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FAHR SYNDROME DUE TO HYPOPARATHYROIDISM PRESENTING IN A COMATOUS PATIENT: A CASE REPORT

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INTRODUCTION

Fahr's syndrome has a broad differential diagnosis.[1] An important cause is primary hypoparathyroidism, for which diagnostic evaluation is imperative.[2] Clinical picture may involve acute or subacute presentations, in addition to asymptomatic cases.[3] Here we present a case of primary hypoparathyroidism causing Fahr's syndrome with an acute presentation with excellent therapeutic response.

CASE REPORT

A 76-year-old patient, with previous thyroidectomy, started to complain of tremors for 9 months without medical follow-up. She was admitted to the hospital due to a sudden reduction in the level of consciousness, being admitted to the intensive care unit (ICU) for laboratory tests, neuroimaging and passage of a nasoenteral tube for feeding. The exams took three to five days to get the results (table 1). Brain computadorized tomography showed bilateral gross calcifications (figure 1), suggesting the diagnosis of Fahr syndrome and possible differential diagnoses. Soon after, the result of ionic serum calcium showed significant hypocalcemia, and immediate replacement was initiated with prompt clinical improvement. The patient also underwent a brain magnetic resonance imaging (MRI), showing gross, bilateral and diffuse calcifications (figure 2). In addition, she received the result of low parathyroid hormone, diagnosing primary hypoparathyroidism as the cause of hypocalcemia and Fahr's syndrome. The patient was discharged from the ICU with complete clinical improvement.

Variables	Patient	Reference Values
Serum tests		
Hemoglobin (g/dL)	10.5	13–17.5
Leukocytes (cells/mm^3)	9.500	4000–11,000
Platelets (number/mm^3)	363.000	150,000–450,000
C-reactive protein (mg/dL)	15.7	< 0.6
Aspartate transaminase (U/L)	36	<37
Alanine transaminase (U/L)	19	<41
Serum toxoplasmosis IgM	Non-reactive	Non-reactive
VDRL	Non-reactive	Non-reactive
Vitamin 25-OH-D (ng/mL)	26.90	>20.0
VDRL	Non-reactive	Non-reactive
Thyroid stimulating hormone (microUI/mL)	19.8	0.48-5.6
Free thyroxine (ng/dL)	0.94	0.85-1.5
Parathyroid hormone (pg/mL)	4.6	18.5-68.0
Potassium (mM/L)	2.8	3.5–5.1
Calcium ionized (mg/dL)	0.36	8.6-10.3
Magnesium (mg/dL)	1.0	1.6–2.6
Phosphorum (mg/dL)	6.4	2.3-4.3
Blood Urea Nitrogen test (mg/dL)	23	15–50
Creatinine (mg/dL)	1.2	0.5–1.3

Table 1. Results of laboratory tests.

