

Seizure-related head injury with rare etiology-Fahr's syndrome: a case report

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ABSTRACT

A 49-year-old woman presented with scalp laceration following an unsupervised generalized tonic-clonic seizure. Non-contrast head computed tomography performed for mild traumatic brain injury revealed incidental finding of bilateral symmetric striopallidodentate calcifications consistent with Fahr's syndrome. Clinical findings include positive Trousseau's sign, prolonged QT interval, bradykinesia, and shuffling gait. Laboratory evaluation confirmed hypoparathyroidism, hypocalcemia, and hyperphosphatemia. A history of total thyroidectomy 30 years earlier suggested chronic postoperative hypoparathyroidism as the underlying etiology. The patient was managed with antiseizure medication, wound care, and supportive treatment, with good short-term outcome. This case highlights Fahr's syndrome as an under-recognized metabolic cause of seizures and emphasizes the importance of neuroimaging and biochemical evaluation in seizure patients, particularly when head trauma or atypical neurological features are present.

Keywords: Seizure, craniocerebral trauma, brain calcification, hypoparathyroidism, hypocalcemia

INTRODUCTION

Head injuries are common in seizure-related falls, especially in unsupervised patients.¹ Seizure etiologies can vary from idiopathic, metabolic, to structural brain lesions. Fahr's syndrome (FS) is a secondary bilateral calcification of striatum, basal ganglia, and dentate nucleus, with most common etiology from hypoparathyroidism.^{2,3}

FS is a rare disease with prevalence of less than 1 in 1,000,000 people.³ Manifestation of FS includes cognitive disorders, movement disorders, psychiatric symptoms, and seizure.^{2,3} FS can be asymptomatic and often an incidental finding on a brain computed tomography (CT) scan for other indication.³

This case report aims to highlight FS as an under-recognized etiology of seizures and to highlight the importance of neuroimaging in seizure patients, especially when head injury is involved.

CASE

A 49-year-old woman presented to our hospital's emergency room (ER) with scalp laceration after an episode of seizure 30 minutes before. She was unsupervised when the seizure started, hence she fell and hit her head on the floor. Seizure described as generalized tonic-clonic. The patient was aware during the seizure but could not control her body. After the

seizure, patient had postictal headache with slight confusion, initially unaware of the scalp laceration. Upon arrival at the ER, seizure had already stopped and patient was able to walk, complained of headache, scalp laceration, and mild cramps in both arms and legs.

She had her first tonic-clonic seizure in 2020, hospitalized for several days at a local hospital and discharged with daily medications of oral Carbamazepine 200 mg, Vitamin B6, and Folic Acid 400 mcg. From 2020 to 2025, seizure was controlled with medication. In the last 3 months prior to this episode, patient took her medication inconsistently and started to have seizures about 3 times per month, and increased to about 3 times per week. Each seizure resolved spontaneously without medication. In 1996, patient was diagnosed for thyroid gland hypertrophy with airway obstruction. Total thyroidectomy surgery was performed and she was discharged without hormone therapy.

During initial assessment, she showed no disorientation, and was able to follow instruction. Vital signs are normal, but during blood pressure examination, she exhibited a positive Trousseau's Sign (**Figure. 1**). We noticed that she had a shuffling gait. We conducted further neurological exam and found spasticity and bradykinesia. On physical examination, there was scalp laceration measuring 3 cm by 1 cm with





Figure 1. Positive Trousseau's sign

active bleeding in left parietooccipital region (Figure 2). The wound was stitched, covered with sterile gauze and pressure bandage.



Figure 2. Scalp laceration in left parietooccipital region

Due to suspicion of skull fractures and intracranial bleeding, we performed non-contrast head CT-Scan. The results were unexpected: abnormal, bilateral, symmetric strio-pallido-dentate calcification (Figure 3). Electrocardiography (ECG) showed a prolonged QT Interval of 0.56 second (Figure 4). Complete blood count (CBC) and renal function test showed normocytic anaemia (Haemoglobin of 84 g/L, normal range 120-160 g/dl), elevated serum ureum (7.3 mmol/L, normal range 2.5-7.1 mmol/L), high serum creatinine (164 μ mol/L, normal range 49-90 μ mol/L).

