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KALA-AZAR IN A NIGERIAN: REPORT OF A CASE WITH A FATAL OUTCOME

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A case of visceral leishmaniasis (Kala-azar) in a 60-year-old Nigerian female is presented. The clinical findings were fever, weight loss, lympadenopathy, hepatomegaly, and self-healing cutaneous ulcers. Laboratory findings included severe anaemia, lymphocytosis and amastigotes in the blood smear. The patient died before she could be commenced on the pentavalent antimonial specific for the disease.

INTRODUCTION

Leishmaniasis is caused by protozoa of the leishmania species. It is usually zoonotic and involves rodents, canines and various forest mammals. It is transmitted by the phlebotomine sand flies with incubation period of several months. The disease in man is usually cutaneous, mucocutaneous or visceral (Kala-azar). In Africa there are only few accurate statistics on visceral leishmaniasis (1). It is endemic in rural Sudan, and Kenya. The disease is rare in sub-Saharan West Africa. We therefore report here the first documented case of Kala-azar in Nigeria.

CASE REPORT.

A.D (Hospital No. 185654) was a 60-year old housewife and a farmer from one of the Southwestern states. She was admitted with a 9-month history of high-grade intermittent fever, excessive sweating, and difficulty in swallowing. Other symptoms included recurrent self-healing crusting lesions on the face and upper trunk. She also had progressive weight loss. She had not noticed similar skin lesion in her local community. Examination revealed a wasted elderly woman. She was pale and febrile (T-38.8°c). There were multiple ulcers on the face and upper trunk. Some were fresh, some were covered with

scabs and some already healed leaving hypopigmented patches on the skin. There were both axillary and cervical adenopathy. The respiratory rate was 20 breaths/min and the lung fields were initially clear. The pulse rate was 88 beats/min and the blood pressure was 110/70mmHg with normal first and second heart sounds. The abdomen was soft and non-tender and there was hepatomegaly of 5cm below the right coastal margin. The spleen and kidneys were not palpably enlarged. A tentative diagnosis of disseminated tuberculosis was made with differential diagnoses of leishmaniasis (visceral and cutaneous) and deep mycotic infection. Her laboratory results were as follows: PCV-17%, WBC-10.3 x 10^9/L (neutrophils-30%, lymphocytes-70%) and ESR-27mm/hr. Her electrolytes, urea and creatinine estimation were within the normal ranges. The chest X-ray was normal; barium swallow without fluoroscopy monitor was normal. Antibody against human immune deficiency virus infection (HIV) using Enzyme Linked Immunosorbent Assay (ELISA) technique was negative. Buffy coat blood smear was teaming with amastigotes on two occasions.

Fig 1 is a Giemsa stained buffy coat blood smear from our patient as seen under oil immersion of a microscope (X1000). The macrophages contained numerous amastigotes.

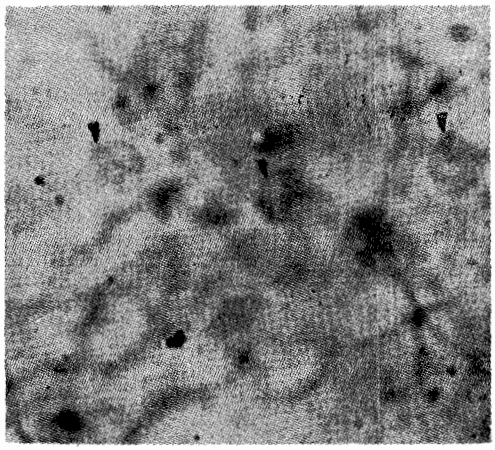

Fig 1

Cutaneous ulcer scraping and swabs were negative for amastigotes as well as for acid and alcohol fast bacilli.

Treatment outcome

The treatments given were both supportive and specific. She was transfused with 3 units of packed red cells with 80 mg of intravenous Frusemide (Lasix) preceding each unit over 4-6 hours. She also had high protein diet with multivitamins supplementation. She had oral Levamisole 120 mg stat, oral

Mebendazole 100 mg twice daily for 2 weeks and oral Biltricide 2.4 gm stat. These were given pending the availability of Antimony Sodium Stilboglucuonate (10mg/kg daily for 3 weeks), which she never had due to non-availability coupled with financial constraints. Pentostam was very expensive and not readily available locally. The patient succumbed to superimposed chest infection before the arrival of the Pentostam ordered from abroad. This was 2 months after the diagnosis was established. A request for postmortem examination was denied by the relatives.

