

A case report of visceral leishmaniasis in the Tharaka District, Kenya

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Abstract. Leishmaniasis is a parasitic disease that is transmitted through a bite of some species of sandflies. It is caused by obliged intra-cellular protozoa of the genus *Leishmania* and is responsible for a broad spectrum of clinical syndromes: cutaneous leishmaniasis (CL), visceral leishmaniasis (VL) and mucocutaneous leishmaniasis (ML). The visceral disease (classically known as “kala azar”) is the most aggressive form and if undervalued is fatal. Here we describe the first case of visceral leishmaniasis in the Tharaka District reported in literature. (www.actabiomedica.it)

Key words: Leishmaniasis, kala azar, leishmania

Introduction

Visceral leishmaniasis results in systemic infection of the liver, spleen and bone marrow. The classical symptoms are: fever, pallor, food refusal, weight loss, vomiting, hepatosplenomegaly, ecchymoses and gingival bleeding. Laboratory tests usually reveal thrombocytopenia, anemia and leukopenia. The parasite presence may be shown through direct evidence in the peripheral blood, bone marrow or splenic aspirates. The pentavalent antimony compounds represent the standard anti-leishmania treatments, but new approaches with amphotericin B are also highly effective against leishmaniasis.

Case report

We report the case of G.H., a six year old child from Kenya. The patient came to S. Horsola hospital, in the Tharaka District, with a history of recurrent fever, abdominal pains, nasal and gingival bleeding, fecal and urinary blood, and joint pains lasting for about four months. In the past, the child showed recurrent episodes of malaria.

On admission the child presented severe malnutrition, anemia, jaundice and swelling of the limbs; the liver and the spleen were palpable 4 and 5 cm below the right and left costal margin, respectively; moreover a 2/VI cardiac murmur was shown.

Laboratory tests gave the following results: haemoglobin 3.7 g/dl, total bilirubin 7.3 mg/dl, SGOT 64 U/L, SGPT 134 U/L, positive blood smear for malaria; the thin blood film showed severe anemia, low platelet cells and poichilocytosis.

The patient was immediately treated with the following therapy: intramuscular Artemisina for five days, oral antibiotic therapy with Cefalexine for seven days and intravenous rehydration treatment. A blood transfusion was also performed due to anemia. After the first Artemisina administration, we decided to change the treatment with intravenous Quinine because of an important bleeding in the site of the injection.

Due to the persistency of anemia, on the 4th and 9th day after admission, two further blood transfusions were necessary.

During the first ten days no clinical improvement was reported and the patient showed recurrent nasal and gingival bleeding, and blood presence in the uri-

ne and stools. The search for *Ancylostoma* or *Schistosoma* infection in the stools was negative. Laboratory exams obtained the following values: creatinina 0.87 mg/dl, total protein 3.4 gr/dl, total bilirubin 13.9 mg/dl, WBC 2400/mm³; the blood smear for malaria at the end of the Quinine treatment was negative.

In the second week an important worsening of the clinical conditions with high fever (BT 39-41°C), tremors and anorexia was shown. We decided to start an antibiotic treatment with Benzatin-Penicillina and Gentamycin but the child continued with high fever and food refusal. The symptoms were highly suggestive for malaria, thypoid fever, Brucella's infection or visceral leishmaniasis; the blood smear for Plasmodium and the Widal-Wright test were negative; we decided to ask for another thin blood film that demonstrated numerous intra and extra-cellular *Leishmania* amastigotes.

We sent a car to the nearest city to find sodium stibogluconate (Pentostam; GlaxoSmithKline, Uxbridge, Middlesex, UK). In the meantime the patient suddenly had an important respiratory distress associated with bradycardia; an anaesthesiologist proceeded with nasopharyngeal intubation and administered Atropine, followed after a few minutes by Adrenaline. When the drug arrived the child was still intubated; we started the therapy with 20 mg/Kg daily, but some hours after the first dose the child died, probably due to cardiac arrest.

Discussion

Leishmaniasis is a parasitic disease that is transmitted through the bite of some species of sandflies. It is caused by obliged intra-cellular protozoa of the genus *Leishmania* and is responsible for a broad spectrum of clinical syndromes: cutaneous leishmaniasis (CL), visceral leishmaniasis (VL) and mucocutaneous leishmaniasis (ML).

Visceral leishmaniasis has a focal distribution in dry, hot areas below 1500 metres; it is endemic only in Baringo District (Rift Valley Province). Several foci of cutaneous leishmaniasis occurred in central Kenya (Samburu, Isiolo, Laikipia, Nakuru and Nyandarua) (1-4).

This case of visceral leishmaniasis is the first one reported in literature in the Tharaka District.

The visceral disease (classically known as "kala azar") is the most aggressive form and if undervalued is fatal. It results in systemic infection of the liver, spleen and bone marrow; in this syndrome the amastigotes replicate in macrophages of the mononuclear phagocyte system and then spread to the reticuloendothelial system. The classical symptoms are: fever, pallor, food refusal, weight loss, vomiting, hepatosplenomegaly, ecchymoses and gingival bleeding (5-7). Laboratory tests usually reveal thrombocytopenia, anemia and leukopenia. The parasite presence may be shown through direct evidence in the peripheral blood, bone marrow or splenic aspirates (8, 9).

The pentavalent antimony compounds meglumine antimonate (Glucantime; Aventis Pharma, Bridgewater, New Jersey, USA) and sodium stibogluconate (Pentostam, GlaxoSmithKline, Uxbridge, Middlesex, UK) are the standard anti-leishmania treatments, at doses of 20 mg/Kg daily for 30 days. However, the resistance to visceral infection in India has suggested new treatment approaches: amphotericin B (Amphotec; Sequus Pharmaceuticals Inc., Menl Park, California, USA) and liposomal amphotericin B (Ambisome; Gilead, Foster City, California, USA), which are highly effective against visceral leishmaniasis (10-13).

Conclusion

Visceral leishmaniasis is a very aggressive disease. The peculiarity of this clinical case depends on the fact that it was the first case reported in the Tharaka District; the diagnosis was difficult since the symptoms were highly suggestive for other more common diseases in Kenya like malaria, thypoid fever and Brucella's infection and since we did not have the possibility to ask for bone marrow or splenic aspirates.

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