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Case Report

Macrophage Activation Syndrome Secondary to Visceral Leishmaniasis: A Case Report

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Abstract: ***Introduction*:** Macrophage activation syndrome (MAS) secondary to visceral leishmaniasis is a rare clinical entity. We report the case of a 4-year-old boy with no prior medical history, who had presented with fever and pallor for three weeks. Clinical examination revealed a temperature of 39°C, hepatomegaly, and splenomegaly. Laboratory tests showed bicytopenia, hypertriglyceridemia, and elevated lactate dehydrogenase (LDH). Bone marrow examination revealed Leishman bodies and hemophagocytic images. The patient was hospitalized and received treatment. The clinical and biological course was favorable. ***Conclusion*:** Visceral leishmaniasis associated with hemophagocytic syndrome is severe and may be life-threatening. Physicians should consider this diagnosis in children presenting with fever and splenomegaly. Urgent complementary investigations are needed to confirm this association and initiate appropriate treatment.

Keywords: Visceral Leishmaniasis, Macrophage Activation Syndrome, Hemophagocytosis.

INTRODUCTION

Visceral leishmaniasis is a parasitic zoonosis that remains a public health issue in Morocco. Its association with macrophage activation syndrome (MAS) a rare clinicopathologic entity that is difficult to diagnose and of variable severity poses a diagnostic challenge [1].

The clinical overlap of these two conditions makes diagnosis difficult and carries a poor prognosis, especially since treatment guidelines remain poorly defined [2].

We report the case of a child presenting with severe macrophage activation syndrome secondary to visceral leishmaniasis.

The aim of this work is to highlight a rare complication of visceral leishmaniasis to improve the recognition and management of macrophage activation syndrome.

CASE PRESENTATION

We report the case of a 4-year-old boy born of a non-consanguineous marriage, vaccinated according to the national immunization program, with no particular medical history. The illness began three weeks prior to admission, with the onset of fever prompting several consultations and symptomatic treatments, without improvement. Due to persistent fever, full laboratory investigations were performed. Based on the clinical and biological findings, MAS was suspected, and the patient was referred to a university hospital for further management.

On admission, the child was conscious, febrile (38.2°C), pale, with abdominal tenderness, hepatomegaly measuring 5 cm, and splenomegaly measuring 3 fingerbreadths below the costal margin. No lymphadenopathy was noted.

Laboratory tests revealed profound bicytopenia with normocytic normochromic anemia (hemoglobin 5.2 g/dL), reticulocyte count of 20,000/mm³, leukocyte count of 1,280/mm³ with neutropenia at 600/mm³, and normal platelets (145,000/mm³). Blood smear showed no abnormalities.

Additional tests revealed hepatic cytolysis, elevated LDH at 2,420 U/L, hyperferritinemia at 828 µg/L, hypertriglyceridemia at 4.55 g/L, and hypofibrinogenemia at 0.54 g/L. Prothrombin rate was 71%, and activated partial thromboplastin time was prolonged at 67 seconds.

Infectious work-up showed CRP at 157 mg/L, and serologies for hepatitis A, B, C, HIV, syphilis, and toxoplasmosis were all negative.

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Bone marrow aspiration revealed hypocellular marrow with megakaryocytes, 3% blasts, numerous hemophagocytic images, and intracellular and extracellular amastigote forms of *Leishmania* (Leishman bodies) (Figure 1).

