





Unilateral Hyperkeratotic Lesions With a Sporotrichoid **Pattern**

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Received: 5 August 2025 | Revised: 16 October 2025 | Accepted: 10 November 2025

Funding: The authors received no specific funding for this study.

Keywords: dermoscopy | follicular keratosis | phrynoderma | vitamin A

1 | Case Presentation

A 65-year-old man, with no relevant medical history aside from moderate alcohol consumption, presented with asymptomatic lesions on his left leg that had developed over several months and had not responded to topical corticosteroids or antibiotics. On physical examination, erythematous-brownish, hyperkeratotic papules and plaques were observed, distributed along the left lower extremity (Figure 1).

Dermoscopy revealed follicular keratotic plugs on a brownish background, dotted vessels, and shiny white interfollicular areas (Figure 2).

Blood count and biochemical tests were within normal limits, except for a vitamin A deficiency (0.13; normal range 0.3-0.6). A biopsy of one lesion was performed, and histopathological examination showed superficial folliculitis with formation of infundibular cysts (Figure 3). PAS and Grocott stains were negative for fungal elements.





FIGURE 1 | (A) Hyperkeratotic papules arranged in a linear distribution on the left lower extremity. (B) Close-up view of the lesions.

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FIGURE 2 | Dermoscopy. On a brownish background, follicular keratotic plugs (black arrow), structureless white areas (yellow arrow), and evenly distributed dotted and glomerular vessels (black star) can be observed.

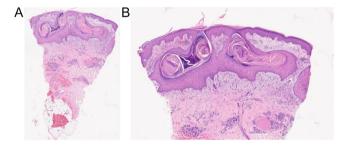


FIGURE 3 | Biopsy from the left leg with hematoxylin and eosin (H&E) stain. (A) Epidermal inclusion cysts with acanthosis and superficial dermal lymphocytic infiltrates ($\times 10$). (B) Epidermal inclusion cyst with adjacent lymphocytic infiltrate ($\times 20$).

2 | What is the diagnosis?

2.1 | Phrynoderma

3 | Discussion

Phrynoderma is a dermatosis classically associated with vitamin A deficiency, although it has also been linked to deficiencies in B vitamins, vitamin E, and essential fatty acids (EFAs). It is now considered a manifestation of generalised malnutrition rather than a result of isolated micronutrient deficiency [1].

This condition is more commonly seen in paediatric populations, individuals with chronic alcohol use like our patient, those undergoing haemodialyses, and patients with intestinal malabsorption such as in inflammatory bowel disease or after gastrointestinal surgery [2, 3]. Chronic alcohol consumption is known to precipitate a decline in hepatic retinoid levels through

increased breakdown, making it the most plausible cause of the deficiency in our patient despite the absence of other nutritional deficits [4].

Clinically, phrynoderma presents as hyperkeratotic follicular papules and plaques with keratin plugs. It typically affects extensor surfaces of the limbs and the buttocks, though other body sites may be involved [5]. The differential diagnosis includes other follicular keratotic disorders such as keratosis pilaris, pityriasis rubra pilaris, follicular psoriasis, and follicular lichen planus [6]. In our case, the sporotrichoid distribution of the lesions also raised suspicion for fungal or mycobacterial infections, as well as cutaneous leishmaniasis and nocardiosis [7]. The patient presented with only a few lesions that were clinically prominent, an uncommon presentation that may reflect localised factors influencing lesion development or progression. A possible explanation for this distribution could be scratching and local trauma, as described in acquired perforating dermatoses, although our patient did not report pruritus [8].

Dermoscopy is a valuable, noninvasive tool to guide diagnosis. A prospective study of 30 patients analysed the most frequent dermoscopic features in follicular keratotic dermatoses. In cases of phrynoderma, the most specific findings were keratin plugs, perifollicular and interfollicular pigmentation, and coiled hairs. These correspond histologically to keratin-filled follicular infundibula, epidermal acanthosis with hyperkeratosis, and melanin deposition, respectively [6].

In our patient, dermoscopy showed follicular keratotic plugs on a brownish background, as well as white structureless areas and dotted vessels, features not described in the aforementioned study. Histologically, these may correspond to epidermal acanthosis and vascular dilatation.

Treatment is based on correcting nutritional deficiencies through dietary measures and micronutrient supplementation, while avoiding vitamin A toxicity. Our patient was treated with vitamin A and E 50,000 IU every 15 days for 6 months, combined with topical calcipotriol and betamethasone dipropionate cream, which resulted in normalisation of vitamin A levels and complete resolution of the lesions. Skin lesions usually resolve within 1–4 months. In some cases, phototherapy may be beneficial [1].

This case highlights the importance of recognising cutaneous signs of nutritional deficiencies and underscores the key role of dermoscopy as a noninvasive diagnostic tool. Its use allows for the identification of patterns that, in the appropriate clinical context, may help avoid more invasive procedures such as biopsy, facilitating early diagnosis and management.

Author Contributions

José González Fernández: manuscript preparation and supervision, final manuscript approval. Mariano Ara Martín: intellectual participation in the diagnostic and therapeutic management of the case, manuscript review. Diego Larrosa-Martínez: concept and planning of the study, literature review. Francesc Felipo-Berlanga: intellectual participation in the diagnostic of the case. Javier Sánchez-Bernal:

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manuscript preparation, final manuscript approval, final decision on the diagnostic and therapeutic management of the case.

Ethics Statement

All patients in this manuscript have given written informed consent for participation in the study and the use of their deidentified, anonymized, aggregated data and their case details (including photographs) for publication. Ethical Approval: not applicable.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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