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REVIEW ARTICLE **OPEN ACCESS**

Unmasking hypothyroidism through skin: A case report on acquired ichthyosis in an adult

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ABSTRACT

A 26-year-old male presented to us with widespread dryness and ichthyotic scaling of whole of the skin, generalized weakness, difficulty in getting up from on squatting and accompanied with noticeable change in the voice. The clinical suspicion of hypothyroidism and polymyositis was confirmed by biochemistry reports. We considered it prudent to report this case for rekindling due to its rarity and association with myositis.

***Key words**: Hypothyroidism; Acquired Ichthyosis; Myopathy; L-thyroxine*

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INTRODUCTION

Ichthyosis, deriving its name from clinical resemblance to scales of a fish, is a disorder of keratinization with dry, rough, scaly skin. More commonly, its mode of transmission is hereditary presenting in early childhood in various syndromic and non-syndromic forms.[1]However, acquired form of ichthyosis may occur in adults suggesting an autoimmune, inflammatory, infective, malignant, endocrinal or drug related cause.[2] Acquired ichthyosis (AI) is a consequence of impaired homeostasis of the most superficial layer of epidermis, i.e., stratum corneum. It is hard to distinguish AI from the congenital form, ichthyosis vulgaris (IV), both on clinical and histopathological grounds [1].

CASE REPORT

A 26-year-old male, labourer by occupation, came to our Dermatology out patient department with complaints of dryness and scaling of whole of the skin, increased skin thickness of the legs, slight gain in weight, constipation without passage of blood and generalized weakness for more than one and a half years. He easily fatigued on his day to day work and it was very difficult for him to stand up from a sitting and squatting position. This led him to eventually leave his job. It was noted during history taking that the patient's speech was unusually slow and hoarse in nature. On enquiring, it was found that the change in voice began simultaneously with the onset of aforementioned complaints. He had no phototoxicity. The patient didn't report similar complaints in other family members. He reported to not have suffered from any symptoms suggestive of diabetes mellitus, hypertension, tuberculosis, leprosy. He has neither received nor was not on any medications, namely, allopurinol, lipid lowering agents or anticancer therapy.

The examination revealed a young, sedate man with puffy face. His voice was hoarse, skin pale, dry, edematous, waxy looking, scaly, and devoid of hair. Scalp hair were coarse and lusterless along with the finger nails. The scales were brownish and adherent, present on non erythematous skin present all over the body except antecubital fossae, popliteal fossae and axillae (Figure 1a and 1b). The edema involving the lower legs was non pitting (Figure 2a and 2b); the skin was cold to touch. The tongue appeared swollen and the palpebral conjunctiva was pale.

The pulse was 56 per minute, regular and low in volume; oral temperature 36°C and blood pressure 130/100 mm of Hg with no lymphadenopathy or hepato-splenomegaly. All the deep tendon reflexes were slow to relax. On routine laboratory investigations, hemoglobin was 9.1 gm/dl (14-18 g/dl) with normocytic anemia, serum direct bilirubin 0.6 mg/dl (less than 0.3 mg/dl), alanine aminotransferase (ALT) 82 IU/L (0 to 45 IU/L) aspartate aminotransferase (AST) 62 IU/L (0 to 35 IU/L), gamma glutamyl transferase 37 U/L (5- 40 U/L) and alkaline phosphatase 82 IU/L (30-120 IU/L). Thyroid stimulating hormone (TSH) was 480 µIU/ml (0.35 to 4.5 mIU) while free thyroxine (T4) and triiodothyronine (T3) were undetectable and antithyroglobulin antibodies were not detected. Antinuclear antibodies (ANA) and rheuma-

