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Case report

Keratoderma climactericum: an under-reported condition successfully treated with an estriol cream

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ABSTRACT

Climacteric keratoderma is a specific cutaneous disorder often underreported, potentially misdiagnosed, or undervalued. Although the pathogenesis remains undetermined, hormonal changes during menopause heavily affect the trophism and normal cellular function of the epidermis and dermis of the palmoplantar regions. We present the case of a 55-year-old woman referred to the Dermatological Clinic of Cagliari for acquired bilateral palmoplantar hyperkeratosis not responding to common topical treatments. Based on the medical history, clinical appearance of the lesions, and laboratory tests, keratoderma climactericum was diagnosed, as confirmed by histopathological examination. An overlap with allergic contact dermatitis was also detected through patch testing, and its synergic effect contributed to the perpetuation of the dermatosis on the hands. A daily high-potency corticosteroidal topical therapy, combined with a 50% urea-based cream at night, was only partially effective. However, adding estriol-based cream at a concentration of 0.125% led to a significant clinical improvement. Climacteric keratoderma is an acquired condition that deserves further clinical trials to assess topical hormonal treatment protocols, as the current approach is largely empirical.

INTRODUCTION

Climacteric keratoderma (CK) is an acquired palmoplantar hyperkeratotic (PPK) disorder described by H. Hauxthausen in 1934, who first noted a strong association with menopause (1). Few cases have been reported in the literature, and the treatment is empirically based on the application of topical urea and estrogens (2). It is generally an underrecognized pathology; the diagnosis is clinical, although it can be supported by skin biopsy and histological examination, excluding major skin conditions (3) that can manifest themselves with similar clinical features. Of course, hormonal deficiency might complicate and be associated with several associated causes of keratoderma, representing a diagnostic and treatment challenge for the dermatologist (4).

CASE REPORT

A 54-year-old woman was referred to the outpatient clinic complaining of a 4-month medical history of skin thickening on her palms and soles, associated with persistent itching and painful fissures (Fig. 1). She had sought consultations with several dermatologists. She had undergone treatments with antifungals, topical steroids, and keratolytic creams without consistent improvement. Physical examination revealed bilateral palmoplantar hyperkeratosis, primarily on pressure points, exhibiting well-defined margins and a yellow-brown coloration. Additionally, a few painful fissure-shaped lesions on some fingers were noted. Her medical history included type 1 diabetes mellitus treated with insulin therapy since adulthood, and she started menopause 9 months before reaching out for our service, as confirmed by her gynecologist.

Blood tests yielded the expected results. Direct microscopic examination (KOH) ruled out a dermatophyte infection, and a skin biopsy was conducted to exclude major dermatitis, particularly psoriasis. Histopathological analysis revealed diffuse orthokeratotic hyperkeratosis, slight lymphocytic exocytosis, and superficial perivascular lymphocytic exudate (Fig. 2).

Based on the patient's medical history, clinical presentation, and histological findings, keratoderma climactericum was diagnosed. Topical therapy was initiated with estriol 0.125% vaginal cream twice daily, and urea 50% cream once daily, both applied at palms and soles, resulting in consistent improvement after one month (Fig. 3). The patient reported relief from itching and improved functionality, particularly during walking.

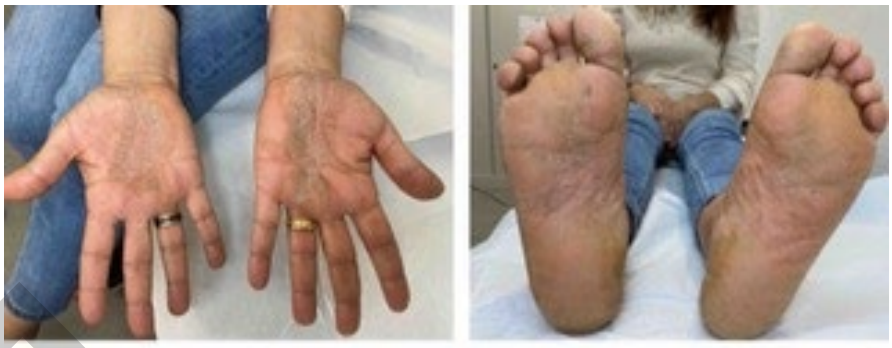

Fig. 1. *Bilateral skin thickening on the palms and soles, mainly on pressure points, with well-defined margins and yellow-brown color; few but very painful fissure-shaped lesions were also associated with some fingers.*

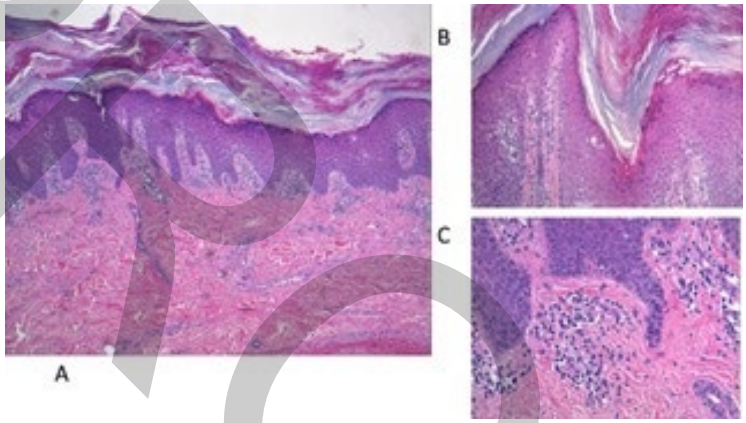

Fig. 2. *Histopathology of a plantar biopsy shows diffuse ortho-keratotic hyperkeratosis, slight lymphocytic exocytosis, and superficial perivascular lymphocytic exudate (Haematoxylin & Eosin stain, inset A magnification 10x; inset B and inset C magnification 20x).*

