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Primary Hypoparathyroidism associated with chronic plaque psoriasis; A case report & review of literature

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Introduction

Psoriasis is a chronic autoimmune dermatosis characterized by the formation of inflammatory plaques on the skin. Calcium and vitamin D play important roles in the pathogenesis of psoriasis. Calcium regulates keratinocyte differentiation, proliferation, and barrier function, while vitamin D deficiency is associated with increased inflammation and severity of psoriasis [1-3]. Hypoparathyroidism, a condition causing low calcium levels, can exacerbate psoriasis by further disrupting calcium homeostasis and depleting active vitamin D. The coexistence of hypoparathyroidism in psoriasis patients has a negative impact on the disease. Understanding the relationship between calcium, vitamin D, and psoriasis can help in managing the condition effectively.

Case Presentation

A 67-year-old female with 3-year history of chronic plaque psoriasis admitted due to worsening of the cutaneous lesions over two weeks. She was treated as an exacerbation of chronic plaque psoriasis with weekly intramuscular methotrexate, oral antihistamines, cetrimide shampoo and local application of steroids mixed with aqueous cream. However, the response to the treatment was unsatisfactory. During her hospital stay, she experienced a generalized tonic-clonic seizure, which was found to be associated with a critically low serum ionized calcium level of 0.26 mmol/L (1.15 – 1.33). Interestingly, we could elicit some chronic symptoms attributable to long-standing hypocalcaemia such as vague muscular pains and cramps for over a decade, in her past history.

She exhibited positive Trousseau's sign and Chvostek's sign. The electrocardiogram (ECG) showed prolonged QT interval (Figure 1). Her serum phosphate level was 2.3 mmol/L (0.97- 1.45). The optimally measured serum intact parathyroid hormone (PTH) level was 4.22 pg/mL (15-75). Furthermore, her brain CT showed widespread intracranial calcifications, supporting the chronicity of hypocalcaemia (Figure 2). So, in the presence of low calcium, high phosphate and low PTH, the diagnosis of hypoparathyroidism was established. In addition, she had marginally low serum magnesium level which was 0. 6mmol/L (0.65-1.05). And she was vitamin D deficient, with the 25-hydroxyvitamin D level being 13.07 ng/mL (less than 20).

Her hypomagnesemia was not severe enough to cause PTH secretory defect [4]. This patient did not have a family history of hypoparathyroidism. She had not undergone any neck surgeries. Other aetiologies for hypoparathyroidism like infiltrative (iron overload and sarcoidosis) were excluded. So, autoimmune-mediated primary hypoparathyroidism was suspected, but specific antibody testing was not available to confirm the autoimmunity. Absence of coexisting adrenal insufficiency and hypothyroidism excluded the possibility of polyglandular autoimmune syndromes.

The initial severe hypocalcaemia was managed with intravenous calcium gluconate followed by oral alfacalcidol 0.5 μg thrice a day (tds) and calcium carbonate 1.5 g tds to maintain serum calcium levels. Oral magnesium citrate (800 mg tds) and vitamin D replacement (vitamin D3 60000 IU weekly for 8 weeks followed by 2000 IU daily for 1 month) were also administered. The patient's serum ionized

calcium gradually increased and a week after normalizing calcium status, her cutaneous lesions showed a remarkable response to the same anti-psoriatic medications (Figure 3). She was discharged after improvement of clinical symptoms and stabilized calcium levels with oral calcium carbonate 1 g twice a day (bd) and alfacalcidol 0.5 µg bd. Follow-up with an endocrinologist was scheduled, and dietary recommendations were provided.

