs, and infection can occur when eggs containing fully developed larvae are swallowed. Our patient appears to have contracted the infection either via stray dogs of the neighborhood or infected soil. The visceral organs affected include the liver, lungs and the central nervous system. Hepatomegaly is a frequent finding in VLM. Focal hepatic lesions, periportal lymph node enlargement and splenomegaly have been reported in patients evaluated sonographically [10].

After hatching in the intestine, the larvae penetrate the intestinal wall, flow through the portal vein and reach the liver, lungs, orbit and brain. Some larvae move slowly in the liver (VLM) or become encapsulated and remain in that state with no further growth for an indefinite period [11]. Our patient was not imaged at intervals and hence the migratory nature could not be documented. There are various non-specific symptoms of VLM including general malaise, cough and liver function disorders [12]. Our patient also had malaise.

The chronic production of parasite antigens, continuous stimulation of host immune system and concomitant production of eosinophils can cause systemic complications; liver being the most common site for these lesions because of its portal drainage [13].

The mechanism of liver infiltration is believed to be an allergic response to the larva [14]. The lesions are most conspicuous in portal venous phase and tend to be in the periphery of the liver and along the portal vein branches. Eosinophil count and percentage in the peripheral blood are considerably higher in the patient group with hepatic lesions than in the group without hepatic lesions. However, eosinophil count and percentage do not correlate with the number of hepatic lesions on CT [15]. Some malignant neoplasms such as lymphoma, leukaemia and carcinoma are often associated with eosinophilia [16].

The liver is the visceral organ most commonly affected and this is what happened with our patient. To our knowledge, CT findings of hepatic toxocariasis were first described by Dupas B et al. in 1986 [17-19]. As hepatic toxocariasis has a nonspecific appearance on a CT scan, it can be mistaken for another diagnosis. The differential diagnosis for multiple low-density liver nodules also includes micro abscesses, other granulomatous diseases (i.e. sarcoidosis), fibrolamellar hepatocellular carcinoma or liver metastases [20]. Stool

examination for ova and parasites is generally not helpful because the infective juvenile larvae do not multiply in paratenic hosts and samples containing intact *Toxocara* larvae are rarely found and are not required for a diagnosis to be made.

Conclusion

We present an unusual case of hepatic VML presenting with a large mass, occupying the right hepatic lobe with hepatic damage and radiological findings suspicious of carcinoma, warranting surgery for treatment and diagnosis.

Hepatic Visceral Larva Migrans and other parasitic liver infections should be included in the differential diagnosis of a low-density liver lesions associated with fever and chills. We all should be aware of hepatic *toxocariasis* when we face an ill-defined hepatic lesions, biliary dilatation, and sludge and periportal lymph node enlargement. Eosinophilia is a useful clue but is not completely specific for VLM. This will help in protecting the patient from unnecessary surgery [21-22].

References

- Brandborg L. Parasitic disease of the liver. In: Zahim DB, TD, editors. *Hepatology*. Philadelphia: Saunders; 1982.p.1010-35.
- Gayotto L, DaSilva L. Ascariasis, visceral larva migrans, capillariasis, strongyloidiasis and pentastomiasis. In: McIntyre N, Benhamou J, Bircher J, editors. *Oxford Textbook of Clinical Hepatology*. Oxford: Oxford Medical Publications; 1991.p.730-39.
- Holland CV, O'Lorcain P, Taylor MR, Kelly A. Sero-epidemiology of toxocariasis in school children. *Parasitology*. 1995;110 (5):535-45.
- Kimmig P, Naser K, Frank W. [Seroepidemiologic studies of human toxocariasis]. *Zentralbl Hyg Umweltmed*. 1991;191(4):406-22.
- Despommier D. Toxocariasis: clinical aspects, epidemiology, medical ecology, and molecular aspects. *Clin Microbiol Rev*. 2003;16(2):265-72.
- Takata I. Experimental infection of man with *Ascaris* of man and the pig. *Kitasato Arch Exp Med*. 1951;23(4):151-9.
- Stoicescu RM, Mihai CM, Giannakopoulou AD. Marked hypereosinophilia in a toddler: a case report. *J Med Life*. 2011;4(1):105-8.
- Glickman LT, Schantz PM. Epidemiology and pathogenesis of zoonotic toxocariasis. *Epidemiol Rev*. 1981;3:230-50.
- Nichols RL. The etiology of visceral larva migrans. I. Diagnostic morphology of infective second-stage *Toxocara* larvae. *J Parasitol*. 1956;42(4):349-62.
- Baldisserotto M, Conchin CF, Soares Mda G, Araujo MA, Kramer B. Ultrasound findings in children with toxocariasis: report on 18 cases. *Pediatr Radiol*. 1999;29(5):316-9.
- Baever PC, Jung RC, Cupp EW. *Clinical parasitology*, 9th edn. Philadelphia, PA: Lea & Febiger;1984.p.320-9.
- van Knapen F, Buijs J, Kortbeek LM, Ljungström I. Larva migrans syndrome: toxocara, ascaris, or both? *Lancet*. 1992;340(8818):550-1.
- Kayes SG. Human toxocariasis and the visceral larva migrans syndrome: correlative immunopathology. *Chem Immunol*. 1997;66:99-124.
- Sakai S, Shida Y, Takahashi N, Yabuuchi H, Soeda H, Okafuji T, et al. Pulmonary lesions associated with visceral larva migrans due to *ascaris suum* or *toxocara canis*: imaging of six cases. *AJR*. 2006;186(6):1697-702.
- Chang S, Lim JH, Choi D, Park CK, Kwon NH, Cho SY, et al. Hepatic visceral larva migrans of *toxocara canis*: CT and sonographic findings. *Am J Roentgenol*. 2006;186(6):W622-9.
- Wasserman SI, Goetzl EJ, Ellman L, Austen KF. Tumor-associated eosinophilotactic factor. *N Engl J Med*. 1974;291:420-4.
- Duprey ZH, Schantz PM. Toxocariasis and Baylisascariasis. In: Richardson DJ, Krause PJ, editors. *North American Parasitic Zoonoses*. Boston, MA: Springer US; 2003.p.23-40.
- Dupas B, Barrier J, Barre P. Detection of *Toxocara* by computed tomography. *Br J Radiol*. 1986;59(701):518-9.
- Hirata T. [Ultrasonographic findings of hepatic granuloma by *Toxocara cati* migration]. *Nihon Igaku Hoshasen Gakkai Zasshi*. 1987;47(12):1606-8.
- Azuma K, Yashiro N, Kinoshita T, Yoshigi J, Ihara N. Hepatic involvement of visceral larva migrans due to *Toxocara canis*: a case report--CT and MR findings. *Radiat Med*. 2002;20(2):89-92.
- Jackson G, Kathuria M, Abraham B, Schnadig VJ. Fine needle aspiration diagnosis of necrotizing eosinophilic abscess clinically mimicking hepatic neoplasia: a case report. *Acta Cytol*. 2010;54(1):60-2.
- Rohilla S, Jain N, Yadav R, Dhaukhandi DB. Hepatic visceral larva migrans. *BMJ Case Rep*. 2013;2013.