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Case Report

Dermatology Section

Hereditary Diffuse Transgradient Palmoplantar Keratoderma in a 57-year-old Male: A Case Report

SWATHI GANESH SHENOY[1], K SURESH KUMAR[2], TS RAJASHEKAR[3], YERRAGONDA SUSMITHA[4]

ABSTRACT

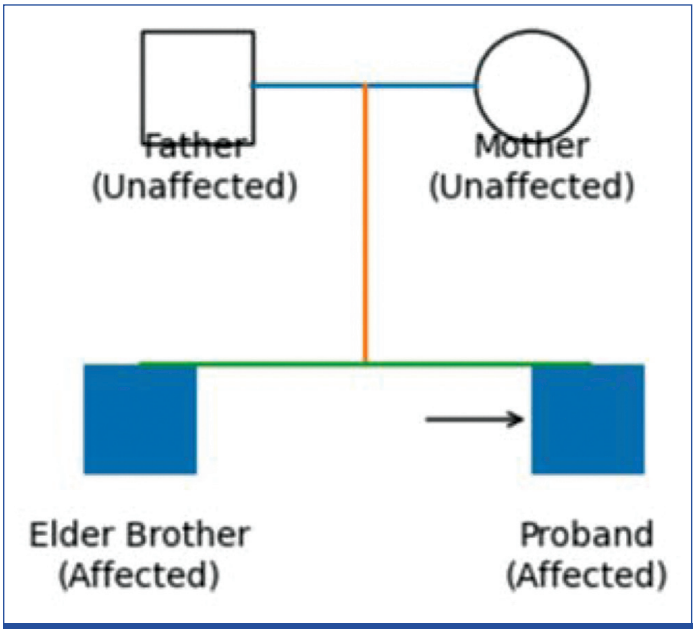
Palmoplantar keratoderma refers to a heterogeneous group of disorders characterised by abnormal thickening of the stratum corneum of the palms and soles. Transgradient palmoplantar keratoderma is a distinctive subtype in which the hyperkeratosis extends beyond the palmar and plantar margins onto the adjoining dorsal surfaces of hands and feet, wrists, ankles, or adjacent skin. Present case is that of a 57-year-old male farmer who presented with thickening of the skin over both palms and soles, associated with fissuring and pruritus. The lesions were first noted in early childhood, initially involving the soles and gradually progressing to the palms over a few years. A positive family history was noted, with similar complaints in his elder brother, suggesting a hereditary pattern. On examination, diffuse palmoplantar hyperkeratosis with transgradient extension onto the dorsal surfaces was observed. Histopathology revealed compact orthokeratotic hyperkeratosis, prominent hypergranulosis, and acanthosis with elongated rete ridges. Transgradient palmoplantar keratoderma should be suspected in patients presenting with early-onset diffuse palmoplantar hyperkeratosis extending onto the dorsal surfaces, especially with a positive family history.

Keywords: Genodermatosis, Hereditary keratoderma, Hyperkeratosis, Pseudoainhum

CASE REPORT

A 57-year-old male, farmer by occupation, presented with thickened skin on both palms and soles associated with multiple cracks and itching. The patient recalls that his parents noted the lesions in early childhood, starting on his soles and gradually progressing to involve his palms over a few years. No history of excessive sweating, deafness, or consanguinity. History of similar complaints present in his elder brother. Patient was also a case of right-eye pseudophakia with peripheral posterior capsular opacification, left-eye posterior subcapsular cataract, nuclear sclerosis, and cortical cataract, with no other significant co-morbidities. No history of similar lesions elsewhere on the body.

Pedigree chart showing affected siblings born to unaffected parents, suggestive of an autosomal recessive inheritance pattern in transgradient palmoplantar keratoderma (Mal de Meleda). The proband is indicated by the arrow [Table/Fig-1].



