VISCERAL LEISHMANIASIS IN HAZARA

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Abstract: Seven cases of visceral leishmaniasis are reported. They were seen during 1988 and were between the ages of 1—1/2—8. One patient left without treatment, another left while the treatment was just started. One died before treatment could be started due to late availability of the drug. Four remaining patients were treated with pentavalent antimony compounds, blood transfusion and general supportive treatment. Two out of four patients were treated successfully while the other two are under treatment and improving.

Introduction

The disease was thought to be non-existing in our part of the country after the separation of East Pakistan (now Bangladesh) in 1972. But cases were reported as early as 1979 from Northern Areas[1] of Pakistan by Burney. Recently in 1986 14 patients were reported by Saleem from Azad Kashmir, adjoining areas of Pakistan and Northern Areas.[2] These cases were collected in 1983 to 1985 from Armed Forces Institute of Pathology and Pathology department of Army Medical College, Rawalpindi. Three patients were treated: by Ashfaq (unpublished) one a 3 years old Afghan refugee from an Afghan refugees camp in Peshawar and two from Hangu in Kohat division. One case reported from the hilly area of village Yaka Ghund near Warsak Dam. We have seen seven patients in less than one year. The disease is present in the foot hills, scattered all over the NWFP (Kohistan, Nathiagali, Peshawar, Kohat) Azad Kashmir and adjoining areas of Punjab and Northern Areas. Only scattered cases are reported having no epidemics due to the nature of the disease as explained below. The increase may be genuine or may be more awareness on the part of Physicians and Pathologists.

The LD (Leishman Donvan) bodies are usually intracellular (inside the macrophages), or both intra and extracellular; but in our case No. 1, they were scanty and totally extracellular. This may be due to rupture of the cells during slide preparation. But the intact cells too were without LD bodies. Two of four treated cases are being reported here.

Case Report No. 1

A 2 years 8 months old boy from Kala Bagh, Nathiagali admitted to DHQ Hospital, Abbottabad on 3rd April, 1987 with a 7 months' history of ill health, pallor, enlarged abdomen, cough and epistaxis. He was wasted but had good appetite. On examination he was found to be wasted, severely anaemic and had early cancrumoris, generalized lymphadenopathy and hepatosplenomegaly

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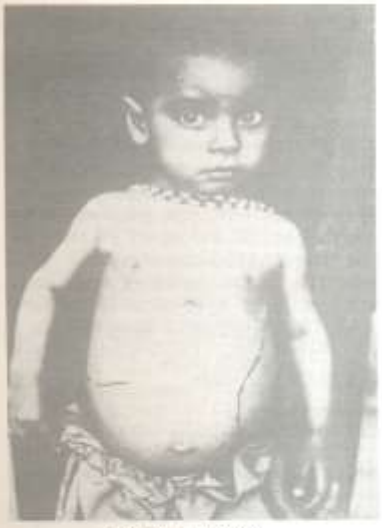
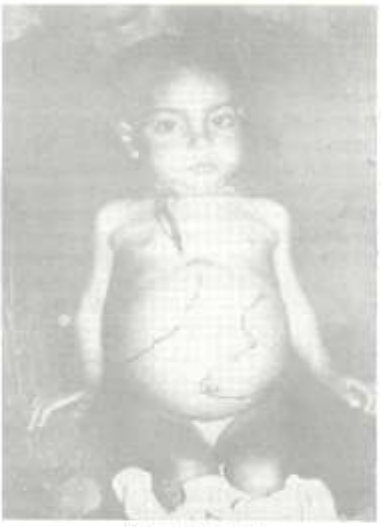


Before treatment



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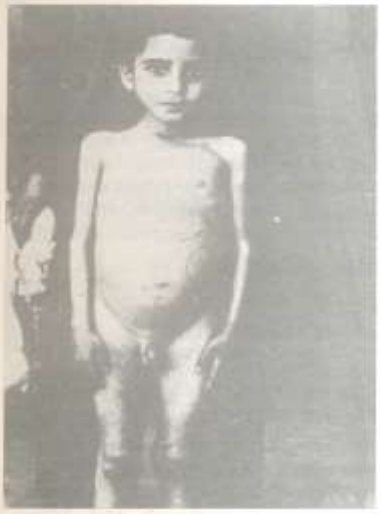

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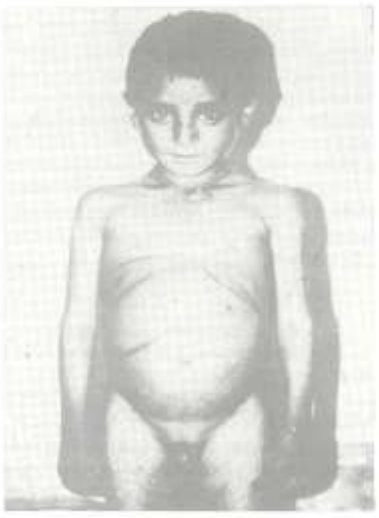

After treatment

His blood picture showed Hb. 3.4gm%, TLC 2300/ Cmm, low RBC and platelets count. His serum proteins were 9.9gm%, albumin 4.8gm%, globulin 5.1gm% with reversed A/G ratio. Bone marrow done thrice was unproductive. Trephine biopsy done showed a hypo plastic picture with only few extracellular LD bodies. Formal gel test was positive. He received Pentavalent Antimony compound (Pentostan) at a dose of 20 mg/kg 1/M daily for 4 weeks with supportive treatment and blood transfusion.

