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Kala-azar in Zambia: first report of two cases

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Summary

Two autochthonous cases of kala-azar, the first such report of the disease from Central and Southern Africa, are described. Both patients presented with generalized macules, papules and nodules without ulceration and both also had tuberculosis. Amastigotes were cultured from blood and identified in skin, bone marrow, liver and spleen.

Introduction

Two cases of kala-azar diagnosed in Zambia are presented. Both also had tuberculosis. This is the first report of kala-azar from Zambia, or indeed from Central and Southern Africa. Certain peculiar features of the presentation of the disease, which contrast with the known clinical features of kala-azar described from other parts of the continent were observed and are discussed.

Case reports

CASE 1

A Zambian male peasant, aged 60 years or older, presented with a history of small nodules and dark spots on the skin for six months and recurrent, unproductive cough for two months. He was born in Katete in the Eastern Province of the country, did a short spell of construction work during the building of the Kariba dam wall between Zambia and Rhodesia in 1955–58 and thereafter lived at Magoye in the Southern Province, where he worked seasonally as a farm labourer.

The patient was febrile (38·9°C), pale and malnourished. He had a cataract in the left eye. Numerous circumscribed, hyperpigmented cutaneous macules, papules and nodules, 2 mm to 30 mm in diameter were present on the face, trunk and limbs (Fig. 1). There was no ulceration of any of the lesions. His skin was generally dry, atrophic, wrinkled and finely scaly (Fig. 1), but he had bilateral pitting oedema of both legs. The superficial cervical, axillary and inguinal lymph nodes were palpable and described as discrete and shotty. Neither liver nor spleen was enlarged. He had a left basal pneumonia.

The skin biopsy of a representative macule revealed oval to round organisms, identified morphologically as amastigotes of *Leishmania sp.*, mainly in macrophages (Fig. 2). Smears from aspirate from the edge of skin nodules showed typical amastigote forms extracellularly and bone marrow smears showed mainly intracellular amastigotes, indicating conclusively that the species was *L. donovani*. Many thick and thin blood films stained with Giemsa were negative. Promastigotes were, however, cultured from blood inoculated on to NNN medium and from bone marrow and skin aspirate. Haemaglobin was 7·5 g per 100 ml, the ESR (Westergren) 75 mm (1 hour) and the total white cell count was 2,800 per mm^3 (90% neutrophils and 10% lymphocytes). Total serum proteins were 6·1 g (albumin 1·8 and globulin 4·3) per 100 ml. Blood urea was 25 mgm per 100 ml. The formol-gel test was strongly positive.

A chest X-ray showed some pleural thickening and patchy consolidation in the left lower zone. Acid-fast bacilli were absent on microscopy and a culture of sputum revealed no pathogenic organisms.

Liver biopsy was performed (results described below). The patient deteriorated rapidly and died on the 16th day after admission. Though supportive treatment was instituted the day after admission, specific therapy was not possible as neither a trivalent nor a pentavalent antimony compound was available in the country.

Post-mortem examination

Only the significant necropsy findings are presented. There was a severe degree of emaciation. The skin showed lesions as already described. There was oedema of both feet and legs. Superficial cervical, axillary and inguinal nodes were enlarged and caseous. There was miliary tuberculosis involving the lungs, pleurae, peritoneum, liver, spleen and lymph nodes. There was mild hepatomegaly (1,720 gm) and splenomegaly (400 gm).

Histopathology

A biopsy of one of the skin lesions revealed the typical pattern of mild cellular infiltrate consisting mainly of plasma cells and lymphocytes. The predominant feature was the accumulation of histiocytes laden with amastigotes (Fig. 2). Extracellular organisms were also noted.

Similar histological changes were observed in the liver, where there was marked proliferation and focal accumulation of Kupffer cells laden with organisms (Fig. 3). The bone marrow (Fig. 4), spleen and lymph nodes all showed similar lesions.

Aggregations of histiocytes with large numbers of amastigotes were also present in the interstitial tissue of kidneys, lungs, pleura, pericardium, adrenal medulla and intestinal mucosa.

Active caseous tuberculous lymphadenitis and miliary tuberculosis of lungs, pleura and pericardium was also present. There was, in addition, moderately severe haemosiderosis of all tissues.

