

## CASE REPORT

# Fahr syndrome secondary to hypoparathyroidism: a three-case series

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### ABSTRACT

**Background:** Fahr disease and Fahr syndrome are two clinical conditions linked by their shared clinical and radiological findings. They are characterized by progressive, bilateral calcium deposits in various brain regions, which are often discovered incidentally. Patients may present with a wide spectrum of symptoms. Treatment is individualized and is based on the patient's symptoms.

**Case presentation:** This is a case series of three patients presenting with neurological symptoms and Fahr syndrome secondary to hypoparathyroidism who were admitted to the hospital and managed symptomatically. The first patient is a 22-year-old female, a known case of hypoparathyroidism, who presented with headache, numbness, and dizziness. She was found to have hypocalcemia, and head computed tomography (CT) showed bilateral, symmetrical, dense calcifications. She was managed with intravenous calcium during admission and discharged on oral calcium supplementation. The second patient is a 37-year-old female who presented with abnormal body movements, with findings of Fahr disease on head CT. The patient was treated with intravenous calcium during admission and was discharged with close follow-up. The third patient is a 39-year-old female, a known case of Fahr syndrome, who presented with headache, dizziness, and a fall. She was found to have severe hypocalcemia and was admitted to the intensive care unit for calcium replacement and monitoring.

**Keywords:** Fahr syndrome, hypoparathyroidism, hypocalcemia, basal ganglia calcification.

### Introduction

Fahr disease and Fahr syndrome are rare related clinical and radiological conditions characterized by progressive bilateral calcium deposition in various brain regions, most commonly affecting the basal ganglia. In some cases, the thalamus, hypothalamus, hippocampus, cerebral cortex, and subcortical white matter may also be involved [1]. Fahr disease was first identified by Fahr in 1930 in a 55-year-old patient who experienced severe seizures [2]. These conditions can be distinguished based on their underlying causes. Fahr disease typically refers to the primary or genetic form, now more precisely known as primary familial brain calcification. Fahr syndrome, on the other hand, describes secondary forms associated with identifiable causes such as vascular, neoplastic, inflammatory, and particularly endocrine disorders of calcium-phosphate metabolism, such as hypoparathyroidism [3]. Fahr disease is managed symptomatically, whereas Fahr syndrome is treated by addressing the underlying etiology.

Fahr disease and Fahr syndrome present a wide range of clinical manifestations, including neuropsychiatric symptoms, cognitive impairment, speech dysfunction,

and involuntary movements. Interestingly, incidental findings on imaging can be observed in 15%-20% of patients [3]. The severity of psychiatric symptoms has been correlated with the extent of calcification compared to neurological symptoms [4].

In this article, three cases of Fahr syndrome will be presented. These cases will include descriptions of the symptoms, associated laboratory data, and imaging findings. Additionally, the management and follow-up for each case will be discussed.

### Case Report

#### Case 1

The first case involves a 22-year-old female with a past medical history of primary hypoparathyroidism

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diagnosed during childhood. She also has epilepsy, which started at the age of seven with approximately one attack every 6 months; her last attack occurred 1 month prior to the current presentation. At that time, she was started on levetiracetam 500 mg twice daily, alongside vitamin D and calcium supplements.

The patient presented to the emergency department (ED) with a one-day history of right occipital headache, drowsiness, and bilateral upper limb numbness. She reported that these symptoms typically occur as a prodrome to her seizure episodes. On physical examination, neurological examination revealed a Glasgow Coma Scale score of 15/15. She was conscious, alert, and oriented to time, place, and person. Speech was clear and coherent. Pupils were equal and reactive to light. Visual fields were intact on confrontation testing. Extraocular movements were intact with no evidence of nystagmus. Facial sensation to pinprick was preserved bilaterally, and no facial asymmetry was observed. No dysarthria was noted on examination. Muscle power, tone, deep tendon reflexes, and sensation were otherwise normal. The remainder of the systemic examination was unremarkable.

