

Case Report

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Hyperpigmentation-Ashy dermatosis

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Abstract

Ashy dermatosis-like hyperpigmentation Erythema dyschromicum perstans (EDP), also known as ashy dermatosis, is a hypermelanotic condition characterised by the development of a slate-grey macula in otherwise healthy individuals. Here, we describe sudden-onset EDP in a 19-year-old male patient. His lesions had started 3 months ago and increased in the last 2 weeks. Dermatological examination revealed pigmented macules and patches of different sizes, which tended to coalesce, on the back and anterior aspect of the trunk. The lesions did not regress after local treatment with a prescribed corticosteroid cream of moderate potency. The patient was free from systemic disease, was not taking any drugs and had no relevant family history. Histopathological examination of lesional biopsy tissue from the back and anterior of the trunk revealed EDP, characterised by vacuolar degeneration of the basal layer, mild lymphohistiocytic inflammatory cell infiltration around the vessels in the upper dermis, melanophages, pigment incontinence, and low-level lymphocyte exocytosis in the epithelium. Dapsone (100 mg/day) controlled the lesions. EDP is a rare dermatosis, for which differential diagnosis and treatment may thus sometimes be inadequate.

Keywords: ashy dermatosis, hyperpigmentation, pigment incontinence, dapsone

1. Introduction

Ashy dermatosis-like hyperpigmentation Erythema dyschromicum perstans (EDP), also known as ashy dermatosis, is a hypermelanotic condition characterised by the development of a slate-grey macula in otherwise healthy individuals (1). The disease is not characterised by systemic or internal organ involvement; instead, it affects the skin only. Patients often request treatment due to cosmetic concerns (2). The EDP rate is the same in men and women (1, 3). Here, we describe sudden-onset EDP in a 19-year-old male patient.

2. Case Report

A 19-year-old male patient applied to the outpatient clinic with discoloration on the trunk and back. His lesions had started 3 months ago and increased in the last 2 weeks. Dermatological examination revealed pigmented macules and patches of different sizes, which tended to coalesce, on the back and anterior aspect of the trunk (Fig. 1 and 2).

The patient reported that the lesions had previously been more erythematous and purplish. The lesions did not regress after local treatment with a prescribed corticosteroid cream of moderate potency. The patient was free from systemic disease, was not taking any drugs and had no relevant family history. The complete blood count, liver and kidney function, erythrocyte sedimentation rate, and thyroid function were normal, as were all urine analyses. The anti-HBs, anti-HCV, anti-HIV, VDRL, and ANA tests were all negative.



Fig. 1. Hyperpigmented macular eruption on the anterior aspect of the trunk



Fig. 2. Diffuse pigmented macules and patches on the back

Histopathological examination of lesional biopsy tissue from the back and anterior of the trunk revealed EDP, characterised by vacuolar degeneration of the basal layer, mild lymphohistiocytic inflammatory cell infiltration around the vessels in the upper dermis, melanophages, pigment incontinence, and low-level lymphocyte exocytosis in the epithelium (Fig. 3). Dapsone (100 mg/day) controlled the lesions.

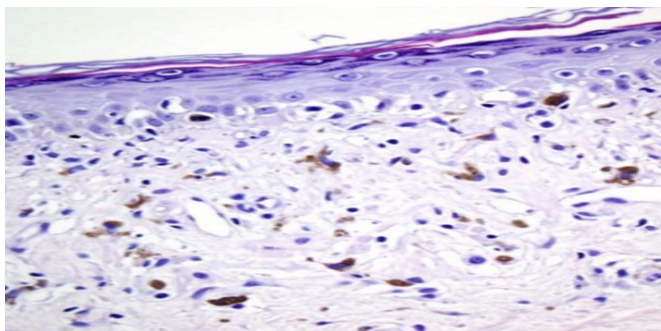


Fig. 3. Mild interphase dermatitis and pigment incontinence with perivascular lymphocytic infiltrate in the dermis (H&E 40X)

3. Discussion

Ashy dermatosis (EDP) is a rare dermal melanosis first described by Ramirez in 1957, and represents an example of acquired pigmentation (4, 5). Although the cause of EDP is unknown, Tlougan et al. (6) suggested that triggers might include infections (parasites and HIV), drugs (penicillin, ethambutol, and benzodiazepines), radiocontrast agents, ammonium nitrate, cobalt and fungicides. It is possible that thyroid dysfunction and endocrinopathies (such as diabetes mellitus, atopy and dyslipidaemia) may also cause the disease. Immunopathological examinations of active lesions suggested that immune-mediated mechanisms might play roles in the pathogenesis of EDP (5, 6). Hyperpigmentation refers to pigment deposition in the epidermis and/or dermis. As well as EDP and postinflammatory hyperpigmentation (PIH), primary cutaneous amyloidosis (PCA), neuralgia paresthetica (NP) and

certain drug reactions may trigger hyperpigmentation (7). Few studies on EDP treatment have appeared. Silverberg et al. (8) found that sun screen, chemical peeling agents, antibiotics, and topical and systemic steroids were ineffective. Odom et al. (9) described partial responses to griseofulvin, clofazimine, and dapsone. EDP is a rare dermatosis, for which differential diagnosis and treatment may thus sometimes be inadequate. More clinical studies are needed.

Conflict of interest

None to declare.

Acknowledgments

None to declare.

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