A Case of Localized Psoriasis Following Treatment with Iodine$^{131}$ for Hyperthyroidism: A Rare Entity

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ABSTRACT

Psoriasis is a T-cell mediated inflammatory skin disease, characterized by circumscribed erythematous plaques, covered by silvery micaceous scales. It is known to be triggered by a number of factors including drugs. Drugs may either cause de novo psoriasis or responsible for aggravating preexisting psoriasis. The morphological types can vary from localized or generalized plaque psoriasis to pustular psoriasis and even erythroderma. In this work, we report the case of a 45-year-old male patient who developed localized psoriasis following treatment with iodine$^{131}$ for hyperthyroidism. Based on history, clinical pictures, localized examination and histopathological findings with psoriasiform changes favoured the diagnosis. He was treated with topical steroid with clobetasol propionate (0.05%) and salicylic acid (6%) combination for 1 month following which lesions resolved. We suspected that the mechanism behind this is due to activation of dihydrofolate reductase by radioactive iodine. Further study about the folic acid pathway in psoriasis and the connection between radioactive iodine with psoriasis may be necessary.

Keywords: Localized psoriasis, Radioactive iodine$^{131}$, Hyperthyroidism

Introduction

Psoriasis is a T-cell mediated inflammatory skin disease, characterized by circumscribed, erythematous well-defined plaques covered by silvery micaceous scales. Radioactive iodine ($^{131}$I) is an important isotope used in hyperthyroidism (Graves’ disease, Toxic multinodular goiter and autonomously functioning thyroid nodule). The most significant cutaneous adverse effect of $^{131}$I is iododerma. Here we report a case of localized psoriasis following treatment with radioactive iodine for hyperthyroidism.

Case Report

A 45 years old male shopkeeper by occupation attended skin OPD with itchy plaque over dorsum of feet which was gradually increasing for last 1 month. He is a known case of thyrotoxicosis for which he was on oral carbimazole for last 6 years and not responding and recently treated with 9.2 mili curie of oral radioiodine$^{131}$. He developed erythematous papular eruption over dorsum of both feet after 20 days of $^{131}$I therapy and it is slowly increasing in size. On examination symmetrical hyperkeratotic plaque was present over dorsum of both feet (Figure 1). Auspitz’s sign was positive. Oral radioiodine therapy had been stopped. No others cutaneous or mucosal sites were involved. He had no history of similar episode in the past. General, systemic and routine blood examinations were unremarkable other than thyroid profile. Histopathology showed hyperkeratosis, parakeratosis acanthosis, suprapapillary thinning, club shaped rete ridges and collection of neutrophils in stratum corneum (Munro’s microabscess) and in dermis there was dilated blood vessels and perivascular lymphocytic infiltrates in papillary dermis (Figures 2, 3). A final diagnosis of $^{131}$I induced localized plaque psoriasis was made. Patients was treated with topical steroid with clobetasol propionate (0.05%) and salicylic acid (6%)
combination for 1 month following which lesions resolved and there was only hyper pigmentation (Figure 4). Radioiodine therapy had not been restarted and he had been started on oral methimazole. Informed consent has been taken from the patient.

**Discussion**

Psoriasis is a T-cell mediated common, chronic and recurrent inflammatory skin disease, characterized by circumscribed, erythematous sharply demarcated papules and plaques covered by silvery micaceous scales. Many factors contribute its pathogenesis like genetic, immunological and environmental factors. Several other factors like trauma, stress, infections and medications might exacerbate psoriasis [1]. Radioactive $^{131}$ is an important isotope used in hyperthyroidism (Graves’ disease, Toxic multinodular goiter and autonomously functioning thyroid nodule). Cutaneous adverse effect of $^{131}$ include iododerma, which is characterized by acneiform eruption with inflammatory follicular pustules, or may present as urticaria or bullous lesion with ulceration and crust. Iododerma may occurs in the face, neck, extremities and trunk [2]. There have been many drugs documented to directly trigger the eruption of psoriasis (antibiotics, non-steroidal anti-inflammatory drugs, angiotensin-converting enzyme inhibitors, interferon, amiodarone, etc.)
terbinafine, benzodiazepines, digoxin, clonidine, quinidine, gold, potassium iodide, imiquimod etc.) and many others exacerbate the existing psoriasis (acetazolamide, aminoglutethimide, amiodarone, antibiotics, terbinafine, diltiazem, hydroxychloroquine, lithium, potassium iodide, propanol etc.) [3,4]. It is very difficult to explain the exact mechanism, they may affect the psoriatic process at different stages but with same results. It has been seen that iodine/iodides specifically activate the enzyme dihydrofolate reductase and Kang and Kim [5] reported a case of psoriasis exacerbated by radioactive iodine therapy. We suspected that the mechanism behind this, is due to activation of dihydrofolate reductase through radioactive iodine as there is no other significant drug history. Further study about the folic acid pathway in psoriasis and the connection between radioactive iodine with psoriasis may be necessary. This article has been presented to highlight the rarity of such condition.

**Ethics**

**Informed Consent:** Informed consent has been taken from the patient.

**Peer-review:** Internally peer-reviewed.

**Authorship Contributions**


**Conflict of Interest:** No conflict of interest was declared by the authors.

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**References**