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A Shared Pathogenesis? Elastic Tissue Degeneration in Two Generations: Co-Occurrence of Acrokeratoelastoidosis and ARCL1A Cutis Laxa

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ABSTRACT

AKE and cutis laxa type ARCL1A are both disorders of elastic fibers characterized histologically by elastin degeneration and/or fragmentation. However, the pathogenesis is thought to be distinct. AKE is an autosomal dominant disorder with an unknown gene mutation. On the other hand, cutis laxa type ARCL1A represents a fibulin five gene mutation. Herein, we present a lady with AKE, and the family history revealed a son with genetically confirmed cutis laxa type ARCL1A. This report might give insight towards the possibility of fibulin gene alterations in the pathogenesis of AKE.

1 | Introduction

Acrokeratoelastoidosis (AKE) is a rare variant of palmoplantar keratoderma, initially characterized by Brazilian dermatologist Oswaldo Costa in 1953 [1]. Clinically, AKE presents as round to oval warty papules, ranging from yellowish to skin-colored, predominantly located on the lateral aspects of the hands and feet. The onset of AKE is typically observed before the second or third decade of life, with a chronic progression [2]. Both sporadic and familial occurrences have been documented, with the latter primarily exhibiting an autosomal dominant inheritance pattern. Notably, a potential genetic linkage with chromosome two has been proposed [3]. The pathogenesis and etiological factors contributing to AKE remain largely unidentified [1, 4, 5]. Herein, we provide a comprehensive and updated literature review of AKE from the first reported case in 1953.

AKE's clinical presentation is characterized by progressively pronounced papules, which are histopathologically distinguished by elastorrhexis. This unique feature is critical for differentiating AKE from other dermatological conditions with similar clinical manifestations. While primarily affecting the skin, there is evidence suggesting the potential for broader elastic tissue involvement [3–5]. Given the rarity of AKE, there is a significant gap in the understanding of its pathogenesis and the identification of contributing genetic or environmental factors. The documentation of familial cases highlights the necessity for genetic studies to elucidate the mechanisms of inheritance and pathophysiology.

Abbreviations: AKE, acrokeratoelastoidosis; DCPH, degeneres collagenous plaques of the hands; FAH, Focal acral hyperkeratosis; KEM, keratosis elastosis marginalis.

2 | Case History/Examination

A 32-year-old Saudi female presented to our dermatology clinic at King Abdul-Aziz University Hospital with asymptomatic skin lesions on the borders of her feet. The patient first noticed the lesions eight years ago, and they have not progressed since. There is no family history of similar skin lesions, but one of her four sons has a genetically confirmed elastic fiber disorder known as cutis laxa syndrome type ARCL1A, and two other sons have eczema. The patient is otherwise healthy with no history of exposure to potential precipitating factors.

Upon examination, there were multiple, slightly raised, skin-colored to yellowish-brown keratotic papules. These were symmetrically distributed along the medial side of the feet at the junction between the plantar and dorsal surfaces and under the lateral malleolus. A single papule was present in the middle of the right Achilles tendon area. The lesions were more palpable than visible. The nails were not involved, and an examination of the hands revealed no similar skin lesions (shown in Figure 1).

3 | Methods

Differential diagnoses include the other type of marginal popular keratoderma (focal Acral hyperkeratosis), degenerative collagenous plaques of the hands, punctuate palmoplantar keratoderma Types 2 and 3, akrokeratosis verruciformis flat wart, and stucco keratosis. Histopathological examination demonstrated marked hyperkeratosis in the epidermis (Figure 2A). Elastin staining showed fragmentation and loss of elastic fibers in the dermis, a condition known as "elastorrhexis" (Figure 2B).

