Rocks in the head: extensive intracranial calcification

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Basal ganglia calcification is one of the features of chronic hypocalcaemia.¹ However, extensive intracranial symmetrical calcifications outside the basal ganglia have been reported rarely in patients with chronic hypocalcaemia secondary to post-surgical hypoparathyroidism.

A 62-year-old woman who underwent subtotal thyroidectomy 15 years ago presented with long-term paraesthesia in the distal extremities. Progressive cognitive impairment and unstable gait were noted by her family but she had no tetany, cramps or seizures. On examination, she had disorientation to time, place and person, and her vital signs were stable. Chvostek’s and Trousseau’s signs were negative and neurological examination was normal. Plasma biochemical analysis was as follows: urea nitrogen 4.6 mmol/L, creatinine 71 µmol/L, calcium ions 0.68 mmol/L, magnesium 0.90 mmol/L, phosphorus 1.26 mmol/L and intact parathyroid hormone less than 9.5 ng/L. Other analytes were within normal range. Noncontrast computed tomography of the brain is shown in Figure 1. After 10-day therapy with oral calcium carbonate 1.5 g and calcitriol 0.5 µg daily, her chronic paraesthesia resolved and plasma calcium rose to 0.90 mmol/L.

Although pathologic intracranial calcification occurs with tumours, aneurysms, arteriosclerosis and several infectious diseases, the finding of bilateral symmetrical calcification of the basal ganglia, dentate nuclei and corona radiata is characteristic of chronic hypocalcaemia associated with hypoparathyroidism.² A similar pattern of intracranial calcification may also be seen in chronic renal failure with hypocalcaemia and secondary hyperparathyroidism. The pathogenesis of intracranial calcification remains unknown. Possible determinants of extra-osseous calcification include the calcium phosphate product, metabolic acidosis, local pH change, expression of osteopontin and matrix Gla protein, and inflammation or tissue injury.³

Although chronic hypocalcaemia due to hypoparathyroidism may appear to be asymptomatic or, as in our patient, to be associated with relatively mild symptoms (paraesthesias, with cognitive and gait disturbances), replacement therapy with calcium and vitamin D supplements is warranted to prevent progression to more severe manifestations (extrapyramidal signs, muscle spasm, seizures, psychosis). The calcium level should be kept at or slightly below the normal range to avoid potential side-effects including hypercalciuria and nephrolithiasis.

REFERENCES
Noncontrast computed tomography of brain shows extraordinary, widespread, symmetrical parenchymal calcifications in the common location of (a) basal ganglia, (b) dentate nuclei of the cerebellum and (c) corona radiata.