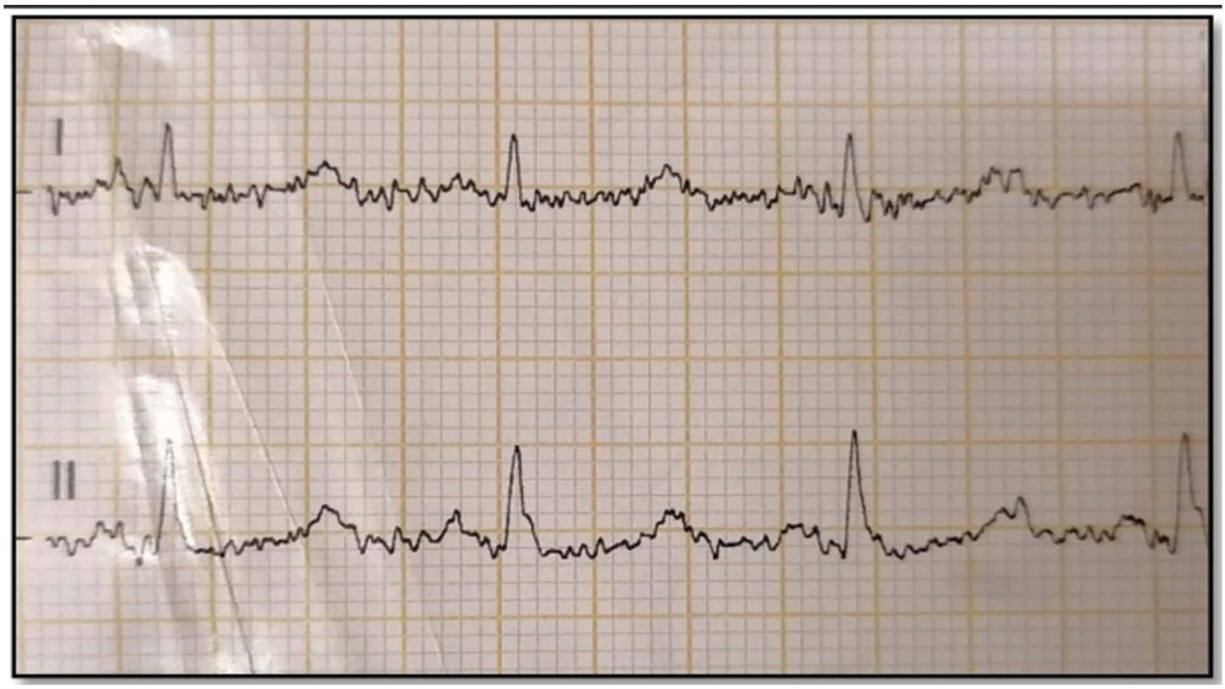

Figure 1 : Corrected QT = 519 msec (>440 msec)

