CLINICAL IMAGES

COMPUTED TOMOGRAPHY FEATURES OF BASAL GANGLIA AND PERIVENTRICULAR CALCIFICATIONS IN CHILDHOOD

There are numerous congenital, inflammatory, endocrine and toxic causes for basal ganglia and periventricular calcification. The commonest causes are listed in Table I.

This pictorial account shows four common causes of basal ganglia and periventricular calcification, namely cytomegalovirus (CMV) and HIV infection, tuberous sclerosis (TS) and pseudohypoparathyroidism.

HIV (FIG. 1)

HIV is probably the most common cause of basal ganglia and periventricular calcification today. Non-enhanced computed tomography (NECT) shows diffuse cerebral atrophy in 90% of cases. Bilateral, symmetrical basal ganglia calcification is seen in 30% of cases, but virtually never before 1 year of age.[1]

CMV (FIG. 2)

CMV has a particular affinity for the developing germinal matrix. This causes widespread periventricular tissue necrosis with subsequent dystrophic calcification. CMV can, however, also cause widespread parenchymal calcification. NECT will

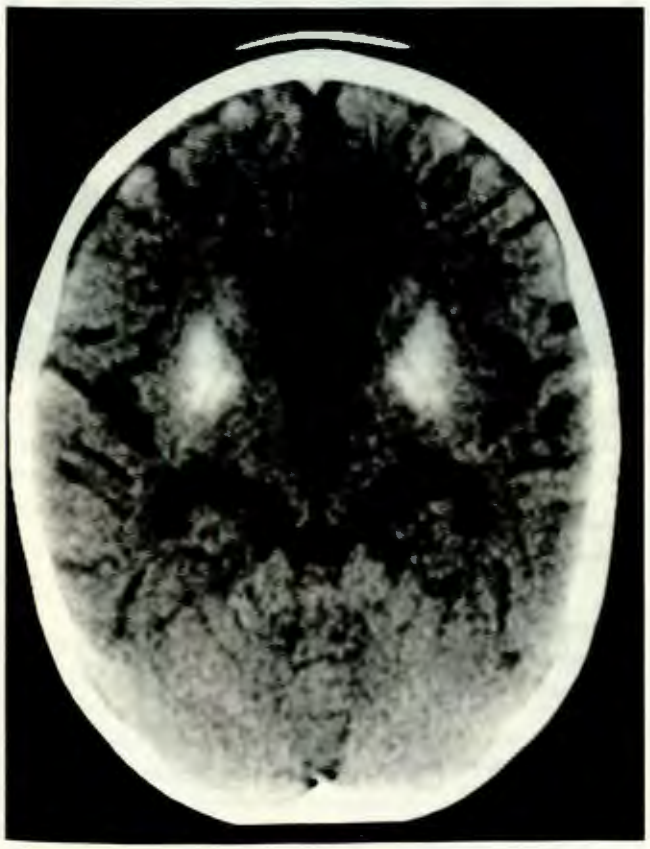

Fig. 1. HIV: bilateral, symmetrical calcification in the basal ganglia with increased central and peripheral cerebrospinal fluid spaces in keeping with cerebral atrophy.

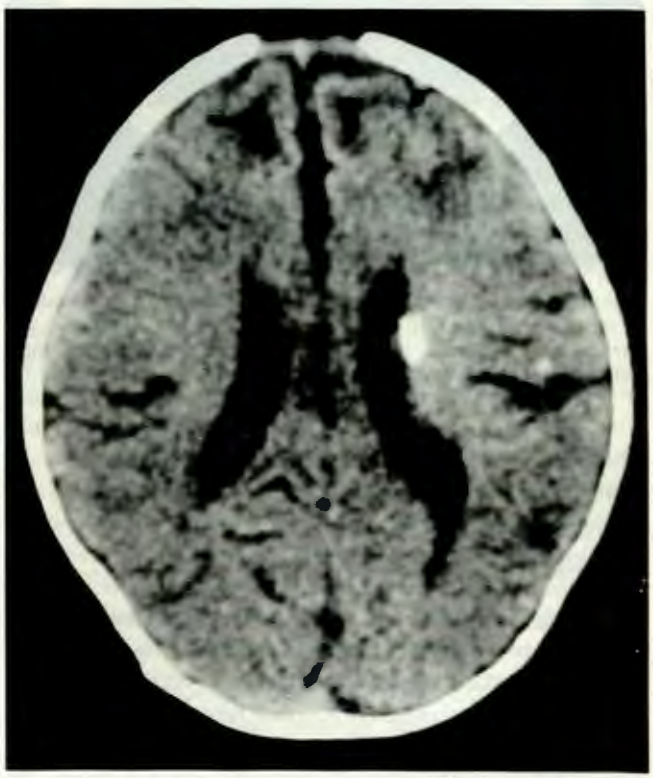

Fig. 2. CMV: a periventricular calcification in relation to the left lateral ventricle and another smaller calcification in the left parietal parenchyma.