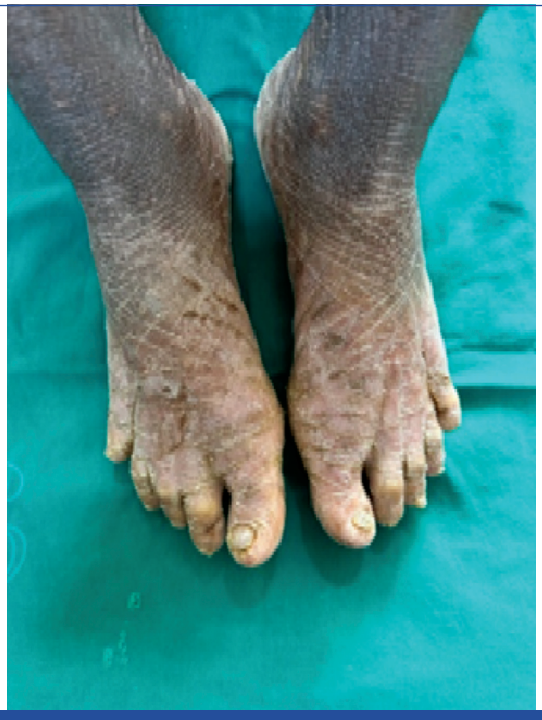
[Table/Fig-1]: Pedigree chart.

Bilateral palms and soles showed diffuse symmetrical transgradient palmoplantar keratoderma with marked yellowish hyperkeratosis, dry rough skin, accentuated dermatoglyphics, and deep fissures, extending onto the dorsal aspects of the hands and feet. Hyperkeratotic plaques with fissuring and desquamation were present over the palmar, plantar, and dorsal surfaces. No starfish-shaped keratoses or constriction bands were noted. All fingernails and toenails showed severe dystrophy with parrot-beak deformity, onychogryphosis, onychauxis, subungual hyperkeratosis, longitudinal ridging, and yellowish-brown discolouration. Flexion deformities of the 3rd, 4th, and 5th distal interphalangeal joints were present bilaterally [Table/Fig-2-5].

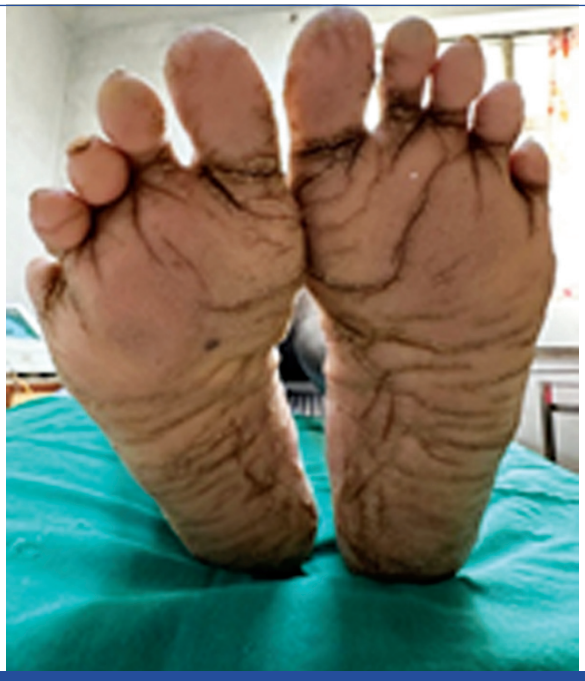
Differential diagnoses considered included Vohwinkel syndrome due to diffuse mutilating keratoderma and flexion deformities, though constriction bands/starfish keratoses were absent. Papillon-Lefèvre syndrome – considered in diffuse PPK with nail involvement, though periodontitis was absent. Epidermolytic palmoplantar keratoderma – ruled out histopathologically by absence of epidermolysis.

[Table/Fig-2]: Shows palmar aspect of both hands with flexion deformity noted over 3rd, 4th and 5th distal interphalangeal joints.

[Table/Fig-3]: Shows the dorsal aspect of both hands with a parrot-beak appearance of all fingernails.



