

Acquired Ichthyosis and Rhabdomyolysis as Unusual Manifestations of Severe Hashimoto's Hypothyroidism: A Case Report

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Abstract

Acquired ichthyosis is an uncommon dermatological manifestation that may occasionally reveal an underlying systemic disorder, including endocrine diseases. We report the case of a 31-year-old man with no significant dermatological or family history, admitted for severe asthenia, diffuse myalgia, dysphonia, and progressive psychomotor slowing evolving over several months. Clinical examination revealed marked acquired ichthyosis characterized by thick polygonal scales predominantly affecting the extensor surfaces, associated with bradycardia and cognitive slowing. Laboratory investigations demonstrated profound hypothyroidism with thyroid-stimulating hormone (TSH) levels >100 mIU/L and markedly decreased free thyroxine (FT4 = 0.45 ng/dL). Creatine phosphokinase (CPK) levels were markedly elevated (7245 IU/L), consistent with rhabdomyolysis, in the absence of trauma, intense physical exertion, or myotoxic drug exposure. Autoimmune work-up showed strongly positive anti-thyroid peroxidase (>1200 IU/mL) and anti-thyroglobulin antibodies (300 IU/mL), confirming Hashimoto's thyroiditis. In the absence of any alternative cause, both the muscular involvement and acquired ichthyosis were attributed to severe thyroid hormone deficiency. Treatment with levothyroxine resulted in progressive clinical improvement, normalization of CPK levels, and marked regression of skin lesions. This case highlights the importance of considering severe hypothyroidism in the diagnostic work-up of acquired ichthyosis associated with muscular manifestations.

Keywords

Acquired Ichthyosis, Rhabdomyolysis, Hashimoto's Thyroiditis

1. Introduction

Acquired ichthyosis is a rare dermatological disorder characterized by generalized scaling resulting from abnormal epidermal keratinization. Unlike inherited forms, it usually develops during adulthood and is often associated with an underlying systemic disease, including malignancies, infectious disorders, autoimmune diseases, metabolic abnormalities, and, less frequently, endocrine disorders. It is classically subdivided according to its underlying cause into paraneoplastic, drug-induced, nutritional, infectious, and endocrine-related forms, each reflecting a distinct mechanism of epidermal barrier disruption. Among endocrine causes, thyroid dysfunction has long been recognized as a potential trigger of cutaneous xerosis, although progression to clinically overt ichthyosiform scaling remains distinctly uncommon and is reported almost exclusively in cases of severe, long-standing hormone deficiency.

Hypothyroidism is a rare but recognized cause of acquired ichthyosis, particularly in its severe forms. Thyroid hormones play an important role in epidermal differentiation, keratinocyte proliferation, lipid metabolism, and maintenance of the skin barrier; consequently, severe hormone deficiency may produce prominent cutaneous abnormalities. However, the coexistence of acquired ichthyosis with significant muscle involvement is rarely described and may provide an important diagnostic clue to an underlying systemic endocrine disorder.

Hypothyroidism is among the most prevalent endocrine disorders worldwide, with autoimmune thyroiditis of the Hashimoto type representing its leading cause in iodine-replete regions. While fatigue, cold intolerance, weight gain, and bradycardia are well known, the musculoskeletal and dermatological expressions of severe hypothyroidism remain comparatively underrecognized, despite their potential to constitute the presenting or occasionally the sole clue leading to diagnosis. Hypothyroid myopathy, ranging from asymptomatic elevation of muscle enzymes to overt rhabdomyolysis, and cutaneous xerosis progressing to acquired ichthyosis are two such peripheral manifestations that, although individually well described, are only exceptionally reported in combination.

We report a case of profound autoimmune hypothyroidism revealed by the unusual association of acquired ichthyosis and rhabdomyolysis, highlighting the importance of considering thyroid dysfunction in the etiological evaluation of acquired ichthyosis, particularly when accompanied by unexplained muscular symptoms or marked elevation of muscle enzymes. This case also emphasizes the value of recognizing seemingly unrelated cutaneous and musculoskeletal manifestations as potential components of a single underlying endocrine disorder.