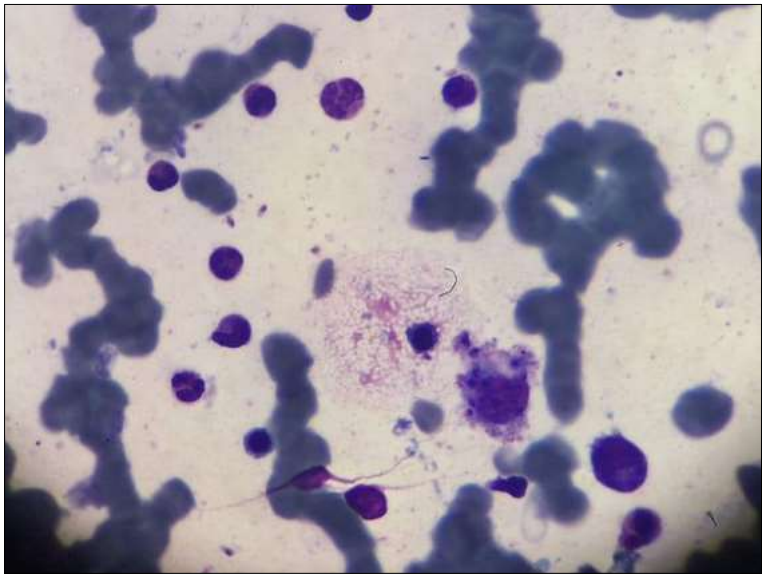

Figure 1 : Image of hemophagocytosis with intra- and extracellular Leishmania bodies (May-Grünwald-Giemsa stained bone marrow smear, 100x objective, hematology lab, CHU Hassan II, Fez)

The diagnosis of MAS secondary to visceral leishmaniasis was confirmed. A rapid test for *Leishmania* using the patient's serum was positive (Figure 2).

Figure 2 : Positive rapid diagnostic test for leishmaniasis (Hematology lab, CHU Hassan II, Fez)

The patient received a transfusion of red blood cells and etiological treatment with meglumine antimoniate (100 mg/kg/day), combined with corticosteroid therapy, leading to favorable clinical and biological improvement.

DISCUSSION

Macrophage activation syndrome (MAS), also known as hemophagocytic syndrome, is a rare but potentially fatal condition characterized by excessive and uncontrolled activation of macrophages and T lymphocytes. This hyperactivation leads to phagocytosis of blood cells, resulting in severe cytopenias and multiorgan failure [3]. MAS can be either primary (genetic) or secondary to various conditions, including infections, autoimmune diseases, or cancers [2].

Infections are responsible for approximately 50% of MAS cases, with viral infections (CMV, EBV, HSV) being the most common, followed by mycobacteria (tuberculosis), intracellular bacteria, pyrogens, and parasites [2]. Among the parasitic causes, visceral leishmaniasis is noteworthy; its association with MAS represents a diagnostic challenge due to the rarity and similarity of clinical and biological features, which can lead to diagnostic and therapeutic delays [4].

Visceral leishmaniasis (VL), caused by protozoa of the *Leishmania* genus, is a parasitic disease

transmitted by female phlebotomine sandflies. It is endemic in several regions of the world, including Morocco, and primarily affects children [5]. The association between VL and MAS is well documented, although relatively rare. This association complicates diagnosis due to the overlapping clinical features of both conditions, such as prolonged fever, splenomegaly, and cytopenias.

The diagnosis of MAS is based on clinical and biological criteria defined by the Histiocyte Society in 2004, known as the HLH-2004 criteria (Table 1). These criteria include, among others, fever, splenomegaly, cytopenias affecting at least two blood cell lines, hypertriglyceridemia, hypofibrinogenemia, and hyperferritinemia. The presence of hemophagocytic images in bone marrow examination, although nonspecific, is also an important diagnostic feature.

Table 1 : HLH-2004 diagnostic criteria for MAS

• Fever
• Splenomegaly
• Cytopenias (≥2 cell lines)
• Hypertriglyceridemia and/or hypofibrinogenemia
• Hemophagocytosis in bone marrow, liver, or lymph nodes
• Low/absent NK cell activity
• Ferritin > 500 µg/L
• Elevated soluble IL-2 receptor (>2,400 U/mL)
The diagnosis of MAS is confirmed when at least five of the HLH-2004 criteria are fulfilled.

The diagnosis of macrophage activation syndrome associated with visceral leishmaniasis is often difficult in the early stages of the disease, and clinicians must persist in establishing it, especially in endemic areas, by repeating biological tests if necessary (bone marrow aspiration with serology), before considering immunosuppressive treatment [6].

In cases of VL associated with MAS, treating the leishmaniasis is essential. Liposomal amphotericin B is considered the treatment of choice due to its effectiveness and relative safety [1]. However, in regions where this treatment is not available, alternatives such as pentavalent antimonial derivatives may be used, although they are less effective and more toxic. The addition of corticosteroid therapy may be considered to control excessive macrophage activation, but this approach should be assessed on a case-by-case basis depending on the severity of MAS and the response to antiparasitic treatment [7].

CONCLUSION

The severity of MAS-VL association, especially in endemic settings, warrants systematic use of serology alongside bone marrow examination, and early adoption of aggressive therapeutic strategies to improve outcomes.

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