Case Report

# Reticulate acropigmentation of Dohi: A rare case

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## ABSTRACT

Reticulate acropigmentation of Dohi (RAD) is a rare genodermatosis characterized by reticulated hyperpigmented and hypopigmented macules, primarily involving the acral areas. Although typically inherited as an autosomal dominant trait, autosomal recessive inheritance has also been reported. We present a rare case of a 36-year-old female with widespread hyperpigmented patches across her body, with disease onset at 16 years of age. Histopathological examination of a skin biopsy revealed basal melanosis, increased melanocytes throughout the epidermis, and dermal melanophages, supporting the diagnosis of RAD. Dermoscopic findings correlate well with histopathological changes. No effective treatment exists, with cosmetic camouflage being the most practical approach. This case is reported due to the rare, sporadic occurrence of RAD without a family history and extensive body involvement.

**Key words:** Adenosine deaminase actin on RNA 1, Basal melanosis, Double-stranded RNA-specific adenosine deaminase gene, Hyperpigmented macules, Hypopigmented macules, Reticulate acropigmentation of Dohi

Reticulate acropigmentation of Dohi (RAD) is a rare genodermatosis. It is inherited as an autosomal dominant trait, although an autosomal recessive trait has also been reported [1]. To determine the gene responsible for this disease, a genome-wide search was performed in three families with dyschromatosis symmetrica hereditaria (DSH), and the DSH locus was mapped to chromosome 1q21.3. The mutations involved in causing DSH have been identified in the gene that encodes double-stranded RNA-specific adenosine deaminase (DSRAD) as the disease gene [1,2]. DSH was first described by Toyama in 1929 and is characterized by symmetrical distribution of hyperpigmented and hypopigmented macules on the extremities, over the hand and feet [3].

We present the case of a 36-year-old female who presented with chief complaints of hyperpigmented patches all over the body. The rationale for this case report is that patients with rare genodermatosis like this can present with hyperpigmented skin lesions at various sites, and topical steroids do not work on these patients. It needs to be biopsied for diagnosis, with subsequent treatment specified by dermatologists.

## CASE REPORT

A 30-6-year-old married female presented with a history of hyperpigmented patches all over the body for the past 20 years. She came for her dermatological examination recently because of worrisome cosmetic issues. There were multiple, well-defined hyperpigmented macules and patches in a reticulate pattern with atrophic changes in some places. The patches were present on the bilateral upper limbs, neck, abdomen, axilla, buttocks, and bilateral lower limbs (Fig. 1). No lesions on the face were observed. No mucosal lesions were observed. The patient first noticed the hyperpigmentation on her hand when she was 16 years old. She did not turn up, thinking that it would fade away with time. The local practitioner gave her local steroid ointments without a skin biopsy. The patient had no history of photosensitivity and photophobia. The lesions were itchy, but later they were asymptomatic.

The patient's intelligence was normal, and her systemic examination revealed no abnormality. The past medical history and family history of the patient were insignificant. On gross examination, we received a skin-covered tissue bit total measuring 0.4 × 0.3 × 0.2 cm.

For microscopic examination, a biopsy was taken from the hyperpigmented area, and the section shows mild hyperkeratosis, mild epidermal thinning with an abundance of melanocytes in the basal epidermis. There was a presence of increased melanocytes in other epidermal layers, which taper toward the surface. Perivascular and peri-adnexal mononuclear infiltrate were noted. In dermis, dermal melanophages were noted (Fig. 2).

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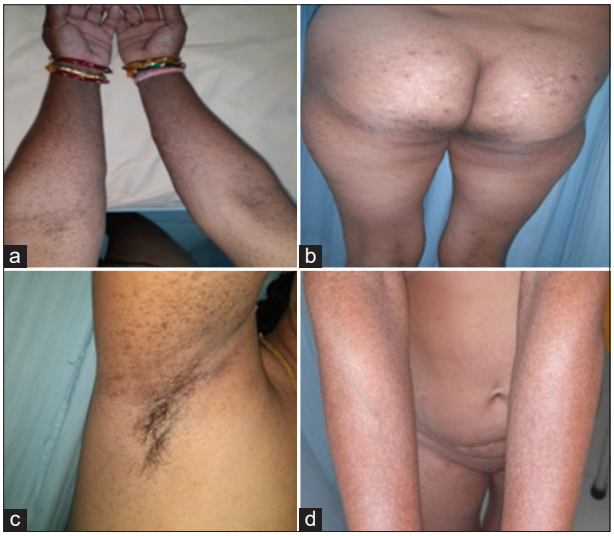


**Figure 1: Hyperpigmented macules on the (a) forearm, (b) buttocks, (c) axilla, and on (d) dorsum of hands**

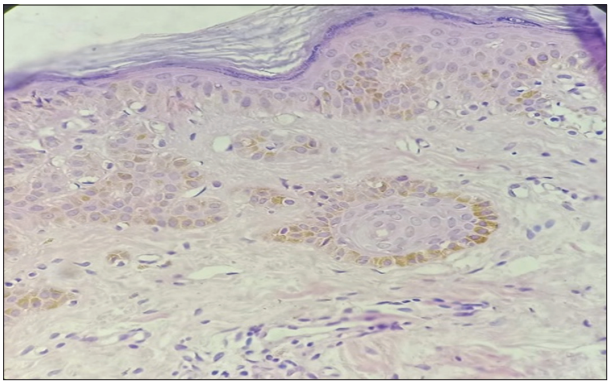

**Figure 2: Biopsy from the hyperpigmented area shows an abundance of melanocytes in the basal layer and the presence of melanocytes in other epidermal layers (Hematoxylin and Eosin, ×400)**

## DISCUSSION

RAD is a rare dyschromatosis characterized by mottled pigmentation developing on acral areas [4]. The exact pathogenesis is not yet understood, though a resemblance to reptilian skin, and an evolutionary process mediated by embryonic neural reflexes expressed only in genetically predisposed individuals has been suggested [5].

