Dystrophic calcification in an old cerebral infarction

Ian C Duncan
FF Rad (D) SA

Basil J Sher
FC Rad (D) SA

Sunninghill Hospital
Sandton

Case report

A 61-year-old female patient presented with severe mitral regurgitation and left ventricular outflow obstruction, treated by mitral valve replacement and cardiac myomectomy respectively. The immediate postoperative period was complicated by multiorgan failure and a right-sided frontal lobe cerebral infarct probably on a cardioembolic basis (Fig. 1). She made a good recovery from all of these post operative complications. Eighteen months later she presented again with transient bilateral recurrent visual field defects, headache and vertigo with nausea and vomiting. As part of the neurological imaging work-up an unenhanced computed tomographic (CT) scan of the brain was performed which showed no evidence of any recent haemorrhage or recurrent infarction, but which showed the uncommon finding of dystrophic calcification within the old right frontal infarct (Fig. 2).

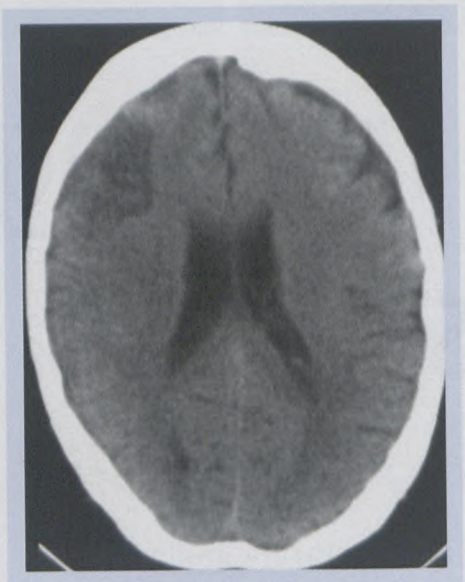

Fig. 1. Unenhanced axial CT scan showing a non-haemorrhagic infarct in the right frontal lobe.

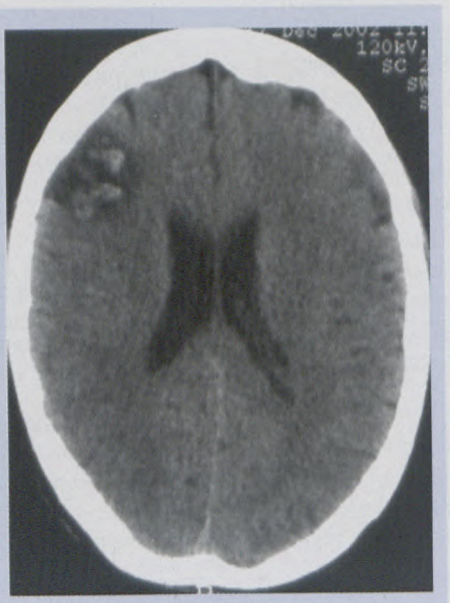

Fig. 2. Unenhanced axial CT scan done 18 months later showing areas of dystrophic calcification following a gyriform pattern.

Discussion

Dystrophic calcification within cerebral infarcts is an uncommon phenomenon. The first reported cases in the radiological literature date back to 1984[1] and 1985[2] where a total of six cases were identified by means of CT scanning. Calcification within the infarcted territories was found on CT scans performed between 9 months and 5 years after the initial ictus. In none of these six cases was the serum calcium level elevated although in two cases a low serum calcium had been recorded on several occasions. In 1988, Parisi *et al.*[3] described a 60-year-old man who presented with a large left frontoparietal infarction. A follow-up CT scan done 18 days after the initial presentation showed areas of hyperintensity within the infarct thought initially to represent haemorrhagic transformation. As a result the anticoagulation therapy was stopped whereafter the patient continued to deteriorate and eventually died 4 days later. Autopsy of the brain showed no evidence of recent haemorrhage but did demonstrate the presence of calcium salts throughout the infarcted area, particularly within the grey matter at the margins of the infarct. Again no significantly elevated serum calcium levels were ever found during the period of hospitalisation. The importance of this particular case was the rapidity of onset of the CT hypertensity and the initial mistaken diagnosis of haemorrhagic transformation of the infarct although such haemorrhagic transformation must still represent the commonest cause of a change within any recently infarcted area to a hyperintense appearance on CT scans, particularly in a patient on anticoagula-

Table I. Causes of intracranial calcification

Physiological

Pineal gland
Habenular commissure
Choroid plexus
Dural, cerebral falx
Idiopathic basal ganglia calcification
Ligamentous calcification

Pathological

- Arteriosclerosis
- Cerebral aneurysm
- Intracranial vascular malformation
- Previous haematoma including subdural haematoma
- Tumour: craniopharyngioma, oligodendroglioma, chordoma, meningioma, choroid plexus papilloma, dermoid, ependymoma, astrocytoma, lipoma of the corpus callosum, pituitary adenoma, medulloblastoma, metastases
- Infections: tuberculoma, cysticercosis, toxoplasmosis, cytomegalovirus, rubella, herpes, HIV, healed abcess
- Fahr syndrome
- Sturge-Weber syndrome
- Tuberous sclerosis, Down's syndrome, MELAS, Cockayne syndrome, neurofibromatosis
- Hypercalcaemia: Parathyroid disease, renal failiure
- Hypoparathyroidism, pseudohypoparathyroidism
- Carbon monoxide, lead poisoning
- Systemic lupus erythematosus
- Post cerebral irradiation or methotrexate therapy

tion therapy.

The exact pathophysiologic mechanism by which this dystrophic calcification occurs remains unknown. Hypercalcaemia does not appear to play any role in its development. CT is far more sensitive than plain film imaging in detecting calcification and many more calcified areas or lesions can thus be identified with CT imaging. Other known causes of cerebral calcification are listed in Table I.

To date no reports concerning the magnetic resonance imaging (MRI) appearance of dystrophic calcification in cerebral infarcts have appeared. Again given the T1 hyperintense signal of so-called 'wet calcium' within a tissue or the T2 hypointense signal sometimes seen in areas of calcification, the MR appearance could also be confused with subacute or chronic haemorrhage within an infarct.

In conclusion, this case represents an example of rare dystrophic calcification within a cerebral infarct. As shown in the literature the onset of such calcification can occur within days of presentation and must be differentiated from haemorrhagic transformation in this setting.

References

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3. Parisi J, Place J, Nag S. Calcification in a recent cerebral infarct — Radiologic and pathologic correlation. *Can J Neurol Sci* 1988; **15:** 152-155.