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# CT Recognition of Fahr's Syndrome: A Radiologic Case Report

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**Abstract** **Case Report**

Fahr's syndrome, or idiopathic basal ganglia calcification, is a rare neurological disorder characterized by bilateral, symmetrical calcifications involving the basal ganglia, thalami, dentate nuclei, and subcortical white matter. We report the case of a 46-year-old man who presented with transient loss of consciousness following minor head trauma. Brain CT revealed a right occipital fracture, left frontal contusions, and scalp swelling, but also multiple symmetric calcifications affecting the lentiform nuclei, thalami, dentate nuclei, hippocampus, caudate nuclei, corona radiata, and centrum semi-ovale—findings consistent with Fahr's syndrome. This case highlights the importance of recognizing typical CT features of Fahr's syndrome, even when detected incidentally in trauma settings, and discusses the role of various imaging modalities in diagnosis.

**Keywords:** Fahr's syndrome; basal ganglia calcification; idiopathic intracerebral calcification; brain CT; neuroimaging.



## INTRODUCTION

Fahr's syndrome, also known as idiopathic basal ganglia calcification or primary familial brain calcification, is a rare inherited or sporadic disorder marked by abnormal calcium deposition in bilateral and symmetrical deep brain structures such as the globus pallidus, putamen, caudate nuclei, thalami, and dentate nuclei [1, 2]. The condition is often linked to mutations in genes including *SLC20A2*, *XPR1*, *PDGFB*, and *PDGFRB* [2]. Computed tomography (CT) remains the imaging modality of choice for detection due to its high sensitivity to calcium compared with MRI, However, MRI findings correlate more closely with the clinical manifestations and are therefore more informative [2, 3]. Although most patients present with movement disorders, psychiatric disturbances, or cognitive decline, incidental findings are becoming more frequent owing to the widespread use of CT and MRI [2]. The following case illustrates a classic CT pattern of Fahr's syndrome discovered incidentally in a patient undergoing imaging for traumatic brain injury.

## CASE PRESENTATION

A 46-year-old man was admitted to the emergency department following a transient episode of loss of consciousness. On arrival, he was fully conscious, oriented, and hemodynamically stable. Neurological examination revealed no focal deficits, cranial nerve abnormalities, or motor asymmetry. There was mild tenderness over the occipital region but no cerebrospinal fluid leakage or signs of basilar fracture.

A non-contrast brain CT scan revealed multiple, dense, and symmetrically distributed intracranial calcifications involving the lentiform nuclei, thalami, dentate nuclei, caudate nuclei, hippocampi, corona radiata, and centrum semiovale. These calcifications were sharply marginated, homogeneous in density, and bilaterally confluent, with no associated edema or cortical laminar necrosis. No vascular or parenchymal malformations were identified, and the overall pattern was highly suggestive of idiopathic basal ganglia calcification (Fahr's syndrome).

In addition, the CT demonstrated a right temporal bone fracture associated with left frontal lobe contrecoup contusions. Scalp swelling was present in both frontal and occipital regions, consistent with post-traumatic soft-tissue edema. No intracranial hemorrhage, midline shift, or mass effect was observed.

Laboratory investigations, including serum calcium, phosphorus, magnesium, alkaline phosphatase, vitamin D, and parathyroid hormone levels, were within normal ranges, effectively excluding metabolic,

endocrine, or toxic etiologies such as hypoparathyroidism or heavy metal exposure. Additional screening for infectious diseases (toxoplasmosis, cytomegalovirus, brucellosis) and autoimmune markers was negative. The coexistence of extensive, bilateral, and symmetrical calcifications with normal biochemical parameters and absence of secondary causes confirmed the diagnosis of idiopathic Fahr's syndrome.

The patient was managed conservatively for his post-traumatic lesions with analgesics and observation. Neurological follow-up was recommended for further evaluation of the incidental calcifications. He remained clinically stable, with no parkinsonian, cognitive, or psychiatric symptoms during the hospital stay and at follow-up.

