International Journal of Research in Dermatology
Bothra SJ et al. Int J Res Dermatol. 2026 Nov;12(6):677-680
https://www.ijord.com

DOI: https://dx.doi.org/10.18203/issn.2455-4529.IntJResDermatol20263749

Case Report

Beyond extensor surfaces: disseminated keratosis pilaris in childhood and its successful management with isotretinoin

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Received: 24 August 2026
Accepted: 10 September 2026

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ABSTRACT

Keratosis pilaris (KP) is a common follicular keratinisation disorder typically limited to extensor surfaces. Disseminated involvement is rare. An 11-year-old girl presented with generalised hyperpigmented follicular keratotic papules involving the face, trunk, and extremities, sparing the palms and soles, associated with mild pruritus. There was no history of atopy, systemic illness, or nutritional deficiency. Histopathology showed orthokeratotic hyperkeratosis with follicular plugging and mild perifollicular inflammation, confirming KP. Dermoscopy revealed keratin plugs with coiled vellus hairs. Laboratory investigations, including serum retinol, were normal. The patient was treated with low-dose oral isotretinoin and topical keratolytics, resulting in marked clinical improvement within six weeks. Disseminated KP is an uncommon presentation, especially in children. Recognition of atypical hyperpigmented variants is important to avoid misdiagnosis. Systemic retinoids may be effective in extensive disease.

Keywords: Keratosis pilaris, Disseminated keratosis pilaris, Follicular keratosis, Paediatric dermatoses, Isotretinoin

INTRODUCTION

Keratosis pilaris (KP) is a common disorder of follicular keratinisation characterised by folliculocentric keratotic papules resulting from abnormal keratinisation of the pilosebaceous unit.[1] It typically manifests during childhood or adolescence, most commonly affecting the extensor aspects of the upper arms, thighs, and buttocks.[1] Although largely asymptomatic, mild pruritus may occur and is often a cosmetic concern.

KP is frequently associated with atopic diathesis, ichthyosis vulgaris, and obesity.[2] Several variants have been described, including keratosis pilaris rubra, keratosis pilaris atrophicans, erythromelanosis follicularis faciei et colli, and unilateral KP. Disseminated keratosis pilaris, characterised by involvement beyond classical sites, is rare and infrequently reported.[3] We report an 11-year-old girl with generalised hyperpigmented follicular papules involving the face and body, with sparing of palms and soles, highlighting an unusual distribution that expands the clinical spectrum of KP.

CASE REPORT

An 11-year-old female presented with multiple brownish-to-black raised lesions over the body for one year. The lesions initially appeared on the upper limbs and progressively involved the lower limbs, trunk, and face over six months, associated with mild pruritus. She also reported redness and watering of both eyes for three months. There was no history of photosensitivity, mucosal involvement, systemic complaints, or similar lesions in family members.

Cutaneous examination revealed multiple discrete to confluent folliculocentric hyperpigmented keratotic papules measuring 1–3 mm distributed over the face,

trunk, and extremities. The lesions imparted a characteristic rough "sandpaper-like" texture (Figure 1). No erythema, scaling, pustulation, scarring, or alopecia was noted (Figure 2). Examination of the hair, nails, mucosae, palms, and soles was normal.



Figure 1: 11-year-old girl came to OPD with (A) Extensor aspect of the upper limb showing numerous discrete to confluent folliculocentric hyperpigmented keratotic papules distributed diffusely, imparting a rough "sandpaper-like" texture. (B) Bilateral forearms demonstrating symmetrical involvement with multiple folliculocentric hyperpigmented keratotic papules, with relative sparing of the palms.



Figure 2: (A) Lateral view of the face showing multiple skin-coloured to hyperpigmented folliculocentric keratotic papules over the cheek with a roughened surface. (B) Frontal view demonstrating diffuse involvement of the forehead and cheeks by similar folliculocentric keratotic papules without significant erythema or inflammatory changes.

Based on the clinical presentation, differential diagnoses considered included keratosis pilaris, phrynoderma, Darier's disease, eruptive lichen planus, lichen scrofulosum, follicular atopic dermatitis, and pityriasis rubra pilaris. To establish a definitive diagnosis, a skin biopsy was obtained from a lesion on the left leg.

Histopathological examination from a lesion on the left leg showed orthokeratotic hyperkeratosis with prominent follicular plugging, mild acanthosis, and a mild perifollicular lymphohistiocytic infiltrate, consistent with KP. There was no suprabasal acantholysis, dyskeratosis, granulomatous inflammation, or interface dermatitis, excluding other differentials (Figure 3).

