Dermatology Practical & Conceptual

Dermoscopic and Reflectance Confocal Microscopy of Galli-Galli disease

Qing Wang[1], Lin Feng[1], Maolin Yao[2], Yin Yu[1]

1 Department of Dermatology, Chongqing Hospital of Traditional Chinese Medicine, Chongqing, China
2 College of Traditional Chinese Medicine, Chongqing Medical University, Chongqing, China

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Corresponding Author: Yin Yu, Department of Dermatology, Chongqing Hospital of Traditional Chinese Medicine. No. 40, daomenkou, Yuzhong District, Chongqing 400011, China. ORCID ID: 0000-0003-4620-5055. Email: 1654637656@qq.com

Introduction

Galli-Galli disease (GGD) is a rare autosomal pigmentation disorder that commonly appears as pink-to-brown pruritic macules and papules as well as reticulated hyperpigmentation, commonly affecting the neck, trunk, and flexor. Herein, we report a case of GGD with dermoscopic and reflectance confocal microscopy (RCM) features.

Case Presentation

A 46-year-old female presented with widespread pruritic erythematous papules and macules over the axillae, neck, and trunk that had been present for nine years. Her son developed similar lesions. Physical examination revealed multiple reticulated erythematous papules and macules over the neck, trunk, and axillae (Figure 1A). No systemic abnormality was found, and complete blood count was normal. Dermoscopy showed scattered tan pigmentation, light white spots, pseudo-grid-like structures, and linear and reticular telangiectasia (Figure 1B). RCM revealed downward proliferation of the epidermis with an unclear border between the lesion and the adjacent normal skin, local damage of the spinous spongy structures, and unevenly distributed bright dendritic cells in the basal layer. An infiltration of unequal high-refractive melanophagocytes and medium-refraction inflammatory cells in the superficial dermis could also be seen (Figure 2A). Biopsy of the lesion showed hyperkeratosis with porokeratosis, epidermal acanthosis, suprabasal acantholysis, and infiltration of lymphocytes in the superficial dermal (Figure 2B). Whole exome sequencing revealed a genetic variation c.10C>T (p.Gln4Ter) in the keratin 5 gene. A diagnosis of GGD was made, and a combination of systemic acitretin (20mg/d), topical mometasone furoate, and ultraviolet B(UVB) phototherapy was supplied. Lesions improved with residual pigmentation after six months of treatment.

Conclusions

GGD is an extremely rare autosomal pigmentation disorder resulting from mutations in the POFUT1, POGLUT1, and KRT5 genes [1]. It is considered an acantholytic variant of

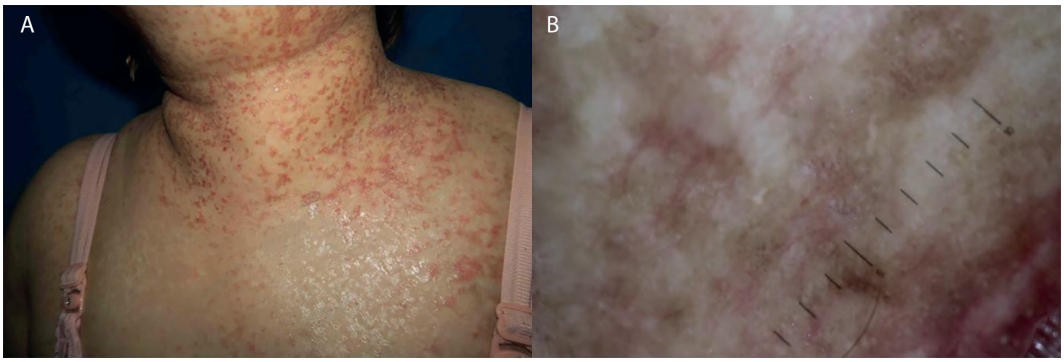

Figure 1. A) Clinical presentation: Multiple reticulated erythematous papules and macules over neck, trunk, and axillae. B) Dermoscopy: Scattered tan pigmentation and light white spots, pseudo-grid - like structures. Linear and reticular telangiectasia are easily seen.

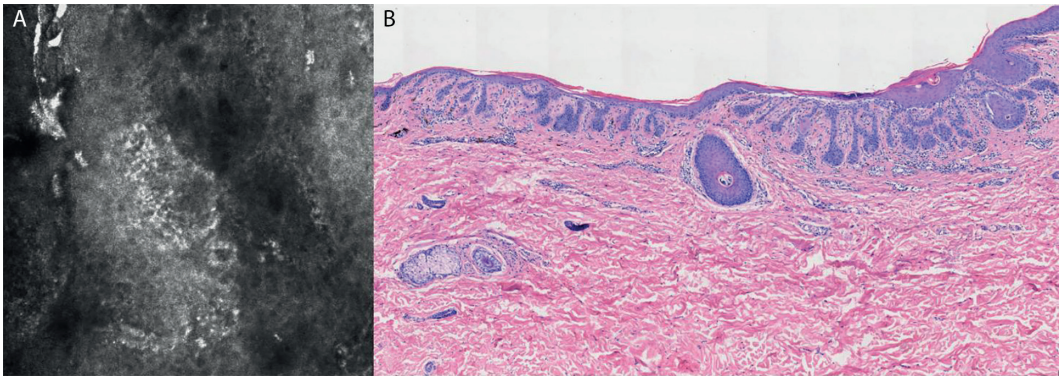

Figure 2. A) Reflectance confocal microscopy: Downward proliferation of the epidermis with an unclear border, and an infiltration of melanophagocytes with unequal high refraction and medium refraction inflammatory cells in the superficial dermis. B) Biopsy of the lesion shows seborrheic keratosis-like hyperkeratosis with porokeratosis, epidermal acanthosis, suprabasal acantholysis, and infiltration of lymphocytes in the superficial dermis (40x).

Dowling-Degos disease (DDD). Histologically, GGD is characterized by elongated, downward filiform proliferation of epithelial strands with acantholysis and basal hyperpigmentation. There have been few reports about dermoscopy and RCM of GGD. We found that dermoscopy showed tan pigmentation spots, pseudo-grid-like structures, and linear and reticular telangiectasia, corresponding to abnormal pigment changes and collagen fibers. RCM showed local damage to the spinous spongy structures, which may correspond to acantholysis. Additionally, unevenly distributed bright dendritic cells in the basal layer may indicate interface failure and infiltrated inflammatory cells.

The main differential diagnosis is DDD, which commonly appears as reticular or dot-like brown macules that particularly affect the skin folds like neck and flexures. Pathologically, DDD is almost similar to GGD except that there is no acantholysis [2]. The treatment of GGD is difficult. Published therapies, including topical and systemic corticosteroids and retinoids, UVB phototherapy, and laser, were with incomplete or temporary effects in most cases [3]. A case of GGD successfully treated with alitretinoin was recently reported, which might reveal a new treatment [4]. The patient in our report was treated with systemic acitretin, topical mometasone furoate, and UVB phototherapy for six months, and responded well. Still, a long follow-up is needed.

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