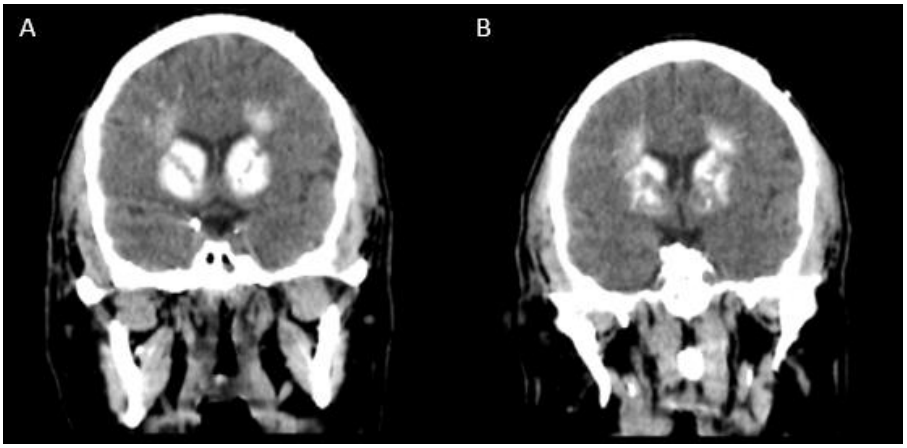


**Figure 1 A and B show NECT coronal reconstructions demonstrating symmetrical intracerebral calcifications, predominantly involving the caudate nuclei, lentiform nuclei, and centrum semiovale**

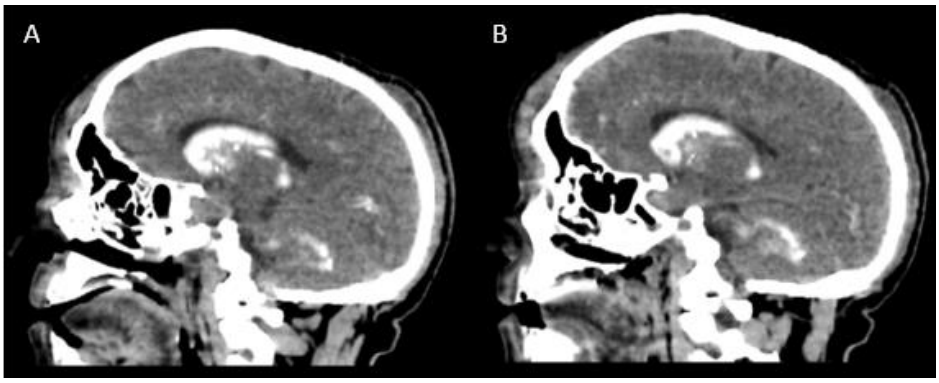


**Figure 2 NECT sagittal reconstructions showing intracerebral and intracerebellar calcifications involving the right (A) and left (B) caudate and dentate nuclei, respectively**

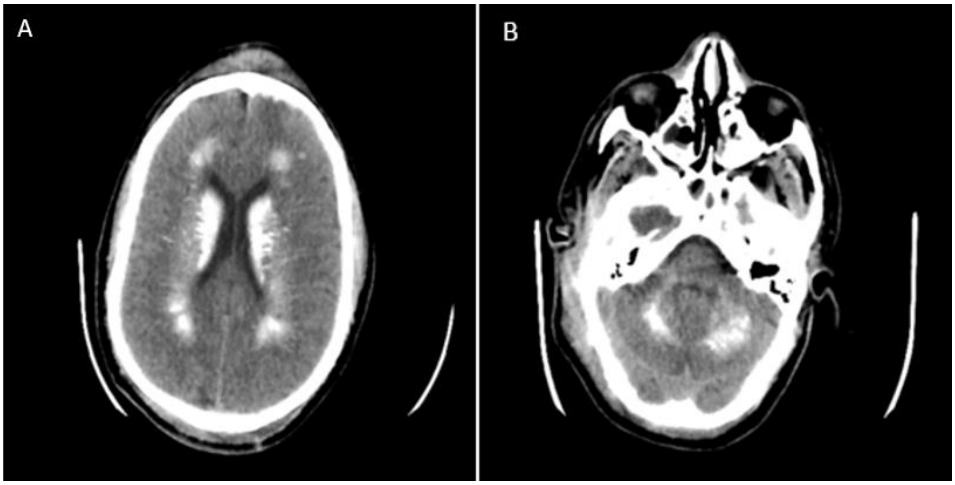


**Figure 3 NECT axial images showing symmetrical calcifications of the caudate nuclei and corona radiata (A), and of the dentate nuclei (B).**

