

## Intracranial Calcification in an Elderly Woman with Carpopedal Spasm

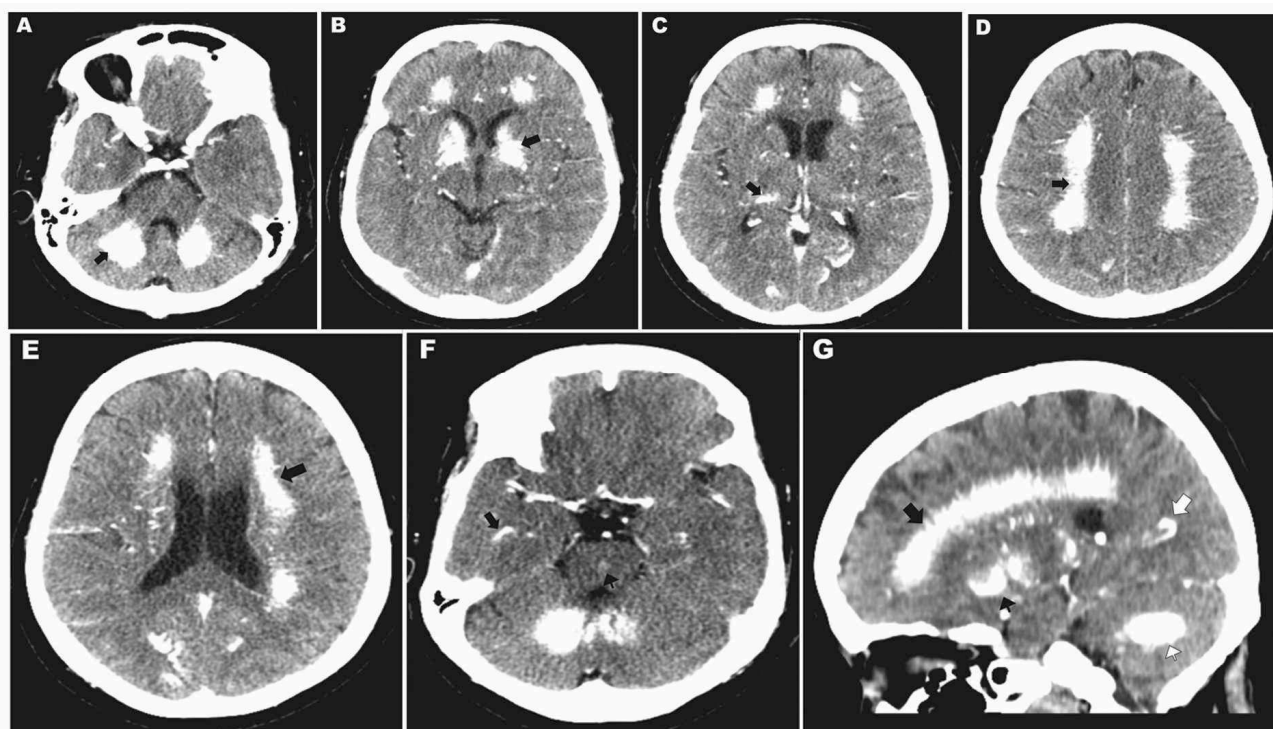
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A 66-year old woman presented to us with syncope and carpopedal spasm. The history of her condition started 10 years ago as recurrent loss of consciousness associated with carpopedal spasm occurring at 2-3 times in a year. A medical consult led to a diagnosis of hypocalcemia, and she has since been maintained on oral calcium supplementation. She was admitted to our center after her most recent syncopal attack which lasted for 20 minutes. At the emergency department, she had spontaneous recovery of consciousness without neurologic sequelae. Laboratory investigation showed normal complete blood count, serum sodium, potassium, creatinine and thyrotropin results. The assays for ionized calcium at 0.6 mmol/L (nv -1.0-1.3) and serum magnesium at 1.7 mg/dL (nv 1.8-2.4) were low. Neither the electrocardiogram

nor electroencephalogram showed any abnormality. Computed tomography scan of the brain demonstrated diffuse bilateral parenchymal calcification most prominent in both cerebro-cerebellar hemispheres and basal ganglia (Figure 1). She was given calcium gluconate intravenously and maintained on calcium and magnesium supplements and calcitriol per orem.

The patient is hypertensive and has a regular angiotensin receptor blocker. The patient underwent a subtotal thyroidectomy in 1975 for nodular goiter. She denies history of diabetes, cardiac, pulmonary, renal or gastrointestinal diseases. She also denies the same medical condition in the family.



**Figures 1A-F** Axial view of a Cranial CT scan showing extensive calcification of both cerebellar hemispheres (arrow) in Panel A, bilateral capsuloganglionic regions (arrow) in Panel B, bilateral thalami (arrow) in Panel C, bilateral centrum semiovale (arrow) in Panel D, bilateral corona radiata (arrow) in Panel E and at temporal (arrow) and pons (arrowhead) in Panel F. **Figure 1G** is a sagittal view showing feathery calcification of centrum semiovale (black arrow), basal ganglia (black arrowhead), occipital (white arrow) and cerebellar regions.

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The clinical and imaging presentation of our case is compatible with the Fahr's syndrome. Fahr's syndrome refers to a rare neurodegenerative entity characterized as symmetric polytopic calcifications in one or more of the following areas: basal ganglia, cerebral white matter, thalami, internal capsulae, and cerebellum, which can lead to pyramidal, extrapyramidal, cerebellar symptoms, alteration of sensitive perception and psychiatric manifestations.<sup>1-6</sup> There is no cure for Fahr's Syndrome, nor is there a standard course of treatment.<sup>7</sup> Treatment addresses symptoms on an individual basis.<sup>3,8</sup>

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