

Short report

# The significance of the incidental finding of basal ganglia calcification on computed tomography

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**SUMMARY** Basal ganglia calcification was found as an incidental finding in 42 out of 7000 patients who underwent computed tomography. The calcification showed on plain skull radiography when the maximum density on computed tomography exceeded 100 Hounsfield units. The 26 patients with basal ganglia calcification detected on computed tomography who were available for follow-up, were investigated with matched controls. No clinical features of basal ganglia calcification were noted. Twenty-four patients had no significant metabolic abnormality and two patients had parathyroid disorder identified.

Basal ganglia calcification identified radiographically has been associated with any one of 24 conditions<sup>1, 2</sup> (table) with treatable parathyroid disease being the most common of these associations.<sup>3</sup> Computed tomography (CT) has identified basal ganglia calcification more sensitively than plain skull radiographs,<sup>2, 4-9</sup> but the incidence of disease including parathyroid disorder in the CT group has been less.<sup>2, 4-7</sup> This study was carried out to correlate radiographically basal ganglia calcification on CT with that on plain skull radiographs; to attempt to define any systemic metabolic mechanism of basal ganglia calcification formation; and to investigate the possibility of early detection of treatable parathyroid disease.

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## Patients and methods

The CT scans of patients who had attended the Institute of Neurological Sciences from 1975 to 1980 were reviewed for evidence of basal ganglia calcification. From approximately 7000 patients, 15 778 scans were obtained and 42 cases of basal ganglia calcification were found. Sixteen had died or were lost to follow-up. The 26 remaining patients were matched for age, sex, clinical diagnosis and drug treatment with controls. All patients and controls had the following investigations: full clinical assessment; a lateral, a 20° AP, and Towne view skull radiograph were taken on a Siemens CRT 7 using the 0.3 mm focal spot. CT was performed on an EMI head scanner using a 160 × 160 matrix (except for the first 1½ years when the matrix was 80 × 80).

The presence or absence of relevant calcification was recorded, its distribution noted and the average and maximum density in Hounsfield units (H) was determined for each region of calcification. Venous blood was taken without haemostasis and casual urine samples were obtained from fasting, rested patients. Serum electrolytes urea, creatinine, calcium, phosphate, total protein

Table 24 conditions described<sup>1, 2</sup> in association with radiographically identified basal ganglia calcification

Idiopathic hypoparathyroidism	Cockayne's syndrome
Secondary hypoparathyroidism	Lipoid proteinosis (hyalinosis cutis)
Pseudohypoparathyroidism	Tuberose sclerosis
Pseudo-pseudohypoparathyroidism	Parkinsonism
Hyperparathyroidism	Vascular disease
Hypothyroidism	Cerebral haemorrhage
Birth anoxia	Radiation therapy
Carbon monoxide intoxication	Methodrextate therapy
Lead intoxication	Cytomegalic inclusion disease
Fahr's syndrome (ferrocalcinosis)	Encephalitis (measles, chicken pox)
Familial idiopathic symmetrical basal ganglia calcification	Toxoplasmosis
Hastings-James syndrome (idiopathic lenticulo-dentate calcification)	Cysticercosis

albumin, bilirubin, alkaline phosphatase, transaminases, gamma GT, creatine kinase, urates, cholesterol and magnesium were measured by standard techniques as were urinary electrolytes, urea, creatinine, calcium, phosphate and magnesium. Serum calcium was corrected for albumin,<sup>10</sup> urine calcium/creatinine ratio and renal threshold phosphate concentration (TmPO<sub>4</sub>/GFR)<sup>11</sup> were derived.

## Results

The diagnosis of the patient group was most frequently epilepsy, headache, cerebral infarction or dementia. No clinical evidence of basal ganglia disease was found in the patient group. Features of idiopathic hypoparathyroidism and pseudohypoparathyroidism were each found in one patient. Of the eight epileptic patients, one had cataracts, macular degeneration, sensori-neural deafness, psychoneurosis and hypertension with negative family history; another two had had birth anoxia. One male with optic atrophy had low intelligence. All but nine patients were receiving drugs: most commonly these were anticonvulsants, diuretics or steroids.

Plain skull radiographs showed basal ganglia calcification in eight out of 26 patients. There was no correlation between the distribution of basal ganglia calcification on CT and underlying disease. Fifteen patients had more or less identical density of basal ganglia calcification in each hemisphere; two more were significantly denser in the right hemisphere and seven more were denser in the left hemisphere (a significant difference being greater than 20H). In general basal ganglia calcification showed on the plain skull radiographs where the maximum density on CT was greater than 100H and where average density was greater than 65H. The only exception was one patient who had a maximum density of 211H and average density of 71H with negative plain skull radiographs; this was explained by the very small area of calcification involved.

