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Case Report

Visceral Leishmaniasis with Renal Involvement: A Case Report

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Abstract

Visceral leishmaniasis (VL) is widespread in tropical and temperate region. It is caused by the protozoan parasite leishmania species and is transmitted through the bite of infected female sandflies. It manifests as pancytopenia, hepatosplenomegaly and fever. Here, we present a case of 20-year-old male presented with difficulty in swallowing for 2 months, abdominal distention for 30 days and shortness of breath for 20 days along with progressive renal impairment. Clinical examination revealed hepatosplenomegaly and laboratory investigations confirmed pancytopenia, nephrotic range proteinuria, hematuria and hypocomplementemia. Bone marrow aspiration and biopsy was performed which showed leishmania donovani (LD) bodies. It'suncommon presentation makes it challenging for physicians as well as pathologist to make a timely diagnosis.

Keywords: *Bone marrow, Kidney disease, Visceral leishmaniasis*

Introduction

One of the neglected tropical diseases (NTDs) is leishmaniasis also known as kala-azar, a protozoan infection [1]. It is caused by an obligate intramacrophage protozoa, an infectious disease that is widespread in tropical and temperate region of many countries including Asia [2]. It can have a variety of clinical presentations in humans, and are often categorized as cutaneous, mucocutaneous, and visceral (VL) [3]. Female phlebotomine sand flies are necessary for the life cycle of leishmania parasites as well as for propagation, or acting as a vector [4]. In the midgut of the insect, the promastigote multiples and matures into an infectious metacyclic form that moves to the proboscis. Sandflies bite mammals to obtain blood, and when they do, metacyclic promastigotes are released into the wound [5]. After entering the host, they soon encounter leukocytes, specifically macrophages, neutrophils and dendritic cells, phagocytized the material via phagocytosis, these cells ingest the parasites which are then transported to lysosomes and endosomes. Then, they instantly go through further morphological change, losing their flagellum and developing an oval form that is known as a 'amastigote' [6].

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Case Report

A 20-year-old male presented to the hepatology out patient department with a complaint of difficulty in swallowing for 2 months, abdominal distention for 30 days and shortness of breath for 20 days. He has no notable medical or surgical history in the past.

On assessment, he had tachycardia, tachypnea and blood pressure was 130/90mmhg. Pallor, dehydration, and periorbital and pedal edema were present. Abdominal examination revealed tenderness over right hypochondrium, epigastrium and left hypochondrium. The patient initially sought treatment at a local health post. Subsequently, the patient received ayurvedic medicine. Regrettably, his symptoms did not abate with the initial treatment, necessitating further intervention and prompting his referral to our center for continued and more specialized management. His peripheral blood smear showed pancytopenia (moderate normocytic normochromic anemia with moderate leukopenia with moderate thrombocytopenia), renal function test showed sodium: 123 mmol/L, urea: 66mg/dl, creatinine was within normal range, liver function test showed aspartate ammino transferase (AST): 50 IU/L, alanine ammino transferase (ALT): 58IU/L, alkaline phosphatase (ALP): 617 U/L and albumin: 2.7 gm/dl, urine examination showed RBC: 80-120/HPF, 24 hours urine protein: 6825.0 mg/24hrs and serum complement level (C3): 84.1 mg/dl. Ultrasonography whole abdomen showed hepatosplenomegaly with dilated splenic vein and portal vein-suggestive of portal hypertension, echogenic bilateral kidneys and moderate ascites. Ascitic fluid cytology was normal. Possibility of infectious glomerulonephritis was kept as differential diagnosis. Patient was kept on intravenous antibiotics (taxim and moximycin), diuretics (Lasix, spironolac), vitamin supplement (thiovit), a therapeutic procedure was carried out (three liters of fluid was taken out in three setting under aseptic condition, two pint of blood transfusion were administered). Prior to the scheduled renal biopsy, a bone marrow aspiration and biopsy were carried out which showed normocellular bone marrow with multiple foci of extracellular as well as intracellular amastigote forms of Trypanosoma (LD bodies).(Figure 1A, B).

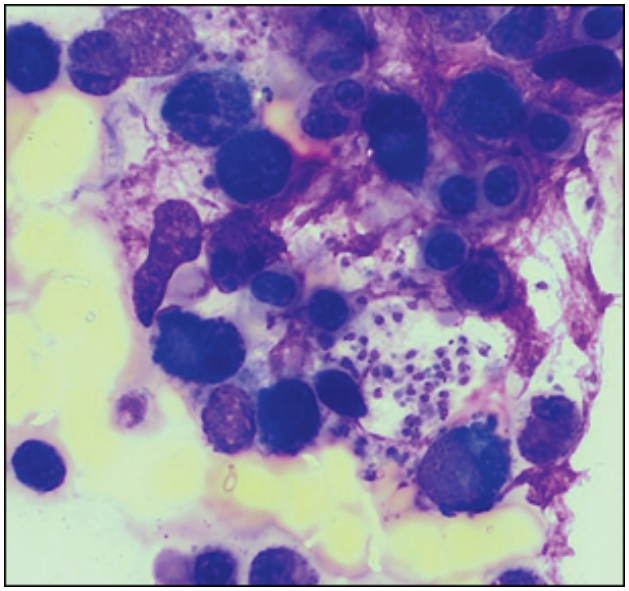

Figure 1(A): Giemsa stain (40x): Intracellular amastigote form of LD bodies (arrow)

