



Images in Clinical Medicine

Idiopathic intracranial calcification

Kalyan Mansukhbhai Shekhda^{a*}, Paul Tobin^b, Surendra Kumar Gupta^b, P. S. Sridhar^b

^aDepartment of Diabetes, Endocrinology and Medicine, Southend University Hospital NHS Foundation Trust, Southend On Sea, England, United Kingdom, ^bDepartment of Medicine, Glangwili General Hospital, Carmarthen, Wales, England, United Kingdom

Submission : 22-May-2020

Revision : 29-Jun-2020

Acceptance : 02-Jul-2020

Web Publication : 25-Aug-2020

A 66-year-old man was admitted to hospital following an episode of hematemesis and collapse. On examination, he was confused, but there was no focal neurological deficit present. Because of this confusion and collapse, a computed tomography (CT) scan of the brain was requested. It showed extensive calcification of intracranial structures, namely, cerebellum [Figure 1a], basal ganglia [Figure 1b], and cerebral hemisphere [Figure 1c]. We investigated him for possible causes of calcification, including full blood count, autoimmune profile, bone profile, urinary ceruloplasmin levels, parathyroid hormone levels, and USS of parathyroid glands. All of the investigations were normal, and there was no family history of note. The patient was diagnosed with Fahr's disease. Fahr's disease is a rare genetic disorder characterized by abnormal calcification of brain structures, most commonly the basal ganglia, cerebellum, and the cerebral cortex [1]. It can be asymptomatic or can present with movement abnormalities,

pyramidal signs, cognitive impairment, and neuropsychiatric symptoms. Before diagnosing Fahr's disease, it is important to exclude other causes of intracranial calcification such as endocrinopathies (Hypercalcemia, hypoparathyroidism, and hyperparathyroidism); Wilson's disease; tuberous sclerosis; mitochondrial myopathies; and infections such as tuberculosis and brucellosis [2]. If any of these are present, then its called Fahr syndrome. There is no specific treatment for Fahr's disease, and so management is focused on symptom control with regular follow-up in the clinic. Treatment of Fahr syndrome consists of treating the underlying cause [3].

Declaration of patient consent

The authors certify that they have obtained appropriate patient consent form. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his

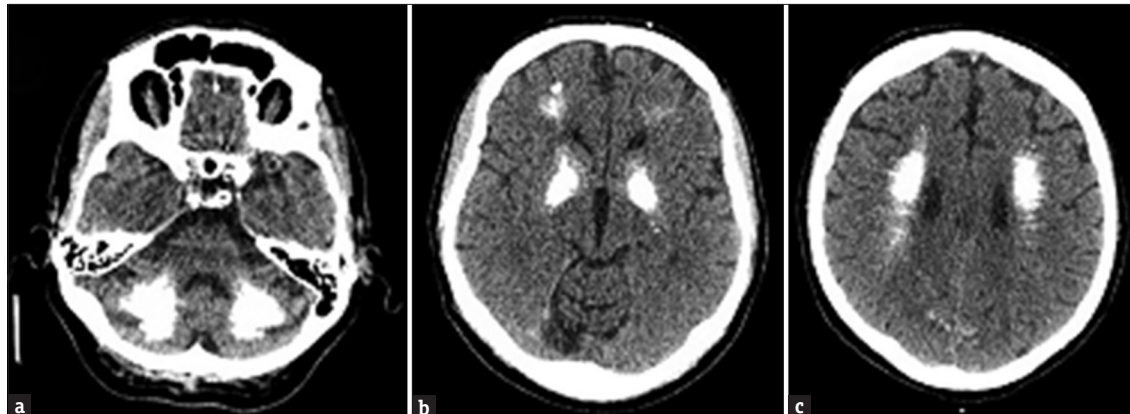


Figure 1: Computerized tomography of the brain images of a patient with calcification of bilateral cerebellum (a), basal ganglia (b), cerebral hemispheres (c)

*Address for correspondence:

Dr. Kalyan Mansukhbhai Shekhda,
Department of Diabetes, Endocrinology and Medicine, Southend University
Hospital NHS Foundation Trust, Southend on Sea, SS00RY, England,
United Kingdom.
E-mail: kalokly@gmail.com

Access this article online

Quick Response Code:



Website: www.tcmjmed.com

DOI: 10.4103/tcmj.tcmj_127_20

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: WKHLRPMedknow_reprints@wolterskluwer.com

How to cite this article: Shekhda KM, Tobin P, Gupta SK, Sridhar PS. Idiopathic intracranial calcification. Tzu Chi Med J 2021; 33(1): 96-7.

name and initial will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Manyam BV. Bilateral striopallidodentate calcinosis: A proposed classification of genetic and secondary causes. *Mov Disord* 1990;5(Suppl 1):94.
2. Perugula ML, Lippmann S. Fahr's disease or Fahr's Syndrome? *Innov Clin Neurosci* 2016;13:45-6.
3. Avrahami E, Cohn DF, Feibel M, Tadmor R. MRI demonstration and CT correlation of the brain in patients with idiopathic intracerebral calcification. *J Neurol* 1994;241:381-4.