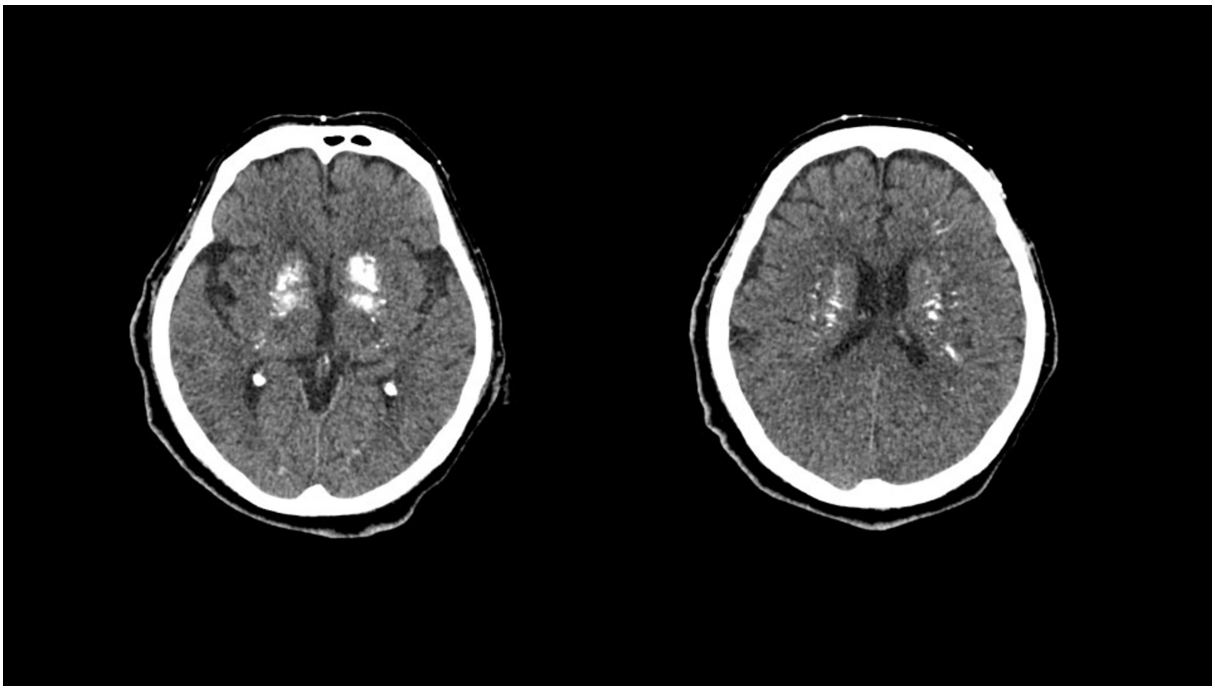

Figure 1a. CT scan.

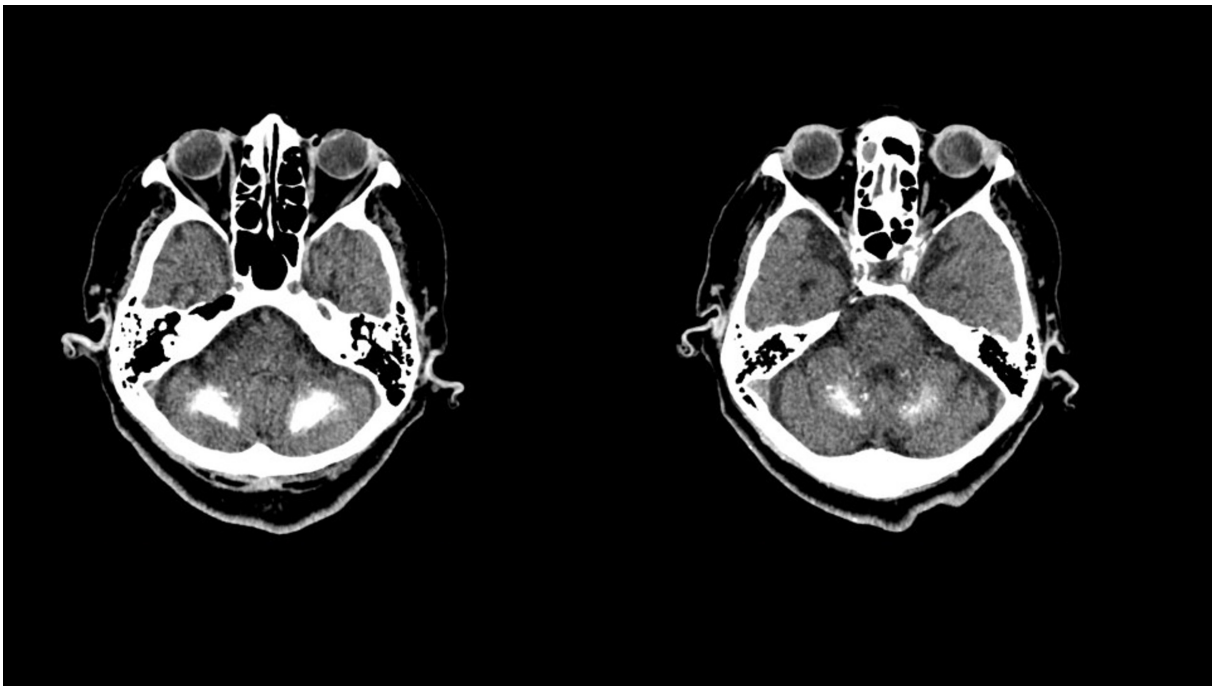

Figure 1b. CT scan.

