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A rare case of visceral larva migrans (VLM)

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Abstract
Visceral larva migrans (VLM) is one of the clinical syndromes of human toxocariasis. We report a case of hepatic VLM presented with pre-prandial malaise and epigastric discomfort with pain in right upper quadrant of abdomen in the past 4 month in a 31-year-old male with history of eating rabbit flesh. The ultrasound imaging studies of the abdomen showed a small hypoechoic area measuring 8-13 mm in right lobe of liver (segment VII). His CT Abdomen revealed well defined hypodense peripherally enhancing lesion in segment VII of liver -likely infective granuloma. His blood picture showed severe eosinophilia. Anti-Toxocara canis immunoglobulin G (IgG) was observed in enzyme-linked immunosorbent assay (ELISA) and western blot. Patient underwent CT guided biopsy which showed liver abscess with eosinophilic infiltrate favouring the possibility of parasitic infestation suggestive of visceral larva migrans and toxocariasis. Patient underwent anthelmintics treatment, the patient complained of newly developed skin rash with eosinophilia. Hepatic lesion was same in size. After the treatment, the patient was free of symptoms such as pre-prandial malaise, epigastric discomfort, pain in right upper quadrant, and skin rash. Laboratory test showed a normal eosinophilic count at 1 month, 6 months. Our case report highlights that a high degree of clinical suspicion for hepatic VLM should be considered in a patient with a history of ingestion of raw food in the past, presenting with severe eosinophilia and a variety of symptoms which reflect high worm burdens. Remission of symptoms and eosinophilia and complete radiological resolution of lesions can be complete with treatment.

Keywords: visceral larva migrans, toxocariasis, ELISA

Introduction
Visceral larva migrans (VLM) or toxocariasis is a zoonotic infection usually caused by dog or cat ascarids of the Toxocara genus. The eggs are ingested and then hatch into larvae, which penetrate the intestine and start migrating. Because humans are not the definitive host, the larvae cannot mature, and so continue migrating for months or years. Most people who are infected with Toxocara spp. are asymptomatic. Symptoms depend on the extent and frequency of infection, the distribution of larvae in tissues, and the inflammatory response of the host. There are two clinical syndromes of infection: VLM and ocular larval migrans (OLM). The symptoms of VLM are related to the organ invaded, most commonly the liver, lung, or other thoracic or abdominal organ VLM usually affects children <5 years old, although it can affect adults. In children with VLM 33-86% had pulmonary symptoms: most commonly, chronic cough, which may be paroxysmal and worse at night, wheezing, and pulmonary infiltrates. On examination, 65% had hepatomegaly, 43% had an abnormal lung (examination (wheezes or rales). VLM is diagnosed by serology and treated with albendazole.[1]

Case Scenario
30 year male patient residence of coorg came to surgery opd with complain of pain in right upper quadrant since 4 month. insidious onset, mild to moderate intensity, intermittent, cramping type, non-radiating, aggravated after meal, no relieving factor Patient also complaints of weight loss of 7 kg in 1 month
H/O constipation on and off since 1 month
H/O passing hard stool
No h/o fever
No h/o vomiting
No h/o Malena /hematemesis

Past History: No comorbidity
H/o Eating raw meat product (pig)

Clinical Examination
Patient is moderately build and well nourished

Afebrile
BP-130/80mmhg (at right hand in supine position)
PR-80bpm,
BMI: 26
No pallor/icterus / cyanosis / clubbing /lymphadenopathy/oedema/dehydration

Systemic Examination
Per Abdomen
Inspection
Shape of abdomen -NAD
All corresponding moves equal with respiration
No visible gastric peristalsis
No engorged /dilated veins
Hernial orifice intact

Palpation
Abdomen soft and Tenderness present in right hypochondria.
No mass palpable.
Liver felt 1 cm below the coastal margin (Hepatomegaly). No splenomegaly.

Percussion
Resonant note heard all over abdomen, No shifting dullness

Auscultation
Bowel sound present.
Respiratory system - B/L air entry equally present. Right sided basal rhonchi present
Cardiovascular system - S1 S2 (+), no murmur
Central nervous system - No focal neurological deficit

Investigation
Complete Blood Count (CBC)

Table 1

Haemoglobin	**16.0**
Total count	7080
Polvmorphs	42.6
Lymphocytes	37.0
Monocytes	4.4
Eosinophils	16.0
Basophils	0.0
Platelets	2.28 lakhs/cumm
ESR (1 hour)	12mm/hr
RBC	5.59
HCT/PCV	46.1
MCV	82.5
MCH	28.5
MCHC	34.6
RDW-CV	14.4
RDW-SD	45.9
MPV	8.4
PCT	0/192

Ultrasound imaging studies of the abdomen showed a small hypoechoic area measuring •8-13 mm in right lobe of liver (segment VIl).