toid factor were negative. Serum lipid profile showed serum total cholesterol as 162.2 mg/dl (125 to 200 mg/dl) done by CHOD-PAP method, serum triglyceride levels as 531.5 mg/dl (less than 150 mg/dl) done by GPO- trinder method, serum low density lipoproteins (LDL) as 107.10 mg/dl (less than 100 mg/dl), serum high density lipoproteins (HDL) as 43.7 mg/dl (40 mg/dl or higher) done by direct measure PEGME/CHOD method. Serum muscle enzymes showed LDH (lactate dehydrogenase) 327 U/L (140 to 280 U/L) and serum creatinine kinase (CPK) as 276 U/L (55 to 170 U/L). Electromyography (EMG) was normal and no abnormality was detected on ultrasonography abdomen. Skin biopsy was not performed due to lack of consent. A clinical diagnosis of hypothyroidism leading to secondary ichthyosis and myositis was made. The patient was sent to the endocrinologist and started levothyroxine in graded dosing with topical moisturizers and general advice on skincare.

DISCUSSION

A patient of severe endocrinological dysfunction with dermatological symptoms attending the skin OPD is a rare occurrence. Hence, suspecting and diagnosing such subjects is of immense importance. One such young patient who developed generalized, dry ichthyotic scaling, and mild edema of the body, severe laziness, weakness and hoarseness of voice, came to us and was diagnosed with severe thyroid deficiency without any underlying cause, and this likely to be autoimmune. Though, antithyroglobulin antibodies were not detected, this positivity is not linked to the diagnosis of autoimmune thyroiditis.

Though acquired ichthyosis has been linked to malignancies namely, Hodgkin's lymphoma, multiple myeloma, cutaneous T cell lymphoma, drugs like allopurinol, cholesterol-lowering drugs, anticancer agents, and autoimmune disorders such as systemic lupus erythematosus, celiac disease, Crohn's disease,[1, 2] this patient had none of the underlying factors.

Ichthyosis at birth or shortly after is most frequently hereditary.[1] Interestingly, Valeria Brazzelli et al[3, 4] in 2005 and 2010 reported cases of acquired ichthyosis in the pediatric age group to autoimmune thyroiditis resolving successfully on supplementation with levothyroxine.Table 1 draws a subtle comparison between ichthyosis vulgaris, the hereditary form, and acquired ichthyosis to hypothyroidism to reach a correct diagnosis, especially at this age [1,3-7]. The significance of the early diagnosis of hypothyroidism in infancy lies in preventing the consequences of associated neurological morbidity [6].

Deranged liver function and lipid profile as seen in our patient may be associated with abnormal thyroid function, as reported by J. Delaleu et al[7]The activity of LDL (low-density lipoprotein) receptors is also decreased resulting in its decreased clearance. Thyroid hormone plays a role in fatty acid metabolism and cholesterol biosynthesis by induction of the key enzyme, 3-hydroxy-3- methylglutaryl CoA (HMG CoA) reductase.Therefore, thyroxine deficiency may result in altered epidermal lipid metabolism and reduced levels of fatty acids, phospholipids, and sterols in the skin, required to maintain the cutaneous barrier function [4, 7, 8].

The derangement in liver function as indicated by the raised hepatic enzymes may also be responsible for disturbed hepatic lipid metabolism leading to hepatic steatosis. As seen in our patient, high CPK and LDH may indicate hypothyroidism-induced myopathy. Even though the patient didn't have fatty liver, a significant association has been found between hypothyroidism and non-alcoholic fatty liver disease (NAFLD) by Piantanida et al [9] It has been ascribed to dyslipidemia, intrahepatic accumulation of triglycerides, insulin resistance, hepatic inflammation, and fibrosis.

In most cases, the clinical features of hypothyroidism are non-specific, delaying the diagnosis and depriving the patients of effective, economical, and readily available treatment. Supplementation with L-thyroxine has been shown to resolve systemic and cutaneous manifestations of hypothyroidism over six months [7]. Lause et al, [6] in their review on the skin in endocrine disorders in 2017, recommend monitoring of TSH at 6-week intervals to alter the dosing according to the therapeutic response.