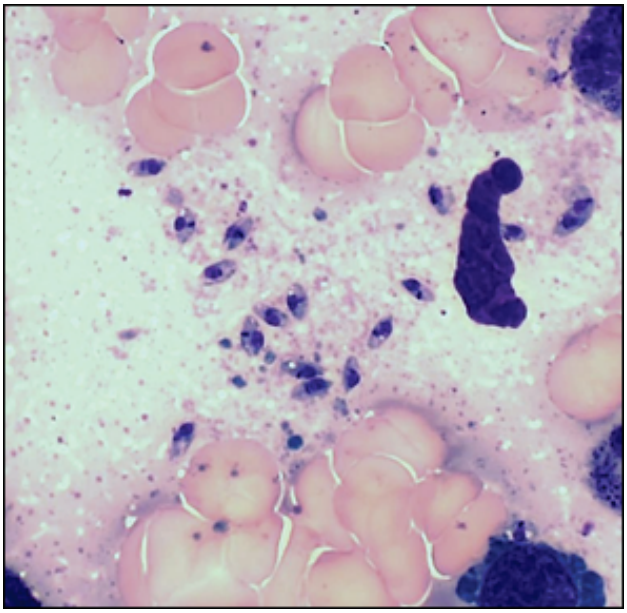

Figure 1(B): Giemsa stain (oil immersion): Extracellular amastigote form of LD bodies (arrow) with kinetoplast imparting a "safety pin like" or "double dot like" appearance.

Thus, renal impairment was also clinically attributed to the possibility of visceral leishmaniasis involving the kidney as well. After the diagnosis, on patient request, they were referred to another center for kala-azar management. On a 2-week follow-up, the renal function test was reverted back to normal, and the patient didn't have any new complaints.

Discussion

When approaching the case scenario as of the current case, visceral leishmaniasis (VL) is one of the crucial differentials to be kept in mind. It

can manifest with uncommon and complex features and perhaps with multi-system involvement [2]. A serological diagnosis can also be made with the rK39 ELISA test, which, however, has a sensitivity of 92% [7]. rK39 ELISA was not performed in the current case since leishmaniasis was not first considered a differential diagnosis clinically. In visceral leishmaniasis, the parasite invades the spleen and triggers an immunological reaction that causes the splenic architecture to become completely disorganized, stromal cells to disappear, and dendritic cells to become excessively activated,resulting in splenomegaly [3]. Although the liver is more efficient at clearing parasites compared to the spleen, it still undergoes significant changes. Granulomas form in the hepatic parenchyma to clear the majority of the parasitic load. These granulomas are organized structures formed by immune cells to wall off and contain the parasites. This immune response can lead to hepatomegaly [3]. Renal impairment in visceral leishmaniasis presents with complex clinical characteristics, alteration in both glomerular and tubular function, abnormalities in urine concentration and acidity, as well as acute and chronic renal insufficiency, poor patient outcomes could be influenced by acute kidney injury [2]. GB silva et al. (2014), in an article,reported that renal impairment was present in 45.9% of cases [8]. The current case also had a deranged renal function test;perhaps in addition to interfering with interferon gamma signaling, leishmania can also induce the production of growth factor beta, a cytokine that inhibits macrophages, and interleukin-10, all of which can impact the cellular immune response and cause polyclonal B-cell activation, which has been linked to kala-azar glomerular disease [9]. Nonetheless, kala-azar therapy may be effective in treating glomerular lesions [2]. The diagnosis of visceral leishmaniasis remains a challenge for most pathologists and physicians. Bone marrow aspirates are an authentic method that is safer and is proposed as a technique of choice for the parasitological diagnosis of visceral leishmaniasis [10]. Bone marrow cytology usually show both intracellular as well as extracellular collection of amastigote forms of parasite along with reactive plasmacytosis [11]. Early suspicion as well as recognition of visceral leishmaniasis are critical for appropriate management and avoiding unnecessary investigations. They are also critical for improving patient outcomes, reducing transmission as infected individuals serve as reservoirs for the parasite, and reducing the duration and intensity of parasitemia, thus lowering the risk of transmission to others through infected sand flies [12]. This is crucial for public health efforts to control the spread of the disease and minimize the socioeconomic burden associated with it [1]. VL can be fatal if untreated, particularly in immunocompromised individuals or those with delayed diagnoses [13]. Early recognition and appropriate treatment significantly reduce mortality rates associated with the disease [14].

Conclusion

The case highlights the importance of considering visceral leishmaniasis as an important differential diagnosis in patients presenting with organomegaly or unexplained fever with multisystem involvement;however, serological tests like rK39, if available, or bone marrow cytology can be the diagnostic modalities of choice to confirm VL.

This will also avoid unnecessary investigation modalities and lessen the financial burden and anxiety for the patient.

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