CT Abdomen revealed well defined hypodense peripherally enhancing lesion in segment VII of liver -likely infective granuloma

Arterial Phase

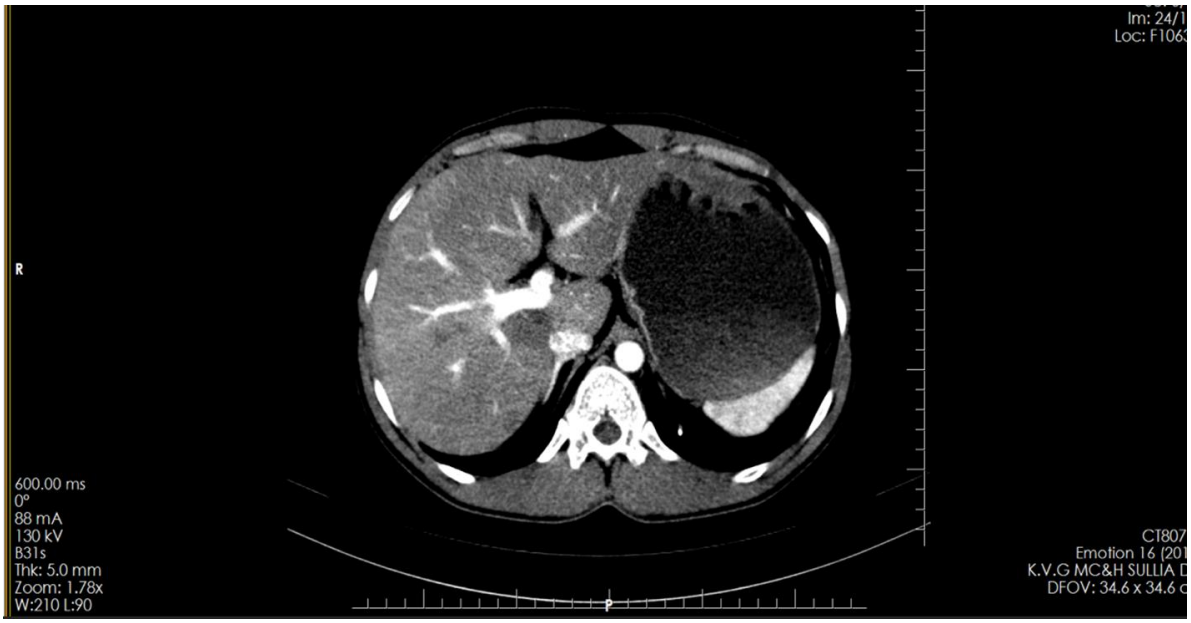

Fig 1

Portal Venous Phase

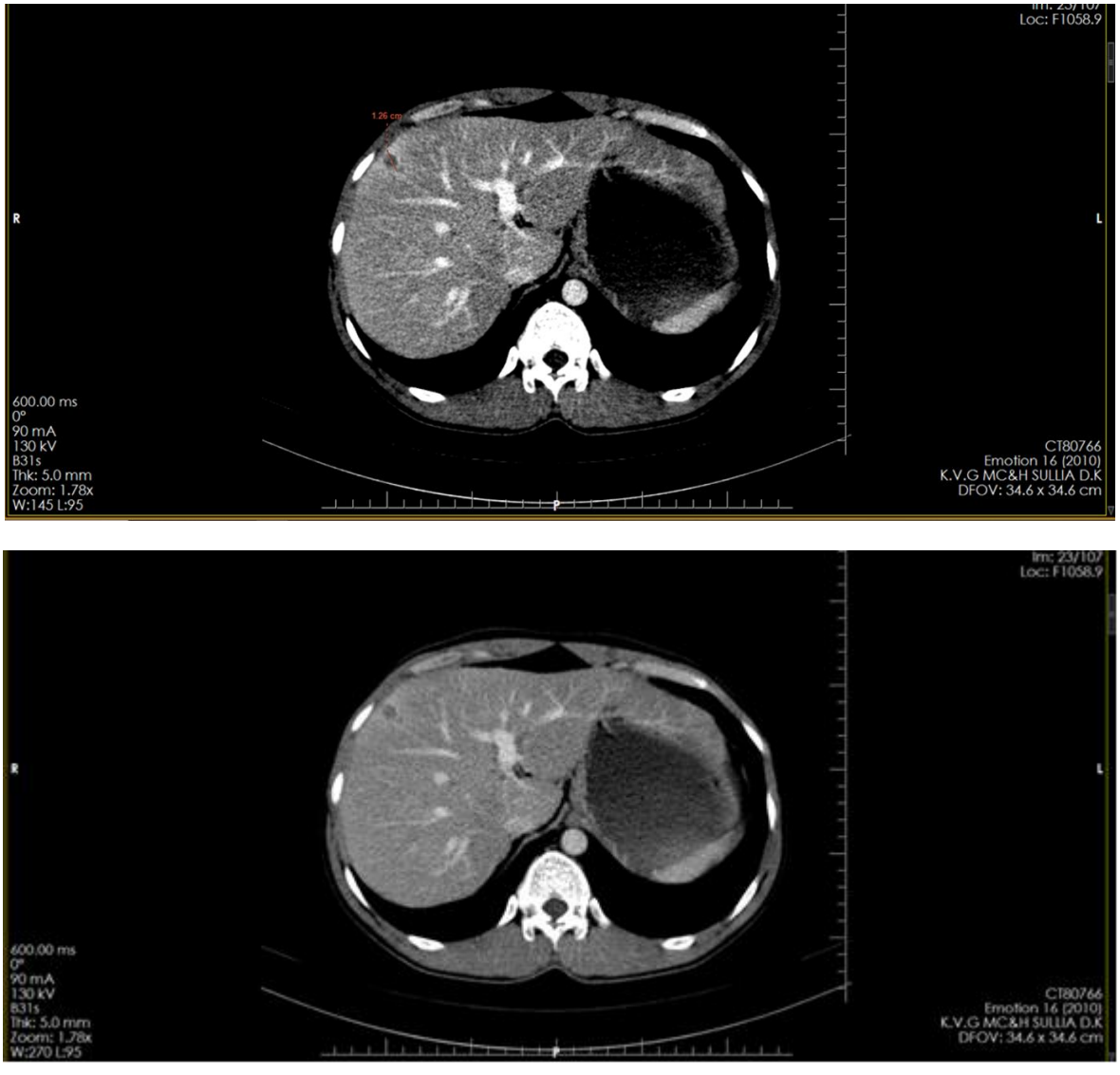

Fig 2

Delayed Phase

Fig 3

Anti-Toxocara canis immunoglobulin G (IgG) was observed in enzyme-linked immunosorbent assay (ELISA) and western blot. Patient underwent CT guided biopsy which showed liver abscess with eosinophilic infiltrate favouring the possibility of parasitic infestation suggestive of visceral larva migrans and toxocariasis.

Treatment

Anthelmintic treatment was started, the patient complained of newly developed skin rash with eosinophilia. Hepatic lesion was same in size. After the treatment, the patient was free of symptoms such as pre-prandial malaise, epigastric discomfort, pain in right upper quadrant, and skin rash. Laboratory test showed a normal eosinophilic count at 1 month, 6 months.

Follow up was uneventful.

Conclusion

Our case report highlights that a high degree of clinical suspicion for hepatic VLM should be considered in a patient with a history of ingestion of raw food in the past, presenting with severe eosinophilia and a variety of symptoms which reflect high worm burdens. Remission of symptoms and eosinophilia and complete radiological resolution of lesions can be complete with treatment. After failure of conservative treatment, surgery such as lobectomy of liver can be done.

Toxocariasis, caused by infection with larvae of Toxocara canis, and to a lesser extent by Toxocara cati and other ascaridoid species, manifests in humans in a range of clinical syndromes. These include visceral and ocular larva migrans, neurotoxocariasis and covert or common toxocariasis. Toxocara canis is one of the most widespread public health and economically important zoonotic parasitic infections humans share with dogs, cats and wild canids, particularly foxes. This neglected disease has been shown through seroprevalence studies to be especially prevalent among children from