With increasing use of EUS as a diagnostic and therapeutic technique, it can provide an unique access to vascular structures in the mediastinum and abdomen. EUS probes provide high quality 2D resolution, color doppler, and pulsed doppler images. Aortic floating thrombus is a rare, life-threatening disease with predisposing abnormal coagulation function, aortic disease, and history of aortic stent implantation. Echoendoscopic evaluation of proximal aorta can confirm the diagnosis in distal embolic phenomenon, endoscopic ultrasonographers to extend the horizons of EUS beyond the limits of current practice

References

1. Tsilimparis N, Hanack U, Pisimisis G, Yousefi S, Wintzer C, Rückert RI. Thrombus in the non- aneurysmal, non atherosclerotic descending thoracic aorta - An unusual source of arterial embolism. Eur J VascEndovasc Surg. 2011;41:450–7.
2. Yves GhislainAbissegue, Youssef Lyazidi, Hassan Chtata, Tarik Bakkali, and Mustapha TaberkantAcute systemic embolism due to an idiopathic floating thrombus of the thoracic aorta: success of medical management: a case report. BMC Res Notes. 2015; 8: 181.
3. Blangetti I, Fenoglio L, Avallato C, Bertora M, Novali C, Peinetti F, *et al*. Transesophagealecocardiography: the correct intraoperative Way to detect the source of peripheral embolism in an emergency. Ann Vasc Surg. 2013;27(8):e13–1185.
4. Oh JK, Seward JB, Tajik AJ. Transesophageal and Intracardiac Echocardiogrphy. In: The Echo Manual, 3rd edition, Lippincott Williams & Wilkins. 2007. p.29-30.
5. American College of Cardiology Foundation Appropriate Use Criteria Task Force, American Society of Echocardiography, American Heart Association, *et al*. J Am Coll Cardiol. 2011; 57:1126.
6. Turley SR, Unger J, Cox MW, Lawson J, McCann RL, Shortell CK. Atypical aortic thrombus: should nonoperative management Be first line? Ann Vasc Surg. 2014;28(7):1610–7.

Hepatic Visceral Larva Migrans with Atypical Manifestations: A Report of Three Cases

Saloni N. Desai[1], Sudheer S. Pargewar[1], Nitesh Agrawal[1], Chhagan Bihari[2], S. Rajesh[1]

[1]Department of Radiology, [2]Department of Pathology, Institute of Liver and Biliary Sciences, New Delhi, India.

Corresponding Author: *Dr Saloni N. Desai*
Email: saludesai@gmail.com

The term visceral larva migrans (VLM) is used to describe the migration of second-stage larvae of certain nematodes through the human viscera.[1] These nematodes pass through the intestinal wall and travel with the blood stream to various organs where they cause inflammation and damage. Affected organs can include the liver, lung, heart and the central nervous system. The larvae are known to move slowly through the affected organs (hence the term migrans), the resulting inflammationcausing multiple oval to cigar-shaped eosinophilic granulomas or abscesses.[2] This entity should be considered in the differential in patients with sustained eosinophilia showing the typical imaging findings. We further report secondary complications of portal vein thrombosis and abscess rupture in these patients which has so far not been documented in literature.

Case 1

A 55-year old female came with the chief complaints of intermittent high grade fever and pain in right upper quadrant since 1 month. Her complete blood count revealed normal white blood count (9400/µL, normal range 4-11) with eosinophilia (18%). Ultrasound showed the presence of multiple conglomerated predominantly hyperechoic lesions in right lobe of liver (**Figure 1A**). Further, MRI (**Figure 1B - F**) was done which revealed multiple diffusion restricting lesions in right lobe of

liver characteristically distributed along portal radicles. These were predominantly T1 hypointense and T2 hyperintense, however, some of them also demonstrated T1 hyperintensity with corresponding hypointense T2 signal. Peripheral enhancement on portal venous phase after administration of hepatobiliary specific contrast agent (Gd-BOPTA, Multihance, Bracco Diagnostics Inc.) was observed. Additionally, complete thrombosis was noted in adjacent segment V branch of right portal vein. A diagnosis of eosinophilic abscesses with adjoining segmental portal vein thrombosis was suggested on the basis of distribution of the lesions and eosinophilia. Fine needle aspiration showed cellular smears containing large number of inflammatory cells with Charcot-Leyden crystals consistent with VLM (**Figure 2**).