Laboratory investigations demonstrated hypocalcemia with a total serum calcium level of 1.37 mmol/l (albumin: 41 g/l; corrected calcium: 1.4 mmol/l). Additional findings included a serum phosphorus of 1.47 mmol/l, a markedly reduced parathyroid hormone (PTH) level of 0.7 pmol/l, and a mildly reduced magnesium level of 0.6 mmol/l. Complete blood count, liver function tests, kidney function tests, and remaining serum electrolytes

were normal. A non-contrast computed tomography (CT) brain revealed: Bilateral symmetrical dense calcification noted in the caudate nuclei, lentiform nuclei, dentate nuclei, cerebellar gray matter, and frontal subcortical white matter to a lesser extent, with faint subcortical calcification in the parietal lobes. The prominent bilateral basal ganglia calcifications are presented in Figure 1. In the context of the patient's findings, the history of hyper/hypoparathyroidism is suggestive of Fahr's syndrome/disease with no signs of recent ischemia, recent hemorrhage, or mass effect. An electrocardiogram (ECG) performed at the ED and showed a normal sinus rhythm.

During the ED stay, the patient received 1 l of normal saline and 1 g of paracetamol for headache relief. Initial laboratory investigations were obtained, followed by a non-contrast CT scan of the brain. After the laboratory results became available, urgent calcium replacement was initiated with calcium gluconate 2 g diluted in 100 ml of 5% dextrose in water, administered over 30 minutes. Serum calcium was then rechecked. While awaiting the repeat calcium level, the neurology team reviewed the patient after the CT brain findings. Their assessment was that the patient was a known case of epilepsy with chronic headaches, and no changes to her antiepileptic regimen were recommended apart from adding magnesium sulfate for headache management. Despite calcium gluconate administration, the repeat calcium level remained low at 1.33 mmol/l. The patient was subsequently admitted under the internal medicine team as a case of Fahr disease with hypocalcemia secondary to primary hypoparathyroidism. The

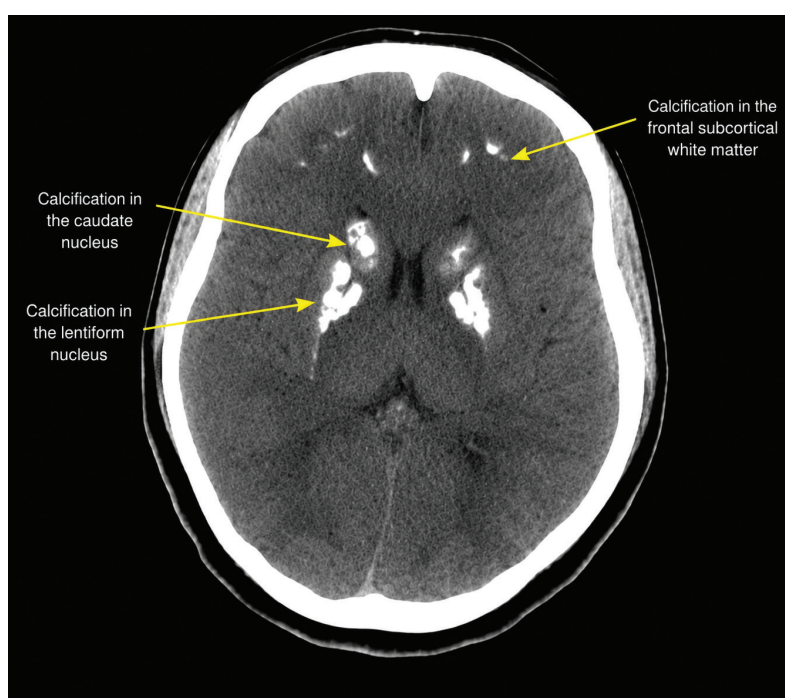


**Figure 1.** Axial non-contrast CT image of the brain demonstrating marked bilateral symmetrical calcification involving the caudate and lentiform nuclei, with smaller calcific deposits in the frontal subcortical white matter. Arrows indicate the main sites of intracranial calcification involving the caudate nuclei, lentiform nuclei, and frontal subcortical white matter.