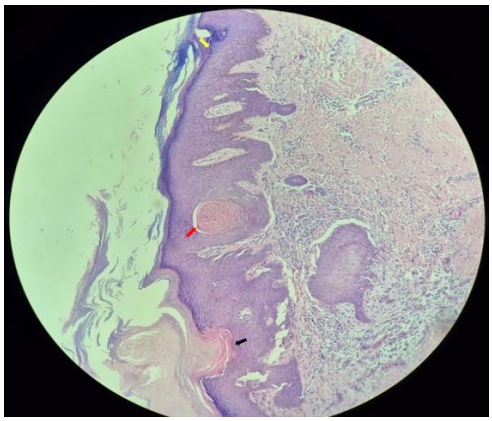


Figure 3: Photomicrograph (hematoxylin and eosin stain at 40x magnification) showing keratin accumulation at the orifice of the hair follicle (black arrow), keratinous plugging of hair follicle (red arrow), and the presence of prominent hypergranulosis (yellow arrow) supporting the diagnosis of keratosis pilaris.

Dermoscopy demonstrated folliculocentric papules on a light-to-medium brown background, each centered by a whitish–yellow keratin plugs with occasional darker cores and coiled or entrapped vellus hairs producing a "corkscrew" appearance, with minimal scaling and no significant vascular features (Figure 4).

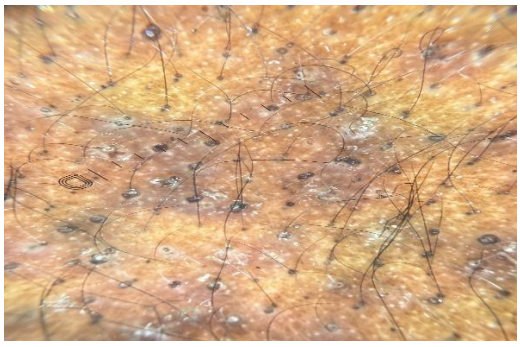


Figure 4: Dermoscopy showing accentuated follicular plugging with perifollicular hyperpigmentation, keratinous debris, along with numerous coiled and entrapped vellus hairs producing a characteristic "corkscrew" appearance, with minimal scaling and absence of significant vascular structures, supporting the diagnosis of keratosis pilaris.

Routine investigations, including complete blood count, liver and renal function tests, thyroid profile, and serum retinol levels, were within normal limits. Based on the

clinical and histopathological findings, a diagnosis of disseminated keratosis pilaris was made. In view of the extensive involvement and significant hyperkeratosis, the patient was initiated on oral isotretinoin 10 mg once daily, along with a topical urea-based keratolytic emollient.

At a six-week follow-up visit, the patient demonstrated a marked reduction in skin roughness and hyperpigmentation with good drug tolerability (Figure 5 and 6). She continues to remain under regular follow-up for further assessment of therapeutic response and adverse effects.

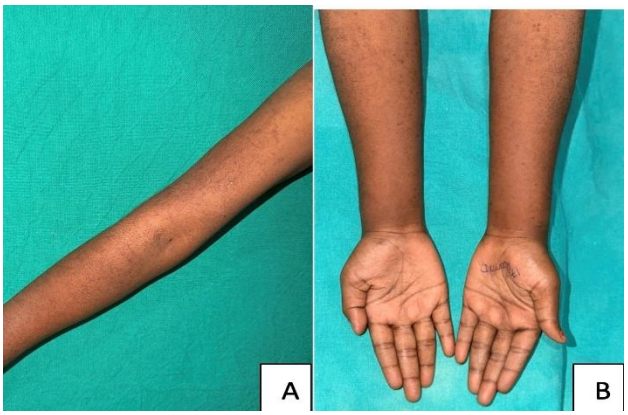


Figure 5: (A) Extensor aspect of the upper limb showing marked reduction in follicular plugging and perifollicular hyperpigmentation after treatment with oral isotretinoin 10mg in 6 weeks. (B) Bilateral forearms exhibiting near complete resolution of lesions with a smoother looking surface after treatment for 6 weeks.



Figure 6: (A) Lateral view of the face showing complete resolution of keratotic papules present over the ear and cheeks post treatment. (B) Frontal view demonstrating noticeable improvement with near complete resolution, leaving minimal residual pigmentation following treatment with oral isotretinoin 10mg for 6 weeks.