DISCUSSION.

Visceral leishmaniasis (Kala-azar) is caused by *Leishmania donovani.* It is endemic in Asia, the Mediterranean, South America, East and North Africa. It is relatively rare in sub-Saharan West Africa with occasional cases from Sene-Gambia (2), Togo (3) and Chad Republic (4). There are about 500,000 new cases of Kala-azar reported annually (5). It is endemic in about 62 countries around the world. It is spreading in several new areas owing to epidemiological changes, such as urbanization and mass migration of people. There are various clinical forms of Kala-azar in different localities. Two forms of transmission have been observed; the urban form where transmission is primarily human-to-human, and the rural form where transmission is primarily zoonotic. In Africa, a rural form of transmission is generally seen. The patient in this report is presumed to have acquired the infection by zoonotic means from rodents or ground squirrel, which are the usual reservoir of the parasites in this part of the world. The infection in her is also a sporadic human infection that is characteristics of zoonotic infection. The Africa Kala-azar differs in several ways from those seen elsewhere. Skin lesions are characteristic. These may represent healed ulcerations at the sites of the initial infection, or in some cases represent parasites reinvasion of the skin producing macular and nodular skin lesions. This is called dermal leishmanoid. Because of the dermatotrophic nature of *L. donovani* strain in this area, leishman skin test is often positive and parasites may be found in these dermal lesions. There were multiple superficial ulcers on the face and upper trunk of

this patient, but amastigotes could not be recovered from them. This may be as a result of paucity of parasites in the superficial lesions. However, in Sudan where a number of oral lesions were associated with visceral leishmaniasis, parasites were found in abundance in the oral lesions, but not in the enlarged spleen or liver. Such cases may represent intermediate condition of parasites virulence and host resistance between typical Kala-azar and cutaneous leishmaniasis (6). This case report presented with the symptoms and signs of African form of Kala-azar i.e. fever, weight loss, anaemia, visceromegaly, polyadenopathy and multiple skin lesions. Some infected patients may remain asymptomatic for a long time until they have their immunity lowered by malnutrition or some other tropical conditions and more recently HIV infection. Indeed overlapping of visceral leishmaniasis and AIDS had been documented (7). The risk of co-infection in Africa is less. Only one case each has been reported in Cameroon and Guinea-Bissau (8). However, this risk may soon be heightened in the sub region because of mass migration of people due to civil wars, famine and high rate of prostitution among the populace. By extension we may start to see more cases in Nigeria. Laboratory diagnosis of leishmaniasis is generally based on smears or histopathology and additional clinical information to characterize the species. Molecular studies with restriction endonucleases and isoenzymes pattern should ultimately provide a sound biochemical basis for identification and differentiation (9,10). Morphologic diagnosis is the most accepted for the identification of leishmania, which are intracellular parasites. They are typically found in the vacuoles of mononuclear cells or macrophages. In tissue section or smear stained with Giemsa, the parasites are identified by the presence of dark staining kinetoplast and a lighter staining nucleus. *L. donovani* is usually diagnosed in specimens from liver, spleen, bone marrow or lymph node. Scrapings from cutaneous or mucocutaneous ulcers must be taken from active margin of the lesions (11). Culture of the blood in Kala-azar aspirates or scraping in cutaneous lesions is definitive (12). Serologic tests may be of value in visceral leishmaniasis, but is of limited value in cuta-

neous disease. The most frequently used serologic test is indirect immunofloresence (IIF). The older complement fixation (CFT) test using mycobacterial antigen is also useful (13). The diagnosis was established in our patient by Giemsa staining of the buffy coat smear, in keeping with the fact that parasites were frequently found in the peripheral blood film of Africans with Kala-azar. For example, parasitaemia was detected in 15 (75%) of the 20 patients with Kala-azar in Kenya (14). The rarity of this condition in our environment is underlined by the non-availability of appropriate medication in most cities in our areas of practice. With increased awareness and diagnosis of Kala-azar in Nigerian patients, it is expected that the relevant drugs will be more readily available. In conclusion, practicing physicians in Africa are advised to consider Kala-azar in cases of pyrexia of unknown origin (PUO) especially now that it has been established to be an opportunistic infection in patients with AIDS.

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