management plan included further calcium replacement with calcium gluconate 2 g in 200 ml of normal saline over 20 minutes, oral calcium carbonate 1,200 mg every 6 hours, correction of potassium and magnesium, and initiation of oral alfacalcidol 1 mcg once daily. Ten hours after admission, serum calcium increased to 1.6 mmol/l. Following endocrinology consultation, aggressive calcium replacement was initiated with 11 g of calcium gluconate in 1 l of normal saline infused at 50 ml/h. Fourteen hours later, the calcium level rose to 2.22 mmol/l. The infusion was then discontinued to confirm stability, and 17 hours later the serum calcium was 2.07 mmol/l. As the patient showed clear clinical improvement, she was discharged on oral calcium, alfacalcidol, and vitamin D supplementation.

The patient was reviewed in the endocrinology clinic 18 days after discharge. Laboratory investigations performed 3 days before follow-up showed a 25-hydroxyvitamin D level of 200.95 nmol/l, fasting glucose of 5.2 mmol/l, and unremarkable thyroid, renal, and liver function tests. At follow-up, the patient was asymptomatic. She was counseled regarding her condition and advised to reduce calcium carbonate to 1,200 mg twice daily and increase alfacalcidol to 1.5 mcg daily.

## Case 2

This is a 37-year-old Saudi woman with a past medical history of primary hypothyroidism diagnosed 4 years earlier and chronic hypocalcemia of unclear etiology previously followed at another facility; however, medical records were unavailable for review. She was on

levothyroxine 75 mcg orally once daily and oral calcium tablets once daily, although she reported poor adherence to the calcium supplementation.

The patient presented to the ED with abnormal body movements, described as bilateral hand contractions, left arm spasms, head deviation, a fixed gaze to the left, tongue biting, and a fall to the left side during the episode. The event was witnessed by her daughter and did not involve loss of consciousness, and the patient was fully aware of her surroundings during the entire episode. She also reported a 3-month history of dysarthria, for which she had not sought medical attention. Additionally, she reported experiencing similar episodes over the past 4 years. During these previous visits, she was prescribed calcium supplements; however, she stopped taking them 6 months prior to presentation. On physical examination, the patient was hemodynamically stable; the neurological examination was remarkable for severe aphasia, limb ataxia, and an inability to perform finger-to-nose and heel-to-shin coordination tests. Muscle power, tone, deep tendon reflexes, and sensation were otherwise unremarkable. The remainder of the physical examination was unremarkable.

Laboratory investigations were significant for hypocalcemia with a calcium level of 0.94 mmol/l. The albumin level was 47 g/l, serum phosphorus level was 2.13 mmol/l, and PTH was markedly low at 0.2 pmol/l. Creatine kinase level was evaluated at 1,085 IU/l. Otherwise, complete blood count, kidney, liver function, and other electrolyte levels, including sodium, potassium, and magnesium levels, were within normal limits. Furthermore, a non-contrast CT brain performed