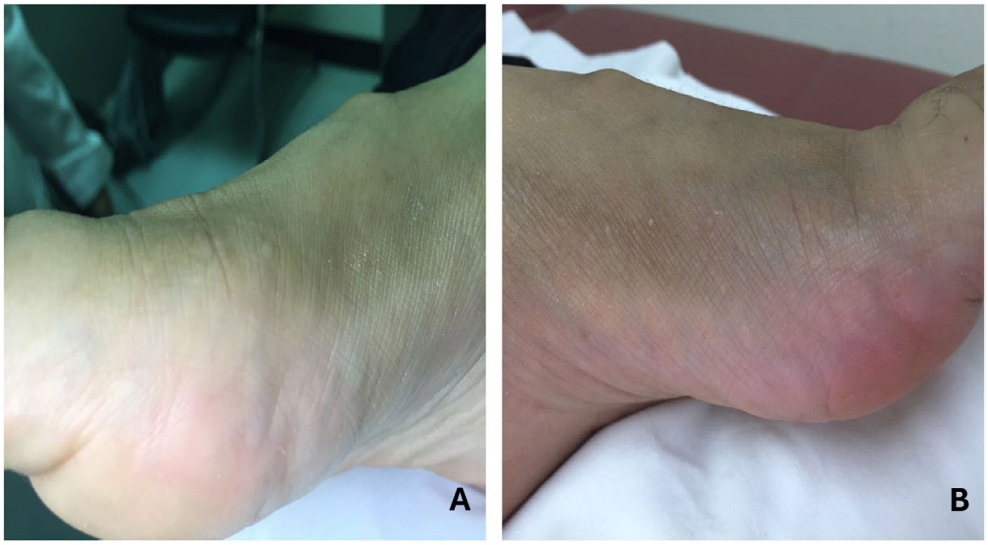

FIGURE 1 | (A) Acrokeratoelastoidosis. Multiple, slightly raised, skin-colored to yellowish-brown keratotic papules, distributed symmetrically along the medial side of the feet at the junction between the plantar and the dorsal surface. (B) Acrokeratoelastoidosis. Multiple keratotic papules distributed symmetrically along the medial side of the feet at the junction between the plantar and the dorsal surface.

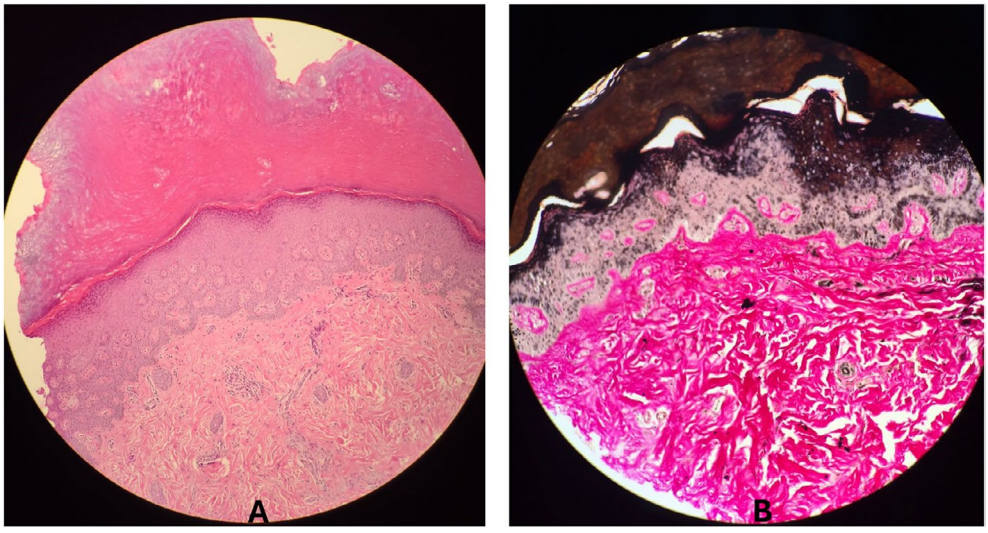

FIGURE 2 | Depicts histopathology of Acrokeratoelastoidosis. (A) The histological section demonstrates an epidermis with marked hyperkeratosis. (B) With Elastin stain, the dermis shows fragmentation and loss of elastic fibers (Elastorrhexis).

The patient was then reassured about his condition. A literature review was made and is displayed in Table 1.

4 | Discussion

Acrokeratoelastoidosis (AKE) is a rare type of marginal papular acrokeratoderma, belonging to a broader group of skin disorders characterized by abnormal palmar and plantar thickening, known as palmoplantar keratoderma (PPK) [6, 7]. The pathogenesis and etiological factors of AKE remain unknown; however, some reported cases have been associated with conditions such as palmoplantar hyperhidrosis [1, 4, 5].

AKE was initially believed to have a predilection for Arabs and Black Africans, but recent findings indicate that AKE can affect individuals of all ages, genders, and races [8, 9]. Although both genders can be affected, most reported cases involve females. Both sporadic and familial forms of AKE have been documented, with the sporadic form being more common. Familial cases typically exhibit an autosomal dominant inheritance pattern with a proposed linkage to chromosome two, suggesting a potential genodermatosis [3]. The disorder can manifest immediately after birth or be acquired later, usually before the age of 30 [2, 4, 10]. While the lesions are generally asymptomatic, they can occasionally be associated with itching [11, 12].

