CASE REPORT

Histoid leprosy in an HIV positive patient taking cART

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Accepted for publication 09 July 2010

It is well known that HIV infected patients are more susceptible to infection with M. tuberculosis, but this is not clear for M. leprae. Only a few studies have reported this coexistence. Co-infected cases have been reported in the past in the whole spectrum of leprosy i.e. tuberculoid,1 borderline,2 and lepromatous.3,4 The deficiency of cell mediated immunity in leprosy is specific to M. leprae antigen and probably, the reason why the clinical course of leprosy is not influenced by HIV infection.5 When HIV positive patients are treated with highly active anti-retroviral treatment (cART) there is treatment-induced recovery of the immune system of the host which may lead to immune reconstitution inflammatory syndrome (IRIS). Various authors have reported tuberculoid leprosy6,7 and Type I lepra reaction8 as a manifestation of IRIS in HIV positive patients. We are reporting an HIV positive patient who developed histoid leprosy in spite of taking cART for 9 months, without any signs and symptoms of IRIS.

Case report: A 42 year old married, male truck driver, presented with genital ulcers to the Department of Dermatology, SP Medical College, Bikaner. He gave a history of multiple unprotected acts of sexual intercourse with many roadside prostitutes in different parts of the country for many years. On examination, there were superficial serpigenous painless ulcers over the glans and prepuce. Clinically he was diagnosed as a case of herpes genitalis and treated with oral and topical acyclovir. A history of high-risk behaviour prompted an HIV test, which was performed after pre-test counselling, and found to be reactive by Tridot as well as ELISA test. The VDRL test was non-reactive. IgG and IgM antibodies against HSV-2 were raised. He refused tests for CD4 + cell count, viral load and cART due to financial constrains. Post test counselling was then done. After 2 months the patient returned with herpes genitalis along with oral candidiasis. He was counselled and cART was started with a combination of zidovudine, lamivudine and nevirapine, which was well tolerated. After 9 months of cART therapy he returned with generalised skin coloration, shiny, non-itchy, painless and non-tender, discrete,
papulo-nodular lesions distributed symmetrically all over the body including oral mucous membrane and tongue which had been present for the last one and half months (Figures 1 and 2).

The lesions were not associated with fever, joint pains and lymphadenopathy. The peripheral nerves were not thickened, and there was no glove or stocking anaesthesia. Systemic examination was normal. VDRL and TPHA tests were again non-reactive. An X-ray on his chest was normal, and PCR for *Mycobacterium avium intracellulare* was negative in a skin biopsy specimen. The CD4 + cell count was 400 cells/mm$^3$. The slit skin smear from a nodule was highly positive for *Mycobacterium lepra*, and the histopathological findings were consistent with Histoid leprosy (Figure 3).

MB-MDT was started along with cART, and Efavirenz was substituted for Nevirapine. The lesions had completely regressed after a year, but MDT was continued for 1 more year. The patient was lost to follow up after 2 years.

**Figure 1.** Papulo-nodular lesions of histoid leprosy over the back in an HIV positive patient.

**Figure 2.** Nodules of histoid leprosy over the tongue in an HIV positive patient.
Leprosy in HIV infected patients have been reported, and most of them were either in untreated HIV positive patients,1–4 or as a manifestation of IRIS,6–8 while this patient developed histoid leprosy during anti-retroviral treatment without showing any signs and symptoms of IRIS. In our case neither HIV infection nor cART influenced the course of leprosy, as the patient developed histoid leprosy in spite of taking cART for 9 months. This observation is consistent with the study by Jacob et al.5 who reported that the clinical course of leprosy is not influenced by HIV infection. In the present case even after increase in CD4+ counts from 150 to 400 cells/mm³,3 the patient developed histoid leprosy rather than reversal reaction, which may reflect the poor specific host immune response to M. leprae in HIV co-infection. We excluded Mycobacterium avium intracellulare infection which may mimic histoid leprosy by PCR in skin biopsy specimens.9 Ethical approval was not required for this study.

References

8 Kharkar V, Bhor UH, Mahajan S, Khopkar U. Type 1 lepra reaction presenting as immune reconstitution inflammatory syndrome. Indian J Dermatol Venereol Leprol, 2007; 73: 253–256.