LETTERS

Post Kala Azar dermal leishmaniasis in a 15-month-old Greek boy

Dear Editor,

Post–Kala Azar dermal leishmaniasis (PKDL) is a chronic dermatosis. The geographical distribution of PKDL is restricted to certain regions such as the Indian subcontinent and East Africa, where *Leishmania donovani* is endemic, whereas this entity is almost unknown in Europe. We report the case of a patient who was successfully treated for PKDL, suggesting the emergence of such a rare complication of visceral leismaniasis (VL) in a non-endemic area.

A previously healthy 15-month-old Greek boy of Roma origin was diagnosed with VL, based on the identification of Leishmania bodies on microscopic examination of the specimen obtained by bone marrow aspiration. No travel history to any other country was reported. Treatment with Liposomal Amphotericin B was initiated for VL, but on the 7th day of treatment, the patient developed a papular and nodular rash, with few lesions in the face, trunk and extremities. He was afebrile, spleen was not palpable and no lymphadenopathy or mucous membrane involvement was observed. A second bone marrow aspiration, performed after the eruption of the rash, did not reveal any Leishmania amastigotes. Nevertheless, a biopsy from a skin nodule showed a cellular infiltration that consisted of lymphocytes, macrophages and plasma cells, while parasites were also identified. Identification of the *Leishmania* species was unfortunately not attainable. Treatment with Liposomal Amphotericin B was continued, at a total dose of 30 mg/kg. After a few days, an improvement of the rash was noted and one month later it had completely healed. The child is in good health without any evidence of VL or PKDL relapse for more than 3 years of follow-up.

PKDL is characterized by a macular, maculopapular and nodular rash, which usually develops following the treatment of VL. Diagnosis is usually made clinically, especially in endemic areas, but a definitive diagnosis should be made upon demonstration of Leishmania bodies within macrophages. Although some cases may heal spontaneously, many patients require prolonged treatment[1,2]. The skin lesions usually develop after all signs and symptoms of VL have resolved, with a usual interval of 6-12 months or even decades later (Indian type) or soon after and sometimes during treatment (African type) as in our patient[2]. Agents as pentavalent antimonials and amphotericin B are the recommended medications with satisfactory cure rates, but sometimes the relapse rate rises up to 64% requiring long-term treatment[1,3].

Consequently, certain questions arise: why do some parasites spread to skin during VL and survive there despite treatment, and, on the other hand, why PKDL is not a common manifestation in all VL cases? There is accumulating evidence, that (impaired) immune responses play a major role. The immune system seems to be more effective in killing parasites in patients who do not develop PKDL. It is unclear whether a different and more effective treatment of VL, such as Liposomal Amphotericin B, would prevent PKDL[3] – the latter had not been proven in the patient reported.

References

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Conflict of interest

None.

Doganis D[1], Mavrikou M[1], Tsolia M[2]

[1]First Department of Pediatrics
[2]Second Department of Pediatrics, School of Medicine, National and Kapodistrian University
P. and A. Kyriakou Children's Hospital, Athens, Greece

Corresponding author: Doganis Dimitrios, Thivon & Levadias Str, 11527 Athens, Greece, tel: +302107452211, Fax: +302132009104, e-mail: doganisd@gmail.com