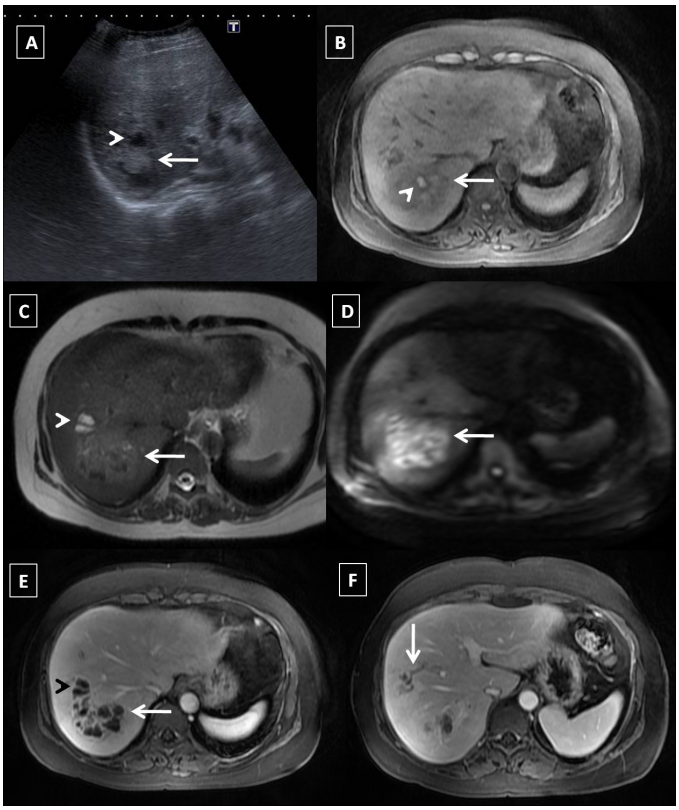

Figure 1: A 55-year old female came with fever and right upper quadrant pain. (A) Gray-scale ultrasound image showing heterogeneous, predominantly hyperechoic mass lesion in the right lobe of liver (arrow) with few cystic areas (arrowhead). (B) T1-weighted axial MR image showing a hypointense mass (arrow) with few hyperintense foci (arrowhead). (C) T2-weighted axial MR image demonstrating a hyperintense mass (arrow) with cigar-shaped cystic lesions within (arrowheads). (D) The lesion shows diffusion restriction (arrow). (E) Peripheral enhancement is seen within this conglomerate mass (arrow). (F) Segmental thrombosis of segment-V portal venous radicle is seen secondary to the lesions (arrow).

Case 2

A 53- year old female presented with intermittent fever with chills since 20 days. Her routine blood tests were unremarkable except for eosinophilia (14%). Preliminary ultrasound revealed multiple confluent hypoechoic lesions consistent with features of abscesses in right lobe of liver with perihepatic fluid collection along the dome of liver. Contrast MRI (**Figure 3**) demonstrated multiple confluent loculated peripherally enhancing lesions along portal tracts in right lobe of liver. Also, capsular breach with T1 hypo/T2 hyperintense subphrenic collection and mild right sided pleural effusion were noted. A possibility of VLM with a differential diagnosis of amoebic abscess with contained rupture and reactionary right pleural effusion was suggested. Serological investigation (with ELISA) for Entamoeba (IgG) was negative while that

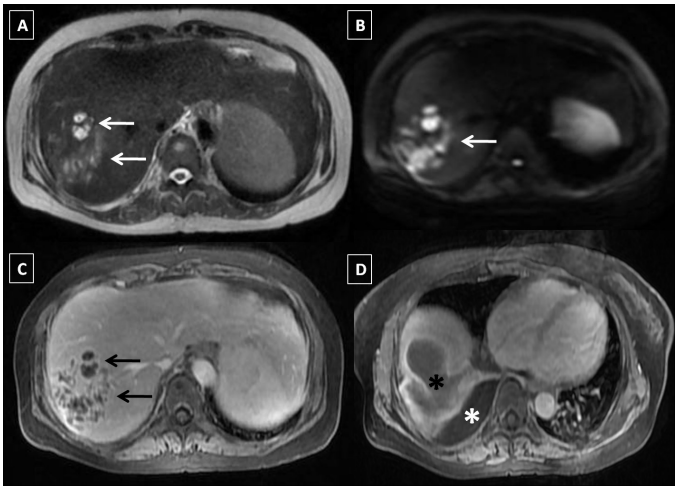

Figure 2: 53-year old female with intermittent high grade fever. (A) Axial T2-weighted MR image shows conglomerate hyperintense cystic lesions (arrows) showing diffusion restriction (arrow in B). (C) Peripheral enhancement is seen in these lesions (arrows). (D) Subcapsular rupture of the lesions is seen (black asterisk) with right pleural effusion (white asterisk).

for toxocaracanis (IgG antitoxocaracanis) came positive and a confimatory diagnosis was made on fine needle aspiration cytology.

