A Case of Guttate Psoriasis in Iodine Induced Hypothyroidism Patient

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ABSTRACT

Psoriasis is a chronic inflammatory skin disorder that has been increasingly associated with autoimmune comorbidities, including thyroid dysfunction. Guttate psoriasis, a less common variant, is often triggered by infections or medications but rarely by endocrine disorders. Hypothyroidism may play a role in exacerbation in predisposed individual. A 29-year-old male with history of thyrotoxicosis treated with iodine radiation presented with a two-month history of pruritic, erythematous, scaly skin changes over his body. The rash appeared 3 weeks after the patient discontinued Levothyroxine therapy. Clinical examination and biopsy confirmed guttate psoriasis. Laboratory investigations revealed elevated TSH, high thyroglobulin and thyroid peroxidase antibodies, with negative infectious and autoimmune markers. Initial treatment with topical agents and narrowband UVB therapy showed limited response. Methotrexate was later initiated, resulted in significant improvement. Clinician should consider thyroid dysfunction as a possible trigger for guttate psoriasis, especially in patient with history of autoimmune thyroid disease. Early recognition and management are essential for optimal outcome.

Keywords: Guttate psoriasis, hypothyroidism, iodine therapy, autoimmune disease.

INTRODUCTION

Guttate Psoriasis is a distinct clinical variant of psoriasis characterized by the acute onset of multiple, small droplet-shaped erythematous papules, often accompanied by scaling. This condition typically occur in younger individuals and is frequently triggered by infections, mostly streptococcal pharyngitis. While the exact pathogenesis remains unclear, guttate psoriasis is believed to result from an interplay of genetic predisposition, immune dysregulation and environmental factors. It accounts for approximately 2% of all psoriasis cases.

This case presents a rare case of guttate psoriasis associated with iodine induced hypothyroidism, highlighting an unusual trigger and emphasizing the need for awareness of atypical presentation in clinical practice.

THE CASE

A 29-year-old male known case of iodine induced hypothyroidisim, presented to the clinic complaining of a rash that began 2 months ago. He first noticed it on his elbows and knees before it spread to involve his trunk and abdomen. The rash is painless but pruritic, affecting his sleep. It spares his palms and oral cavity. The patient denied having sore throat, fever, recent travel, sexual contact or the use of new medications before the eruption of the rash. His past medical history included thyrotoxicosis, which was treated with Iodine radiation leading to a hypothyroid state thereafter. He was prescribed lifelong thyroid replacement therapy with Levothyroxine but was not compliant. He discontinued the medication on his own 3 weeks before the onset of the rash. Additionally, his 55-year-old mother had been diagnosed with Psoriasis 1 year ago .

On clinical examination, patient was hemodynamically stable and not in acute distress. Dermatological findings revealed scattered erythematous papules and small plaques with silvery scales on some lesions, distributed over the trunk, elbows and knees. A scalp exam showed hyperkeratotic plaque over occipital region. The nails were normal. There was no evidence of lymphadenopathy or signs of secondary bacterial infection. Examination of the oropharynx was unremarkable with no peritonsilar erythema or exudates.

A few investigations were ordered to determine the etiology of the lesions. A throat swap was negative for streptococcus. Serology tests for HIV and syphilis were also negative. ANA CTD and Anti-dsDNA were negative. TFT results was as follows: TSH 33.4 (high), FT3 3.1, FT4 14.2. Thyroglobulin antibodies were 4000 (high), and thyroid peroxidase antibodies were 600 (high). Skin biopsy shows hyperkeratosis, lose of angular layer, intraepithelial neutrophilic infiltrate, elongation of rete ridges, perivascular lymphocytic infiltrate with no fungal elements.

Based on the clinical findings and investigations done patient was diagnosed with guttate psoriasis. Patient agreed to start on topical corticosteroid and topical calcipotriol, in addition to narrow band UVB therapy. The initial dose of UVB was 350 mJ and was gradually increased to reach a target dose of 1600 mJ. After multiple sessions of UVB, the patient became depressed as his condition did not show significant improvement. A decision was made to discontinue UVB therapy and initiate methotrexate at a dose of 10 mg weekly was taken while continuing the other topical medications. Once the new medication was started, the patient showed an excellent response and his lesions began to heal. Currently, the patient is following up in dermatology clinic until full remission of his condition is achieved.

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DISCUSSION

Psoriasis is a chronic, inflammatory, autoimmune, multisystem condition affecting up to 2% of the population[1,2]. While it predominantly impacts the skin, it can also involve the nails and joints. Psoriasis often co-exists with other autoimmune conditions. Such as inflammatory bowel disease, systemic lupus erythematosis and thyroiditis[3]. There are various types of psoriasis, including plaque psoriasis, postural psoriasis, erythrodermic psoriasis and guttate psoriasis. Plaque psoriasis is the most common type, accounting for 90% of cases[4]. It is commonly recognised by skin lesions characterized by round erythematous plaques with loosely adherent silvery scales over the extensor surfaces[5].