socio-economically disadvantaged populations both in the tropics and sub-tropics and in industrialised nations. Human infection occurs by the accidental ingestion of embryonated eggs or larvae from a range of wild and domestic paratenic hosts. Most infections remain asymptomatic. Clinically overt infections may go undiagnosed, as diagnostic tests are expensive and can require serological, molecular and/or imaging tests, which may not be affordable or available. Treatment in humans varies according to symptoms and location of the larvae. Anthelmintics, including albendazole, thiabendazole and mebendazole may be given together with anti-inflammatory corticosteroids. The development of molecular tools should lead to new and improved strategies for the treatment, diagnosis and control of toxocariasis and the role of other ascaridoid species in the epidemiology of Toxocara spp. Molecular technologies may also help to reveal the public health importance of T. canis, providing new evidence to support the implementation of national control initiatives which have yet to be developed for Toxocara spp. A number of countries have implemented reproductive control programs in owned and stray dogs to reduce the number of young dogs in the population. These programs would positively impact upon T. canis transmission since the parasite is most fecund and prevalent in puppies. Other control measures for T. canis include the regular and frequent anthelmintic treatment of dogs and cats, starting at an early age, education and enforcement of laws for the disposal of canine faeces, dog legislation and personal hygiene. The existence of wild definitive and paratenic hosts complicates the control of T. canis. Increasing human and dog populations, population movements and climate change will all serve to increase the importance of this zoonosis. This review examines the transmission, diagnosis and clinical syndromes of toxocariasis, its public health importance, epidemiology, control and current research needs.[4]

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