Case 3

A 31-year old male presented with complaints of upper abdominal pain, off and on fever and loss of appetite for 1 month. Laboratory investigations revealed raised white blood cell count (14000/µL, normal range- 4-11) with marked eosinophilia (58%). Ultrasound revealed multiple, relatively well-defined, discrete as well as coalescing heteroechoic lesions involving the liver. Contrast-enhanced computed tomographic scan (CECT) demonstrated multiple fluid-attenuating round to oval conglomerate lesions involving the right liver lobe (**Figure 4A and B**) for which imaging differentials of eosinophilic or amoebic abscess were suggested. Fine needle aspiration confirmed the diagnosis and subsequent serological investigations (with ELISA) for toxocaracanis (IgG antitoxocaracanis) came positive. He was treated with albendazole leading to a significant regression in the size of the lesions (**Figure 4C and D**).

Discussion

Toxocaraspecies, the ascarid of dogs and cats, is the most common culprit causing VLM in humans.[1] Infection can be acquired by ingesting food material contaminated by eggs of Toxocara and primarily occurs in children aged 1-4 years, but can occur at any age. The larvae hatch in the small intestine, invade the mucosa, and enter the portal system. The liver traps some larvae, but other larvae proceed to the lungs and the circulatory system, where they can disseminate to virtually every organ. In particular, the larvae deposit in the liver, lungs, eye, heart, and brain.[2-5] However, the parasite cannot complete its life cycle in humans. Larvae persist in tissues, provoking a granulomatous reaction and eventually dying. As a result, abscesses or granulomas form.

Liver is the most involved site, and presents in the form of eosinophilic granulomas or abscesses usually mimicking tumoral processes.[6] Eosinophilic granulomas

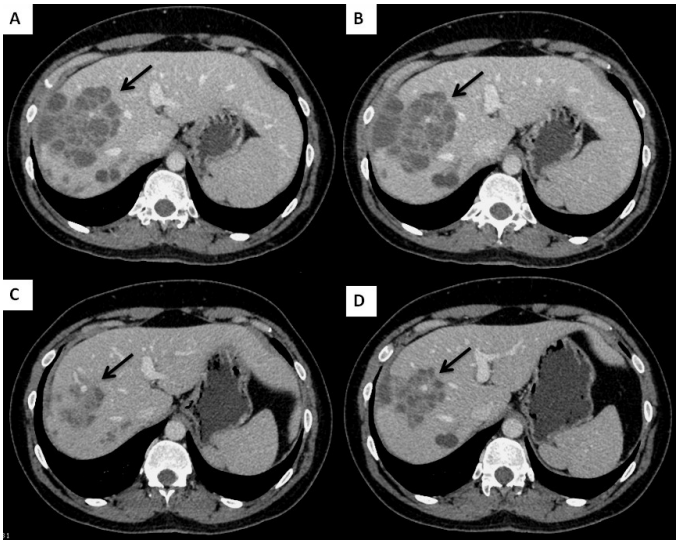

Figure 3: Axial CECT images portal venous phase before (A,B) and 6 months after therapy (C,D) demonstrating significant regression in the size of the clustered cystic lesions (black arrows).