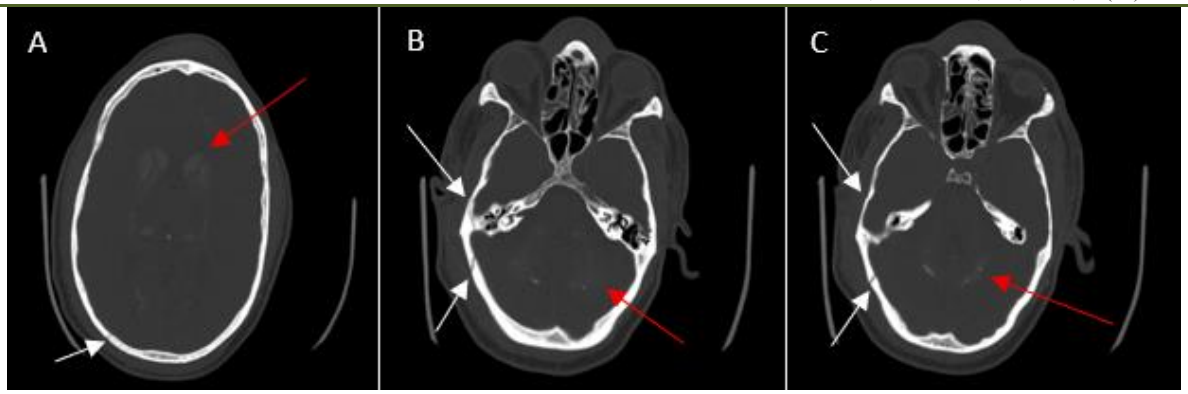


**Figure 4 NECT axial images showing a right temporal bone fracture (white arrow) and symmetrical intracerebral and intracerebellar calcifications (red arrows).**

