

Short reports

Computed tomography in non-specific mental retardation and idiopathic epilepsy

S LINGHAM AND B E KENDALL

Hospital for Sick Children, Great Ormond Street, London

SUMMARY In 29 children with mental retardation and infantile spasms, cranial computed tomography findings were abnormal in 75% and this finding may lead to modification of treatment or influence genetic counselling. In 41 children with mental retardation and other forms of epilepsy abnormal computed tomography findings were much less frequent and were not relevant to treatment.

A recent study¹ indicated that cranial computed tomography (CT) is unhelpful in the investigation of children with isolated non-specific mental subnormality. The CT findings did not modify treatment and in only 8% of children was a specific abnormality shown so that the investigation added disappointingly little to improving diagnosis in this condition. Children with non-specific mental subnormality and a history of non-focal epilepsy without obvious clinical or genetic explanation form another easily definable group in which we thought assessment of the value of CT would be of interest.

Patients and methods

We examined the clinical records of all children presenting to the neurological department of this hospital over a period of 18 months with mental subnormality and epilepsy without focal neurological deficit. Children with documented birth asphyxia (10), previous intracranial haemorrhage (1), encephalitis (2), prolonged metabolic abnormalities known to cause cerebral damage (2) (hypoglycaemia (1) and jaundice (1)), clinical evidence of a phacomatosis (1), a head circumference of less than the 2nd (12) or greater than the 98th percentile (5), and those with gross dysmorphic features (Pierre-Robin syndrome) (1) were excluded.

Children with mild dysmorphic features that occur not infrequently in the population such as clinodactyly, mild cleft lip (1), and minor hypoplasia (1) were included. There were no karyotype

abnormalities. No child had received adrenocorticotrophic hormone, steroid, or other treatment associated with brain shrinkage.

Epilepsy was classified according to the international classification and grouped according to the predominant type of seizure. Children with infantile spasms were included in the study. The 70 patients who met these criteria and also had good quality CT scans were studied. There were 36 boys and 34 girls whose ages ranged from 3 months to 17 years (mean 5 years). All were moderately or severely retarded with an intelligence or development quotient below 70 and each child aged more than 5 years was educationally subnormal and attended a special school.

An EMI CT 1010 machine was used in the true axial plane. Sections of 0.5 cm were used in babies up to 4 months of age and through the middle fossae in all children. In children aged more than 4 months and in other regions 1 cm sections were used. Plain scans were performed in all children. Unless a radiological abnormality requiring further elucidation was shown on the plain scan, rescanning after intravenous contrast medium was performed only when clinically requested. In this study it was given in one third of the patients. No further radiological studies were performed.

Results

The 3 children (4%) suffering from petit mal and 2 (3%) from myoclonic seizures all had normal CT scans. Thirty six children (51%) aged 3 months to 17½ years (mean 6 years 8 months) had grand mal epilepsy. CT scans were normal in 21 (58%) of these, generalised cerebral atrophy was present in 12 (33%), and there was 1 child with porencephalic cyst and 1 with agenesis of the corpus callosum. One child had diffuse but asymmetrical low density of the hemispheric white matter which progressed to cerebral atrophy and was subsequently shown at necropsy to have a diffuse non-inflammatory

neuronal degeneration, but the aetiology was not established.

Twenty nine children (41%) aged 3 months to 2½ years (mean 9 months) had infantile spasms. Of these, only 7 (24%) had normal scans, 14 (48%) had generalised cerebral atrophy, 3 including 2 with Aicardi's syndrome had agenesis of the corpus callosum, and 2 had tuberous sclerosis. In 3 cases communicating hydrocephalus was found, but ventriculoperitoneal shunting was not indicated.

Discussion

The CT scan was normal in all children with petit mal and myoclonic seizures and is not considered to be indicated in these cases. In grand mal plus retardation the CT scans showed possible aetiological features in 3 children: 1 with porencephalic cyst, 1 with agenesis of the corpus callosum and 1 with cerebral degeneration, but failed to show a treatable pathology or influence treatment. We found that in children with infantile spasms plain CT scans were worth while. Again lesions requiring specific treatment are uncommon, but genetic counselling is required if tuberous sclerosis is shown. In no case in our series did contrast enhancement give additional

information and it is rarely indicated unless an unexpected pathology is shown on plain CT scan.

Conclusion

When infantile spasms occur with non-specific mental retardation CT scanning will show an abnormality in about 75% of cases. Specific abnormalities occur in under one third of these children, few will require definitive treatment, but genetic counselling may be initiated. Some 60% of retarded children with other forms of non-focal epilepsy have normal CT scans and uncommonly show specific, treatable, or genetically transmitted abnormalities.

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References

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Correspondence to Dr B Kendall, The Hospital for Sick Children, Great Ormond Street, London WC1 N3JA.

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Hypophosphataemic rickets in the preterm infant; hypocalcaemia after calcium and phosphorus supplementation

I Z KOVAR, P D MAYNE, AND I ROBBÉ

Department of Child Health and Chemical Pathology, Westminster Medical School and Hospital, London SW1

SUMMARY A preterm infant with hypophosphataemic rickets became hypocalcaemic when given milk specially formulated for preterm infants that contained increased phosphorus and calcium. The rickets resolved spontaneously. Routine calcium and phosphorus supplementation for preterm neonates should be investigated further.

Rickets and poor bone mineralisation in the preterm infant is common and of uncertain aetiology.¹ Human milk, as the sole source of nutrition for these infants, may not provide adequate mineral substrate and supplementary calcium and phosphorus have been advocated.² We report on an infant with rickets of prematurity secondary to phosphorus depletion. Symptomatic hypocalcaemia developed when phosphorus supplementation was

given, while the rickets resolved spontaneously on an un-supplemented diet.

Case report

A baby girl, the second of twins, was born at 30 weeks' gestation, after an unbooked delivery with no antenatal care. She weighed 1420 g. Her sibling was stillborn. The neonatal period was complicated by mild hyaline membrane disease and hyperbilirubinaemia. Feeding from the first week was with mother's expressed breast milk supplemented daily from week 3 with 400 IU of calciferol (BP).

Plasma alkaline phosphatase activity, measured at 4 weeks of age to screen for rickets,³ was 2600 U/l (childhood reference range (CRR), 170-850 U/l). Radiographic examination of the long bones showed no evidence of active rickets, although a