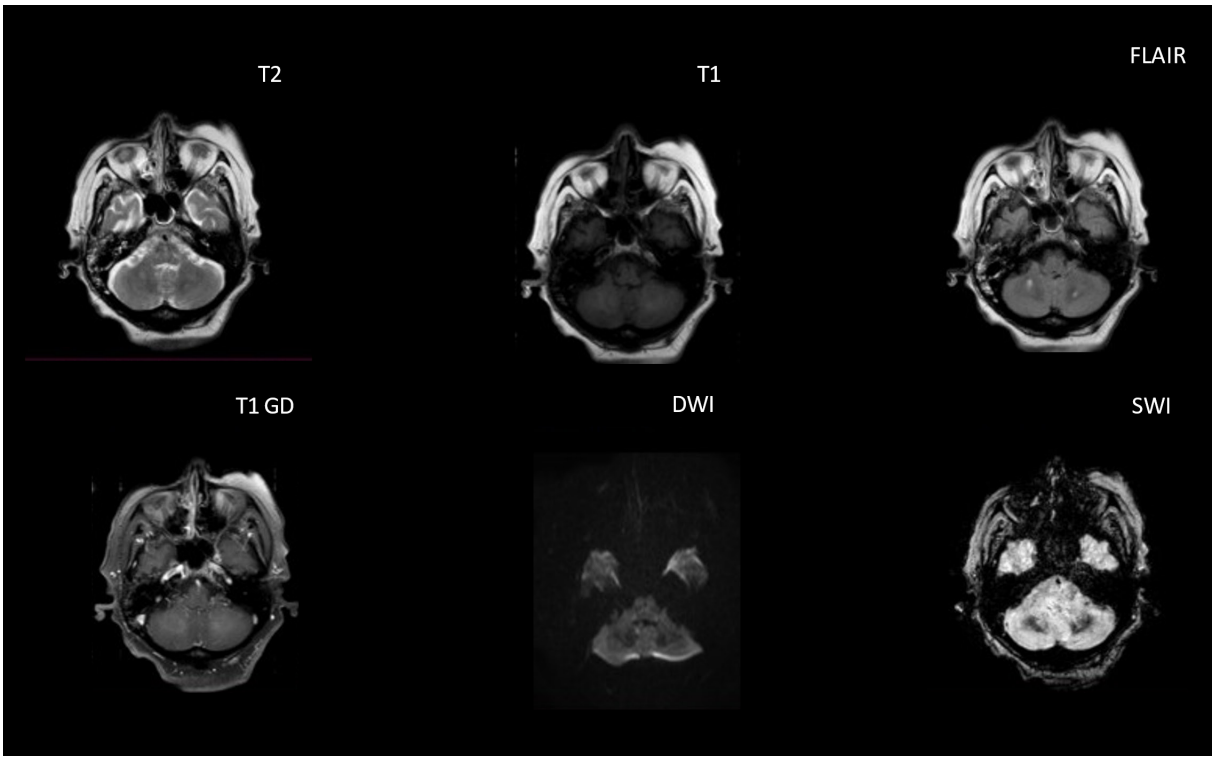

Figure 2a. Brain MRI sequences respectively of axial slices in unenhanced T2, T1, FLAIR, T1 GD diffusion and SWI.