CASE 2

A Zambian male, about 65 years old, was admitted to the Chipata General Hospital (Eastern Province) with a

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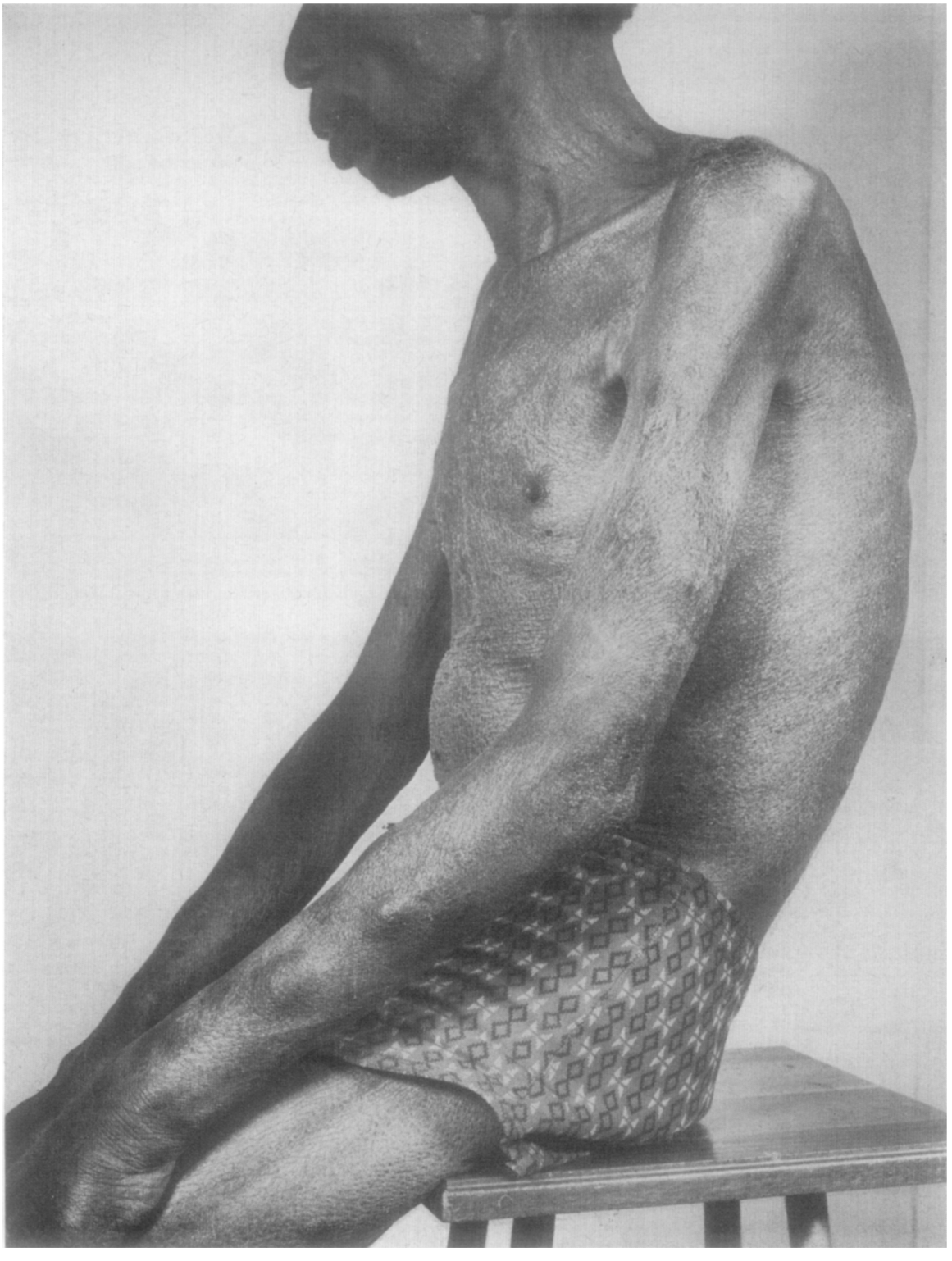

Fig. 1. The macular papular raised skin lesions are seen on both forearms, left upper arm, thigh, trunk and malar area of face.