2. Case Report

We report the case of a 31-year-old man with no significant personal or family medical history who was admitted for severe asthenia, diffuse myalgia, dysphonia, and progressive psychomotor slowing that had developed insidiously over several months. The symptoms had gradually worsened, with increasing fatigue and muscular complaints, without any identified precipitating event. There was no history of intense physical exertion, trauma, alcohol consumption, or medication use, including statins or other potentially myotoxic drugs. The patient had no previous history of dermatological disease, and the onset of the cutaneous manifestations was recent.

History taking revealed the progressive development of marked skin dryness, initially perceived as unusual by the patient, followed by generalized scaling that gradually extended to most of the integument. Dermatological examination showed severe acquired ichthyosis, characterized by diffuse xerosis and thick polygonal scales, predominantly involving the extensor surfaces of the lower limbs (**Figure 1**). The cutaneous abnormalities were not associated with any previous personal or family history suggestive of inherited ichthyosis. On admission, the patient was hemodynamically stable, with a blood pressure of 130/70 mmHg, a body temperature of 37°C, and a heart rate of 54 beats/min. Electrocardiography confirmed sinus bradycardia. No peripheral edema was observed, and deep tendon reflexes were preserved. The clinical examination also revealed cognitive and psychomotor slowing.

Given the combination of diffuse acquired ichthyosiform scaling and prominent muscular symptoms, a systemic cause was considered. Laboratory investigations revealed a marked elevation of creatine phosphokinase (CPK), reaching 7245 IU/L, meeting the biochemical criteria for rhabdomyolysis. Serum creatinine was 16 mg/L (1.6 mg/dL), with an estimated glomerular filtration rate of 57 mL/min/1.73 m² and blood urea of 0.25 g/L. Electrolytes were within normal ranges, including sodium 140 mmol/L, potassium 4 mmol/L, and corrected calcium 96 mg/L (2.40 mmol/L). Urine culture was sterile, while myoglobin was not measured. AST and LDH were elevated at 131 IU/L and 708 IU/L, respectively, whereas ALT was 40 IU/L. The association with severe xerosis and ichthyosiform scaling further supported the possibility of an underlying systemic metabolic disorder.

Several potential alternative causes of rhabdomyolysis were evaluated. There was no history of intense physical exercise, trauma, alcohol consumption, or medication use. Infectious investigations were negative, and no significant electrolyte abnormality was identified. Antinuclear antibodies were negative, and the myositis-specific antibody panel was negative, arguing against an underlying inflammatory myopathy. C-reactive protein was 1, the complete blood count was normal, and no other precipitating factor for rhabdomyolysis was identified. The association with severe xerosis and ichthyosiform scaling further supported the possibility of an underlying systemic metabolic disorder.

Thyroid function testing demonstrated profound hypothyroidism, with thyroid-stimulating hormone (TSH) levels greater than 1000 mIU/L and markedly decreased free thyroxine (FT4) at 0.45 ng/dL. The autoimmune etiology was confirmed by strongly positive anti-thyroid peroxidase antibodies (>1200 IU/mL) and anti-thyroglobulin antibodies (300 IU/mL), establishing the diagnosis of severe autoimmune hypothyroidism due to Hashimoto thyroiditis.

Alternative systemic causes of acquired ichthyosis were also investigated. A cervico-thoraco-abdomino-pelvic CT scan did not reveal any underlying malignant process. Nutritional deficiencies were not identified, with normal vitamin B12, folate, and ferritin levels. The complete blood count was normal, serum albumin was 38 g/L, and serum protein electrophoresis was normal. Infectious investigations were negative. The patient reported no previous medication use, making a drug-induced acquired ichthyosis unlikely.

In the absence of another identified precipitating factor, the muscular involvement was attributed to severe thyroid hormone deficiency. Similarly, the acquired ichthyosis was considered a cutaneous manifestation of the underlying hypothyroid state. Thus, the simultaneous occurrence of acquired ichthyosis and rhabdomyolysis represented the two main peripheral manifestations leading to the diagnosis of previously unrecognized profound hypothyroidism.