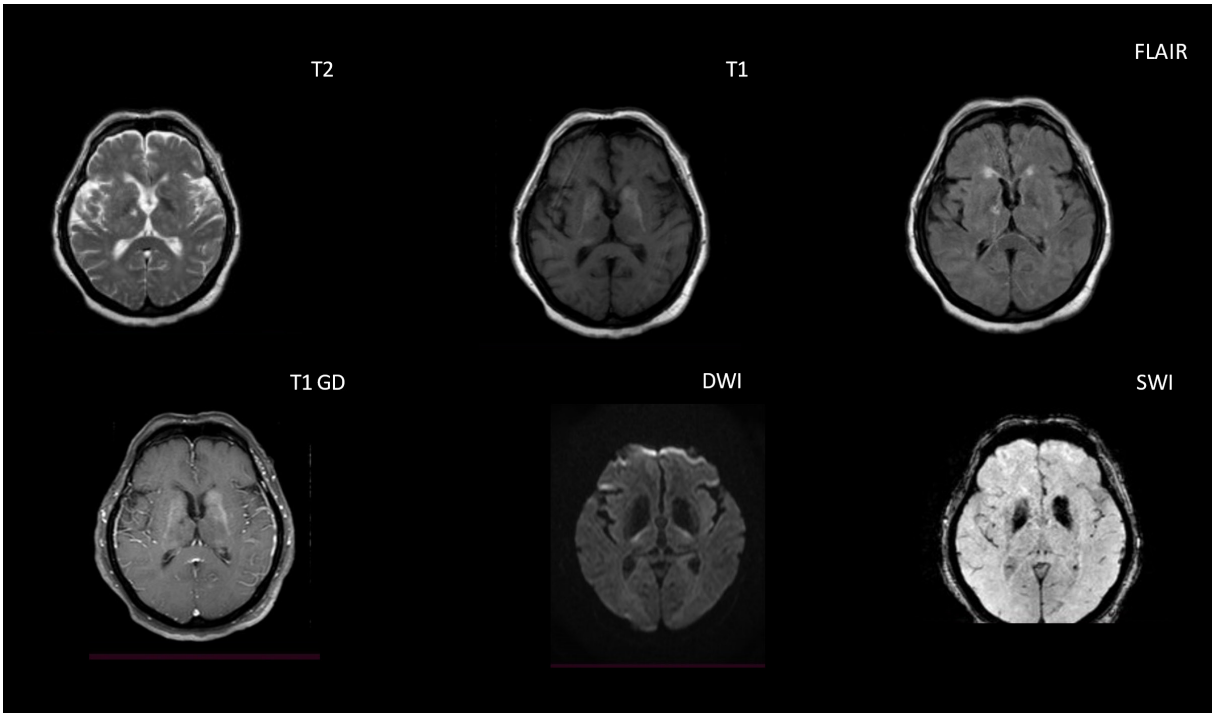

Figure 2b. Brain MRI sequences respectively of axial slices in unenhanced T2, T1, FLAIR, T1 GD diffusion and SWI.

DISCUSSION

Present report demonstrates a favorable evolution of a patient with primary hypoparathyroidism causing Fahr's syndrome with an acute presentation with excellent therapeutic response. Similar cases have been described in literature, but with an unfavorable outcome.[4] Nonspecific clinical and radiological presentation makes early diagnosis difficult, requiring an adequate clinic suspicious. Presence of reduced PTH with reduced calcium and neuroimaging with gross calcifications suggests the diagnosis.[2] This case report emphasizes early treatment importance in suspected.

AUTHOR CONTRIBUTIONS

Conceptualization, JWLTJ, PPdAC, methodology, JWLTJ writing original draft preparation, JWLTJ, writing- review and editing, JWLTJ, PPdAC, visualization JWLTJ, supervision JWLTJ PPdAC, project administration JWLTJ, PPdAC. All authors have read and agreed to the published version of the manuscript.

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INSTITUTIONAL REVIEW BOARD STATEMENT

The study was conducted according to the guidelines of the Declaration of Helsinki.

INFORMED CONSENT STATEMENT

Written informed consent has been obtained from the patient to publish this paper.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

ABBREVIATIONS

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