Toyama in 1929 was the first one to describe DSH, which is characterized by symmetrical distribution of hyperpigmented and hypopigmented macules on the extremities, over the hand and feet [3]. A genetic mutation has been identified on chromosome 1q11-1q21 as responsible for the production and distribution of melanin. Miyamura *et al*. (2003) were the first to identify the autosomal dominant form of DSH due to a mutation in the DSRAD or adenosine deaminase actin on *RNA 1* gene [3].

The onset is normally reported to occur in the first decade, but occasionally onset can occur at a later stage [5], as seen in our case.

RAD is characterized by the presence of hyperpigmented and hypopigmented macules, symmetrical, irregular in size and shape, forming a reticulate pattern over the dorsa of hands and feet and occasionally on the arms and legs [4]. Our case also showed the involvement of the dorsa of hands and forearms. There are isolated reports of association with neurofibromatosis type 1, thalassemia, polydactyly, and torsion dystonia [6]. The involvement of palms and soles is unusual [5], and our case also did not show the involvement of palms and soles.

Biopsy is diagnostic on clinical correlation and shows basal melanosis in the hyperpigmented lesion, as in our case [6]. Dermoscopy is a new investigative tool that gives specific characteristic changes [2]. Dermoscopy of the pigmented areas shows reticulated hyperpigmented spots, monotonous pigmented spots, reticulated hypopigmented spots, or monotonous hypopigmented spots [7].

Dermoscopically, reticulate pigment network in "honeycomb pattern" against pinkish to brownish background corresponds with peri-vascular infiltrate and pigmentary incontinence in dermis with presence of rete ridges. Focal hyperpigmentation and hypopigmentation correspond to increased and normal melanin in the basal layer and were constructed of connected and unconnected pigmented spots, respectively [7], while monotonous pigment may reflect the hyperpigmentation of basal keratinocytes without formation of rete ridges [7]. Dermoscopy was not done at our setup due to the lack of infrastructure.

In our case, the patient was given topical 20% azelaic acid followed by sun-screen lotion with advice of monthly follow-up. Our patient responded to this "consistent" topical treatment after 6 months of follow-up with the waning of her skin lesions. Topical retinoids and hydroquinone as adjuvant therapy can be tried in such cases, which were not given in our case. Fractional $CO_2$ laser and miniature punch grafting combined with a 308 nm excimer laser light have been tried [8-10]. Split-skin grafting can benefit the patients, but is normally not carried out in case where medications do not work. Camouflage is the most reasonable approach if acceptable. The clinic-pathological correlation helps to clinch the diagnosis.

Our RAD case had well-defined hyperpigmented macules and patches in a reticulate pattern on the bilateral upper limbs, neck, abdomen, axilla, buttocks, and bilateral lower limbs. However, the differential diagnoses for RAD are described as below [11-15]: (a) Dowling–Degos disease (DDD): Reticulate hyperpigmentation mainly in flexural areas (e.g., axillae, groin); comedone-like lesions, pitted perioral scars. RAD is acral and may have hypopigmented macules. (b) Galli-Galli disease: It is a variant of DDD with suprabasal acantholysis seen on histopathology. RAD shows basal layer pigmentation changes without acantholysis. (c) DSH: Autosomal dominant condition that presents in early childhood with mixed hyper- and hypopigmented macules, especially on dorsal hands/feet and face. RAD is similar, but with the lack of facial involvement/hands/feet. Our case was

a 36-year-old female without skin lesions on the face, feet, or hands. Further genetic testing was not done in our case due to cost restraints. (d) Dyskeratosis congenita (DC) has a triad of reticulate pigmentation (often on neck and chest), nail dystrophy, and oral leukoplakia. DC has systemic findings (e.g., bone marrow failure), which are absent in RAD. (e) Xeroderma pigmentosum photosensitivity, freckling, and skin cancer susceptibility; DNA repair defect. RAD lacks photosensitivity and malignancy risk. (f) Naegeli–Franceschetti–Jadassohn syndrome: It is reticulate pigmentation that fades in adulthood, dental anomalies, hypohidrosis, and absence of dermatoglyphics. RAD is persistent without systemic findings and does not fade away without ideal treatment. (g) Amyloidosis cutis dyschromica: Reticulate pigmentation with amyloid deposits in the skin on histology. RAD lacks amorphous amyloid on biopsy.

## CONCLUSION

This case is reported due to its rarity and lack of familial involvement. The presence of melanocytes in most epidermal layers reaching the surface, with abundant basal melanocytes in hyperpigmented macules, confirms the diagnosis as RAD. A strong clinicopathological correlation is needed between dermatopathologists and dermatologists for its diagnosis.

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