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CASE AND RESEARCH LETTERS

Dermoscopy of Erythromelanosus Follicularis Faciei et Colli



Dermatoscopia de erythromelanosus follicular faciei et colli

Dear Editor:

Erythromelanosus follicularis faciei et colli (EFFC) is a rare pigmentary disease of unknown etiopathogenesis typically affecting the face/neck of children or young adults, which is clinically characterized by the combination of bilateral/symmetrical brownish pigmentation and erythema, associated with more or less evident follicular plugging.¹ Of note, such a condition is often associated with keratosis pilaris on the arms and shoulders, thereby letting some authors speculate that EFFC could be a variant of this latter dermatosis.¹ Not uncommonly, EFFC is mistaken for other similar pigmentary/erythematous dermatoses involving the aforementioned districts, with consequent diagnostic errors/delays and prescription of inappropriate therapies.¹ Over the last few years, several studies have shown that dermoscopic examination may be useful to assist the diagnosis of general skin diseases.^{2–5} We here describe for the first time the use of dermoscopy as a noninvasive diagnostic aid in a case of EFFC, comparing its dermoscopic findings with those detectable in other conditions which classically enter into the differential diagnosis.

A 33-year-old man presented with a 6-year history of progressively worsening, asymptomatic, reddish-brown pigmentation associated with slight roughness on the cheeks, temples, lateral aspects of the nose, and frontal area (Fig. 1). Polarized light dermoscopic examination (carried out with DermLite DL3×10; 3Gen, San Juan Capistrano, CA, USA) revealed whitish scales and numerous whitish follicular keratotic plugs over a reddish-brown background; moreover, several gray-blue granules (peppering) were also evident in the perifollicular and interfollicular areas (Fig. 2). Histological examination showed slight orthokeratosis, follicular hyperkeratosis, increased basal layer pigmentation, perivascular and periadnexal lymphocytic infiltrate, and pigmentary incontinence with dermal melanophages (Fig. 2b), thus leading to the diagnosis of EFFC. Topical tacalcitol was prescribed and the use of sunscreen was recommended, with significant improvement of the clinical picture after eight weeks.

Dermoscopic findings seen in our instance of EFFC are related to the peculiar histological features which characterize this condition,^{1,6} with follicular plugging, scaling, peppering, and reddish-brown background respectively corresponding to hyperkeratotic hair follicles, orthokeratosis, pigmentary incontinence/dermal melanophages, and dermal vasodilation/hyperpigmentation of the basal layer.^{1,6} Such a dermoscopic picture is similar to that reported in a recent case of erythrosis pigmentosa peribuccalis, a pigmentary dermatosis presenting as brownish-red pigmentation and small papules around the mouth and nose, which displayed erythema, scaling, yellowish follicular keratotic plugs, and perifollicular grayish globules/dots.⁷ These similarities are easily explained by the fact that both such disorders may share several histological features, so much so that they are considered to be part of the same condition spectrum by some authors.⁷ Interestingly, the detection of the above-mentioned dermoscopic features might come in handy in the noninvasive distinction of EFFC from its main differential diagnoses as the latter typically show different features. In particular: lichen planus pigmentosus usually displays diffuse, structureless, brownish pigmentation and/or fine/coarse, gray-blue/brown dots/globules⁸; Riehl's melanosis constantly features brownish pseudonetwork, gray dots/granules and telangiectatic vessels⁹; poikiloderma of Civatte commonly shows structureless brownish pigmentation and telangiectatic vessels, with or without whitish areas (personal observations); melasma typically presents light yellow-brown uniform patches, with or without dark brown patches and capillary network¹⁰; keratosis pilaris rubra atrophicans faciei frequently displays whitish follicular plugs over a reddish background with or without telangiectatic vessels (personal observations); demodicidosis mainly shows the so-called "Demodex tails" (creamy/whitish gelatinous threads representing the presence of the mite itself under magnification) protruding out of follicular openings, "Demodex follicular openings" (round and coarse follicular openings containing light brown/grayish plugs surrounded by an erythematous halo), erythema and whitish scaling³; keratosis pilaris often features coiled/twisted hair embedded in the horny layer, sometimes associated with perifollicular erythema and vascular ectasia (keratosis pilaris rubra)⁴; and follicular lichen planus reported to show follicular keratotic plugs without broken or twisted hairs.⁴

In conclusion, this paper emphasizes that dermoscopy might be used as an auxiliary tool in the noninvasive differential diagnosis of EFFC. Further studies are obviously needed to confirm our preliminary observations.

