Article published online: 2022-06-08

Case Report

Remarkable Improvement of Palmar Hyperkeratosis to Thyroxine Replacement in a Patient with Severe Undiagnosed Severe Hypothyroidism

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Abstract

Hypothyroidism can present with a wide range of nonspecific symptoms, some of which are cutaneous. However, hypothyroidism presenting as isolated palmar hyperkeratosis is rarely described in the literature. Here, we describe a 30-year-old male who presented with hyperkeratosis and painful fissures of both palms for 1½ years' duration. He had minimal relief with keratolytic agents. On physical examination, he was found to have bradycardia and when investigated he was found to have severe hypothyroidism. A diagnosis of autoimmune hypothyroidism was made after further laboratory studies. He was treated with thyroxine, and a gradual improvement of hyperkeratosis was noted over a period of 3 months. Although extremely rare, clinicians should consider hypothyroidism as a cause of hyperkeratosis, especially when it is refractory to treatment and/or there are other possible symptoms of hypothyroidism.

Keywords: Hypothyroidism, palmar hyperkeratosis, secondary palmoplantar hyperkeratosis

Introduction

Hypothyroidism is the second most common endocrine disorders after diabetes mellitus. It affects nearly 3.7% of the population.[1] Hypothyroidism can present with a wide variety of symptoms, most of which are nonspecific. It can even be asymptomatic, with a prevalence of 3%–10% of subclinical hypothyroidism in the general population.[2] The dermatologic manifestations of hypothyroidism are of a wide range[3] and are mostly nonspecific.

Palmoplantar hyperkeratosis can either be hereditary or acquired. The causes of acquired palmoplantar hyperkeratosis may be keratoderma climactericum, chemical induced, systemic disease, malignancy associated, dermatoses, medication associated, nutritional deficiency, psoriasis, lichen planus, pityriasis rubra pilaris, eczema, Reiter's syndrome, fungal infections, trauma, or idiopathic.[4-6] Cases of hypothyroidism presenting as palmoplantar hyperkeratosis were described rarely in English literature;[7-9] After an extensive search of literature, we were able to find only a single case of isolated palmar hyperkeratosis as the sole presentation of hypothyroidism[10] Here, we describe a case of a 30-year-old male patient who presented with palmar hyperkeratosis which improved significantly after thyroid hormone replacement, suggesting that the undiagnosed severe hypothyroidism was the underlying cause of his palmar hyperkeratosis.

Access this article online

Quick Response Code:

Website: www.jdeponline.com

DOI: 10.4103/jdep.jdep_6_18

Case Report

A 30-year-old male presented with complaints of thickening of the skin in his palms with painful fissures between thickened skin [Figure 1]. The complaint started 1½ years before and was worsening over the first 6 months. He consulted a skin specialist and was given keratolytic medications (40% urea cream and 2% salicylic acid), which gave only minimal relief of his symptoms. During the past 2 months, the symptoms worsened, and he now reports inability to hold objects due to pain. Medical history is significant for hypercholesterolemia

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How to cite this article: Thomas A, Robert A, Kumar AS. Remarkable improvement of palmar hyperkeratosis to thyroxine replacement in a patient with severe undiagnosed severe hypothyroidism. J Diabetes Endocr Pract 2018;1:12-4.

