Large Asymmetric Pigmented Nodule in a 27-Year-Old Female

Nódulo pigmentado asimétrico de gran tamaño en una mujer de 27 años

To the Editor:

A 27-year-old female presented with an asymptomatic nodule on the left thigh. She reported a smaller pigmented lesion since birth, with significant growth in the last 5 years. The patient had no personal or family history of skin cancer. She was otherwise healthy and had no systemic symptoms. She was doing oral contraception and no other medications.

Physical examination revealed a smooth nodular dome-shaped lesion measuring 1 cm × 2 cm, with central hyperpigmented and peripheral erythematous areas, located on the medium third of the anterior left thigh (Fig. 1). There was no regional lymphadenopathy.

Dermoscopically there was a central dark hyperpigmented area surrounded by a rim of thin regular pigment network (Fig. 2). In the central area we observed a predominant globular pattern, an eccentric thickened pigment network and a blue-whitish-veil.

Excision was performed. Histological examination revealed a compound (predominantly intradermal) melanocytic nevus with congenital-like growth pattern and a dermatofibroma, with no signs of malignancy (Fig. 3). Nevus cells involved deep dermal appendages and neurovascular structures and they became progressively smaller in deeper portions of the lesion suggesting maturation and benignity. Intermingled with melanocytes, there was a proliferation of spindle fibro-histiocytic cells characteristic of dermatofibroma.

Collision tumors result from the combination of two or more benign and/or malignant neoplasms in a single lesion.[1] As it is unknown whether the association is a random event or whether there is, in fact, a pathogenic relationship in the development of two distinctive neoplasms together, some authors propose the designation of compound tumor instead of collision tumor because the last one suggests the association is incidental.[2]

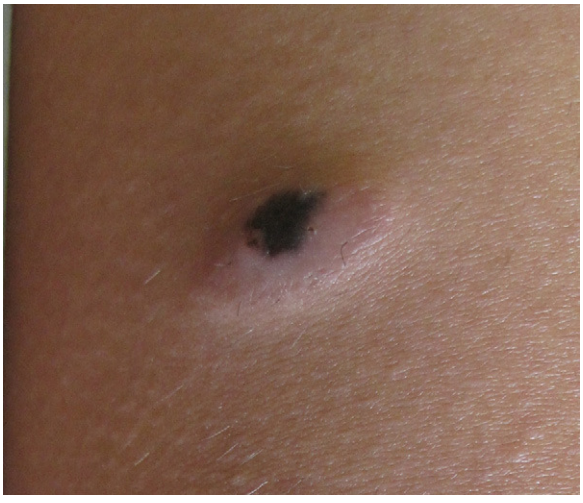

Figure 1 Asymmetric pigmented nodule (1 cm × 2 cm) on the left anterior thigh.

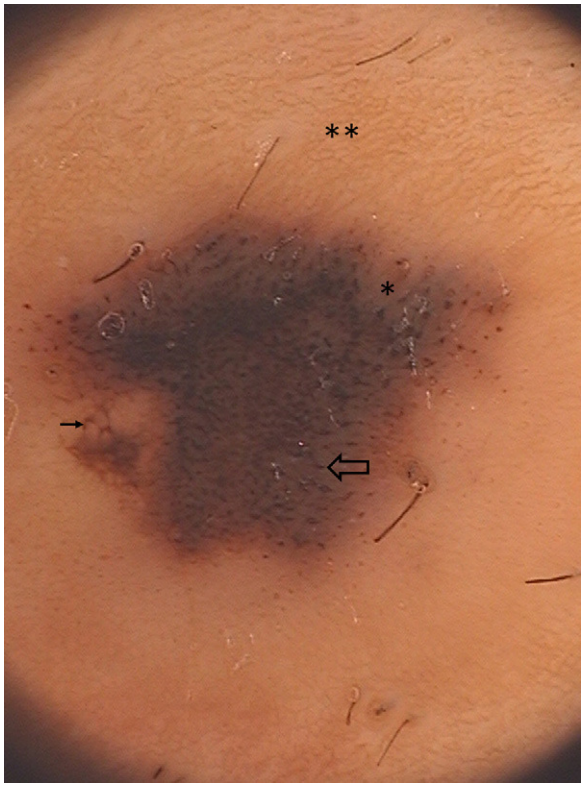

Figure 2 Dermoscopic picture (20×): central dark hyperpigmented area with a predominant globular pattern (thick arrow), an eccentric thickened pigment network (thin arrow) and a blue-whitish-veil (*), surrounded by a rim of thin regular pigment network (**).

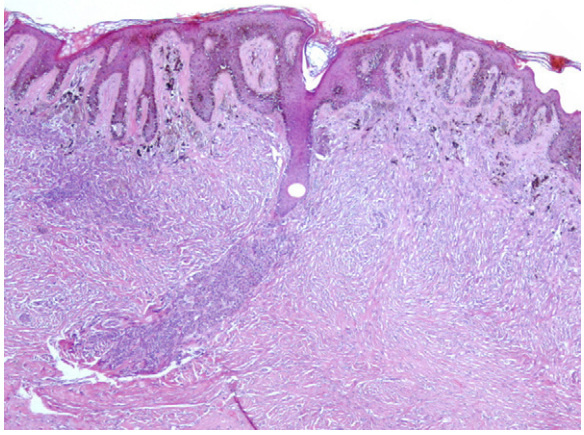

Figure 3 Histological picture (H&E, 4×): collision tumor of congenital nevus and dermatofibroma.