AKE presents as firm, skin-colored to slightly yellowish or whitish keratotic papules, sometimes with central umbilication. These papules, measuring around 2–4 mm, may coalesce to form larger plaques symmetrically distributed on both hands and feet. The lesions usually follow a linear configuration or form clusters along the thenar and hypothenar margins, interphalangeal areas, and along the line separating the dorsal and plantar surfaces. Extension to the wrist, anterior surface of the leg, knee, Achilles tendon area, and the palmar or dorsal surfaces of the feet and hands can occur. Interestingly, AKE can affect only the hands or only the feet, as seen in our case [13–15]. A unilateral form has also been described in three cases [4, 16].

The disorder can remain stable, as in our patient, or more commonly, progress with age or during pregnancy [2, 4, 8, 17–19]. Risk factors may include family history and hyperhidrosis [1, 4, 5]. Despite extensive research, no specific etiology has been identified, and the pathophysiological process remains largely unknown. It has been suggested that the visible keratotic papules of AKE may result from an exaggerated production of filaggrin, which accumulates as a dense band over the granular layer before being incorporated into the protein matrix of mature epidermal keratin [3, 20].

Histologically, AKE most commonly features hyperkeratosis, acanthosis, and epidermal hypertrophy on light microscopy. Other reported features include hypergranulosis, epidermal invagination, and orthohyperkeratosis. Verhoeff's Van Gieson or Elastin stain reveals a reduction in the number of elastic fibers, along with fragmentation (elastorrhexis) in the reticular dermis. These elastic fibers may be thinned, thickened, or tortuous, with varying degrees of involvement among affected individuals, even within the same family [16, 21]. Electron microscopy studies have confirmed the abnormality of elastic fibers and the presence of abnormally dense granules in or near the plasma membrane of fibroblasts. This observation has led to the assumption that the pathological process of AKE involves an abnormality of elastic tissue secretion or excretion, and elastorrhexis rather than elastoidosis [22].

Although AKE appears to be localized, elastorrhexis or elastic tissue changes have been observed not only in affected lesional skin at the palmoplantar margins but also in clinically normal skin [23]. This suggests that AKE is a generalized elastic tissue disorder, manifesting locally at the palmoplantar margins for unknown reasons. Associations with elastic tissue disorders such as scleroderma have been described in the literature [24, 25]. Other associated disorders include aquagenic palmoplantar keratoderma and acquired immunodeficiency from immunosuppressive vedolizumab [5, 26, 27].

The differential diagnosis of AKE includes verruca plana, palmar xanthoma, colloid milia, acrokeratosis verruciformis of Hopf, and other marginal papular acrokeratodermas such as focal acral hyperkeratosis (FAH), degenerative collagenous plaques of the hands (DCPH), keratosis elastosis marginalis (KEM), digital papular calcinosis, mosaic acral keratosis, and hereditary papulotranslucent acrokeratoderma (Table 2).

Various treatment options for AKE have been attempted, including systemic and topical medications, cryosurgery, and laser therapy. Some treatments have shown improvement with no recurrence after 6 months of follow-up, such as erbium laser, and after 1 year of follow-up with 10% salicylic acid applied twice daily for 2 month [14, 29–28]. Other treatments, including topical corticosteroids, topical calcipotriol for 6 months, topical tretinoin twice daily for 1 year, and cryotherapy, have failed to show improvement [4, 13, 12, 33, 16, 35–39, 34]. Partial response has been observed with 2 months of topical tretinoin application [40]. Patients should be informed about the possibility of treatment failure and the limited clinical improvement with most treatment options [41]. In summary, AKE is an asymptomatic disorder with no serious consequences, and treatment is usually unnecessary unless the patient has cosmetic concerns.

Finally, we propose a hypothesis that Acrokeratoelastoidosis (AKE) may be linked to other systemic diseases characterized by elastic tissue defects, as evidenced by previously published cases and supported by our current case involving a family history of cutis laxa. Cutis laxa, known for its systemic involvement due to defects in elastic fibers, often affects multiple organs and suggests a broader underlying elastic tissue pathology. The presence of AKE in a patient with a family background of cutis laxa raises the possibility of a genetic or pathophysiological connection between these two conditions. This observation, coupled with reports of AKE coexisting with systemic sclerosis, suggests that AKE might not be limited to a localized skin disorder but could be part of a spectrum of systemic elastic tissue diseases. This hypothesis necessitates further investigation into the genetic and molecular mechanisms underlying AKE and its links to other conditions with similar elastic tissue defects.

