

Hypersensitivity Reaction Over Lesions of Post-Kala-Azar Dermal Leishmaniasis Mimicking Type 1 Reaction in Leprosy

TO THE EDITOR:

Post-kala-azar dermal leishmaniasis (PKDL) is an infrequent complication of visceral leishmaniasis (kala-azar). Usually it develops 1 to 5 years after apparent cure of visceral leishmaniasis⁽⁷⁾. The disease manifests as hypopigmented macules and papules, plaques and nodules affecting the face, limbs and trunk⁽²⁾. Clinically, the morphology and distribution of the lesions closely simulate that of leprosy, creating confusion in differentiating it from the latter. Histopathologically, the epithelioid cell granuloma may simulate tuberculoid leprosy, particularly when the parasites are scanty and cannot be identified in the infiltrate⁽⁴⁾. The occurrence of neuritis in the lesions of PKDL has made the issue even more complex^(6,8).

We have recently observed a peculiar phenomenon of hypersensitivity reaction over the lesions of PKDL in a patient following treatment with sodium stibogluconate injection which we herein describe.

A 40-year-old male presented with erythematous elevated lesions and hypopigmented patches over the face of 2 years duration. The lesions started as hypopigmented macules which gradually increased in size and number. Subsequently erythematous elevated lesions developed. There was no history of fever, constitutional symptom(s) and neuralgic pain along the limbs. None of his family members had a similar skin disease.

Examination revealed dimorphic lesions, e.g., hypopigmented macules and erythematous papules and plaques distributed over the chin, cheek and forehead. There was neither any hypesthesia over the hypopigmented macules nor any nerve thickening. A diagnosis of PKDL was made.

A slit-skin smear repeated several times from both the infiltrated papules and hypopigmented macules did not yield any acid-fast bacilli or Leishman-Donovan

(LD) bodies. A culture of the skin tissue in NNN agar media was also noncontributory.

However, a hemagglutination test (HT) and counter current immunoelectrophoresis (CIEP) tests were positive.

The patient was treated with injection of sodium stibogluconate. However, after 3 days he complained of pain and swelling over the skin lesions. Examination revealed increased erythema, edema and tenderness over the lesions. The sodium stibogluconate was stopped and the pain and swelling subsided with a nonsteroid antiinflammatory drug after 2 days. However, a similar episode occurred with the restart of the injections.

For confirmation of PKDL, the finding of parasites in the slit-skin smears, a skin biopsy or both and a positive differential agglutination test is required. However, finding LD bodies in a skin biopsy requires a thorough search over a prolonged period of time and at times may not even yield LD bodies⁽²⁻⁴⁾. In the present case also LD bodies could not be demonstrated.

In the majority of PKDL cases no hepatosplenomegaly is found⁽²⁻⁴⁾. Only very few patients present with hepatosplenomegaly and lymphadenopathy apart from the skin lesions. However, the antigen load is too scanty to be detected in these organs like skin. Very rarely, painful hepatosplenomegaly and lymphadenopathy have been found in cases of PKDL on treatment with sodium stibogluconate. This is either due to a delayed hypersensitivity reaction or due to deposition of immune complex caused by antibodies targeted against leishmania antigen^(1,5). Our present observation is quite akin to the earlier observation. The hypersensitivity reaction here manifested over the skin lesions instead of the liver, spleen or lymph nodes. This, in turn, proved the presence of leishmania antigen in the skin lesion(s), and thereby indirectly confirmed the diagnosis of PKDL. This hypersensitivity reaction was quite similar to the type 1 reaction seen in leprosy.

The rarity of this phenomenon prompted us to report this case. We like to share our experience with that of others in the field.

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A Lost Talisman: Catastrophic Decline in Yields of Leprosy Bacilli from Armadillos Used for Vaccine Production

TO THE EDITOR:

The editorial by Meyers⁽⁷⁾ in the March 1998 issue of *IJL* addresses timely and important issues on the role of the armadillo in leprosy research. He states that “it was most unfortunate that deep-seated controversy surrounded research on this animal model and, as a result, the armadillo was soon largely relegated to an industrial role—namely, the manufacture of large numbers of leprosy bacilli for *in vitro* biochemical, immunologic, chemotherapeutic, and eventually molecular biologic studies.” He also states that armadillos yield 10^{13} leprosy bacilli each. This is a misconception. Using present production methods, yield is only 10^{11} to 10^{12} *Mycobacterium leprae* per animal⁽⁵⁾. At the Gulf South Research Institute (GSRI), New Iberia, Louisiana, U.S.A., yields were as high as 10^{14} *M. leprae* per animal. This difference of 2 to 3 orders of

magnitude had a profound effect on the World Health Organization (WHO) Immunology of Leprosy (IMMLEP) program, and will handicap any future programs dependent upon armadillo-derived bacilli.

As Principal Investigator on projects that produced high-yielding armadillos at GSRI and low-yielding armadillos at the Florida Institute of Technology (FIT), Melbourne, Florida, U.S.A., I am uniquely qualified to piece together the details of what happened. In late 1973 we sent 4 g of infected tissues from GSRI to Tore Godal of WHO. He estimated they contained 10^{11} acid-fast bacilli (AFB). This prompted him to send a 2 g sub-sample to R. J. W. Rees of the National Institute for Medical Research (NIMR), London, U.K. Rees found that they contained 1.1×10^{12} AFB per g (wet weight), and that one g of bacilli (dry weight) contained 1.4×10^{14} *M. leprae*. By June of 1974, Rees had found that seven tissue