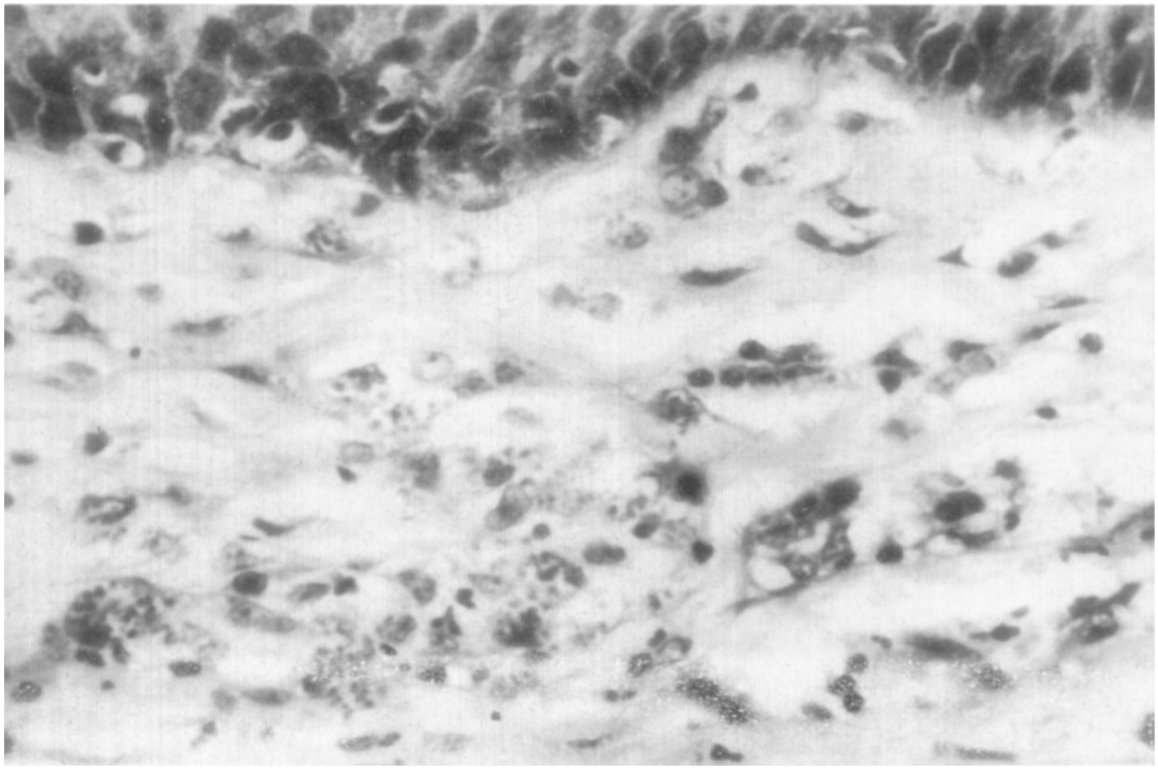

Fig. 2. Biopsy of a skin nodule reveals aggregates of histiocytes packed with amastigotes of leishmania. Inflammatory cells are scarce. *HE×950.*