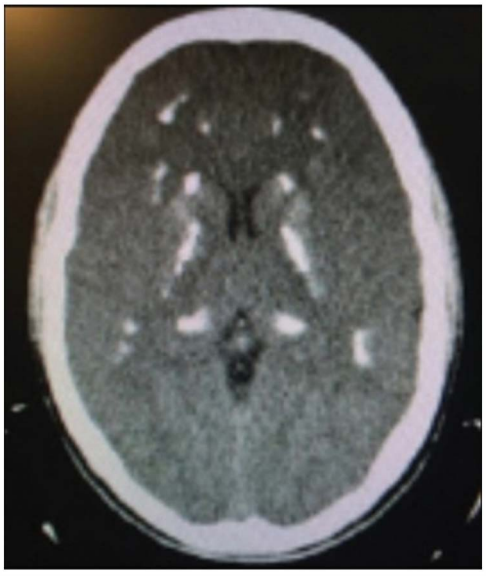

Figure 2 : Intracerebral calcifications

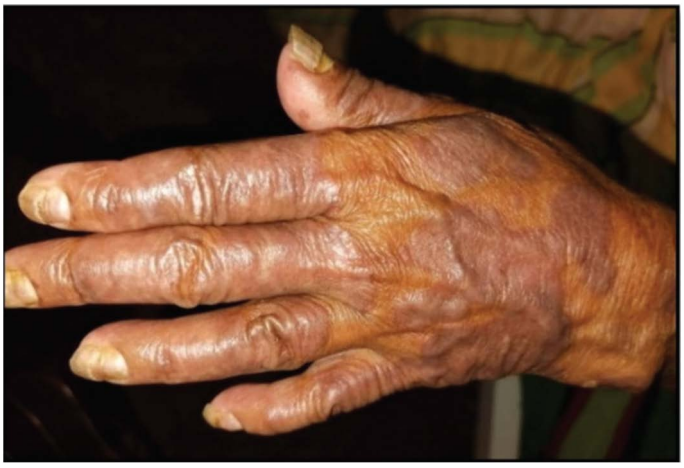

Figure 3 : Healed psoriatic lesions

Table 1

Case Report	Patient's age & sex	Sub-type of psoriasis	Temporal association of psoriasis to hypoparathyroidism	Etiology of primary hypopara-thyroidism	Response to calcium normalization
Vickers H R and Sneddon I B (5) - 1963	51 y, F 43 y, M	Extensive ? Plaque	2-3 weeks after 2 years after hypoparathyroidism	Postsurgical (following total thyroidectomy)	Improved in 3-6 weeks
Montegomery P R (6)- 1964	63 y, F	Not mentioned	1-2 months after hypoparathyroidism	Postsurgical	Great improvement
Risum G (7)- 1973	29 y, M	Pustular psoriasis	Psoriasis after hypoparathyroidism	Primary (? Idiopathic)	Improved within days
Stewart A F, et al. (8) – 1984	F	Pustular psoriasis	Psoriasis after hypoparathyroidism	Postsurgical	Complete resolution in 1-3 weeks
Tercedor J, et al. (9) - 1991	71 y, M	Pustular psoriasis	Inconclusive	Idiopathic	Not mentioned
Kawamura, et al. (10) - 1999	36 y, F	Pustular psoriasis	Psoriasis after hypoparathyroidism	Primary (? Idiopathic)	Complete resolution
Ashkevari S, et al. (3) - 2014	17 y, F	Pustular psoriasis	Psoriasis after hypoparathyroidism	Idiopathic	Complete resolution in 6 months
De Moura, et al. (11) - 2015	73 y, F	Pustular psoriasis	10 years after hypoparathyroidism	Postsurgical	Complete resolution
Rezayi A, et al. (12) - 2021	17 y, M	Plaque psoriasis	Hypoparathyroidism diagnosed 3 years after psoriasis diagnosis, but hypocalcaemic symptoms for 7 years prior to onset of psoriasis	Primary	Complete resolution in 1 month
Masson L, et al. (13) - 2021	28 y, M	Pustular psoriasis	Hypoparathyroidism diagnosed 1 year after psoriasis diagnosis	Primary	Complete resolution in 2 weeks

Discussion

We have discussed here a case of long-standing symptomatic hypocalcaemia due to primary hypoparathyroidism causing exacerbation of chronic plaque psoriasis. Table 1 presents a summary of the previously reported cases of psoriasis which got precipitated by the concurrent hypoparathyroidism.

All the previous cases, except two, were pustular psoriasis. So, this case becomes the third to report the association of plaque psoriasis with hypoparathyroidism. All of those case reports are highlighting the obvious negative influence of hypocalcaemia to the pathogenesis of psoriasis characterized by disordered keratinocyte differentiation and proliferation as well as disruption of cell adhesions caused by the depletion of calcium dependant cadherins. In our case, we would like to point out the contributory effect of vitamin D deficiency to the immunopathogenesis of psoriasis, in addition to the hypocalcaemia caused by the hypoparathyroidism.

Similar to all the previously reported cases, we also observed an excellent response of psoriatic lesions to the prompt normalization of calcium and vitamin D levels. So, correction of hypocalcaemia in patients with psoriasis, specially in pustular and plaque subtypes, can be identified as a key therapeutic measure in addition to the conventional anti-psoriatic medications.

Conclusions

There is an evident association between primary hypoparathyroidism and psoriasis. Prompt management of hypoparathyroidism and vitamin D deficiency with calcium and vitamin D supplementation resulted in regression of psoriatic lesions. Thus, we suggest considering hypocalcaemia as an underlying causative factor especially in cases of treatment resistant psoriasis.

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