An observant eye to the rare dermatological features accompanying the systemic symptoms of malaise, intolerance to cold, and weight gain with normal appetite may help physicians settle the clinical doubt of hypothyroidism.[10]Whether the ichthyotic skin changes occur only secondary to hypothyroidism or autoimmunity also contributes to its development has to be discerned and discerped.

CONCLUSION

Where the mainstay of treatment of hereditary ichthyosis aims at hydration, exfoliation and prevention of secondary infections and eczema with spontaneous resolution over age in most, it is essential to note that acquired ichthyosis may keep failing general topical management if the underlying etiology remains to be unidentified.

Table 1: Differences between hereditary (IV) and acquired ichthyosis (in hypothyroidism)

Characteristics	Ichthyosis Vulgaris[1, 5]	Acquired Ichthyosis to Hypothyroidism[3, 4, 6, 7]
Mode of transmission	Hereditary	Non-hereditary
Pathophysiology	Gene mutation: Loss of function of profilaggrin/filaggrin	Impaired epidermal lipid metabolism and/or presence of anti profilaggrin antibodies
Onset	In the early months of life	Adulthood
Skin involvement	Chest and abdomen are not commonly involved	Most parts of the body
Associated cutaneous features	Atopic dermatitis; palmer hyper linearity; keratosis pilaris	Xerotic, cold skin with mottling; palmoplantar keratoderma; yellowish discoloration of the skin (carotenemia); localized and/or generalized edema; macroglossia; brittle hair and nails
Investigations	Serum IgE levels may be raised.	Abnormalities in thyroid function test. Anti-thyroid peroxidase and antithyroglobulin antibodies may be detected.
Treatment	Topical moisturizers, topical retinoids, and alpha hydroxy acid-based preparations.	Hormone replacement therapy with L- thyroxine.

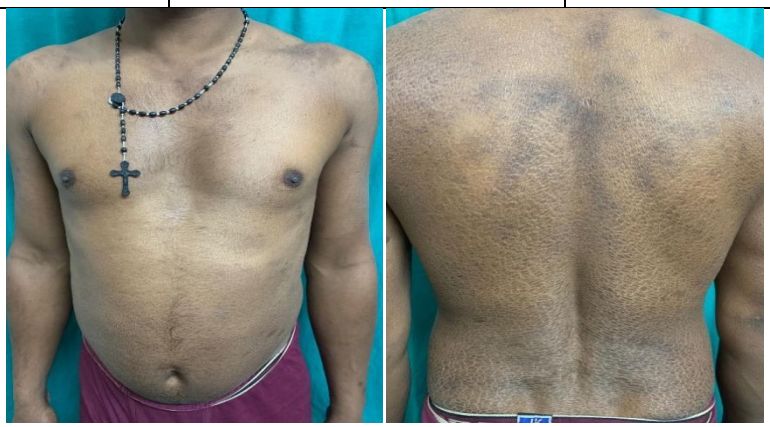

Figure 1 (a) and 1 (b): Fish like scales with involvement of the trunk

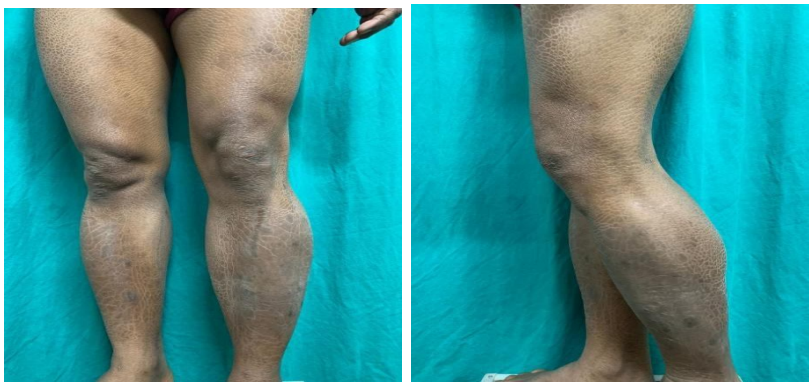

Figure 2 (a) and 2 (b): Pretibial myxedema

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