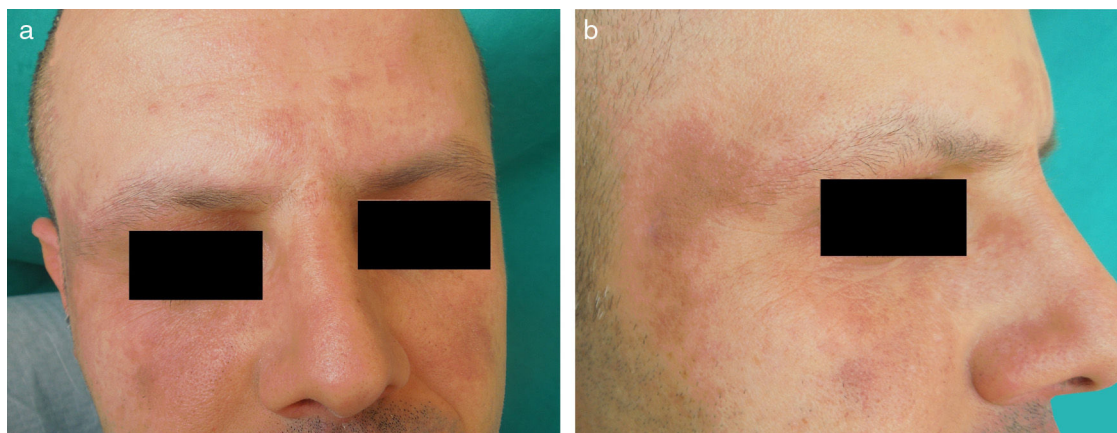


Figure 1 Clinical examination shows irregular areas of reddish-brown pigmentation of the cheeks, temples, lateral aspects of the nose, and frontal area (a and b).

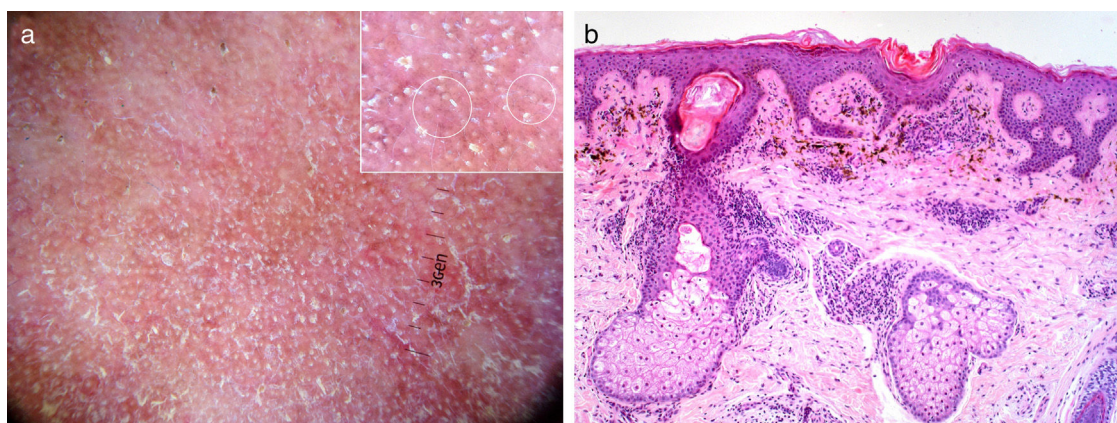


Figure 2 Polarized light dermoscopic examination displays whitish scales and numerous follicular keratotic plugs over a reddish-brown background; moreover, several perifollicular and interfollicular gray-blue granules (peppering) are also evident in the box (a). Histology reveals findings consistent with a diagnosis of erythromelanosis follicularis faciei et colli, i.e. slight orthokeratosis, follicular hyperkeratosis, increased basal layer pigmentation, perivascular and periadnexal lymphocytic infiltrate, and pigmentary incontinence with dermal melanophages (hematoxylin and eosin stain 200 \times) (b).