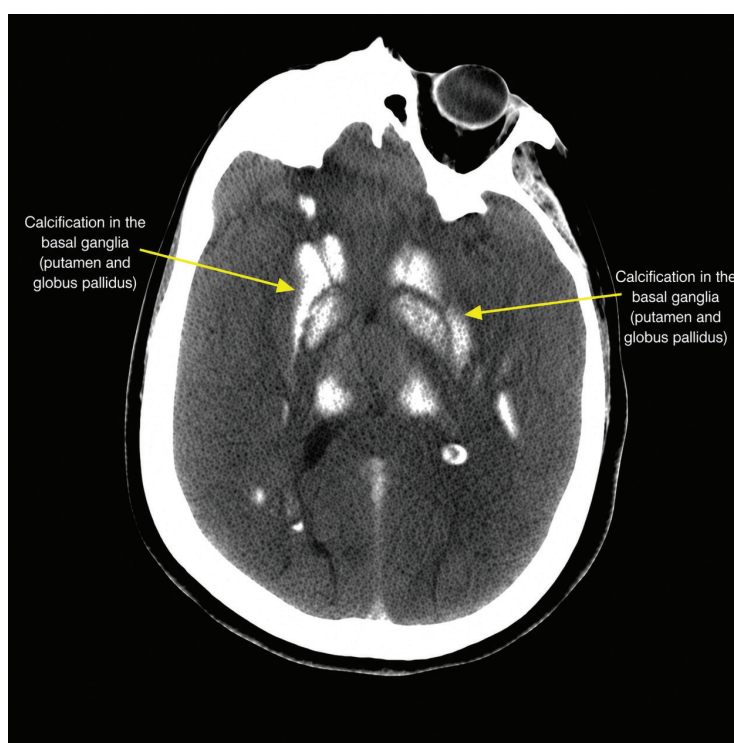


**Figure 2.** Axial non-contrast CT image of the brain demonstrating extensive bilateral symmetrical calcifications involving the basal ganglia. Arrows indicate the prominent bilateral basal ganglia calcifications.

in the ED demonstrated extensive bilateral symmetrical supratentorial and infratentorial calcifications predominately involving the centrum semi-oval, corona radiata, basal ganglia and pons as well as dentate nuclei findings were suggestive of Fahr syndrome with otherwise no large territorial infarction or acute intracranial hemorrhage. Representative bilateral basal ganglia calcifications are demonstrated in Figure 2. ECG performed at the ED was normal sinus rhythm.

In the ED, the patient was placed on continuous cardiac monitoring and initiated on intravenous calcium replacement. She received intravenous calcium gluconate starting with 1 g administered over 10 minutes, followed by a repeat dose. Subsequently, 4 g of calcium gluconate diluted in normal saline was administered over 3 hours. The patient demonstrated significant clinical improvement following initiation of treatment. Neurology was consulted for further evaluation. After partial correction of serum calcium levels, reassessment revealed that the patient was conscious, alert, and oriented, with normal cranial nerve function and intact motor strength, sensory examination, and reflexes. The neurology team concluded that the clinical history and presentation were not suggestive of an epileptic seizure. Instead, the episode was considered most consistent with neuromuscular irritability secondary to severe hypocalcemia. The patient's chronic dysarthria was thought to be likely related to the underlying metabolic disturbance.

She was subsequently admitted for continued calcium correction and further evaluation. Normalization of calcium level and complete resolution of the symptoms were achieved within 1 day of treatment. Additional

investigations performed during hospitalization including 25-hydroxyvitamin D level 118 nmol/l, thyrotropin level 1.81 mU/l, and thyroxine (T4) 17.24 pmol/l. The endocrinology team was consulted and recommended discharge on oral calcium supplementation 1,200 mg every 6 hours and alfacalcidol 2 mcg once daily while continuing the previous dose of levothyroxine. The patient was instructed to follow up in the endocrinology outpatient clinic after 1 month for reassessment and ongoing management. However, the patient did not attend the scheduled follow-up appointment.

### Case 3

The third case involved a 39-year-old woman with a history of osteoporosis, rheumatoid arthritis, hypoparathyroidism, and a recent diagnosis of Fahr syndrome 1 year earlier at another hospital. Her medications included omega-3, vitamin D supplements, sulfasalazine, prednisolone, and an antiepileptic drug started 1 year earlier, the name of which she could not recall.

Her history of presenting illness consisted of a 1-day history of worsening frontal headache and dizziness, followed by a fall. There was no loss of consciousness, seizure-like activity, tongue biting, or urinary incontinence. There was no reported head trauma. She also reported muscle spasms, numbness involving the upper and lower limbs, perioral numbness, slowed speech, and mild shortness of breath. The headache was chronic in nature but worsened prior to presentation and was associated with visual changes. In addition, she reported daily episodes of vomiting before sleep, consisting of food particles or gastric contents, with

