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Cutaneous Lymphoid Hyperplasia on a Preexistent Melanocytic NevusA. Dadban^a, F. Truchetet^b^aDepartment of Dermatology, Amiens University Hospital, Amiens, and ^bDepartment of Dermatology, Metz-Thionville General Hospital, Metz, France**Key Words**

Cutaneous lymphoid hyperplasia • Cutaneous pseudolymphoma

Introduction

Cutaneous lymphoid hyperplasia (CLH) is defined by a polymorphic proliferation of B and T cells, organized in structures similar to lymphatic follicles [1]. In the published cases, there have not been any preexistent lesions. We describe the first report of CLH on a preexistent melanocytic nevus.

Case Report

An 83-year-old man had had a painful growth on his back for a month. He did not remember any trauma, insect bites or preexistent lesions at the site. His medical history was significant for type II diabetes, hypercholesterolemia, myocardial infarction and coronary bypass surgery. He had been taking gliclazide for 2 months, and sotalol, potassium, furosemide, aspirin and simvastatin for several years. Clinically, there was a 5-cm inflammatory, erythematous nodule on the back. The nodule was homogenous, and no pigmented lesion was noticed clinically. Incisional biopsy showed a normal epidermis, a dermal cell population made of small nests of pigmented cells, and a dense dermal infiltrate composed of small lymphocytes (fig. 1, 2). The pigmented cells were S-100 protein positive, and the lymphocytes were predominantly CD20-positive (B) cells. There were also a few small, CD3-positive lymphocytes at the periphery. The lymphocytic infiltrate did not dissociate nevus nests. The nevus had been completely excised with the biopsy. *Borrelia burgdorferi*-specific antibodies were negative. Nevertheless, in the hypothesis of borreliac lymphocytoma, a 15-day course of doxycycline was prescribed. The remaining nodule disappeared within 2 months.

Comments

CLH can be idiopathic or arise subsequent to a number of antigenic stimuli, of which the most frequently cited are insect bites, ectoparasitoses (as scabies), tattoos, vaccinations, foreign bodies (as piercing material) and drug ingestion, including herbal medi-

Fig. 1. Incisional biopsy of a back lesion. $\times 2.5$.

Fig. 2. Nevus nests with dense lymphocytic infiltrate. $\times 40$.

cine [2]. Depending on the geographic zone, *B. burgdorferi* infection may be a common etiology. Although a borreliac etiology (as may be suggested by the response to antibiotics) or an idiopathic CLH with a purely coincidental adjacent nevus could not be eliminated, the presence of a melanocytic nevus in the biopsy mate-

rial, and the complete regression of CLH after the excision of the nevus suggest a cause-and-effect relationship. We postulate that nevus cells had become antigenic, in a similar way as in Sutton's phenomenon [3] or in melanoma-associated vitiligo [4], albeit with a different histological reaction in CLH. In both phenomena, a preexistent melanocytic lesion becomes antigenic with no apparent reason. The role of antimelanocyte T cells [5] could be suspected. Finally, the drug gliclazide as well as its family, started a month before the appearance of the lesion, are not known to induce CLH-like lesions. The lesion subsided while gliclazide was continued. In the event of relapse, this drug's etiological role should be considered.

Conclusion

We believe our report to be the first describing coexistent nevus and CLH. This rare event, as well as being troublesome from the histological viewpoint, could represent a peculiar form of immunological reaction to melanocytes, hitherto unknown.

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Dr. Ali Dadban
Service de Dermatologie, Centre Hospitalier Universitaire d'Amiens
Avenue René-Laënnec
FR-80054 Amiens Cedex 1 (France)
Tel. +33 3 22 45 58 41, Fax +33 3 22 45 57 94
E-Mail alidadban@gmail.com