

LARVA CURRENS - A MARKER OF HIV ?

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A 41-year-old man with larva currens was diagnosed to have HIV infection. There was no evidence of hyperinfection with *Strongyloides stercoralis*. The effect of coexistent HIV infection in the natural course of strongyloidiasis is discussed.

Key words : Larva currens, Strongyloidiasis, HIV infection

Larva currens (racing larva) is the cutaneous lesion produced by the wandering larva of *Strongyloides stercoralis* during the autoinfection cycle. It is characterised by recurrent pruritic serpiginous urticarial bands starting in the perianal region and moving to the trunk or thighs at a rate of several centimeters per hour.¹ Strongyloidiasis is the only helminthic infestation significantly associated with HIV infection.

Case Report

A 41-year-old promiscuous truck driver reported to our OPD with pruritic urticarial bands, with a few papules and erosions on the trunk and thighs of 3 days duration. He had recurrent attacks of such lesions at intervals of 15-20 days for the previous one year. The lesions started on the buttocks and spread to the back, abdomen and thighs within

hours. As it extended in curvilinear pattern, the initial lesions used to disappear. Each episode used to persist for about a week. He also had generalized, firm, discrete, non tender lymphadenopathy. There were no constitutional features. The systems were normal. Urinalysis and haemogram were normal. Stool smear did not show any ova or parasite and was repeatedly negative for strongyloides larvae. Modified Harada - Mori culture of stool² was attempted, but did not yield the larvae. LFT, RFT and chest x-ray were normal. Blood ELISA for HIV was repeatedly positive. FNAC of the lymph nodes showed a florid reactive hyperplasia.

He was treated with tab. albendazole 400mg bid for 3 days, which cleared the lesions. He was symptom free on review after 4 months and apparently healthy, except for the lymphadenopathy, suggestive of persistent generalized lymphadenopathy (PGL).

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Discussion

The characteristic skin lesions suggested the diagnosis of larva currens. The prompt thera-

peutic response to albendazole supported it. It may not be possible to demonstrate larva of *S.stercoralis* in all cases, as direct smear will be positive only in 72% of cases even after repeated tests.³ This is because of the sporadic and often scanty excretion of the larvae.⁴ The culture will be successful only if the strain of the parasite tested is capable of a free living cycle of development in soil.⁴ Depression of cell-mediated immunity (CMI) is reported to result in hyperinfection with *S. stercoralis*.⁵ This is a disseminated disease characterised by a massive increase in the number of the parasites invading various internal organs, including lungs, liver and central nervous system, often with a fatal course. When AIDS was recognised, disseminated strongyloidiasis was included as one of the indicators of the disease.⁶ But till recently very few cases of hyperinfection have been reported, inspite of the high prevalence of HIV in areas endemic for strongyloidiasis. This has probably led to the exclusion of disseminated strongyloidiasis in the recent CDC classifications of AIDS.⁷

It is possible that the depression in CMI associated with HIV infection led to repeated autoinfections with *S.stercoralis* in our patient. However he did not show any evidence of hyperinfection. This may support the view that hyperinfection with *S.stercoralis* is rare in HIV infection.

It is also possible that the relatively intact CMI status of our patient with PGL prevented a massive strongyloidiasis. This indicates that the natural course of strongyloidiasis may not be altered at least in the early stages of HIV infection.

To the best of our knowledge, this is the first report of larva currens associated with HIV from India. As *S.stercoralis* is common and HIV is on the increase, more reports are to be expected. This should further clarify their relationship.

References

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