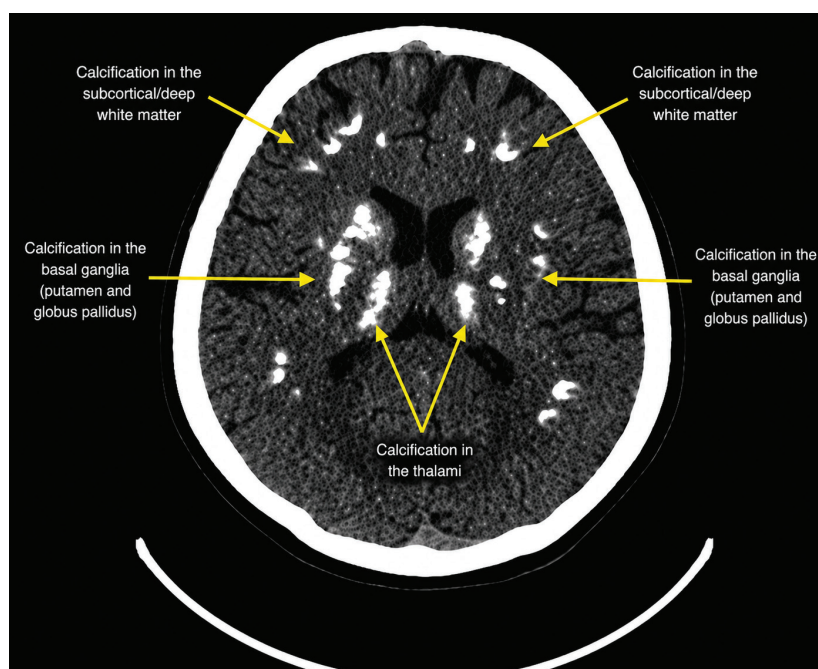


**Figure 3.** Axial non-contrast CT image of the brain demonstrating diffuse bilateral symmetrical intracranial calcifications involving the basal ganglia, thalami, and subcortical/deep cerebral white matter. Arrows indicate the prominent sites of calcification in the basal ganglia, thalami, and subcortical/deep white matter.

relief afterward. She also had chronic watery, non-bloody diarrhea associated with generalized abdominal pain, with the most recent episode occurring on the morning of presentation. Her history was notable for a previous hospital admission ~1 year earlier for hypocalcemia.

In the ED, she received 1 l of intravenous normal saline, 1 g of paracetamol, and 10 mg of metoclopramide. Laboratory investigations revealed hypocalcemia with serum calcium of 1.55 mmol/l and corrected calcium of 1.6 mmol/l in the setting of albumin 37.1 g/l. Additional abnormalities included phosphorus 0.64 mmol/l, PTH 0.5 pmol/l, magnesium 0.57 mmol/l, and potassium 3.23 mmol/l. Sodium, creatinine, creatine kinase, complete blood count, and liver function tests were unremarkable. After the laboratory results were reviewed, urgent calcium replacement was initiated with calcium gluconate 2 g in 100 ml of 5% dextrose in water over 30 minutes, followed by a repeat calcium measurement. A non-contrast CT scan of the brain demonstrated diffuse bilateral symmetrical dense parenchymal calcifications involving the subcortical deep white matter, basal ganglia, dentate nuclei, thalami, and cerebellum. No acute territorial infarction or intracranial hemorrhage was seen. These findings were considered suggestive of Fahr syndrome in correlation with the metabolic profile. Representative bilateral basal ganglia, thalamic, and subcortical/deep white matter calcifications are demonstrated in Figure 3.