Collision tumors are not rare. Boyd and Rapini performed a retrospective evaluation of approximately 40,000 cutaneous biopsies, yielding 69 collision tumors.[3] Cascajo and colleagues found 54 collision tumors of malignant cutaneous neoplasms associated with seborrheic keratoses in 85,000 biopsies.[2]

The most common combinations found by Boyd and Rapini included basal cell carcinoma and nevus (14), basal cell carcinoma and seborrheic keratosis (8), nevus and seborrheic keratosis (14), actinic keratosis and nevus (7), and basal cell carcinoma and neurofibroma (4).[3] Another commonly described association is that of melanocytic nevus with desmoplastic trichoepithelioma.[1] Our patient had a compound tumor of congenital nevus and dermatofibroma. As far as we know, this is the first reported case of such a combination.

Furthermore this tumor clinically and dermoscopically mimicked malignant melanoma. Although in some cases dermoscopy can help with collision tumor diagnosis,[4] this was not the case. In fact, according to Giorgi and colleagues cutaneous collision tumors are extremely difficult to diagnose preoperatively, even with the help of dermoscopy, in particular when one of the lesions is melanocytic.[5] The presence of a pigment network, the pathognomonic sign of the melanocytic lesion, allows dermatologists to use dermoscopic algorithms, which in this case ended up in the wrong preoperative diagnosis of a malignant lesion.

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F. Osório[a,*], A. Margarida Barros[a], O. Ferreira[a,b], F. Azevedo[a]

[a] *Department of Dermatology and Venereology, Centro Hospitalar de São João EPE, Porto, Portugal*
[b] *Faculty of Medicine of University of Porto, Portugal*

[*] Corresponding author.
E-mail address: filipaosorio@gmail.com (F. Osório).

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Hipertricosis generalizada adquirida por diazóxido

Acquired Generalized Hypertrichosis Due to Diazoxide

Sr. Director:

La hipertricosis (HTC) se define como el crecimiento excesivo del pelo en cualquier parte del cuerpo[1]. Se diferencia del hirsutismo en las características y distribución androgénica de los folículos pilosos. Clínicamente la HTC se clasifica en función de su extensión y de su etiología. Así, encontramos formas localizadas o generalizadas que pueden ser congénitas o adquiridas. La HTC generalizada adquirida puede presentarse como una manifestación idiopática, iatrogénica o secundaria a procesos sistémicos[2] como el hipotiroidismo, las porfirias, la enfermedad celíaca, la dermatomiositis, la infección por el VIH o tumores. La aparición de lanugo de forma generalizada en el adulto puede ser un marcador de una neoplasia interna o presagiar el desarrollo de la misma; sin embargo, no se ha descrito ningún caso en edad pediátrica[3].

Presentamos el caso de un varón de 6 años derivado a nuestra consulta por la aparición de pelo terminal en la cara, el tronco y las extremidades (fig. 1) de inicio en el primer año de vida y evolución lentamente progresiva. Entre sus antecedentes personales presentaba hipoglucemia hiperinsulínica de la infancia desde los 5 meses de edad, en tratamiento con diazóxido a la dosis de 20 mg/kg/día con un adecuado control. No refería antecedentes familiares de interés. El paciente no presentaba retraso del crecimiento, afectación neurológica ni otras enfermedades de base o tratamientos habituales. En la exploración se observaba una hipertricosis generalizada, más acentuada en la frente, el bigote y las cejas, de inicio de varios meses tras la introducción del diazóxido a dosis plenas (fig. 2). No mostraba predilección por zonas fotoexpuestas. No se apreciaban otras lesiones cutáneas, mucosas, ni alteraciones dentarias. Se realizó un hemograma y una bioquímica básica con hormonas tiroideas, LDH, anticuerpos antigliadina, antiendomisio y antitranglutaminasa, encontrándose todos

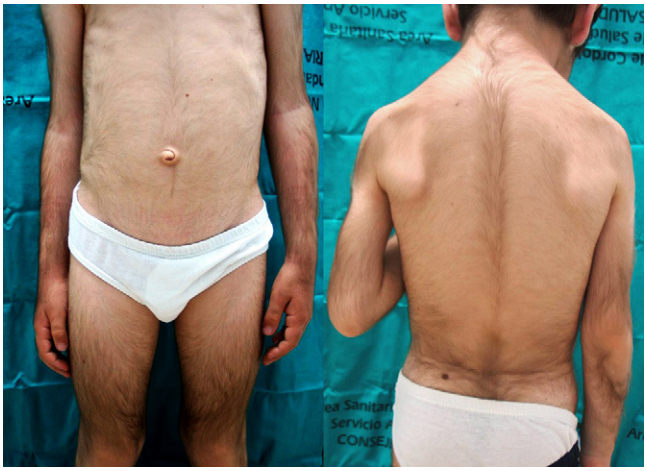

Figura 1 Aumento difuso de pelo terminal en el tronco y los miembros superiores.