in a non-diabetic patient. Liver cirrhosis, impaired glucose tolerance and diabetes mellitus are common disorders with overlapping pathophysiologic mechanisms. It is surprising that HHNS has not been described previously in patients with liver cirrhosis.

References


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Post-Kala-Azar Dermal Leishmaniasis Manifesting after Initiation of Highly Active Anti-Retroviral Therapy in a Patient with Human Immunodeficiency Virus Infection

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Key words: kala-azar, dermal leishmaniasis, human immunodeficiency virus, immune reconstitution, highly active anti-retroviral therapy

The spectrum of illness involving patients infected with the human immunodeficiency virus is constantly changing. Some of these changes may be attributed to the new highly active anti-retroviral therapy. Restoration of immune function in patients responding to this therapy has resulted in the augmentation of the inflammatory response to a variety of HIV-related diseases [1]. This phenomenon, termed the ‘‘immune reconstitution syndrome,’’ has recently been reported in the context of atypical mycobacterial disease, cryptococcal adenitis, cytomegalovirus retinitis, and autoimmune hyperthyroidism. We report here the occurrence of post-kala-azar dermal leishmaniasis in an HIV-infected patient following the initiation of HAART.

Patient Description

A 32 year old man was referred to our institution because of a rash of 3 months duration. The patient had immigrated to Israel from Ethiopia 4 years earlier and HIV infection was subsequently diagnosed. He was then lost to follow-up. His medical history was remarkable for a disease consistent with visceral leishmaniasis (kala-azar) at a younger age, but no additional data were available.

Three months prior to admission he had attended the infectious disease clinic at our institution. The CD4 cell count was 150/μl and the viral load 10^6 HIV-RNA copies/ml. HAART was initiated, including zidovudine, lamivudine and indinavir at a standard dosage.

Two weeks later the patient began complaining of a rash that had started around the mouth and rapidly spread to other parts of the face as well as the torso [Figure]. He was afebrile and the rest of the physical examination was unremarkable, without sign of lymphadenopathy or splenomegaly. A skin biopsy revealed a lymphocytic infiltrate with Leishmania amastigotes, consistent with the diagnosis of grade III post-kala-azar dermal leishmaniasis. Treatment with intravenous stibogluconate 20 mg/kg/day for 4 weeks led to a full recovery.

HIV = human immunodeficiency virus

HAART = highly active anti-retroviral therapy
Numerous papular skin lesions of post-kala-azar dermal leishmaniasis affecting the entire face with additional involvement of the torso (not shown).

Comment

Visceral leishmaniasis is endemic in African countries and is caused primarily by *Leishmania donovani*. PKDL is a frequent complication of this condition, occurring in over 50% of such cases in certain geographic areas [2], especially when visceral leishmaniasis is inadequately treated. The clinical presentation most commonly includes papular lesions starting around the mouth and spreading to other parts of the face and occasionally the torso and limbs [3]. However, maculopapular, papulonodular, and micropapular (measles-like) lesions may also be seen [3]. PKDL usually manifests months to years following visceral leishmaniasis but may rarely occur without or simultaneously with it. Diagnosis of PKDL is usually based on clinical grounds, since skin biopsy is highly specific but positive in only 20% of cases [3]. Although visceral leishmaniasis is an emerging complication of HIV infection in endemic areas, PKDL has rarely been reported in HIV-infected patients [4].

The immune response to leishmanial infection is mediated primarily via the Th1 pathway. Alteration of immunological mechanisms during HIV infection damages the protective immune response against parasites and may lead to disseminated disease [5]. In the patient described here, early immune reconstitution following the initiation of HAART probably resulted in the restoration of previously compromised Th1 immune response and the resultant PKDL. This report broadens the clinical spectrum of the immune reconstitution syndrome. Clinicians caring for HIV-infected patients should be alert to atypical illnesses that may be the result of successful anti-retroviral therapy.

References


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**Capsule**

**A milk protein lactoferrin enhances human T cell leukemia virus Type I and suppresses HIV-1 infection**

Human T cell leukemia virus type I (HTLV-I) and HIV-1, causative agents of adult T cell leukemia/lymphoma and AIDS, respectively, are transmitted vertically via breast milk. Moriuchi et al. demonstrate that lactoferrin, a milk protein that has a variety of antimicrobial and immunomodulatory activities, facilitates replication of HTLV-I in lymphocytes derived from asymptomatic HTLV-I carriers and transmission to cord blood lymphocytes in vitro. Transient expression assays revealed that lactoferrin can transactivate HTLV-I long terminal repeat promoter. In contrast, lactoferrin inhibits HIV-1 replication, at least in part, at the level of viral fusion/entry. These results suggest that lactoferrin may have different effects on vertical transmission of the two milk-borne retroviruses.

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