Due to the severe hypocalcemia, the patient was admitted under internal medicine to the intensive care unit (ICU) as a case of Fahr syndrome complicated by severe hypocalcemia, with mild hypokalemia and hypophosphatemia. Repeat calcium remained low at 1.6 mmol/l despite treatment. Endocrinology therefore recommended calcium gluconate 9,400 mg diluted in 900 ml of normal saline and infused at 50 ml/h, in addition to sodium phosphate 30 mmol diluted in 250 ml of normal saline over 8 hours. The treatment plan included continuation of calcium and phosphate infusions, oral calcium carbonate 1,200 mg every 8 hours, alfacalcidol 1 mcg once daily, and correction of magnesium and potassium. Seven hours after ICU admission, serum calcium increased to 2.1 mmol/l and the calcium infusion was discontinued. However, 8 hours later the calcium level had decreased again to 1.64 mmol/l. Endocrinology therefore advised resuming the calcium infusion and increasing alfacalcidol to 2 mcg once daily. By the following day, calcium had improved to 1.9 mmol/l with symptomatic improvement, and the endocrine team recommended stopping the infusion and continuing oral therapy. The discharge plan was calcium carbonate 1,200 mg every 8 hours for 1 week, then every 12 hours thereafter; alfacalcidol 2 mcg once daily; and vitamin D 50,000 IU once weekly. The patient was transferred from the ICU to the regular ward for further observation. On the following day, discharge was initially planned, but repeat laboratory investigations showed recurrent abnormalities: calcium 1.67 mmol/l, corrected calcium 1.6 mmol/l, potassium 3.13 mmol/l, magnesium 0.53 mmol/l, and phosphorus 0.88 mmol/l. Discharge was therefore postponed, and she received magnesium sulfate 2 g in 200 ml of normal saline over 4 hours together with calcium gluconate 4 g in 100 ml of normal saline over 2 hours. Repeat testing the next day demonstrated improvement, with calcium 2.08 mmol/l,

corrected calcium 2.1 mmol/l, potassium 4.13 mmol/l, magnesium 0.7 mmol/l, and phosphorus 1.09 mmol/l. She was then discharged on the previously recommended endocrine regimen and scheduled for follow-up 12 days later.

At her endocrine follow-up visit, she reported taking calcium carbonate only twice daily and alfacalcidol 1 mcg once daily. Laboratory evaluation showed recurrent hypocalcemia, with calcium 1.77 mmol/l and corrected calcium 1.7 mmol/l, as well as potassium 3.35 mmol/l and magnesium 0.53 mmol/l. Vitamin D was normal. Although she denied symptoms of hypocalcemia, she was advised to attend the ED for correction of calcium, magnesium, and potassium, but she refused. Her medications were therefore adjusted to calcium carbonate three times daily, alfacalcidol 2 mcg once daily, and vitamin D3 50,000 IU once weekly. At the next clinic visit 1 month later, she was asymptomatic and reported treatment adherence. Laboratory investigations showed normalization of calcium, corrected calcium, potassium, and magnesium, although phosphorus remained mildly low. She continues to be followed regularly in the endocrinology clinic.

## Result and Conclusion

This case series describes three female patients with Fahr syndrome secondary to hypoparathyroidism who presented with diverse neurological manifestations, including headache, paresthesia, neuromuscular irritability, seizure-like episodes, dysarthria, ataxia, and falls. All patients demonstrated marked hypocalcemia with inappropriately low PTH levels, while brain CT consistently revealed extensive bilateral symmetrical intracranial calcifications involving the basal ganglia and other characteristic regions.

Acute management focused on prompt correction of hypocalcemia using intravenous calcium replacement, followed by oral calcium supplementation and active vitamin D therapy. Clinical improvement paralleled normalization of serum calcium in all three cases, although one patient experienced recurrent hypocalcemia related to poor treatment adherence, emphasizing the importance of long-term follow-up and compliance.

Collectively, these cases reinforce that hypoparathyroidism is an important and treatable cause of Fahr syndrome. In patients presenting with unexplained neurological manifestations accompanied by characteristic bilateral intracranial calcifications, prompt evaluation of calcium-phosphate metabolism and parathyroid function is essential, as early recognition and correction of the underlying metabolic disturbance can result in significant clinical improvement and may prevent recurrent symptoms and disease progression.

