

VISCERAL LEISHMANIASIS IN A SIX MONTH OLD CHILD: IS CONGENITAL TRANSMISSION OF DISEASE POSSIBLE?

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Infantile kalaazar is a sporadic disease and occurs in many countries of the Mediterranean region, Middle East and China. The causative organism of this disease is *L. infantum* and is primarily a zoonosis, occurring in dogs and other wild carnivores which serve as reservoir. Transmission of leishmaniasis occurs by the bite of the female phlebotomine sandflies and although numerous species are found in endemic areas, only a few have so far been proven as vectors. *P. alexandri* in China, *Ph. rnaritini* in the Sudan and *Ph. argentipes* in India are the confirmed vectors of *L. donovani* infections. For *L. infantum* infections, *Ph. ariasi*, *Pb. perfeliewi* and *Pb. perniciosus* have thus far been incriminated¹.

First reports of visceral leishmaniasis from Pakistan came in the early sixties^{2,3}. Cases mostly children, were described from the valleys of Gilgit and Skardu situated at a height between 7500 to 8500 feet above sea level. Later studies revealed that the disease was also present in Kashmir and its adjoining regions⁴. The disease is sporadic and clinically resembles the Mediterranean type of visceral leishmaniasis. In at least one isolate obtained from bone marrow of a 2 year old male child from Murree hills the organism was typed as *L. infantum* s.s. by isoenzyme characterization⁵. Different species of sandflies exist not only in this area³, but are also found in the neighbouring areas of Kashmir valley⁶. The disease affects mainly children although adults are not entirely immune. In north-west China 95% of cases were children below the age of 10 years⁷ and those reported in Pakistan have all been above one year, although the disease does occur occasionally in younger children. We describe a case of 6 month old boy from Bagh in Azad Jammu and Kashmir.

CASE REPORT

A six month old male child was admitted in the hospital with complaints of irregular high grade fever and cough for about a fortnight. Three days prior to admission he developed generalized purpuric rash all over the body. He had severe bleeding tendency especially from puncture sites. He was the first issue of his parents and was immunized upto date. On physical examination he was febrile and pale, with liver enlargement of 3 cm below right costal margin and a big spleen 5.5 cm below left costal margin. There was no lymphadenopathy, and auscultation of chest revealed occasional scattered crepitations bilaterally. A provisional diagnosis of septicemia/DIC was made.

Laboratory investigations revealed a total white cell count of $3.7 \times 10^9/l$, with 36% neutrophils and 64% lymphocytes. Total red cell count was $3.7 \times 10^{12}/l$, haemoglobin 10.5g/dl, PCV 0.3051/l, MCV 82.7fl, MCH 28.5pg and MCHC 34.4g/dl. The platelet count was $23 \times 10^9/l$ and his bleeding and clotting times were measured as 10 and 6 minutes respectively. Although his serum albumin: globulin ratio was reversed, the serum immunoglobulins were within normal limits. Bone marrow examination revealed moderate depression of all the normal cell lines, with slight increase in the number of histiocytes. Scanty amastigote forms, mostly extracellular were seen. Aspirate from bone marrow was also inocu-

lated on NNN medium and leishmania promastigotes were grown after 11 days.

The child was treated with sodium stibogluconate (pentostam), 20 mg/kg intramuscular, once daily for thirty days. During his hospital stay the patient developed series of complications such as bronchopneumonia,

gastroenteritis, epistaxis and bleeding from puncture sites. He was managed with intravenous broad spectrum antibiotics, intravenous fluids, blood and platelet infusions and nasogastric feeding. He finally made a full recovery and was discharged from the hospital after a stay of 44 days.

DISCUSSION

Infantile visceral leishmaniasis may be of insidious or acute onset with irregular febrile episodes, anaemia, cachexia, hepatic and splenic enlargement as main symptoms. Malnourished children are more prone to acquire active infection⁸ Majority of the cases reported from the northern areas of Pakistan are under the age of 10 years³, although in the wilderness of northwestern China almost 90% of the diseased children are under 2 years⁷. The incubation period of this disease averages between 3-5 months, although it can vary and be very long. In two cases following voluntary inoculation of promastigotes isolated from dogs, the incubation period was 3.5 and 5 months respectively⁷. The same was observed in two cases who acquired their infection through blood transfusion⁹. Experimental infection in volunteer following bites of infected sandfly is reported to occur between 134 to 166 days¹⁰.

Congenital transmission of disease was first suspected in 1926¹¹, but no clear evidence of this is documented. Napier and Das Gupta reported infection in a new born child where the incubation period was clearly under 3 months¹². Two children in China developed disease at the age of 3 and 3.5 months⁷. Another report describes the disease in a four month old child born prematurely in Kenya who was sick from the sixth day of his life¹³. It has been postulated that the mode of infection the baby can be (i) direct transmission from mother to offspring, or (ii) acquired at the time of birth from perineal hemorrhages with swallowing of maternal blood or secretions or through abraded skin. This case report describes the youngest victim of infantile kala-azar in Pakistan thus far reported. The mother of the child was healthy before and during her pregnancy and at no time complained of symptoms suggestive of visceral leishmaniasis. She was negative for anti-leishmania antibodies when tested by indirect fluorescent antibody technique. In this case the infection was probably acquired postnatally.

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