
# Dermatology Reports

https://www.pagepress.org/journals/index.php/dr/index

**eISSN 2036-7406**





*Please cite this article as:*

*Vargu M, Dedej S, Vargu P, et al*. The role of calcium serum level in clinical manifestation and progress of psoriasis vulgaris in a patient with Fahr's syndrome. *Dermatol Rep 2025 [Epub Ahead of Print] doi: 10.4081/dr.2025.10614*





Submitted 18/09/25 - Accepted 21/10/25

# The role of calcium serum level in clinical manifestation and progress of psoriasis vulgaris in a patient with Fahr's syndrome

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**Key words:** psoriasis vulgaris; Fahr's syndrome; hypocalciemia; hypoparathyroidism; basal ganglia calcification; psychiatry.

**Contributions:** MV and PV contributed to the patient's diagnosis and management, and to data acquisition; SD, AN, and SV contributed to the writing, analysis, and interpretation of data; EK analyzed the pathology report; MV conceived and designed the study. All authors have read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

**Conflict of interest:** the authors have no conflict of interest to disclose.

**Ethics approval and consent to participate:** this case report has been approved by the Institutional Ethics Committee for publication. Informed consent was obtained from the patient included in this study.

**Consent for publication:** the patient gave his written consent to use his personal data for the publication of this case report and any accompanying images.

**Availability of data and materials:** the data presented in the manuscript are available from the corresponding author upon reasonable request.

**Abstract**

Fahr's syndrome is a rare entity characterized by symmetrical and bilateral non-atherosclerotic intracerebral calcifications in the basal ganglia. This entity is usually asymptomatic or may present mainly with neuro-neuropsychiatric and dermatological manifestations. Secondary bilateral striopallidodentate calcinosis (BSPDC) presentations are associated with various diseases. We describe a case of psoriasis vulgaris associated with secondary BSPDC due to hypoparathyroidism. Laboratory tests revealed hypocalcemia, hyperphosphatemia, and a low parathormone serum level; in cerebral computed tomography (CT), bilateral basal ganglia calcification was observed. Correction of calcium deficiency restored the calcium-phosphate balance and led to an improvement in psoriatic lesions. Psoriasis vulgaris is a common condition that may be associated with disturbances in calcium metabolism. Dermatologists should be aware that investigating cases resistant to conventional therapies can uncover underlying systemic disorders that significantly affect patients' quality of life.

**Introduction**

Bilateral striopallidodentate calcinosis (BSPDC) is a rare disorder characterized by symmetrical and bilateral intracerebral calcifications of the basal ganglia with or without deposits in the dentate nucleus, thalamus, and white matter. Radiological studies reported an incidence of basal ganglia calcifications ranging from 0.24% to 2%. Approximately 20% of patients remain asymptomatic until accidental diagnosis through neuroimaging.[1]

We present a rare case of Fahr's syndrome accompanied by psoriasis vulgaris diagnosed for the first time in our service. The significance of this report lies in highlighting the crucial role of dermatologists in investigating the triggering factors of psoriatic lesions resistant to conventional treatments. Furthermore, it emphasizes the importance of dermatological signs in the early detection of systemic diseases. Our study investigated the relationship between serum calcium levels and both the severity and treatment response of psoriasis vulgaris in patients exhibiting bilateral basal ganglia calcification, a hallmark of Fahr's syndrome.

**Case Report**

A 27-year-old male suffering from psoriasis vulgaris for several years was hospitalized in the Dermatology Department. Generalized erythro-squamous plaques were observed in the trunk and extremities. The patient was treated with methotrexate for a long time, but never reported complete remission. His personal history included behavioral disorders and mild mental retardation in childhood.

The family history was negative for psoriasis vulgaris. The routine biochemical and hematological investigations were normal, except for low calcium (5.07 mg/dL). Further biochemical tests were performed to identify the cause of hypocalcemia. The results are shown in Table 1.

The primary hypoparathyroidism diagnosis was confirmed based on laboratory findings and a negative history of congenital anomalies, thyroid surgery, neck radiation, and autoimmune disease. Chvostek's and Trousseau's signs were negative. The neck magnetic resonance imaging (MRI), electrocardiogram (ECG), abdominal echography, and ophthalmologic evaluation revealed no abnormalities.

Brain computed tomography (CT) showed symmetrical bilateral basal ganglia and focal parenchymal calcifications in the frontal lobes (Figure 1). Finally, Fahr's syndrome was diagnosed due to metabolic disorders (primary hypoparathyroidism).

The patient was treated with calcium supplements and emollients. After 1 month, an optimal serum calcium level and complete remission of the skin lesions were observed (Figure 2).

