THE EDITORIAL COMMITTEE welcome original papers. They should be addressed to the Editor, Journal of Neurology, Neurosurgery, and Psychiatry, BMA House, Tavistock Square, London WC1H 9JR. Papers are accepted on the clear understanding that the subject matter has not been and will not be published in any other journal. Papers should deal with original matter and the discussion should be limited to relevant questions. Manuscripts should be typewritten in double spacing on one side of the paper only. Three copies (including Figures and Tables) should be submitted of which only one need be a top copy. A summary of about 50 words should appear at the head of each paper. On the paper the name of the author should appear with initials (or distinguishing forename) only, and the name of the hospital or laboratory where the work was performed. Full postal address should also be provided for correspondence and reprints. Receipt of manuscripts will be acknowledged.

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Letter

Idiopathic familial basal ganglia calcification associated with juvenile hypertension

Sir,—We describe a family in which two sisters with idiopathic basal ganglia calcification had severe hypertension, detected at an early age for which no cause was found. The parents (who were not consanguineous) and two brothers, did not have hypertension or basal gangliar calcification. None showed any neurological abnormality.

The younger sister was noted to have a blood pressure of 180/140 mmHg when admitted unconscious during an attack of hypertensive encephalopathy at the age of 16 years. The cerebrospinal fluid was slightly blood stained. Electroencephalogram and carotid angiogram were normal. The skull x-ray showed bilateral dense basal ganglia calcification and this prompted us to investigate the other family members of whom only the sister showed similar skull x-ray changes.

The elder sister was noted to have a blood pressure of 180/120 mmHg at the age of 13 years when admitted for typhoid fever. Both sisters had no neurological deficits and had been healthy with normal physical and mental development and no history of fits or tetany. No obvious cause for the hypertension was found in either case. Renal causes, renal artery stenosis, and coarctations of aorta were excluded. Investigations including intravenous pyelogram, aortogram, renal arteriogram, presacral air insufflation, and retrograde pyelogram were normal. Twenty-four-hour urine excretion of vanillylmandelic acid was normal and rottine test negative. Renal functions were normal. There was no cardiomegaly, and fundoscopy was normal. Renal biopsy done recently showed the changes, due to arteriosclerosis.

Causes for basal ganglia calcification such as hypoparathyroidism and pseudohypoparathyroidism were excluded in both sisters by several years of clinical observation and the absence of physical stigmata such as short metacarpals, cataracts and subcutaneous calcification. Biochemical investigations showed normal calcium metabolism.

Case 1: Serum calcium was 2.52 mmol/l; phosphate 1.5 mmol/l; alkaline phosphatase 42.5 iu/l. Twenty-four-hour urine calcium excretion was 1.01 mmol.

Case 2: Serum calcium was 2.27 mmol/l; phosphate 0.99 mmol/l; alkaline phosphatase 134 iu/l. Twenty-four-hour urine calcium excretion was 2.42 mmol.

Toxoplasma haemagglutination test was negative in both cases. The cases have been followed up for 14 years.

Idiopathic familial basal ganglia calcification is a rare condition. Only about 10 families have been reported. All these families had extrapyramidal symptomatology such as parkinsonism and athetosis associated with epilepsy and mental retardation, but the family described above was unaffected neurologically.

The interesting association of this condition with a peculiar severe hypertension in childhood in the same members of the family has not been described. Whether the hypertension could be related to such calcification is not known. It is possible that this represents a genetic disorder of some unknown metabolic type where hypertension and basal ganglia calcification are associated.

References

1 Bennett JC, Maffly RH and Steinbach HL. Radiology 1952; 72:368.

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Notice

12th World Congress of Neurology to be held 20 September–25 September 1981. Address for further information: Secretariat, 12th World Congress of Neurology, c/o Simul International, Inc., Kowa Bldg. No. 9, 1-8-10, Akasaka, Minato-ku, Tokyo 107, Japan.
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