Conflicts of interest

The authors declare that they have no conflicts of interest.

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<http://dx.doi.org/10.1016/j.ad.2017.04.016>

0001-7310/

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Hyperkeratotic lesions on legs



Lesiones hiperqueratósicas en piernas

Dear Editor,

Skin hyperkeratosis is a common pathology in dermatological consultation. From localized pathology, such as corns or common warts, to diseases with a more diffuse effect such as psoriasis and ichthyosis, diagnosis is usually easy, in response to the history and location of lesions. But sometimes histological or analytical study is essential.

We report a 71-year-old male with multimorbidity (hypertension, diabetes mellitus, ischemic heart disease, mitral regurgitation, left bundle branch block, hepatic steatosis, fibrosing alveolitis, COPD, hypercholesterolemia, hyperuricemia and glaucoma) who presented hyperkeratotic lesions on the legs (Fig. 1). The patient had worked ten years



Figure 1 Hyperkeratotic lesion on the legs.

in a photographic laboratory and twenty as carpenter. The lesions were asymptomatic and he had them for more than twenty years. They were located only on the front side of the legs and other skin was respected. Lesions were 2–6 mm in diameter and were very adherent. Some had peripheral reinforcement. The patient showed no other symptoms and denied use of cosmetics or chemicals in the area. He had not suffered weight loss or anorexia. He had no history of contact dermatitis, burns or trauma. There was low actinic exposure in the area. No family member had similar injuries.

The patient had a history of working in a carpentry workshop for more than twenty years.

With suspicion of perforating skin disease, actinic keratosis, or porokeratosis, we carried out a 4 mm punch of one of the lesions. Histology showed orthokeratotic papillomatosis with focal lymphoid infiltrate in papillary dermis. The sample showed no atypia. The pathology guided us to keratotic lesions with a 'church spire' pattern (Fig. 2). This

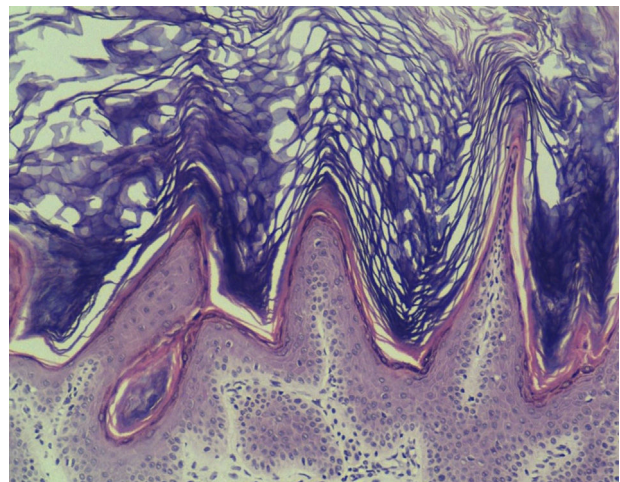


Figure 2 Hematoxylin-eosin (×100). Orthokeratotic papillomatosis with focal lymphoid infiltrate in papillary dermis and church spire pattern.

pattern suggests multiple diagnostic possibilities: arsenic poisoning or tars, nutritional deficits (phrynoderma), digitata keratosis, Hopf verruciformis acrokeratosis, or stucco keratosis. Chest radiography was normal but analytical research showed urinary arsenic levels of 317 µg/g creatinine (normal levels in occupationally exposed people <100 µg/g creatinine). The patient was diagnosed with chronic arsenic poisoning.

Arsenic poisoning has, as its main cause, ingestion of contaminated water.¹ This is a significant public health problem in areas of South and Southeast Asia (India, China, Taiwan, Philippines, Thailand, Bangladesh) and the Americas (Argentina, Chile, Mexico, and the USA) where arsenical products are detected either naturally or occasionally by industrial waste. As a second etiologic factor there is workplace exposure. Health damage results from inorganic arsenic (the toxic form) in pesticides, herbicides, mining and galvanized microchips. Occasionally wood preservatives contain arsenic derivatives.² This could explain our case. Our