Mediastinal Lymphadenopathy in Visceral Leishmaniasis

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Abstract

Visceral leishmaniasis (VL) usually presents with malaise, fever, weight loss, and hepatosplenomegaly. Lymphadenopathy is rarely reported in patients with VL from India. We report an interesting case of VL, presenting with malaise, fever and weight loss along with isolated mediastinal lymphadenopathy and mimicked tuberculosis. Absence of spleen because of previous splenectomy for symptomatic portal hypertension led to difficulty in establishing the correct diagnosis that was eventually established by endoscopic ultrasound (EUS)-guided fine-needle aspiration (FNA).

Introduction

Visceral leishmaniasis (VL) or Kala-azar is a disseminated protozoan infection that spreads via the bite of phlebotome sand flies and is caused by Leishmania sp.¹ The disease onset is usually insidious and typical manifestation of VL include slow progression of malaise, fever, weight loss, and hepatosplenomegaly.¹ Kala-azar (“black fever”) refers to the dark color of skin seen in patients with VL and is usually seen in patients in South Asia but not elsewhere.² Lymphadenopathy is usually reported in cases of VL from East African countries and is rarely reported from India.³ VL is usually diagnosed by demonstrating parasites in the bone marrow or splenic aspirate, and fine-needle aspiration (FNA) from enlarged lymph nodes has been occasionally reported for its correct diagnosis.¹,³ We report an interesting case of VL presenting with malaise, fever and weight loss along with isolated mediastinal lymphadenopathy and mimicked tuberculosis. Absence of spleen because of previous splenectomy for symptomatic portal hypertension led to difficulty in establishing the correct diagnosis that was eventually established by endoscopic ultrasound (EUS)-guided FNA.

Case Report

A 34-year-old male, who had undergone splenectomy with proximal lienorenal shunt 12 years ago for variceal bleeding because of extrahepatic portal venous obstruction, was admitted with a 2-month history of fever, fatigue, and weight loss. The general physical examination revealed a thin built patient with no organomegaly or lymphadenopathy. The hematological investigations revealed hemoglobin of 8.2 g/dL, total leukocyte count of 5,200 cells/μL, and platelet count of 1.3 lac/μL. Blood cultures as well as tests for human immunodeficiency virus (HIV) were negative. Chest X-ray did not reveal any abnormal findings. Liver function tests revealed conjugated hyperbilirubinemia (total/conjugated: 2.5/1.9 mg/dL), elevated alkaline phosphatase (3,326 U/L; normal: <128 U/L), low-serum albumin (2.8 g/dL; normal 3.4–4.8 g/dL), and elevated serum globulins (4.6 g/dL; normal 2.0–3.5 g/dL). Abdominal ultrasonography revealed dilated intrahepatic biliary radicles due to portal cavernoma cholangiopathy (PCC). There were no gastroesophageal varices on gastroscopy.

EUS done for evaluation of PCC revealed enlarged hypoechoic subcarinal as well as aortopulmonary lymph
nodes with areas of necrosis (►Fig. 1, arrows). EUS-guided FNA was performed using 22G EUS FNA needle (EchoTip, Wilson-cook Medical, Winston-Salem, North Carolina, United States) (►Fig. 2), and cytological examination of lymph node aspirate revealed collection of foamy histiocytes, showing numerous amastigote forms of *Leishmania donovani* (►Fig. 3: MGG ×40). Higher magnification revealed multinucleated histiocyte, showing *Leishmania donovani* (LD) bodies (►Fig. 4) and safety pin-shaped LD bodies with kinetoplasts within a histiocyte (►Fig. 5; arrows [MGG ×100]). RK 39 serological test was positive. Patient was successfully treated with liposomal amphotericin B.

**Discussion**

Patients with VL in India usually present with fever, weight loss, hepatomegaly, splenomegaly, pancytopenia, and hyperglobulinemia. Enlarged firm spleen in patient with febrile illness from an endemic area raises suspicion of VL. Our patient had undergone splenectomy for symptomatic extrahepatic portal venous obstruction and did not reside in the endemic area for VL. Therefore, Kala-azar was not considered as a differential diagnosis in the index case. Also, the clinical presentation as well as EUS morphology of mediastinal lymph nodes mimicked lymph nodal tuberculosis.\(^5\)
Lymphadenopathy due to Kala-azar is rare in India and isolated mediastinal lymphadenopathy in the absence of HIV infection is very rarely reported in VL. VL is commonly diagnosed by demonstrating parasites in the bone marrow and splenic aspirate and lymph node FNA has been rarely used for its diagnosis. Most of the cases of lymph nodal leishmaniasis have been diagnosed by aspiration of cervical lymph nodes or ultrasound-guided FNA of mesenteric lymph nodes. The diagnosis of VL by EUS-guided FNA of mediastinal lymph nodes is rarely reported in the literature.

In conclusion, leishmaniasis should be included in differential diagnosis of unexplained lymphadenopathy, especially in endemic areas. EUS-guided FNA is an useful modality for correctly diagnosing unexplained mediastinal lymphadenopathy.

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**References**