KALA AZAR IN A SINGAPOREAN

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SYNOPSIS

A Singaporean Chinese patient (born and bred in Singapore) presenting with pyrexia and gross hepatosplenomegaly was found to have kala azar. This is the first reported case of kala azar in a Singaporean. The patient responded well to treatment with Pentostam.

INTRODUCTION

Kala azar is an infectious disease caused by the protozoa Leishmania Donovani. It is endemic in many parts of the world. Singapore, however, is a non-endemic area. Nair et al documented the first case in Singapore which occurred in an immigrant worker from the Indian subcontinent where the disease is endemic (1). Kala azar in a Singaporean, born and bred in Singapore, has never been reported.

CASE HISTORY

A 32-year-old Chinese man presented in July 1984 with a two weeks' history of intermittent fever. The fever usually occurred in the evening and resolved in the morning with perspiration. Chills and rigor preceded the fever during the first few days. There were no other significant complaints. He was born in Singapore and resided all his life in Singapore except for a short vacation in Spain. He went to Spain (Majorca) one year previously and had stopped over in New Delhi Airport in transit.

Clinical examination revealed increased skin pigmentation and anaemia. The liver edge was 2 cm below the right costal margin and the spleen 3 cm below the left costal margin. Temperature recordings showed an irregular fever with bimodal distribution at times.

Investigations showed Hb 10.7 G/DL, WBC 2.7 × 10^9/L, platelets 118 × 10^9/L and a raised ESR of 80 MM/HR. Serum iron was 40μG/DL and TIBC 190μG/DL. Bone marrow aspiration and trephine biopsy showed reactive changes. The liver function tests were abnormal with total protein 8.2 G/DL, albumin 3.5 G/DL, bilirubin 1.2 MG/DL, alkaline phosphatase greater than 350 U/L, SGPT 95 U/L, and SGOT 115 U/L. Serum electrophoresis showed reduced albumin and polyclonal rise in gammaglobulin. IgG level was raised at 2218 MG/DL. There was also significant proteinuria (1.02 G/24 HR). The abdominal X-ray, ultrasound scan and isotope liver scan confirmed the hepatosplenomegaly but gave no clue as to the cause. Negative or normal investigations included serum folate, B_{12}, direct Coombs' test, haemoglobin electrophoresis, clotting studies, urea and electrolytes, glucose, plasma cortisol, alpha foeto protein, hepatitis B surface antigen, blood film for malaria parasite, blood culture, urine culture, stool culture and examination for ova and cyst, bone marrow culture for tuberculosis, Widal Weil Felix test, amoebic antibodies, VDRL, FTA/ABS, rheumatoid factor, antinuclear antibodies, T cell helper: suppressor ratio and chest X-ray.