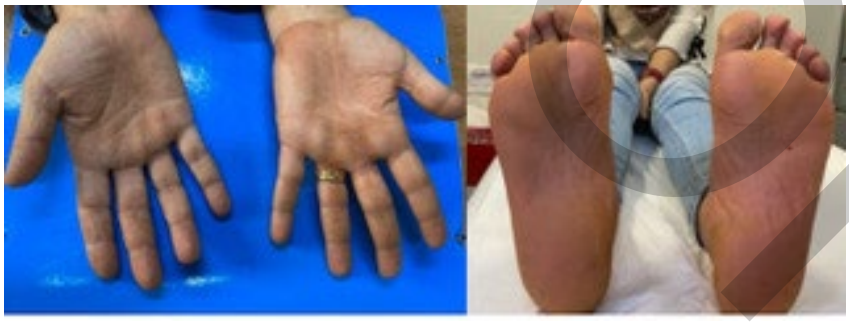

Fig. 3. *Hyperkeratotic palmoplantar region improvement after 4 weeks of applying 0.125% estriol-based cream twice daily.*

However, at the 6-month follow-up, the patient reported a slight worsening of clinical manifestations on her hands, presenting eczema of the fingers and palmar hyperkeratosis. At the same time, complete healing of her feet was maintained. The patient worked at school as a janitor and mentioned that she does not use protective equipment when cleaning. Patch tests were conducted, revealing positivity to cobalt and mixed perfumes. Consequently, a concurrent diagnosis of allergic contact dermatitis was made, and treatment was supplemented with hydrocortisone butyrate 0.1% cream once daily, resulting in complete remission of the clinical picture.

DISCUSSION

This case underscores the importance of recognizing menopause-associated disorders, as climacteric keratoderma may be more prevalent than reported in the literature, particularly considering the dated nature of many existing reports (1-12). The concurrent onset of menopause and the development of skin lesions, characterized by noninflammatory thickening starting centrally on pressure points that slowly extend to the rest of the palms and soles, prompted consideration of this diagnosis. However, several causes of PPK should be excluded (4, 5). While a thorough medical history can often rule out hereditary forms, significant conditions such as psoriasis and contact dermatitis require specific assessment. In the present case, histopathology was promptly performed, given the patient's prior consultations with multiple dermatologists and her request for a definitive diagnosis. After ruling out psoriasis, we felt confident in the diagnosis and initiated treatment with estriol cream based on recent reports (5, 11), which supported our hypothesis "*ex adiuvantibus*." However, the precise mechanisms linking ovarian hormone decline to keratoderma induction remain undocumented. Experimental models are limited and outdated. A study on human female epidermis (13), comparing abdominal to plantar biopsy samples, found lower estrone (E1)-sulfatase activity in plantar epidermis, suggesting that plantar regions may be more susceptible to a decrease in circulating estrogens.

Additionally, xerosis and loss of elasticity on palmoplantar surfaces significantly impact daily functioning and may be exacerbated by detergents, water contact, and friction. Hormonal decline also impairs skin microcirculation. Our patient did not undergo hormonal replacement therapy and was not keen to do so, as she was otherwise in good health and did not report menopausal neurovegetative or psychoemotional symptoms.

Another noteworthy aspect of our case was the sudden worsening of hand manifestations, raising suspicion of additional contact injuries. Other patients may have psoriasis or concomitant infections that could exacerbate the course of climacteric keratoderma. When selecting a treatment strategy, physicians must consider all differential diagnoses and recognize that treatment may be ineffective if the climacteric component is not adequately addressed. In our case, previous cycles of keratolytic and local corticosteroidal topical treatments were ineffective until 0.125% estriol cream was added. Even though oral treatment with etretinate has been reported as effective in 10 patients with climacteric keratoderma (3), our patient preferred to continue with topical treatments only.

CONCLUSION

There are several forms of palmoplantar keratoderma, both congenital and acquired (4, 6), and diagnosing the menopause-related form can be challenging. The presence of other causes of keratoderma may obscure recognition, but treatment resistance is a red flag that should raise clinical suspicion in menopausal women. Empirical treatment with topical estrogens was effective in our patient, underscoring the need for structured clinical trials to establish protocols and recommendations for managing CK.

Authorship: All authors contributed to the case observation and management. Dr TA was responsible for data accuracy and manuscript writing. Dr PL is the pathologist responsible for histology examination. Prof. AL is responsible for supervision and final editing. All authors agree to be listed and have approved the submitted version of the manuscript.

Consent: The patient provided written consent to her history and image publication.

Conflict of interest statement: no conflicts to declare.

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