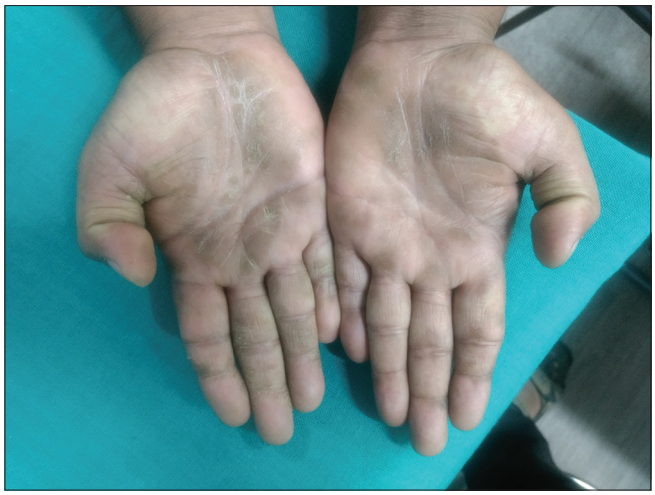

Figure 1: Patient's palms at the time of presentation

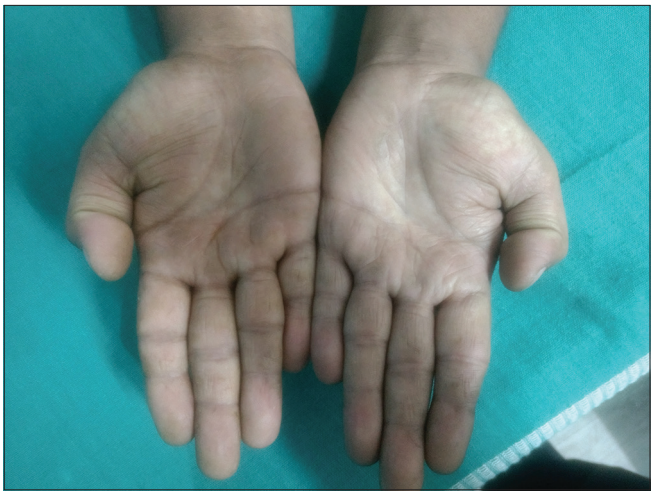

Figure 2: Patient's palms after 2 months of thyroid hormone replacement

diagnosed 2 years back. He has no known allergies. There is no family history of significant dermatologic diseases. He was on atorvastatin 10 mg daily and using the topical keratolytics mentioned above.

Physical examination was significant for bradycardia (52 beats/minute). Electrocardiogram confirmed presence of sinus bradycardia of 52 beats/min. His thyroid hormone levels were analyzed as a part of the workup for bradycardia. Thyroid-stimulating hormone (TSH) level was >100 mIU/ML (0.35–5) with a T3 of 61.95 ng/dl (60–181) and a T4 of 1.25 μg/dl (4.5–12.5). On further laboratory analysis, anti thyroglobulin antibodies were found to be 1188 U/ML (normal, up to 60), and microsomal antibody/thyroid peroxidase antibody was >600 IU/ML (normal up to 34). A diagnosis of autoimmune thyroiditis (Hashimoto thyroiditis) was made based on these investigations.

The patient was started on thyroid hormone replacement with an initial dose of 50 μg/day of thyroxine, which was gradually increased to 125 μg/day. At 2 months follow-up, the palmar hyperkeratosis significantly improved even after discontinuation of keratolytic agents, [Figure 2], and his TSH at the time was 2.14 mIU/ML (0.35–5). Similarly, the blood cholesterol level returned to normal level (167 mg/dl) without statins.

Discussion

Palmoplantar hyperkeratosis secondary to hypothyroidism has been reported rarely in the literature.[7-9] After an extensive search of English literature, we were able to find only one case of isolated palmar hyperkeratosis (without any involvement of the plantar skin) secondary to hypothyroidism.[10] In the reported cases of palmoplantar hyperkeratosis secondary to hypothyroidism, the plantar hyperkeratosis was more extensive, and the palmar involvement was limited, also the keratosis had a yellow hue in most of the reported cases.[7] Although most of the reported cases were in myxedema patients, a few are in asymptomatic patients. In all the cases, the hypothyroidism was due to autoimmune thyroiditis.

Palmoplantar hyperkeratosis can be either hereditary or secondary to systemic or local diseases. Patel *et al*. define acquired keratoderma as a "nonhereditary, nonfrictional hyperkeratosis of the palms and/or soles that involves ≥50% of the surface of involved acral areas and that may or may not be associated with clinical and histologic inflammation."[6] Ruling out the presence of internal malignancy is of utmost importance in the evaluation of secondary hyperkeratosis. Hypothyroidism can only be attributed as the cause of palmoplantar hyperkeratosis retrospectively when the hyperkeratosis refractory to other treatments shows an immediate and apparent improvement upon initiation of hormone-replacement therapy. Previous cases reported 1–9 months of thyroxine therapy for complete remission of symptoms.[8,9] The exact pathophysiology behind hyperkeratosis in hypothyroidism is still largely unknown.

The skin changes commonly associated with hypothyroidism are coarsened, thick or scaly skin, nonpitting edema (myxedema), carotenemia, dry/brittle/coarse hair, alopecia, coarse/dull/thin/brittle nails, dry skin, and loss of the lateral third of eyebrows. The skin changes associated with hyperthyroidism are smooth/thin skin, fine hair, alopecia, onycholysis, and friable nails. Other autoimmune skin changes, such as vitiligo and eczema, can also be seen in association with autoimmune thyroid diseases.[11]

Although extremely rare, clinicians should also consider hypothyroidism as a cause of hyperkeratosis, especially when it is refractory to treatment and/or there are other possible symptoms of hypothyroidism.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Acknowledgment

The authors would like to thank our family clinic in Kumily.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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