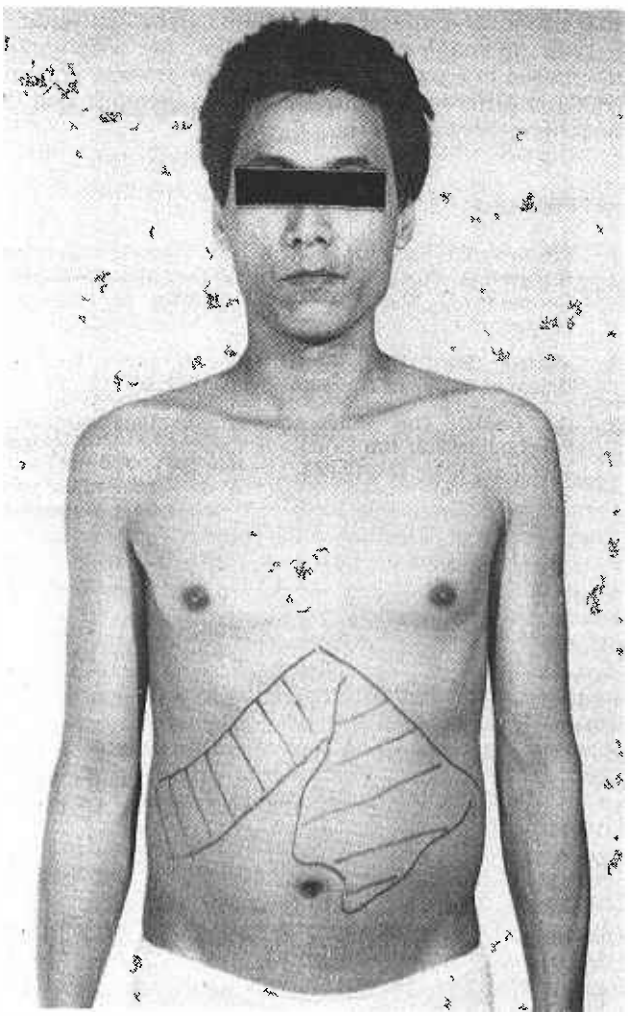

Figure 1 Gross hepatosplenomegaly in kala azar

After two weeks' stay without any apparent progress, the patient discharged himself to seek further medical help. He was seen again one month later when his condition had deteriorated further. He continued to have fever, malaise and easy fatigability and had also developed abdominal distension as well as a skin rash in the groin and back. On clinical examination he was again febrile with gross hepatosplenomegaly (Fig. 1) with his liver edge 6 cm below the right costal margin and spleen 17 cm below the left costal margin. Investigations showed pancytopenia: Hb 6:8 G/DL, WBC 1.5 × 10^9/L, platelet 61 × 10^9/L. ESR 144 MM/HR, total protein 8.2 G/DL, albumin 2.4 G/DL, bilirubin 0.9 MG/DL, alkaline phosphatase 635 U/L, SGPT 98 U/L, SGOT 36 U/L. Computerised axial tomography (CT scan) of the abdomen showed enlarged paraaortic lymph nodes in addition to gross hepatosplenomegaly. Biopsy of the skin rash showed inflammatory reaction.

A repeat bone marrow examination revealed plentiful macrophages, laden with Leishman-Donovan bodies (Fig. 2). A diagnosis of kala azar was made.

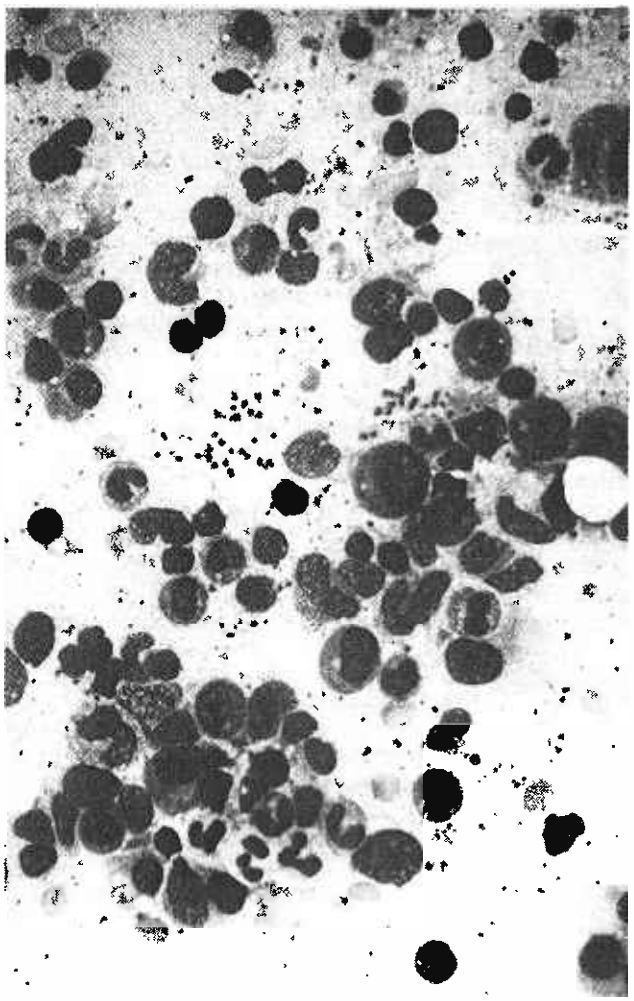

Figure 2 Bone marrow aspiration: macrophages laden with Leishman Donovan bodies

He was started on Pentostam (Sodium Stibogluconate) intravenosly 600 mg daily for 30 days. The response was gradual and the fever subsided after 12