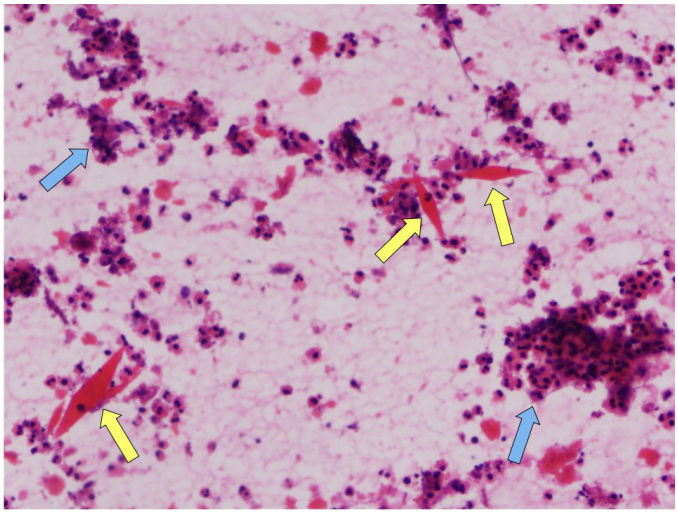

Figure 4: Smear showing degenerated inflammatory cells including eosinophils (blue arrows) and many Charcot Leyden crystals (yellow arrows).

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.[5]

Lesions are usually described as oval, ill-defined nodules, either unique or more frequently multiple. Imaging reveals multiple clustered complex cystic lesions, best appreciated on portal venous phase, with little or no peripheral enhancement or surrounding edema. These are usually small, less than 2 cm in size and likely to be along the portal vein branches or liver periphery with occasional traversing of the portal vein through the larger lesions. However, we encountered two cases with lesions showing diffusion restriction and distinct peripheral enhancement on portal venous phase and can thus mimic amoebic or pyogenic abscesses. Furthermore, imaging revealed portal vein thrombosis and abscess rupture in two of our patients. To the best of our knowledge, these complications have never been reported in literature in cases of hepatic larva migrans. Treatment with albendazole alone or in combination with ivermectin is reported to be effective,[6] although long term therapy is needed for complete eradication.

References

1. Chang S, Lim JH, Choi D, Park CK, Kwon NH, Cho SY, Choi DC. Hepatic visceral larva migrans of Toxocaracanis: CT and sonographic findings. AJR Am J Roentgenol. 2006 Dec;187(6):W622-9.
2. Lim JH. Toxocariasis of the liver: visceral larva migrans. Abdom Imaging. 2008 Mar-Apr;33(2):151-6.
3. Glickman LT, Magnaval JF, Domanski LM, Shofer FS, Lauria SS, Gottstein B, Brochier B. Visceral larva migrans in French adults: a new disease syndrome? Am J Epidemiol. 1987 Jun;125(6):1019-34.
4. Hayashi K, Tahara H, Yamashita K, Kuroki K, Matsushita R, Yamamoto S, Hori T, Hirono S, Nawa Y, Tsubouchi H. Hepatic imaging studies on patients with visceral larva migrans due to probable Ascaris suum infection. Abdom Imaging. 1999 Sep-Oct;24(5):465-9.
5. Mukund A, Arora A, Patidar Y, Mangla V, Bihari C, Rastogi A, Sarin SK. Eosinophilic abscesses: a new facet of hepatic visceral larva migrans. Abdom Imaging. 2013 Aug;38(4):774-7.
6. Raffray L, Le Bail B, Malvy D. Hepatic visceral larva migrans presenting as a pseudotumor. Clin Gastroenterol Hepatol. 2013 Jun;11(6):e42.

Syndrome of Acute Hepatitis and Acute Renal Failure after Raw Fish Bile Consumption: A Case Series

Prasanna K S

Apollo Hospital, Guwahati.

Corresponding Author: *Dr Prasanna K S*
Email: prasannaks19@gmail.com

Rohu (*Labeorohita*) fish is commonly found and consumed in South East Asia (India, Pakistan, Bangladesh, Nepal and Myanmar) as it is abundantly available in this area. It is a common practice among certain groups of people to consume raw gall bladder of fish to prevent malaria, for vision improvement and for good health.[1]

We report one of the rare causes of toxic hepatitis along with acute renal failure which is caused by consumption of fresh fish bile. There are few case reports from Asian countries and one from USA which have reported renal failure along with acute hepatitis after fish bile consumption.[1-3]

Four people from North Assam consumed bile of a Rohu fish. Two of them consumed small quantity of bile (5-10 ml) and had abdominal discomfort and vomiting and the other two had renal and liver injury for which they were referred to our hospital.

Case 1

A young 25 year old male from Lakhimpur, north Assam had consumed about 25 ml fish bile (Rohu fish), 2 -3 hours after consuming bile patient noted upper abdominal pain along with nausea and vomiting. When he was evaluated next day at his place his liver and kidney function tests were abnormal and patient even noted decrease in urine output. With above details he was referred to our hospital. On admission he was hemodynamically stable,