Levothyroxine replacement therapy was initiated at a dose of 50 µg/day. The patient's myalgia gradually improved, with clinical improvement occurring within the first two weeks following treatment initiation. CPK levels progressively decreased from 7245 IU/L at admission to 937 IU/L at month 1 and 166 IU/L at month 2. Thyroid function also progressively improved, with TSH decreasing from > 1000 mIU/L at admission to 142.81 mIU/L at month 1, 77 mIU/L at month 2, 1.62 mIU/L at month 6, and 1.46 mIU/L at month 9. In parallel, a marked regression of the cutaneous abnormalities was observed, with improvement in xerosis and ichthyosiform scaling. The ichthyosis progressively regressed and had disappeared by approximately two months, in the context of thyroid hormone replacement and regular emollient therapy. The parallel improvement of both muscular and cutaneous manifestations following thyroid hormone replacement provided further support for their relationship with the severe hypothyroid state.

Close clinical and biological monitoring was maintained during thyroid hormone replacement, with progressive correction of the severe and longstanding hypothyroidism. Renal function also progressively improved, with serum creatinine decreasing from 16 mg/L at admission to 13 mg/L at month 1, 12 mg/L at month 2, and 11 mg/L at month 9. No worsening of renal function was observed during follow-up. The favorable evolution following levothyroxine therapy, together with the absence of an alternative precipitating factor for the rhabdomyolysis and the absence of a previous dermatological history, strongly supported the interpretation of acquired ichthyosis and muscle involvement as reversible systemic manifestations of profound autoimmune hypothyroidism.



Figure 1. Severe xerosis of the lower limbs with mosaic-pattern scaling suggestive of acquired ichthyosis.

3. Discussion

Acquired ichthyosis is an uncommon dermatological condition characterized by generalized scaling resembling inherited ichthyosis but occurring in adulthood without a prior personal or family history. Unlike congenital forms, acquired ichthyosis is frequently associated with systemic diseases, including malignancies, infectious disorders, autoimmune conditions, metabolic abnormalities, and endocrine diseases [1] [2]. Among endocrine causes, hypothyroidism remains an infrequent but well-recognized etiology, accounting for only a small minority of reported acquired ichthyosis cases in published series, most of which describe isolated cutaneous involvement without concomitant myopathy.

Cutaneous manifestations are common in hypothyroidism and result primarily from reduced epidermal turnover, impaired eccrine gland secretion, and altered lipid metabolism [3] [4]. Xerosis is a frequent finding; however, progression to clinically significant acquired ichthyosis remains uncommon [5]. Thyroid hormone deficiency alters keratinocyte proliferation and differentiation, leading to abnormal keratinization and excessive accumulation of scales [3]. Thyroid hormones act directly on keratinocytes and dermal fibroblasts, promoting normal epidermal turnover, sebaceous and eccrine gland activity, and dermal glycosaminoglycan turnover [4]. Their deficiency favors dermal accumulation of hyaluronic acid and other mucopolysaccharides, which contributes to the classic doughy, myxedematous skin texture of hypothyroidism. In severe and prolonged hypothyroidism, these cutaneous alterations may also be associated with marked xerosis and, rarely, acquired ichthyosiform scaling, although the precise mechanisms underlying this manifestation remain incompletely understood [6] [7].