## Discussion

Fahr syndrome is a rare neuropsychiatric disorder characterized by bilateral intracranial calcifications and may occur secondary to several underlying conditions. Reported secondary etiologies include vascular, neoplastic, inflammatory, and, most commonly,

endocrine or metabolic disorders. Among these, hypoparathyroidism is the most frequently implicated cause, largely because of chronic disturbances in calcium-phosphate metabolism. This association has been demonstrated in previously reported cases showing markedly reduced PTH levels [5]. Hypocalcemia in primary hypoparathyroidism results from deficient secretion of PTH. Reduced PTH activity impairs calcium mobilization from bone, decreases calcium reabsorption in the distal nephron, and diminishes activation of renal 1 $\alpha$ -hydroxylase, the enzyme responsible for converting 25-hydroxyvitamin D to 1,25-dihydroxyvitamin D. Consequently, intestinal calcium absorption is also reduced [6]. The persistent biochemical state of hypocalcemia and hyperphosphatemia promotes calcium-phosphate deposition in various brain regions, producing the characteristic radiological pattern of Fahr syndrome [5].

Fahr syndrome secondary to hypoparathyroidism exhibits a broad clinical spectrum. Case reports have described presentations ranging from acute confusion [5], seizures [6,7], and gait disturbance [7], to other neurological and psychiatric manifestations. In a review of 223 patients with Fahr syndrome secondary to hypoparathyroidism, tetany was the most common presentation, occurring in 114 patients (51.4%). Typical manifestations included muscle cramps, carpopedal spasm, paresthesia, and positive Chvostek and Trousseau signs. Movement disorders were reported in 107 patients (48.4%), seizures, predominantly generalized tonic-clonic, in 104 patients (46.6%), and neuropsychiatric manifestations in 90 patients (40.4%). Only four patients were asymptomatic. Less commonly reported features included dysarthria, dysphagia, muscular rigidity, spastic gait, headache, and hemiparesis [8].

Laboratory abnormalities in Fahr syndrome secondary to hypoparathyroidism typically include low PTH levels in association with hypocalcemia, as demonstrated in multiple case reports [5,9]. These findings highlight the importance of biochemical evaluation in patients presenting with suggestive neurological symptoms and characteristic intracranial calcifications.

The diagnosis of Fahr syndrome is primarily based on characteristic neuroimaging findings. CT is the preferred imaging modality for detection, as it provides superior assessment of the presence, distribution, and extent of intracranial calcifications, and it was the diagnostic method used in the reported cases. Magnetic resonance imaging may also be used as an adjunctive modality [10].

Management of Fahr syndrome is mainly directed toward early recognition and treatment of the underlying cause. In cases secondary to hypoparathyroidism or other metabolic disturbances, correction of hypocalcemia and vitamin D deficiency constitutes the mainstay of therapy [10].

### Learning points

When a patient presents with unexplained neurological manifestations associated with bilateral basal ganglia calcification on CT head, Fahr disease/ syndrome should

be suspected, with ruling out secondary causes, most commonly hypoparathyroidism.

Any patient with intracranial calcifications should undergo evaluation regarding serum calcium, phosphate, magnesium, and PTH, as hypoparathyroidism is one of the likely treatable causes of Fahr syndrome.

The clinical features of Fahr syndrome may include seizures, movement disorders, cognitive dysfunction, psychiatric symptoms, or incidental radiological findings despite severe calcifications.

Early detection and management of the underlying metabolic disturbance with calcium and active vitamin D improve the symptoms and limit further progression.

To achieve an accurate diagnosis, establish a long-term management plan, and prevent recurrent hypocalcemic complications, a multidisciplinary collaboration involving endocrinology, neurology, and radiology is crucial.

### List of abbreviations

|     |                            |
|-----|----------------------------|
| CT  | computed tomography        |
| ECG | electrocardiogram          |
| ED  | emergency department       |
| IRB | Institutional Review Board |
| PTH | parathyroid hormone        |

### Conflict of interests

The authors declare that they have no conflicts of interest regarding the publication of this case report.

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None.

### Consent for participation

Written informed consent was obtained from all the participants.

### Consent for publication

Written informed consent was obtained from the patient.

### Ethical approval

Informed consent was secured from patients involved in this study. All data mentioned in the article were de-identified. Forms and consent were submitted to the Institutional Review Board (IRB) in the Dammam Medical Complex. The manuscript, concepts, and forms were reviewed by the IRB and were approved for submission.

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*Supplementary content (If any) is available online.*

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