At the time of discharge, he was afebrile. His serum proteins were 6.2 gm%, Albumin being 3.9gm%, Globulin 3.3gm% and A/G ratio reverting back to normal. The haemoglobin 10.6gm% and bone marrow became nor- mocellular with no LD bodies at the time of discharge. Liver and spleen regressing gradually and only few lymph nodes were palpable.

Case No. 2

An 8 years old male child from Dasu (Kohistan) was admitted to DHQ Hospital on 3rd June, 1988 with six weeks' history of fever and abdominal distension. There was a daily rise of temperature with rigors and sweating. He also had loose stools with mucous. He was found to be anemic, febrile with hepato-splenomegaly and generalized lymph-adenopathy.

His Hb being 6.4gm% with normal WBC count and ESR of 110mm 1st hour. Platelet count normal. Serum protein were 11 gm% with globulin of 6.4 gm% and albumen 4.6gm%. Aldehyde and formal gel test were positive. Bone marrow biopsy revealed orderly and active picture with plenty of intra cellular and extra cellular LD bodies. He was treated with pentavalent antimony compound (sod. Stibugluconate) 20 mg/kg 1/M body weight for 4 weeks with blood transfusions and general supportive treatment.

On discharge from the hospital anaemia was corrected, patient was afebrile, Liver, Spleen regressing. Serum protein were 6.7gm% with A/G ratio gradually reverting to normal. Bone marrow became normal with no LD bodies.

Discussion

After a bite by the sand fly (Phlebotomus in the old world and Lutzomiyia in the new world) a local lesion may be noticed,[6] from 10 days to a few weeks' time in the non-immune victims, the parasite spreads to the reticulo-endothelial system, e.g., lymph nodes, liver, spleen, intestinal mucosa, bone marrow and skin. It affects the young, but adults from a non-endemic area are also affected being non-immune.

The patient reacts with fever which may have a double rise in 24 hours, lymphadenopathy, hepatosplenomegaly, intestinal symptoms and bone marrow involvement leading from hyperplasia to severe hypoplasia which may result in thrombocytopenia causing bleeding and neutrophnia leading to infection, e.g., chest infection, T.B., dysentery, malaria, worm infestation; diseases which are prevalent in the area where leishmaniasis occur. Severe Cancrumoris may occur terminally.

The disease that occurs in this area is of mediterranean[3] (Chinese) type as against the Indian Kala Azar in which there is black pigmentation on extremities and trunk. In the Indian type human beings are the reservoir and phlebotomus argentipes which is anthropophagic lives near the houses and leads to epidemics. In Mediterranean or Chinese type, canine, e.g., jackals, wolves and dogs are the reservoir and epidemics thus do not occur. That is why we see only sporadic cases scattered all over in the foot hills. Specific treatment is with pentavalent antimony compounds if started early is life saving and rewarding.

The response in Indian type is quicker and relapses less common. The dose in children is 20

mg/kg/day for 6—14 days. But it is prolonged in African[4] and Mediterranean types[6] and treatment is to be given for longer periods. In cases who do not respond to proper doses of pentavalent antimony compounds or in the event of relapses pentamidine, a diamidine derivative, can be used but has side effects like hyperglycemia, hypoglycemia and hypotension with kidney damage. Amphotericin-B, an antifungal drug can also be used, especially in relapses which too has serious side effects. Allopurinol is another[5] drug which cannot be an alternative to pentostam but may be used in conjunction with pentostam. Paramomycin (amino-sidine) is being tried in India and Africa alone and in combination with pentostam. The results are encouraging but still under trial. Dose is 16 mg per kg body weight for 10 days.

Leishmaniasis is a treatable disease and can be fatal if not detected and treated early. The population at risk which lives in the hilly areas is usually poor and uneducated which causes delay in specialist opinion, thus leading to delay in diagnosis. Non-availability of the drugs causes further delay in the treatment and moreover the drugs are very expensive and the poor cannot afford it.

The idea of this presentation is to educate both the people in the affected areas and the junior doctors at the periphery that visceral leishmaniasis exists and suspects with above clinical signs be investigated to exclude this disease.

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