RECURRENCE OF KALA-AZAR IN A CASE OF POST KALA-AZAR DERMAL LEISHMANIASIS

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A case of kala-azar following PKDL, in which PKDL lesions and visceral lesions existed simultaneously is being reported. The second attack could have been due to a fresh infection or due to visceralisation of the dermal lesions. The patient responded to a combination of sodium stibogluconate and allopurinol.

Key words : Kala-azar, PKDL, Sodium stibogluconate, Allopurinol.

Post kala-azar dermal leishmaniasis (PKDL) follows clinical cure of kala-azar in a proportion of cases, and such cases are usually considered immune from the visceral disease. Moreover, PKDL lesions are known to remit when visceral disease becomes active again. However, occasional cases in which PKDL was followed by visceral leishmaniasis have been recorded.[1-3] We are reporting a case who had visceral leishmaniasis subsequent to PKDL, and PKDL lesions persisted even during the manifestation of the visceral disease. This report is the 9th such report from India, and first from Bihar, which saw a resurgence of kala-azar from mid seventies and remains an endemic zone for kalaazar even now.

Case Report

A male aged 19 years was admitted in December 1981 with complaints of irregular fever, loss of appetite and bodyache for the last two months and skin eruptions for about 3 years. The patient had kala-azar 4 years back, for which he had received 9 gm of sodium stibogluconate (SSG) over a period of 15 days and got cured clinically and parasitologically (absence of amastigotes in splenic smear). Ten months later, he developed skin eruptions all over the body. He was pale and febrile. There were generalised hypopigmented macular rashes all over the body, confluent at some places and most dense over the chest. There was one hyperpigmented maculo-papular patch over the tip of his nose and nodules on the base of nose, alae nasi and chin. Liver was enlarged 2 cm below the costal margin and spleen 5 cm below the costal margin.

The TLC was 4,800/mm^3, P 60%, L 29%, E 7%, M 4%, haemoglobin 9 gm/dl and aldehyde test was positive. Splenic aspirate smear and skin smear showed presence of amastigotes (LD bodies). Urine, liver function tests and chest X-ray were normal.

The patient was given 6 ml sodium stibogluconate (Stibanate, 1 ml containing 100 mg of Sb) intramuscular daily. As there was no clinical and parasitological response by the 4th week, allopurinol 20 mg/kg daily in 4 divided dosages was added and continued for 3 weeks. Weekly splenic aspirations were done. Remission of fever, regression of splenic size, increase in weight, rise of haemoglobin and TLC occurred by the 36th day and the skin lesions also became fainter. Parasitological cure i.e. absence of amastigotes in splenic aspirate smear and skin smear was achieved at 6th week, the skin lesions improved further, but did not clear up completely. SSG was continued for a total of 60 days after which the patient was discharged.

At 1, 3, 6 months and 1 year follow-up, the patient did not have any recurrence of the symptoms. Skin lesions had disappeared at the first follow-up.

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Comments

PKDL is a sequel to generalised infection with *Leishmania donovani.* Visceral leishmaniasis occurring subsequent to PKDL raises the question as to whether the visceral infection is due to (i) a fresh infection, (ii) reactivation of a latent visceral infection or (iii) visceralisation of infection from dermal lesions.

It is recognised that cure of kala-azar endows a patient with considerable protection[4] and second attack of kala-azar even in highly endemic areas is almost unknown. Sen Gupta and Mukherjee[3] were of the opinion that reinfection did not seem likely to cause kala-azar in cured patients of kala-azar. In the recent Bihar epidemic of seventies, it was observed that PKDL occurred more commonly in the populations where transmission was continuing, whilst in areas where vector control was achieved, PKDL did not occur, suggesting that PKDL is a cutaneous response to reinfection.[5] It is possible that the same reinfection which caused PKDL is responsible for the subsequent visceral leishmaniasis. Alternatively, the second attack of kala-azar may be a fresh visceral infection.

Relapses in kala-azar occur usualy within a year of the initial attack of kala-azar.[6] In our case, the interval between the two attacks of kala-azar was 4 years. It therefore seems unlikely that reactivation of latent infection was responsible for the second attack. PKDL cases do not show amastigotes in spleen, bone marrow or liver,[3] so it seems unlikely that leishmanial infection can persist in viscera for years.

Patients having PKDL, harbouring parasites in the skin for years after the initial attack of kala-azar, and parasites ultimately causing kala-azar, point out that parasites do not change their tropism from viscerotropic to dermotropic or vice-versa. Which of these cured kala-azar patients develop PKDL and which of these are susceptible to a further recrudescence of kala-azar remains enigmatic.

Unresponsiveness of the patient to SSG is not surprising. Four of the 5 cases reported by Sen Gupta and Mukherjee[3] and one case by Bramhchari[1] were also unresponsive to the conventional therapy. Allopurinol is known to have antileishmanial activities and has been used alone and in combination with SSG in Kala-azar in Kenya[7] and India.[8] Our patient, though unresponsive to SSG alone, responded well to a combination of SSG and allopurinol.

References

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