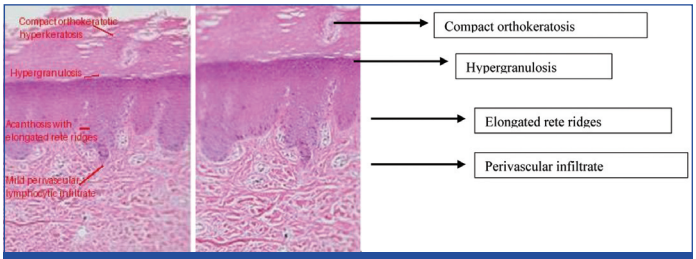
[Table/Fig-4]: Shows the dorsal aspect of both feet with dystrophic toenails.



[Table/Fig-5]: Shows multiple fissures on plantar aspect of both feet.

A punch biopsy was taken from the representative lesion over the lateral aspect of the right foot under local anaesthesia after a test dose, following informed consent from the patient. The tissue was fixed in formalin, routinely processed, and stained with Haematoxylin-Eosin for histopathological examination.

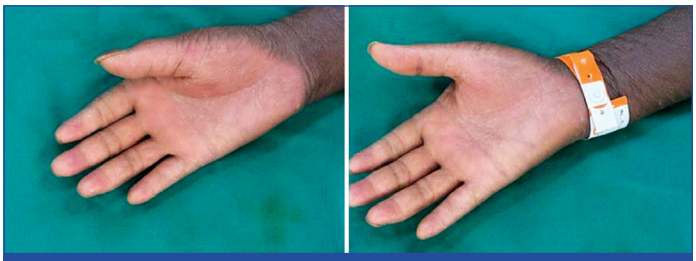
The histopathological section showed features consistent with non-epidermolytic palmoplantar keratoderma, characterised by compact orthokeratotic hyperkeratosis forming a thickened stratum corneum, associated hypergranulosis, elongation of the rete ridges, and a mild superficial perivascular lymphocytic infiltrate within the upper dermis [Table/Fig-6]. All these findings were consistent with non-epidermolytic palmoplantar keratoderma (Mal de Meleda).



[Table/Fig-6]: Photomicrograph of skin biopsy demonstrating compact orthokeratotic hyperkeratosis resulting in pronounced thickening of the stratum corneum (H&E,100x).

The patient was initiated on topical keratolytic and anti-inflammatory therapy, comprising a combination of clobetasol propionate with salicylic acid ointment (Propysalic NF 6% Ointment) applied at night following soaking of the hands and feet in lukewarm water to enhance penetration, along with a morning regimen of emollients containing urea with propylene glycol (Moisturex Cream) and Liquid Paraffin applied four times daily over the entire body for two weeks. This approach aimed to reduce hyperkeratosis, improve skin pliability, and alleviate fissuring and pruritus.

On follow-up after two weeks, the patient demonstrated partial symptomatic improvement, with softening of hyperkeratotic plaques, a reduction in scaling (as shown in [Table/Fig-7-9]), and decreased discomfort from fissures, although complete resolution was not achieved, consistent with the chronic and recalcitrant nature of the disease. Genetic testing could not be performed due to financial constraints and patient unwillingness; however, the clinical and histopathological findings were deemed sufficiently characteristic. The patient was also counselled on avoiding trauma and excessive friction, which may exacerbate lesions.



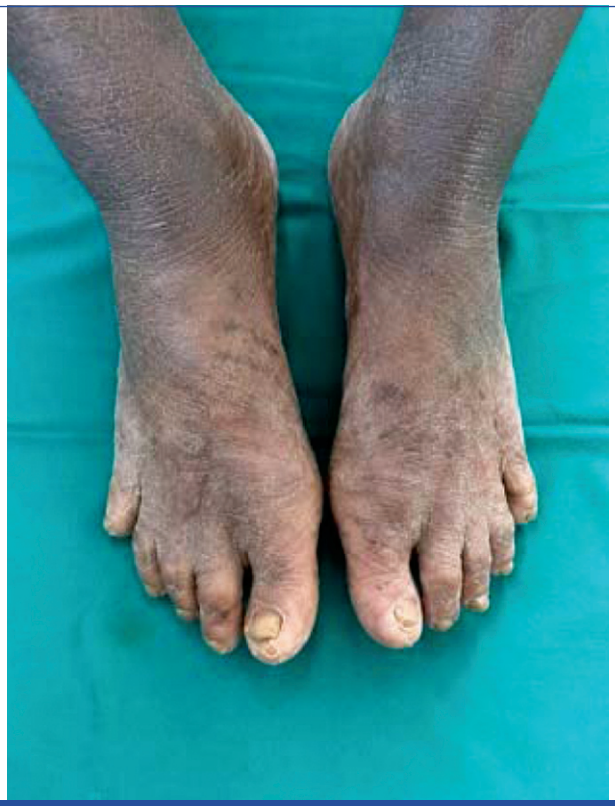
[Table/Fig-7]: Palmar view after two weeks.