There was no significant difference between the patient and control groups in any of the direct and derived biochemistry, except for the serum albumin, where the control group had a lower range than the patient group ( $p < 0.002$ ), but both were within the normal range. Of all other indices, the following were normal: serum electrolytes, urea, creatinine, urate, cholesterol, bilirubin, transaminases and lactate dehydrogenase. Elevated blood glucose was present in five patients with either idiopathic or steroid-induced diabetes. Elevated serum gamma GT was found in five patients who were either on anticonvulsant therapy or were regular alcohol drinkers and the same patients had elevated serum alkaline phosphatase levels (the increased isoenzyme being of

liver origin.) One patient had biochemistry typical of idiopathic hypoparathyroidism and one of pseudohypoparathyroidism. There were isolated mild abnormalities of either serum phosphate, magnesium TmPO<sub>4</sub>/GFR or urine calcium/creatinine ratio in nine patients not suggestive of significant metabolic disease.

## Discussion

Anticonvulsant therapy has been linked with basal ganglia calcification<sup>4</sup>; however, there is no evidence that anticonvulsant therapy *per se* causes such calcification. Of our eight epileptic patients, all taking anticonvulsant therapy, two had confirmed parathyroid disorder and a further three had isolated biochemical abnormalities of doubtful significance. Our control group also received anticonvulsant therapy. Osteomalacia has occasionally been described with anticonvulsant therapy but it is not known to be associated with basal ganglia calcification.

Radiologically, where basal ganglia calcification has been identified by CT, the rate of detection by plain skull radiographs ranges from 7-10%.<sup>5 6 9</sup> Our higher proportion of 30% is probably attributable to the fine focus x-ray tube used. It has been stated<sup>2</sup> that where regions other than the globus pallidus were involved, "pathological calcification" existed. We accept that this is frequently the case where metabolic groups are being studied but we did not find this correlation. Indeed our patient with idiopathic hypoparathyroidism had negative plain films and mild calcification of only the globus pallidus, while one of the most florid examples of basal ganglia calcifications on CT with positive plain skull radiographs was a 43-year-old man with headaches, no drug therapy and normal clinical and biochemical assessment. Owing to the superior sensitivity of CT over plain skull radiographs in the detection of basal ganglia calcification, the patient with idiopathic hypoparathyroidism was undoubtedly diagnosed earlier.

## References

- 1 Reader MM, Felson B. In: Reader MM, Felson B, eds. *Gamuts of Radiology*. Oxford: Pergamon Press. 1975: A40.
- 2 Cohen CR, Duchesneau PM, Weinstein MA. Calcification of the basal ganglia as visualised by computed tomography. *Radiology* 1980;134:97-9.
- 3 Sutton D. In: Sutton D, ed. *A textbook of radiology and imaging*. 3rd ed. Edinburgh: Churchill Livingstone, 1980:1101.
- 4 Brannan TS, Burger AA, Chaudhury MY. Bilateral basal ganglia calcifications visualised on CT scan.

- J Neurol Neurosurg Psychiatry* 1980;**43**:403-6.
- <sup>5</sup> Koller WC, Cochran JW, Klawans HL. Calcification of the basal ganglia: computerised tomography and clinical correlation. *Neurology (Minneapolis)* 1979;**29**:328-33.
- <sup>6</sup> Murphy MJ. Clinical correlations of CT scan detected calcification of the basal ganglia. *Ann Neurol* 1979;**6**:507-11.
- <sup>7</sup> Sachs C, Ericson K, Erasmie U, Bergstrom M. Incidence of basal ganglia calcifications on computed tomography. *J Comput Assist Tomogr* 1979;**3**:339-44.
- <sup>8</sup> Smith D, Bloch S, Al-Rashid RA. Basal ganglia calcifications on CT scanning in children with acute lymphocytic leukaemia. *Neuroradiology* 1980;**20**:91-3.
- <sup>9</sup> Danziger A, Talk W, Sandler M, Forman M. Computed tomography in basal ganglia calcification. *Clin Radiol* 1980;**31**:167-8.
- <sup>10</sup> Imrie CW, Allam BF, Ferguson JC. Hypocalcaemia of acute pancreatitis: the effect of hypoalbuminaemia. *Cur Med Res Opin* 1976;**4**:101-16.
- <sup>11</sup> Walton RJ, Bijvoet OLM. Nomogram for derivation of renal threshold phosphate concentration. *Lancet* 1975;**(ii)**:309-10.