Muscular involvement is another recognized manifestation of hypothyroidism, ranging from mild myalgia to severe rhabdomyolysis [8]. Elevated creatine phosphokinase levels are frequently reported in hypothyroid myopathy, although extreme elevations such as those observed in our patient are uncommon [9]. The mechanisms underlying hypothyroidism-induced rhabdomyolysis are not fully understood but may involve impaired glycogenolysis, mitochondrial dysfunction, and altered muscle energy metabolism secondary to thyroid hormone deficiency [10]. Clinically, hypothyroid myopathy encompasses a spectrum ranging from the pseudohypertrophic form described in adults, known as Hoffmann syndrome, to its pediatric counterpart, Kocher-Debré-Sémélaigne syndrome, both characterized by muscle weakness, stiffness, and muscle enlargement in the setting of elevated CPK levels. However, CPK elevations of the magnitude observed in our patient (7245 IU/L) are distinctly uncommon in conventional hypothyroid myopathy, in which CPK levels are usually only mildly to moderately elevated. [11] Although marked CPK elevations have been reported in severe hypothyroidism, they may clinically and biochemically mimic inflammatory myopathies. Rhabdomyolysis remains an uncommon but recognized manifestation of severe hypothyroidism [12]. Importantly, no alternative precipitating factor such as trauma, strenuous exercise, or myotoxic medication was identified in our case, and the temporal correlation between biochemical improvement and levothyroxine initiation further supported a causal rather than coincidental relationship.

The coexistence of acquired ichthyosis and rhabdomyolysis as revealing manifestations of severe autoimmune hypothyroidism is particularly unusual and constitutes the main originality of this observation. This association may lead to delayed diagnosis because cutaneous and muscular manifestations are often considered separately rather than as part of a unifying endocrine disorder. Both manifestations likely share a common pathophysiological substrate, namely a generalized reduction in cellular metabolic rate and impaired turnover of structural proteins and lipids, which affects the skin and skeletal muscle simultaneously, albeit

through organ-specific mechanisms. However, the precise mechanisms underlying their simultaneous occurrence remain incompletely understood.

Early recognition of hypothyroidism-related systemic manifestations is essential, as appropriate levothyroxine replacement therapy usually leads to significant clinical and biochemical improvement [13]. In our patient, levothyroxine therapy resulted in normalization of CPK levels and marked regression of the cutaneous lesions, further supporting a causal relationship between severe thyroid hormone deficiency and both manifestations [4]. This case underlines the importance of including thyroid function assessment in the etiological work-up of acquired ichthyosis, particularly when associated with muscular symptoms or elevated muscle enzymes. This report has inherent limitations related to its single-case design, which precludes definitive conclusions regarding the true incidence of this association and limits generalizability. Nevertheless, the marked biochemical and clinical improvement following levothyroxine therapy, together with the absence of alternative explanatory factors, lends strong support to a causal interpretation. Larger case series or systematic reviews would be valuable to better characterize the frequency, risk factors, and optimal management of this uncommon dermatomuscular presentation of hypothyroidism. From a practical standpoint, the presence of unexplained xerosis or ichthyosiform scaling associated with myalgia or elevated muscle enzymes should prompt clinicians to include thyroid function testing (TSH and free T4) in the initial diagnostic work-up. This screening is inexpensive, widely available, and can prevent substantial diagnostic delay, given that both the cutaneous and muscular manifestations described here are, unlike many other causes of ichthyosis or rhabdomyolysis, fully reversible under appropriate hormone replacement therapy. Serial reassessment of CPK levels and skin findings after initiation of levothyroxine can further help retrospectively confirm the causal link when the initial presentation is ambiguous or when several potential etiologies coexist.

4. Conclusion

This case illustrates an unusual presentation of severe autoimmune hypothyroidism revealed by the coexistence of acquired ichthyosis and rhabdomyolysis. Although both manifestations are recognized complications of thyroid hormone deficiency, their simultaneous occurrence is exceptional and may delay diagnosis. Clinicians should consider thyroid dysfunction in the diagnostic work-up of acquired ichthyosis, particularly when associated with muscular symptoms or marked creatine phosphokinase elevation, and conversely should screen for cutaneous involvement in patients presenting with unexplained rhabdomyolysis. Early diagnosis and prompt levothyroxine replacement therapy can result in complete clinical and biochemical recovery. A high index of suspicion, systematic thyroid function testing, and timely hormone replacement remain key to preventing diagnostic delay and the potentially severe complications of unrecognized myxedema.

Patient Consent

Written informed consent was obtained from the patient for the publication of this case report and any accompanying data.

Author Contributions

All authors contributed to the management of the patient, the acquisition and interpretation of data, and the drafting and critical revision of the manuscript. All authors read and approved the final version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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