Patient was admitted to regular ward, with medication of IV Ketorolac 30 mg every 8 hours, IV Ranitidine 50 mg every 12

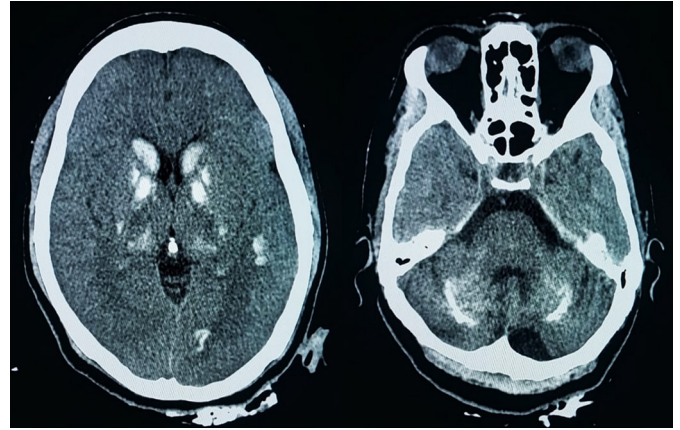


Figure 3. Bilateral and symmetric striopallidodentate calcification

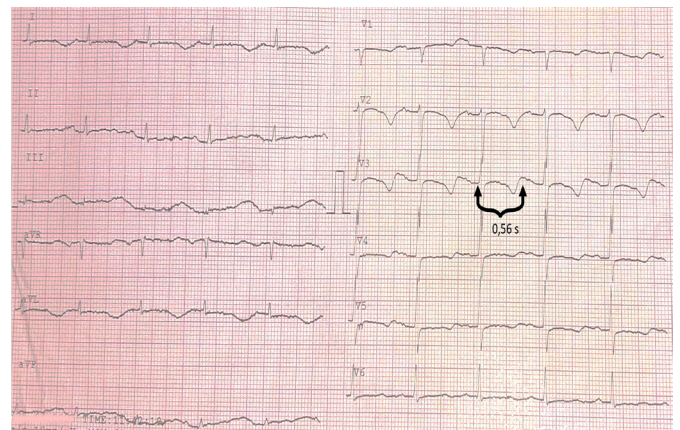


Figure 4. ECG showing prolonged QT interval (0.56 s)

hours, oral Phenytoin 100 mg every 8 hours, oral Piracetam 1200 mg every 8 hours, and 2 bags packed red blood cell (PRBC) transfusion. On daily follow up, there were no further episode of seizure, pain was managed with medication, and wound were clean. The only complain left was about mild cramps. After 2 bags of PRBC transfusion, haemoglobin increased to 94 g/L.

Patient was discharged on day 3 of care with take-home medications of oral Phenytoin 100 mg every 8 hours, oral Piracetam 1200 mg every 8 hours, and Flunarizine 5 mg every 12 hours. Patient returned to Neurosurgery Clinic after 10 days, there were no seizure, headache resolved, and stitches were removed. On follow up, patient returned with laboratory result of low PTH (0.4 pmol/L, normal range 1.6-6.9 pmol/L), low serum calcium (1.55 mmol/L, normal range 2.1-2.60 mmol/L) and high serum phosphate (2.20 mmol/L, normal range 0.8-1.50 mmol/L) and referred to Internal Medicine for hypoparathyroidism, managed with oral calcium carbonate 500 mg every 8 hours and calcitriol 0.5 μ g/day.

DISCUSSION

Unsupervised seizure patient is at high risk of physical injury. After upper extremity fracture, head injury is the second most common injuries in seizure-related injury.¹ Our patient presented with mTBI after an unsupervised seizure. Performing a CT-Scan in mTBI is mandatory when CCTHR indications are met, regardless of the preceding event that causes the injury.⁴ In our institution, neuroimaging is not a

routine procedure in seizure without neurological deficit. We referred to CCTHR to perform CT-Scan in this case, with unexpected finding of bilateral symmetric brain calcifications (Figure 3).

There are various etiologies of brain calcification such as infections and calcified brain tumors, but these calcifications are not bilaterally nor symmetrically configured in striopallidodentate structures.³ Asymptomatic bilateral symmetric basal ganglia calcification are common incidental findings in head CT-Scan of middle-aged patient, but most are confined in globus pallidus in small configuration.⁵ Fahr's Disease (FD) has the same neuroimaging finding as FS. The difference between FS and FD is in the etiology, where FD is associated with idiopathic hereditary conditions such as gene mutations, in the absence of secondary metabolic causes.⁶

Fahr's Syndrome refers to secondary bilateral calcification of striatum, basal ganglia, and dentate nucleus due to metabolic causes.^{2,3} Common etiology of FS in adulthood includes hypoparathyroidism.^{2,6} Parathyroid hormone promotes the release of calcium from bones into bloodstream, increases kidney calcium reabsorption, and increases intestinal absorption.⁶ Seizures are a common clinical manifestation in FS.^{2,3} Cramps, positive Trousseau's sign (carpopedal spasm), and prolonged QT intervals on ECG that are the signs of hypocalcemia.^{2,6} Our patient presented with extensive bilateral and symmetric calcifications on striatum, globus pallidus, and dentate nucleus, with findings of hypoparathyroidism, hypocalcemia, and history of total thyroidectomy, highly supporting diagnosis of FS.