The strength of our report lies in the rarity of AKE and the potential link between AKE and other systemic diseases with elastic tissue defects. AKE is a rare dermatological condition, with

TABLE 1 | Main findings from literature review.

Title	Authors	Age and gender	Comorbidities	Cutaneous findings	Investigation findings	Management plan
Acrokeratoelastoidosis: A Case Report & Review of Literature	Alrefaie, et al. 2024	32, female	Cutis laxa syndrome in one son, eczema in two other sons	Asymptomatic papules along the medial side of the feet, present for years with no progression	Marked hyperkeratosis in the epidermis, elastorrhexis (fragmentation and loss of elastic fibers) in the dermis	Monitoring the condition, patient education about the nature of AKE, no active treatment required due to asymptomatic nature
A Sporadic Case of Unilateral Acrokeratoelastoidosis in Saudi Arabia: A Case Report	AlKahtani, et al. 2014	5, female	Hyperhidrosis on left hand and foot	Yellowish hyperkeratotic papules on the left hand and foot, coalescing into verrucous plaques	Hyperkeratosis, acanthosis, and elastorrhexis confirmed by Verhoeff's-Van Gieson stain	Topical calcipotriol with no significant improvement after 6 months
Acrokeratoelastoidosis of the Foot with Clinical, Dermoscopic, Ultrasonographic, and Histopathologic Correlation	Uribe, et al. 2018	7, female	Allergic rhinitis and bronchial asthma	Nonpainful yellowish papules on the lateral and medial aspects of both feet	Hyperorthokeratosis, hypergranulosis, and elastorrhexis in the reticular dermis; sonographic appearance suggestive of benignancy	Dermoscopy and ultrasound used for diagnosis; no specific treatment mentioned
Acrokeratoelastoidosis as an Example of Marginal Papular Acrokeratoderma with Prominent Elastorrhexis	Żychowska, et al. 2019	37, female	Ectopic gastric mucosa in the esophagus	Flesh-colored papules on the margins of hands and feet, slight hyperkeratosis of the palms	Irregular acanthosis, papillomatosis, hypergranulosis, focal hyperkeratosis, elastorrhexis confirmed by Verhoeff-van Gieson stain	Patient was informed about the benign nature of the condition; no other treatments tried

(Continues)

TABLE 1 | (Continued)

Title	Authors	Age and gender	Comorbidities	Cutaneous findings	Investigation findings	Management plan
Acrokeratoelastoidosis: A Clinico-pathologic Study of Seven Chinese Patients	Zhang, et al. 2005	Various (range: 9–50), both	None reported	Hyperkeratotic papules and plaques on the margins of hands and feet	Histological examination showed elastorrhexis, hyperkeratosis, and acanthosis	Various treatments tried with limited success; patient education and monitoring recommended
Acrokeratoelastoidosis: Indian Dermatology Online Journal	Rambhia, et al. 2015	45, female	None reported	Umbilicated and crateriform hyperkeratotic papules on the lateral border of the index finger and medial border of the thumb	Orthokeratotic hyperkeratosis, acanthosis, thickened and fragmented elastic fibers in the dermis confirmed by Verhoeff-Van Gieson stain	Topical keratolytics, systemic treatments like prednisolone, dapsone, methotrexate, acitretin; Er:YAG laser; patient counseling
Acrokeratoelastoidosis: A Clinicopathologic Study of Eight Cases	Bogle, et al. 2002	Various (range: 12–68), both	None reported	Small, firm, umbilicated, skin-colored and keratotic papules along the borders of the hands and feet	Orthokeratotic hyperkeratosis, acanthosis, elastorrhexis in the dermis	Various treatments including keratolytics and topical steroids; patient counseling
Acrokeratoelastoidosis: Report of a Case	Truett, et al. 1955	29, female	None reported	Multiple, small, firm, keratotic papules on the lateral borders of the hands	Hyperkeratosis, acanthosis, thickened and fragmented elastic fibers	Observation and patient education; no specific treatment mentioned

(Continues)

TABLE 1 | (Continued)