DISCUSSION

Transgradient inherited palmoplantar keratoderma is a rare genetic disorder, with an estimated incidence of around 1 in 100,000 people, characterised by diffuse palmoplantar hyperkeratosis, usually without associated systemic manifestations [1].

The condition typically presents during infancy or early childhood. It represents a distinct clinical subset of palmoplantar keratodermas characterised by extension of hyperkeratosis beyond the palmoplantar margins onto the dorsal surfaces of hands and feet. This transgression of anatomical boundaries is a key diagnostic feature and helps differentiate it from non-transgradient forms of palmoplantar keratoderma [2].

[Table/Fig-8]: Improvement noted on dorsal aspect of both hands after two weeks of treatment given.



[Table/Fig-9]: Improvement noted on dorsal aspect of both feet after two weeks of treatment given.

The mutations in the genes encoding proteins involved in keratinisation process, such as keratins, desmosomes, loricrin, cathepsin C, and gap junction proteins have been implicated in pathogenesis of PPK [3].

Histopathological findings of orthokeratotic hyperkeratosis, hypergranulosis, and acanthosis without evidence of epidermolysis were in keeping with a non-epidermolytic palmoplantar keratoderma, further supporting the diagnosis of Mal de Meleda [4].

On comparing with the case of transgradient Mal de Meleda reported by Khan A et al., the index patient similarly demonstrated early-onset diffuse transgradient palmoplantar keratoderma with fissuring and positive family history, supporting the diagnosis of Mal de Meleda [5].

In relation to the report by Achehboune K et al., the management approach in the index case was largely similar in emphasising symptomatic control and reduction of hyperkeratosis. The published case demonstrated significant improvement with systemic acitretin combined with topical keratolytics and emollients, highlighting the effectiveness of oral retinoids in hereditary palmoplantar keratoderma. In index patient, management additionally focused on care of deep fissures, prevention of secondary infection, and functional limitation caused by flexion deformities and severe nail dystrophy [6].

CONCLUSION(S)

This case highlights a classical presentation of transgradient palmoplantar keratoderma consistent with Mal de Meleda, characterised by early onset, progressive diffuse hyperkeratosis with dorsal extension, and a positive sibling history suggestive of autosomal recessive inheritance. The diagnosis was supported by characteristic clinical morphology and histopathological features of non-epidermolytic keratoderma, in the absence of features suggestive of other syndromic or acquired keratodermas. Although genetic confirmation remains the gold standard, careful clinical evaluation continues to play a pivotal role in the diagnosis and management of rare genodermatoses in resource-limited settings.

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PARTICULARS OF CONTRIBUTORS:
1. Postgraduate Student, Department of Dermatology, Sri Devaraj Urs Medical College, Kolar, Karnataka, India.
2. Professor and Head, Department of Dermatology, Sri Devaraj Urs Medical College, Kolar, Karnataka, India.
3. Professor, Department of Dermatology, Sri Devaraj Urs Medical College, Kolar, Karnataka, India.
4. Senior Resident, Department of Dermatology, Sri Devaraj Urs Medical College, Kolar, Karnataka, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. K Suresh Kumar,
RL Jalappa Hospital, Tamaka, Kolar-563103,
Karnataka, India.
E-mail: skindrsureshk@sduaher.ac.in

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