In contrast, guttate psoriasis is a rare variant, comprising approximately 2% of all psoriasis patients[1,4]. While both types may co-occur with autoimmune conditions and affect individuals with genetic predisposition, guttate psoriasis differs in clinical presentation and triggering factors. It usually affects a younger population and is marked by sudden eruption of small, drop-shaped erythematous papules, typically following infections. Most notable infection is streptococcal pharyngitis.

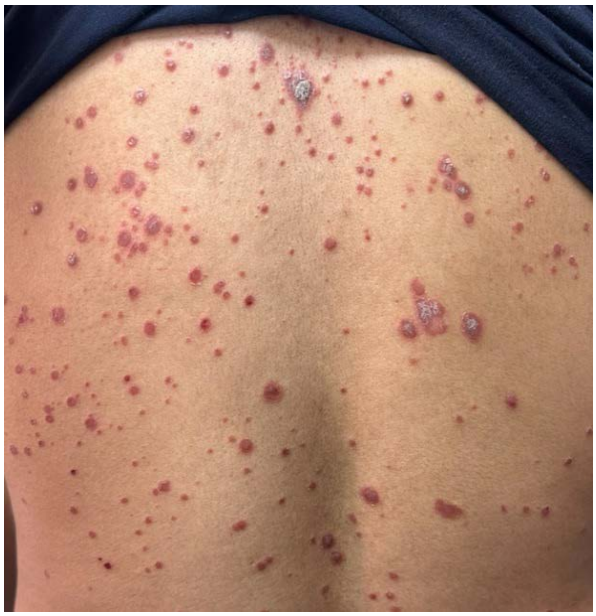
Thyroid hormones play a critical role in maintaining normal skin structure and function. Since guttate psoriasis has multiple predisposing factors, the role of hypothyroidism remains unclear[6,7]. Patients with hypothyroidism often experience dry, rough, and scaly skin, which, creates a fertile environment for the development of psoriasis when accompanied by other factors like genetic predisposition[6].

The diagnosis of psoriasis in general is made clinically by history and clinical exam which includes shapes of the lesions and distribution (Table 1). In this case, however, a skin biopsy was performed to confirm the diagnosis. Since streptococcal infection is known to be the commonest trigger factor, anti streptolysin O is usually requested. Additionally, guttate psoriasis can happen in immunocompromised patients and autoimmune disorders[3]; therefore, serology and ANA CTD preferably requested[3].

The acute presentation of guttate psoriasis characterized by small, erythematous papules over the trunk and proximal extremities is often preceded by a streptococcal infection[1]. Given the overlap with other papulosquamous disorders, skin biopsy is the definite tool to confirm the diagnosis. In this case, the histological findings were consistent with psoriasis.

The treatment of guttate psoriasis involves managing symptoms, addressing triggers, and preventing recurrences. A key component is identifying the underlying causes, such as streptococcal infection, rhematological disease or thyroid disease. The first line treatment is topical treatments, including corticosteroids, vitamin D analogues (e.g., calcipotriol), coal tar and emollients, are used to alleviate inflammation, scaling and dryness. For widespread cases, UVB phototherapy is an effective option. In more severe cases or reluctant to the previous modalities, methotrexate or biological agents (e.g., guselkumab, ixekizumab) can be used[5].

The outcomes of guttate psoriasis are variable. Many patients experience complete remission after treatment of the underlying cause. However, a subset of individuals may experience recurrent episodes or progression to chronic plaque psoriasis, especially if underlying triggers persist or are not addressed[1].



Figures 1 and 2. Scattered Erythematous Papules and Plaques with Silvery Scales on some Lesions

Table 1. Prevalence and characteristics of different types of psoriasis[1,4,5]

Type of psoriasis	Prevalence	Skin characteristics
Plaque psoriasis	80-90%	Well demarcated erythematous plaques with silvery scales, typically on scalp, elbows, knees and lower back
Guttate psoriasis	2%	Small drop-shaped papules with fine scales in trunk and upper extremities
Pustural psoriasis	5%	White pustules surrounded by red skin usually in the hands and feet
Erythrodermic Psoriasis	<1%	Sever redness and shedding of skin over large areas

CONCLUSION

Guttate psoriasis is a rare type of psoriasis that often presents abruptly, typically following streptococcal pharyngitis. However, many conditions can also trigger it like thyroid diseases and SLE[8,9,10]. Early recognition of the cause and prompt management are critical for favourable outcome and permanent resolution.

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