Hypoparathyroidism is common after total thyroidectomy procedure, due to accidental damage in parathyroid gland or its vasculature.^{2,6} This disrupts the calcium-phosphate homeostasis, causing hypocalcemia and pathological calcium deposition in vascular and perivascular of metabolically active regions such as the striopallidodentate structures, which could impair tissue perfusion and impairs neuronal circuit.^{7,8}

After parathyroid gland removal, untreated patient started to develop asymptomatic basal ganglia calcification in a median of 17 years, and hypoparathyroidism diagnosis in a mean of 30 years.^{2,5} In our case, symptoms appear 24 years and full diagnosis was made 30 years after total thyroidectomy. Removal of the micro-calcification is impossible, and treatments are focused on seizure control with ASM while limiting the disease progression by treating the underlying cause.⁶ In postoperative hypoparathyroidism with PTH levels below 10-15 pg/ml, oral supplementation of calcium and calcitriol are recommended.⁷ This patient had a good short-term outcome and seizure was successfully managed with ASM.

CONCLUSION

This case illustrates a clinical cascade from total thyroidectomy leading to unrecognized chronic hypoparathyroidism, hypocalcemia, and subsequent striopallidodentate calcification consistent with FS, ultimately manifesting as recurrent seizures and seizure-related head injury. Because the clinical manifestations of Fahr's syndrome are often nonspecific, the diagnosis may be overlooked. This report emphasizes that

neuroimaging and appropriate biochemical evaluation should be considered in seizure patients, particularly when trauma or atypical neurological signs are present. Management of FS is symptomatic and underlying metabolic disorder should be addressed. Additionally, patients with recurrent seizures require constant supervision to reduce the risk of preventable physical injuries.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient(s) included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The author declare no conflicts of interest.

Financial Disclosure

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Author Contributions

The author is solely responsible for the conception, data collection, analysis, and writing of this manuscript.

REFERENCES

- Mühlenfeld N, Störmann P, Marzi I, et al. Seizure related injuries- Frequent injury patterns, hospitalization and therapeutic aspects. *Chin J Traumatol.* 2022;25(5):272-276. doi:10.1016/j.cjtee.2021.10.003
- Marinković DM, Dragović T, Kiković S, Kuzmić-Janković S, Đuran Z, Hajduković Z. Fahr's syndrome and idiopathic hypoparathyroidism: a case report. *Vojnosanitetski Pregled.* 2026;74(2):184-188. doi:10.2298/VSP150916109M
- Berrabeh S, Messaoudi N, Elmehraoui O, et al. Hypoparathyroidism and Fahr's syndrome: a case series. *Cureus.* 2023;15(6):e40502. doi:10.7759/cureus.40502
- Piwowarczyk S, Oblój P, Janicki Ł, Kowalik K, Łukaszuk A, Siemiński M. Seizure-related head injuries: a narrative review. *Brain Sci.* 2024; 14(5):473. doi:10.3390/brainsci14050473
- Kalampokini S, Georgouli D, Dadouli K, et al. Fahr's syndrome due to hypoparathyroidism revisited: a case of parkinsonism and a review of all published cases. *Clin Neurol Neurosurg.* 2021;202:106514. doi:10.1016/j.clineuro.2021.106514
- Donzuso G, Mostile G, Nicoletti A, Zappia M. Basal ganglia calcifications (Fahr's syndrome): related conditions and clinical features. *Neurol Sci.* 2019;40(11):2251-2263. doi:10.1007/s10072-019-03998-x.
- Shoback DM, Bilezikian JP, Costa AG, et al. Presentation of hypoparathyroidism: etiologies and clinical features. *J Clin Endocrinol Metab.* 2016;101(6):2300-2312. doi:10.1210/jc.2015-3909
- Kaygisiz S. Our cases of Fahr's disease and review of the literature. *Med Sci | Int Med J.* 2025;14(2):325. doi:10.5455/medscience.2025.03.077