Basal ganglia calcification

Rushad D Patell, Rupal V Dosi, Harshal K Joshi, Purav C Shah

Department of Medicine,
Medical College Baroda,
Vadodara, Gujarat, India

Correspondence to
Dr Rushad Dhunrmi Patell,
rushadpatell@gmail.com

DESCRIPTION

A 65-year-old man presented to the emergency room after a fall, sustaining a head injury. A CT of the head showed diffuse, symmetric parenchymal calcifications involving the thalami globus palladi and caudate heads (figures 1 and 2). Detailed evaluation revealed a long-standing history of muscle cramps, involuntary choreiform movements and gradual cognitive decline. His medical history was unremarkable. On examination, Chvostek and Trousseau signs, tremor and rigidity were present. Laboratory studies included 6.3 mg/dL serum calcium (normal range, 8.4–10.5), 0.89 mmol/L ionised calcium (normal range, 1.13–1.32), 6.6 mg/dL phosphate (normal range, 2.7–4.5), 3.2 g/dL albumin (normal range, 3.5–5.3) and 15 pg/mL intact parathyroid hormone (normal range, 10–65). A diagnosis of hypoparathyroidism was made; however, no cause could be identified. Treatment with 0.5 µg calcitriol daily and 1000 mg calcium carbonate was begun. A month later, tetany was no longer evident and serum calcium was to 9.0 mg/dL. A partial recovery of cognitive functioning and improvement of involuntary movements was also noted.

Bilateral basal ganglia calcification has been described with chronic hypocalcaemia, carbon monoxide poisoning, anoxia, tuberous sclerosis, cytomegalic inclusion disease, toxoplasmosis, therapeutic radiation, epilepsy and methotrexate treatment and rarely familial, Fahr's syndrome.¹ Hypocalcaemia due to low parathyroid levels occurs secondary to destruction of parathyroid glands (autoimmune or surgical), abnormal parathyroid development or an altered regulation of hormone production and secretion and may be idiopathic.² Basal ganglia calcification has been

Figure 2 CT scan of the brain shows diffuse, symmetric parenchymal calcifications involving the head of the caudate nuclei, thalami and globus palladi.

correlated with parkinsonism, movement disorders (hemiballismus, chorea, athetosis) or dementia but may often remain asymptomatic.³ Calcium and vitamin D supplementation remain the mainstay of management for parathyroid insufficiency.²

Learning points

- ▶ Chronic hypocalcaemia, especially hypoparathyroidism, can lead to intracranial calcification especially in the basal ganglia.
- ▶ Calcification of basal ganglia may be asymptomatic but can lead to movement disorders and cognitive decline.
- ▶ Calcium and vitamin D supplementation remain the mainstay of management of hypoparathyroidism.

Contributors All authors were involved in patient care as well as in preparation of the manuscript.

Competing interests None.

Patient consent Obtained.

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- 3 Rastogi R, Beauchamp NJ, Ladenson PW. Calcification of the basal ganglia in chronic hypoparathyroidism. *J Clin Endocrinol Metab* 2003;88:1476.

Figure 1 CT scan of the brain shows diffuse, symmetric parenchymal calcifications involving the head of the caudate nuclei, thalami and globus palladi.

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