**Discussion**

Various metabolic, infectious, or degenerative diseases can cause secondary bilateral and symmetric calcifications of the basal ganglia and other brain regions. These include endocrine disorders, mitochondrial myopathies, dermatological disorders, brucellosis, toxoplasmosis, and others. This condition is known as Fahr's syndrome.[2] The most commonly reported metabolic disorders that cause Fahr's syndrome are hypoparathyroidism and pseudohypoparathyroidism.[2]

The association of basal ganglia calcifications with chronic hypoparathyroidism was described for the first time by Eaton *et al.* in 1939.[3] Hypoparathyroidism could mostly be iatrogenic due to surgical removal or radiotherapy, or it could be idiopathic because of genetically inherited and/or autoimmune diseases.[2] The duration, severity, and rate of development of hypocalcemia determine the clinical presentation. Various organs can be affected by calcification, most frequently the kidneys, but also joints, eyes, skin, vasculature, and, rarely, the brain.[4] Association of the disease with pseudohypoparathyroidism was also reported.[5]

There is still no clear explanation for the mechanism linking brain calcification and hypocalcemia.[2] High serum phosphate may also activate the inorganic phosphate transporter Pit1, leading to the expression of osteogenic molecules in the caudate nucleus and gray matter, which could explain basal ganglia calcification.[4] Prolonged hyperphosphatemia may result in downregulation of phosphate transporters in the basal ganglia, thereby promoting colloid precipitation in cerebral blood vessels and subsequent brain calcification.[6]

Different dermatological signs accompany hypoparathyroidism: dry, scaly skin; eczematiform rashes; hyperkeratotic maculopapular rashes; pellagroid hyperpigmentations of Addisonian or melasma type; and exfoliative erythroderma.[7] The link between hypocalcemia and psoriasis is not fully understood.[8] Different studies report that hypoparathyroidism may cause the onset or aggravate psoriasis in patients with surgical hypoparathyroidism and primary hypoparathyroidism. They have indicated that calcium plays a role in controlling the proliferation and differentiation of keratinocytes. Measuring epidermal calcium concentration in patients with psoriasis showed a low level, consistent with parakeratosis.[9] Moreover, vitamin D systemic compounds and oral calcium are recommended to treat psoriasis.[10,11] Some variants of psoriasis have been found to cause disturbances in systemic calcium metabolism.[7,8,12,13] In our case, there is clinical evidence for dermatological and psychiatric manifestations. Calcifications in the frontal lobe explain our patient's psychiatric manifestations since childhood.

**Conclusions**

In Fahr's syndrome caused by hypoparathyroidism, early treatment can prevent calcification and neurophysiological disorders.[4] No specific treatment can remove cerebral calcification,[14] but with every 1% increase in the calcium/phosphorus ratio, the progression of the basal ganglia calcification decreased by 5%.[2] Our experience indicates the importance of a multidisciplinary team to ensure optimal management.

A contrast between clinical polymorphism, diagnostic difficulty, and simple replacement therapy characterizes Fahr's syndrome. Although rare, it should always be considered in the refractory treatment of psoriasis vulgaris, especially when accompanied by other suggestive signs of hypocalcemia. Further investigations may find a significant link between psoriasis vulgaris and metabolic disorders that lead to cerebral calcifications.

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**Figure 1.** CT without contrast shows symmetrical focal parenchymal calcifications in the frontal lobes (yellow arrows) and basal ganglia calcifications (black arrows).