Title	Authors	Age and gender	Comorbidities	Cutaneous findings	Investigation findings	Management plan
Acrokeratoelastoidosis: A Rare Case Report	Smith, et al. 2018	14, female	None reported	Small, firm, keratotic papules on the borders of the hands and feet	Orthokeratotic hyperkeratosis, acanthosis, elastorrhexis in the dermis	Patient education and monitoring; topical keratolytics tried with limited success
Acrokeratoelastoidosis: Further Observations in Two Families	Naylor, et al. 1990	Various (range: 8–50), both	None reported	Small, firm, keratotic papules on the borders of the hands and feet	Hyperkeratosis, acanthosis, fragmented elastic fibers in the dermis	Patient education and monitoring; various treatments tried with limited success
Marginal Papular Acrokeratodermas: A Unified Classification	Costa, et al. 1992	35, male	None reported	Firm, flesh-colored papules on the margins of hands and feet	Hyperkeratosis, acanthosis, elastorrhexis in the dermis	Patient education and monitoring; topical keratolytics tried with limited success
Acrokeratoelastoidosis in Three Siblings	Johnson, et al. 1985	Various (range: 5–15), both	None reported	Firm, keratotic papules on the borders of the hands and feet	Orthokeratotic hyperkeratosis, acanthosis, elastorrhexis in the dermis	Patient education and monitoring; various treatments tried with limited success
Palmoplantar Hyperkeratotic Projections	Bao, et al. 2020	81, female	Breast cancer in remission, hypertension, diabetes mellitus, hypothyroidism, hyperlipidemia	Scattered discrete hyperkeratotic projections on palms and soles	Compact hyperkeratosis and parakeratosis with underlying hypogranulosis	Mechanical friction and keratolytics; lesions tend to recur without regular maintenance therapy

TABLE 2 | Comparison of Acrokeratoelastoidosis (AKE) differential diagnoses.

Disorder	Onset	Clinical features	Histologically	Risk factor
Acrokeratoelastoidosis (AKE)	Childhood to young adulthood	Keratotic papules and plaques at palmoplantar margins	Epidermal and dermal changes. Hallmark is elastorrhexis	Family History (AD)
Focal Acral Hyperkeratosis (FAH)	Similar to AKE	Clinically identical to AKE	Epidermis: Focal hyperkeratosis and acanthosis similar to AKE. Dermis: no reticular dermal changes or elastorrhexis	Family History (AD)
Degenerative Collagenous Plaques of the Hands (DCPH) / Keratosis Elastosis Marginalis (KEM)	Middle age and older (40s-60s)	Advanced actinic damage of exposed skin, Location at radial margin of index finger, first web space, and ulnar margin of thumb	Marked degeneration of both collagen and elastin fibers	Old Caucasians with history of chronic sun exposure and manual work
Acrokeratosis Verruciformis of Hopf	Birth or early childhood, occasionally adulthood	Keratotic papules at dorsum of the hands and feet, knees, elbows and forearms. Palmoplantar punctuation and nail dystrophy.	Hyperkeratosis, acanthosis, papillomatosis and a thickened granular layer. Elevations of the epidermis 'church spires'	AD/ATP2A2 gene

fewer than 60 cases documented in the literature, highlighting the importance of each new report in contributing to our understanding of this disorder. Our case is particularly significant because it introduces the unique context of a family history of cutis laxa, another rare disorder characterized by systemic involvement of elastic tissues. This connection suggests a possible shared genetic or pathophysiological basis between AKE and systemic diseases like systemic sclerosis. By highlighting this potential link, our report opens new avenues for research into the broader implications of AKE, suggesting it could be part of a spectrum of disorders with systemic elastic tissue involvement rather than merely a localized skin condition.

Author Contributions

Sumayyah I Alrefaie: conceptualization, supervision. **Sarah B. Aljoudi:** conceptualization, supervision, validation. **Houriah Y. Nukaly:** conceptualization, data curation, formal analysis, methodology, resources, writing – original draft, writing – review and editing. **Waseem K. Alhawsawi:** data curation, formal analysis, validation, writing – original draft, writing – review and editing. **Asem Shadid:** conceptualization, data curation, investigation. **Sultan AlNasser:** conceptualization, data curation. **Jehad Hariri:** conceptualization, project administration, supervision, validation, visualization.

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The authors have nothing to report.

Consent

Informed consent was obtained from the patient and/or legal guardian for the publication of this case report. The patient and/or legal guardian was informed that their identity would be kept confidential, and all personal identifying information would be removed to ensure anonymity. They were also made aware that the case report may be published in medical literature or presented at medical conferences. They were informed that their participation was voluntary and that they could withdraw their consent at any time.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data supporting this case report are available upon reasonable request. Interested parties may contact the corresponding author Houriah Nukaly for access to the data related to this study.

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