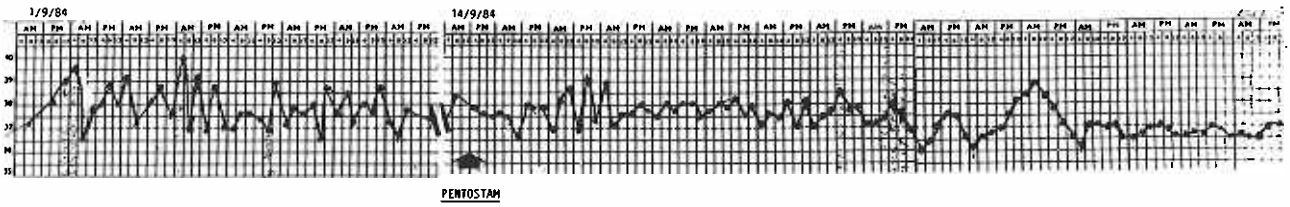

Figure 3 Temperature chart showing effect of treatment with Pentostam

days of therapy (Fig. 3). The hepatosplenomegaly improved slowly. By June 1985, the liver edge was palpable three cm below the costal margin and the spleen not palpable. His full blood count and liver function tests had all returned to normal.

DISCUSSION

Kala azar is a zoonotic infection caused by the protozoa Leishmania Donovani. The usual hosts are rodents and canines but in the Indian subcontinent man is the only host. Transmission is by sandflies (of the genera Phlebotomus in the old world and Lutzomyia in the new world). The disease is endemic in the Mediterranean areas, Africa, Near and Middle East, Southern Russia, China, India and South America (2).

With increasing ease of international travel, people are now more at risk of contracting exotic diseases when travelling from a non endemic area to an endemic one. We believe that our patient had contracted kala azar while holidaying in southern Spain as it is endemic there. The other possibility was that he had contracted the disease at New Delhi Airport. This is less likely as the stop-over in the airport was brief and hopefully there were no sandflies in the airport transit area. The possibility of local transmission of the disease cannot be ignored. The presence of a pool of immigrant workers with clinical or subclinical infections could act as a human reservoir. The sandfly vector (Phlebotomus Argentipes), which transmits kala azar in India, is also found in the Malayan Peninsula (3) from which Singapore is separated only by a narrow strait.

It is obvious and crucial that a correct diagnosis should be made as most patients respond well to specific treatment and the mortality in untreated individuals ranged from 75 to 95 percent within two years. In Singapore kala azar had only been reported once previously in an immigrant worker from the Indian Subcontinent (1). It has never been described in a Singaporean, born and bred in Singapore. Because of its rarity, the diagnosis in our case was not made for about two months. Looking back, this patient had most of the important clinical features and laboratory evidence to suggest the diagnosis of kala azar. The history of travel to Spain one year previously would fit in well with the incubation period of kala azar (three months to two years). The presentation with irregular fever, increased skin pigmentation, rapidly enlarging spleen and a relatively well patient despite the swinging fever is common in patients at the early stages. The presence of significant proteinuria, pancytopenia, grossly raised ESR, hypoalbuminaemia, hypergammaglobulinaemia and polyclonal rise in IgG level are all in keeping with the diagnosis. Obviously, the crucial evidence in the diagnosis was the demonstration of the Leishman Donovan bodies in the bone marrow specimen. Differentiation from histoplasmosis can be difficult but must be made as the treatment in the two conditions are quite different. The excellent response to Pentostam in our patient once again emphasised the need for correct diagnosis in treating patients with pyrexia of unknown origin so that correct and effective treatment can be instituted.

With increasing travel the list of differential diagnoses in a patient with pyrexia of unknown origin (PUO) in different countries tends to increase and is becoming more universal.

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