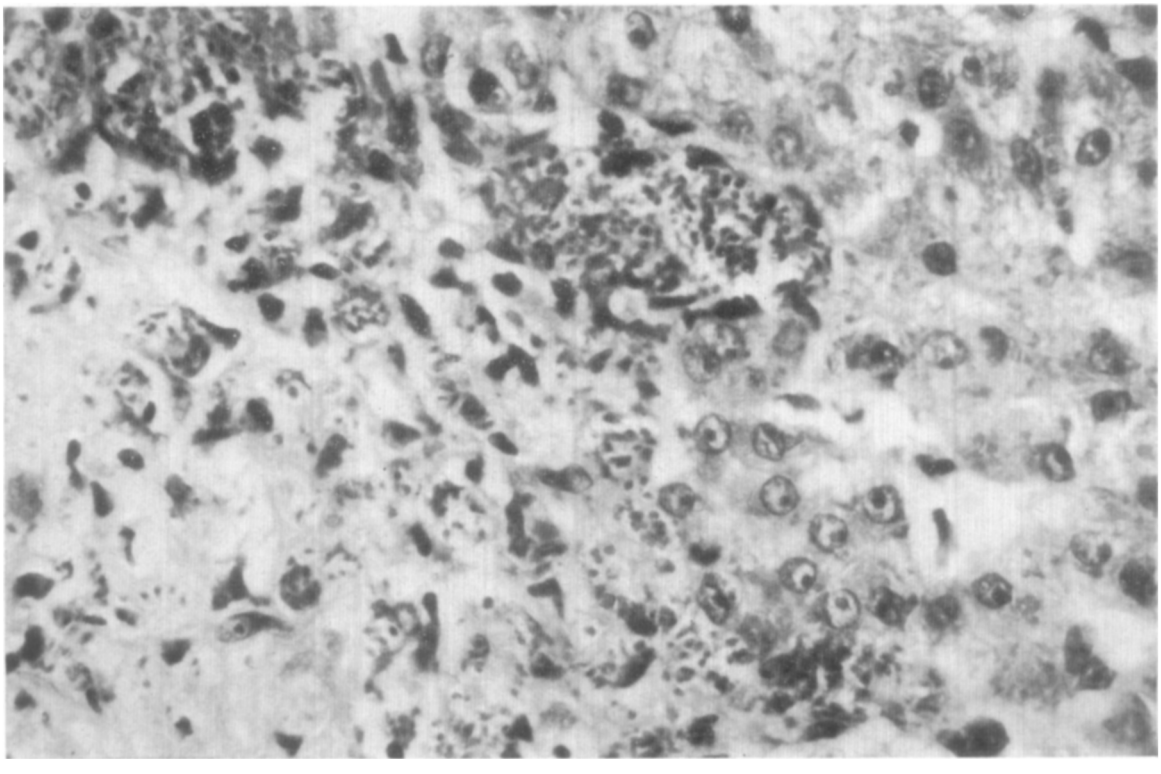

Fig. 3. Histology of liver demonstrates proliferating Kupffer cells packed with amastigotes of leishmania replacing parenchymal tissue. *HE×950.*