DISCUSSION

KP is a common disorder of follicular keratinisation resulting from abnormal keratin deposition within the follicular infundibulum.[1,3,4] It typically presents during childhood or adolescence and remains localised to extensor surfaces of the upper arms and thighs, with gradual improvement over time.[1]

Disseminated or generalised keratosis pilaris is uncommon, particularly in the pediatric population.[3] In such cases, lesions may extend beyond the typical sites to involve the trunk, face, and lower limbs, often posing a diagnostic challenge. Wang and Orlow et al. described the broad clinical spectrum of KP and emphasised its association with conditions such as atopic dermatitis and ichthyosis vulgaris.[2] However, extensive generalised involvement without an associated systemic condition remains relatively rare.

Several reports have described KP-like eruptions occurring in association with genetic or systemic disorders. Lee et al. reported widespread follicular keratotic lesions resembling KP in a patient with Bethlem myopathy.[5] In such cases, abnormalities in dermal connective tissue may influence follicular keratinisation and lead to atypical or persistent lesions. In contrast, our patient did not demonstrate any clinical features suggestive of neuromuscular disease, connective tissue disorder, or inherited syndrome.

Nutritional dermatoses are another important consideration. Phrynoderma, associated with vitamin A deficiency, presents with follicular hyperkeratosis and xerosis.[6] In our case, serum retinol levels were within normal limits, and there were no clinical indicators of nutritional deficiency, effectively excluding this possibility.

An additional distinctive feature in our patient was the hyperpigmented appearance of the lesions. Classical descriptions of KP often highlight erythematous follicular papules; however, in individuals with darker skin phototypes, the lesions may appear predominantly hyperpigmented rather than erythematous. This variation in clinical morphology may lead to diagnostic confusion with other follicular dermatoses such as pityriasis rubra pilaris, follicular lichen planus, or lichen scrofulosorum.

Histopathological examination plays an important role in establishing the diagnosis in atypical presentations. The characteristic findings in KP include orthokeratotic hyperkeratosis with follicular plugging, mild acanthosis, and minimal perifollicular inflammatory infiltrate. The absence of suprabasal acantholysis and dyskeratosis helps exclude Darier disease, while the lack of granulomatous inflammation and interface dermatitis helps rule out lichen scrofulosorum and follicular lichen planus, respectively.

Management of KP is Conventional therapy includes emollients and keratolytic agents such as urea, lactic acid, and salicylic acid.[7] Topical retinoids may also be beneficial in selected cases by promoting normalization of follicular keratinization.[7] Although many cases improve spontaneously with age, extensive variants may persist and require more aggressive treatment.

Systemic retinoids are beneficial in severe hyperkeratotic variants due to their effect on follicular keratinisation.[7] In our patient, low-dose oral isotretinoin combined with topical therapy resulted in significant improvement within a short duration. Recent reports, including that by Auyeung et al have shown response to dupilumab in severe disseminated KP, suggesting a possible immunological component.[8] A comparison of previously reported cases of disseminated keratosis pilaris is summarized in Table 1.

Table 1: Comparison of published reports of generalised keratosis pilaris and the present case.

Author (year)	Age/Sex	Distribution of lesions	Associated condition	Treatment	Outcomes
Wang JF and Orlow et al (2018)	Review	Arms, thighs, occasionally trunk	Atopic dermatitis, ichthyosis vulgaris	Emollients, keratolytic, topical retinoids	Variable improvement
Lee SS et al (2022)	15/F	Generalized follicular papules	Bethlem myopathy	Supportive therapy	Persistent lesions
Auyeung KL et al (2023)	Adult	Widespread pruritic KP	Severe pruritic KP	Dupilumab	Marked improvement
Present case (2026)	11/F	Face, trunk, upper and lower limbs	None identified	Isotretinoin+ keratolytics	Marked improvement

Compared with previously reported cases, our case is notable for the early age of onset, diffuse distribution involving the face and trunk, absence of associated systemic disease, and favourable response to low-dose isotretinoin. Recognition of such atypical presentations is important to avoid misdiagnosis and unnecessary investigations.

CONCLUSION

Disseminated keratosis pilaris is a rare presentation of a common follicular disorder and may mimic several other dermatoses. Clinicopathological correlation is essential for accurate diagnosis. Recognition of such atypical presentations is important to avoid misdiagnosis and guide appropriate management.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Bothra SJ, Biswal A, Begum F, Behera D. Beyond extensor surfaces: disseminated keratosis pilaris in childhood and its successful management with isotretinoin. Int J Res Dermatol 2026;12:677-80.