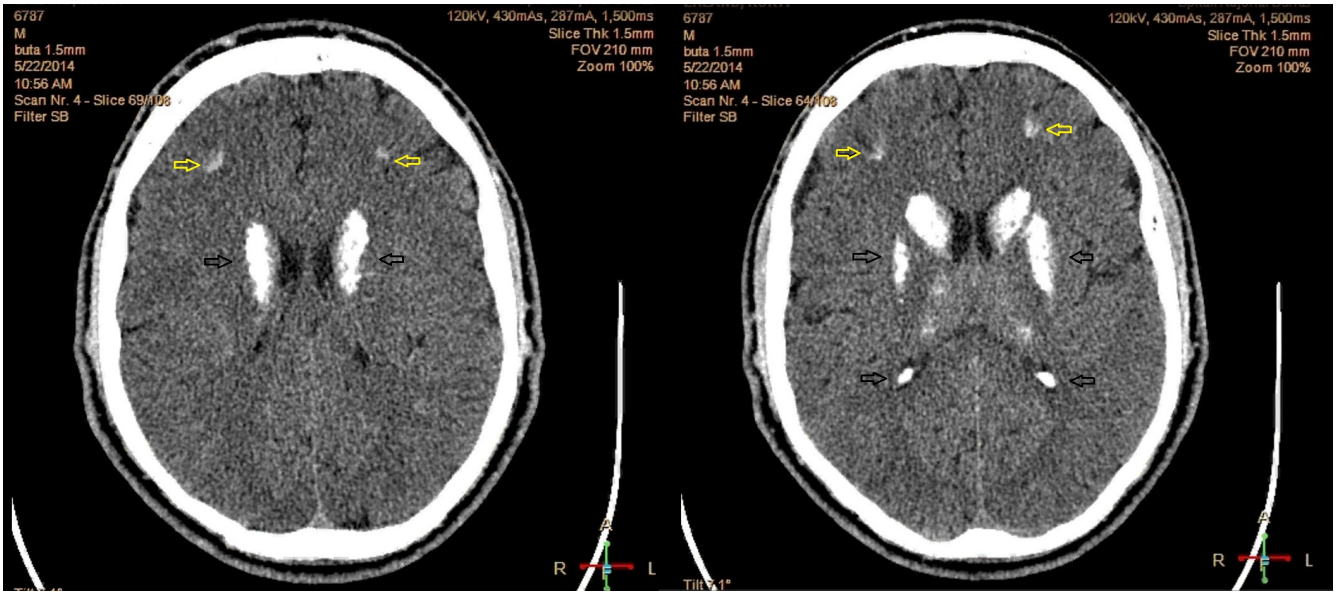


**Figure 2.** Erythro-squamous plaques of psoriasis vulgaris before treatment and improvement of psoriatic lesions after calcium replacement.

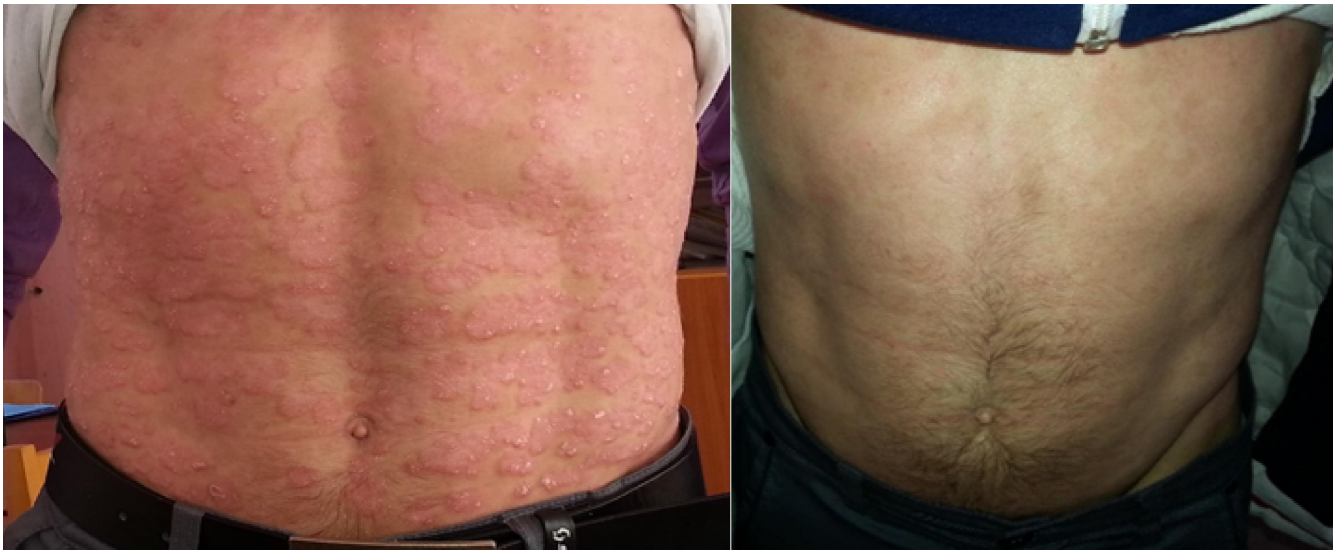

**Table 1.** Results of the biochemical examination.

| **Laboratory examination** | **Result** | **Reference range** |
|---|---|---|
| 1.25$(OH)_2$ vitamin $D_3$ | 42.7 pg/mL | 17.0-53.0 pg/mL |
| 24 hours albuminuria | negative | |
| 24 hours calciuria | 23.85 mg | 50-250 mg/24h |
| PT-hormone | 0.79 mmol/L | 1.59-6.89 mmol/L |
| Phosforemia | 6.21 mg/dL | 2.4-4.2 mg/dL |
| Total calcium | 5.07mg/dL | 8.5-10.5 mg/dL |
| Ionized calcium, $Ca^{++}$ | 0.52mmol/L | 1.05-1.3 mmol/L |
| $Na^+$<br>$K^+$<br>$Cl^-$ | 133 mmol/L<br>3.0 mmol/L,<br>94 mmol/L | 136-145 mmol/L<br>3.5-5 mmol/L<br>95-108 mmol/L |
| TSH | 1.33 μIU/mL | 0.30-5.0 μIU/mL |

PT-hormone, parathyroid hormone; TSH, thyroid-stimulating hormone.