Table I. Common causes of basal ganglia and periventricular calcification

Inflammatory	Congenital	Endocrine	Toxic
HIV	Neurofibromatosis	Pseudohypoparathyroidism	Hypoxia
Toxoplasmosis	Tuberous sclerosis		Lead
Cytomegalovirus	Down's syndrome		Carbon monoxide poisoning

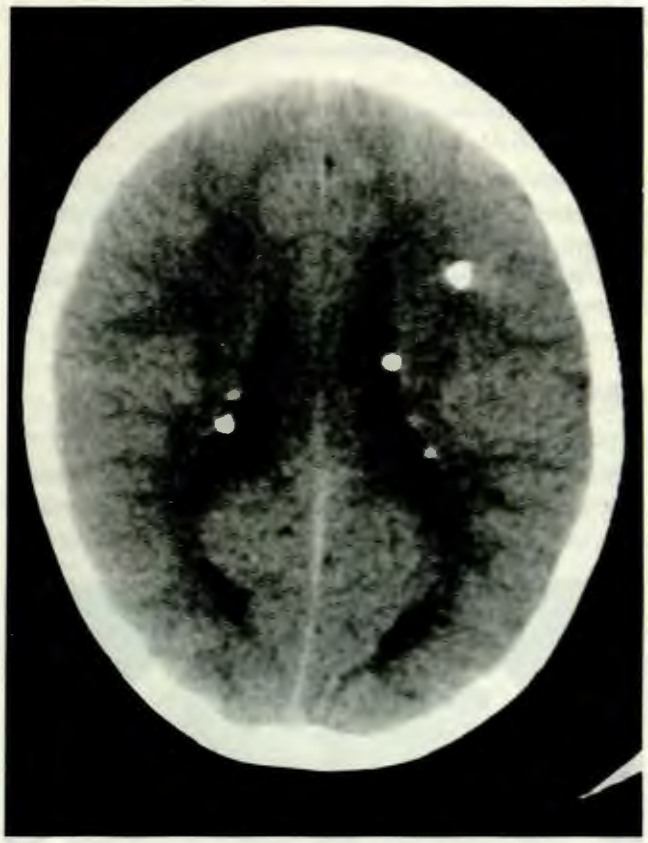

Fig. 3. Tuberous sclerosis: bilateral, round densely calcified nodules in the subependymal area of the lateral ventricles and a single parenchymal calcification which probably represents a calcified white matter lesion.

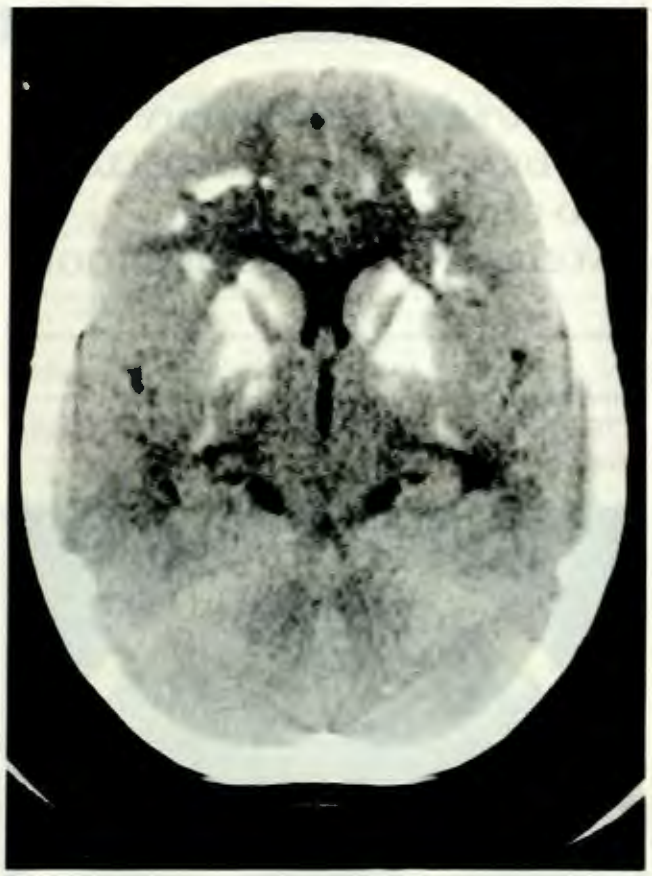

Fig. 4. Pseudohypoparathyroidism: coarse, bilateral, symmetrical basal ganglia, periventricular and parenchymal calcification.

also show atrophy, ventricular enlargement and neuronal migration anomalies.[1,2]

TUBEROUS SCLEROSIS (FIG. 3)

Subependymal hamartomas are found in 95% of patients with TS. Nearly two-thirds are located near the caudate nucleus along the striothalamic groove of the lateral ventricles — just behind the foramen of Monro. Cortical hamartomas are also characteristics of TS. Both subependymal hamartomas and cortical tubers calcify, although rarely before 2 years of age. The calcifications tend to increase in number and density as the patient matures.[2]

PSEUDOHYPOPARATHYROIDISM (FIG. 4)

In this condition there is a lack of response of the end-organs to parathormone (PTH) which results in low serum calcium and high serum phosphate levels. On NECT calcification can be seen in the basal ganglia. These calcifications are bilateral and symmetrical and also occur in the falx cerebri. Calvarial thickening is seen in 30% of cases.[1]

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1. Osborne AG. *Diagnostic Neuroradiology*. St. Louis: Mosby-Year Book, 1994: 93-98, 673-680, 743-745.
2. Barkovich AJ. *Pediatric Neuroimaging*. 2nd ed. Philadelphia: Lippincott-Raven, 1995: 85.