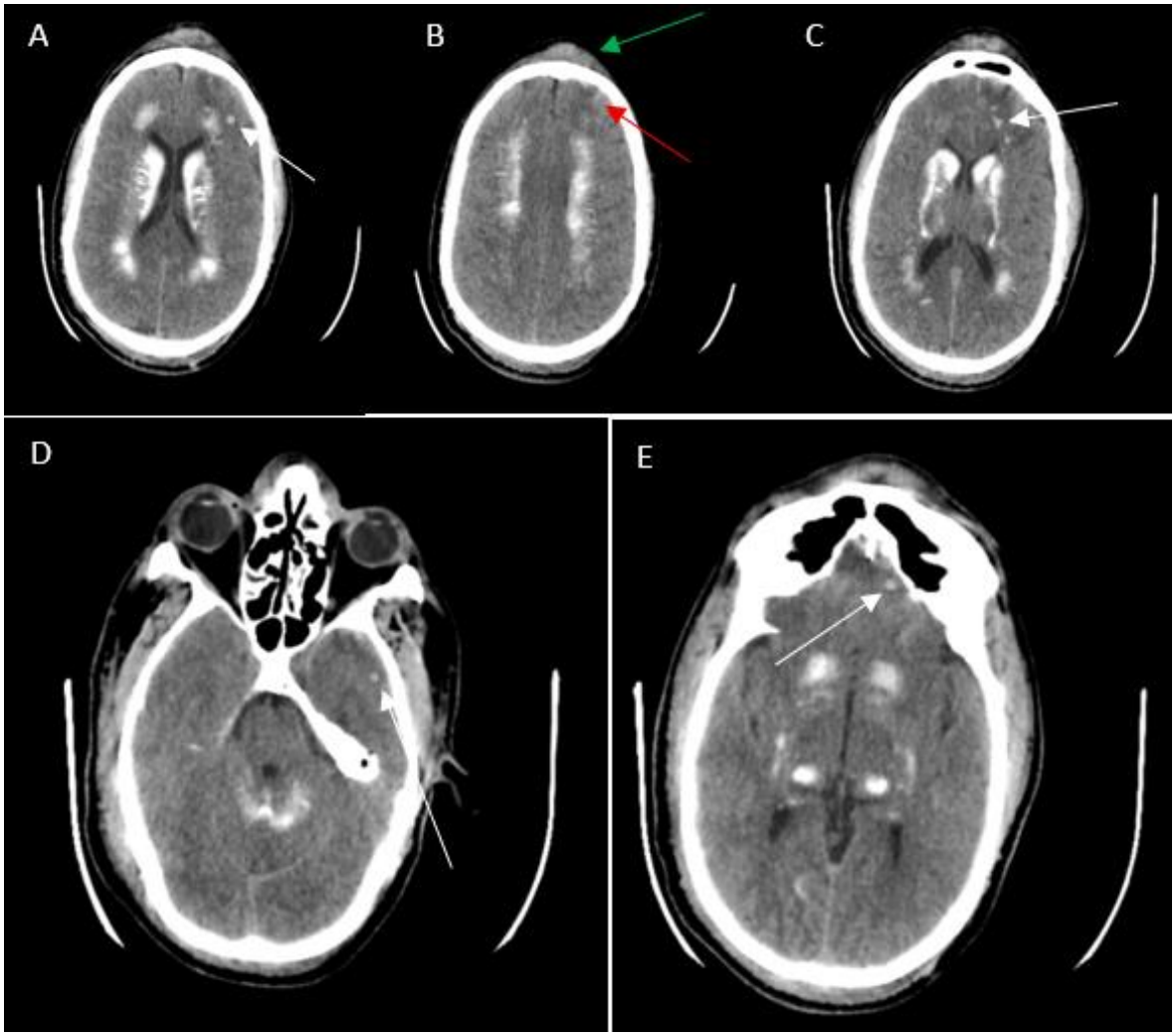


**Figure 5 NECT axial images showing frontal scalp swelling (green arrow), subarachnoid hemorrhage (red arrow), and hemorrhagic foci within edematous parenchyma located contrecoup to the previously demonstrated fracture, to be differentiated from the symmetrical intracerebral calcifications**

## DISCUSSION

Fahr's syndrome typically presents with bilateral and symmetrical calcifications of the basal ganglia, most often involving the globus pallidus and putamen [2]. Other regions frequently affected include the thalami, dentate nuclei, and subcortical white matter, particularly the centrum semi-ovale [4, 5]. On non-contrast CT, calcifications appear as symmetric hyperdense lesions that may be punctate, nodular or confluent. CT remains the imaging modality of choice for detection, owing to its superior sensitivity for calcium compared with MRI [2, 3].

On MRI, calcifications appear as areas of low signal intensity on T2*-weighted and susceptibility-weighted imaging (SWI). Although MRI is less sensitive than CT for detecting small or early deposits, SWI offers complementary diagnostic information by confirming mineral composition and distinguishing calcifications from hemosiderin or hemorrhagic residues [6].

The symmetry and distribution of calcifications are key diagnostic clues distinguishing Fahr's syndrome from secondary causes such as hypoparathyroidism, infections (toxoplasmosis, cytomegalovirus), or metabolic disorders [5, 7]. Age-related calcifications tend to be limited to the globus pallidus and are usually asymmetrical [5].

Advanced imaging modalities such as FDG-PET and SPECT have been shown to detect reduced metabolic activity or perfusion in affected regions, correlating with clinical manifestations [5]. Despite this, CT remains the cornerstone for initial diagnosis [3].

Clinically, patients with Fahr's syndrome may present with parkinsonism, neuropsychiatric symptoms, or seizures, although many cases remain asymptomatic [1, 2]. There is no curative treatment; management focuses on treating reversible metabolic abnormalities and providing symptomatic therapy. Recognizing the radiologic pattern ensures accurate diagnosis, avoids unnecessary investigations, and allows appropriate genetic counselling [2].

## CONCLUSION

This case demonstrates the classic CT findings of Fahr's syndrome discovered incidentally during evaluation for cranial trauma. The identification of symmetrical calcifications in the basal ganglia, thalami, dentate nuclei, and subcortical white matter is diagnostic. While MRI and functional imaging may provide adjunctive insights, non-contrast CT remains the gold standard for detection and characterization. Radiologists should be aware of this distinctive pattern to distinguish it from secondary or age-related calcifications and to ensure appropriate clinical and genetic assessment.

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