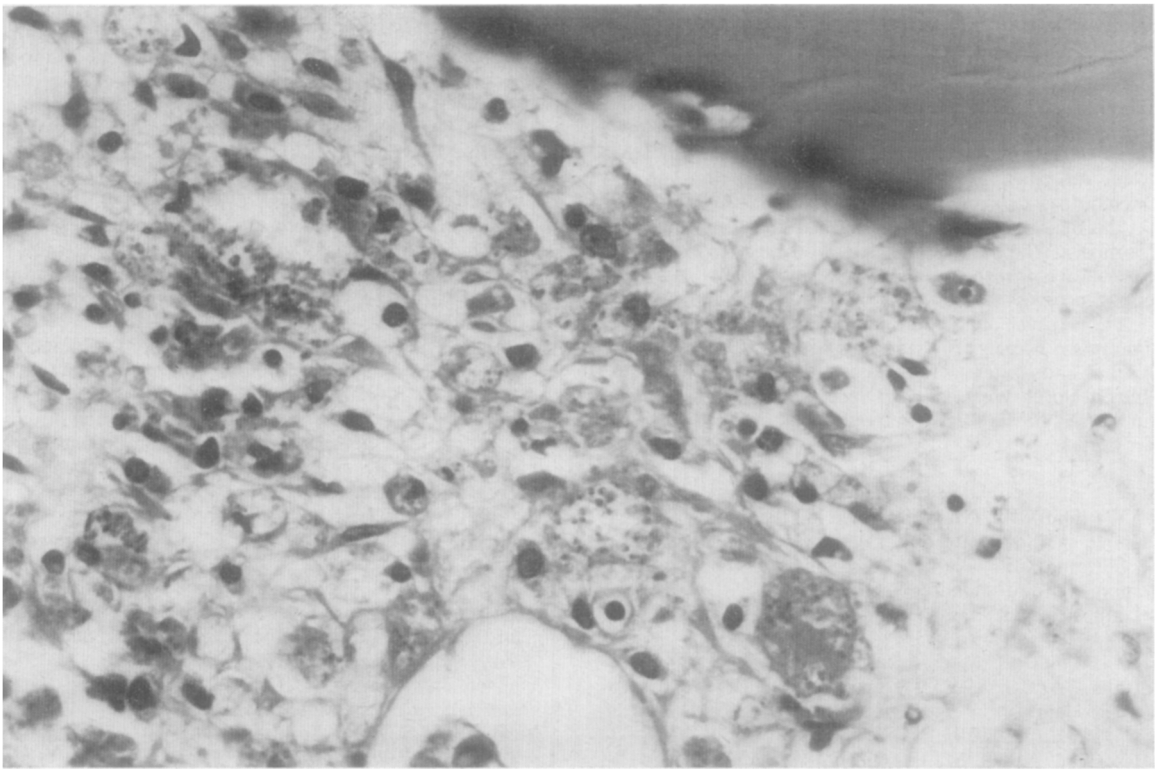

Fig. 4. Large numbers of macrophages filled with amastigotes of leishmania have replaced the normal bone marrow in this area. *HE×950.*

known history of pulmonary tuberculosis and macular-papular skin lesions. His skin lesions had started as small, painful papules on the buttocks about two years earlier and later spread to his extremities. He had worked at Shabani mines in Southern Rhodesia from 1930 to 1966 and was retired because of pneumoconiosis and pulmonary tuberculosis. He returned to Zambia and since then had not ventured from his village near Chipata.

The patient was grossly emaciated and had multiple, generalized, papular, discrete, nonulcerative, lesions on the buttocks, dorsum, palmar and plantar aspects of hands and feet and forearms, legs and both ears. There was hepatomegaly (2 cm below costal margin) but no splenomegaly. Chronic pneumoconiosis with superimposed pulmonary tuberculosis were confirmed. Sputa showed acid-fast bacilli. A skin biopsy showed typical features of cutaneous leishmaniasis but amastigotes were also present in bone marrow smears, confirming that this was a case of kala-azar. The patient died five days after admission. A post-mortem examination was not possible.

Discussion

Clinical presentation

The cases reported in this communication are examples of kala-azar. These are the first proven cases from Southern Africa. The only other report of kala-azar from countries neighbouring Zambia is the case reported from Zaïre (PREVOT, et al, 1968). Cutaneous leishmaniasis has, however, been reported from Mozambique (SATI, 1959), Tanzania (ANDERSON, 1964) and South-West Africa (GROVE, 1970; 1973).

It has been claimed that visceral and cutaneous leishmaniasis do not coexist in the same place (LYSENKO, 1971). This phenomenon is usually explained by the differences in the sandfly fauna in the various regions where leishmaniasis is prevalent. Our cases had visceral leishmaniasis with cutaneous manifestations not unlike those of DCL (diffuse cutaneous leishmaniasis). These findings are difficult to explain. Mirzoian (quoted by MANSON-BAHR, 1971) described skin lesions on the face which preceded visceral leishmaniasis in cases from Central Asiatic Russia. KIRK (1942) described similar lesions from Sudan. These observations were subsequently confirmed experimentally (MANSON-BAHR, 1959) and clinically in American personnel in the Sudan (CAHILL, 1964). WINSLOW (1971) has since described similar cases from China.

In our cases visceral leishmaniasis was accompanied by widespread skin lesions unlike those described by Mirzoian and Kirk.

Whereas the clinical presentation in endemic areas is generally uniform (MOSKOVSKIJ and SOUTHGATE, 1971), the main presenting feature in both our cases of kala-azar was the skin lesions. This is certainly an unusual presentation, although the classical presenting features may have been masked by coexisting tuberculosis in both cases.

Source of infection

Neither of our patients had been out of the country recently. There is no doubt that these are the first autochthonous cases of kala-azar reported from Zambia. Though the patients were referred from different provinces of Zambia, both were born in the Eastern Province. MANSON-BAHR (1971) noted that *Leishmania* sp. have been described from some species of wild animals, including gerbils and ground squirrels in Kenya and rats in the Sudan. In other areas kala-azar is an established zoonosis. Whether a similar situation exists in Zambia is speculative for the moment but further investigations are in progress.

The presence of phlebotomine sandflies in neighbouring countries, namely, *P. katangensis* in Southern Katanga (Zaïre) and Eastern Rhodesia and *P. grovei* from termite hills in South-West Africa (DOWNES, 1971), is significant in view of possible epidemic outbreaks of this disease, as in Kenya.

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