Hypertensive encephalopathy with CT confirmation in four children with acute renal disease

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Abstract

Hypertensive encephalopathy (HE) is a clinical syndrome that occurs infrequently in children and is often underdiagnosed. We review four patients with HE and describe their clinical presentation and radiological findings on computed tomography (CT). Our cases demonstrate typical features on CT and correlate clinically with the syndrome of HE. Prompt recognition of the syndrome aids in earlier diagnosis and treatment, and hence proves beneficial to the patient.

Introduction

Hypertensive encephalopathy (HE) is a clinical syndrome consisting of rapidly progressive symptoms and signs. It occurs in the setting of acute and/or prolonged systemic hypertension that disrupts cerebral autoregulation. Recent studies involving the pathophysiologic mechanisms of HE have postulated that cerebral vascular vasodilatation from high-pressure autoregulatory failure results in increased vascular permeability and cerebral oedema.[1,2] The clinical features include headache, seizures, visual disturbances, altered mental state and focal neurological signs. Delayed diagnosis can prove fatal for the patient, but with treatment there is usually full recovery.

HE is rare in paediatric patients and frequently presents in conjunction with a systemic disorder. The clinical presentation and CT findings of 4 recently identified paediatric cases with HE as a complication of renal disease are reviewed.

Case 1

A 10-year-old boy presented with headaches, facial swelling, visual impairment, vomiting and a right-sided focal seizure which became generalised. His legs were covered with old impetiginous skin lesions, his AntiDNAse B levels were raised at 480 (titre) (normal < 200) and he was diagnosed with acute post-streptococcal glomerulonephritis. Renal function on presentation was fairly well preserved (urea 7.4 mmol/l and creatinine 61 umol/l), but the blood pressure (BP) at admission was raised at 140/91 mmHg (the 95th percentile for his height is 116/78 mmHg). CT demonstrated low density areas in the subcortical white matter of the cerebellar hemispheres and in the posterior parietal parafalcine regions bilaterally (Fig. 1).

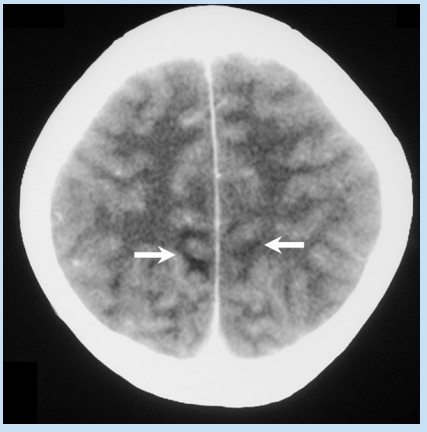

Fig. 1. Axial post-contrast CT scan of the brain shows bilateral parafalcine posterior parietal white matter low densities (arrows) without associated enhancement.

Case 2

A 10-year-old girl presented with acute renal failure and hypertension with a BP of 149/86 mmHg (95th percentile for her height – 118/78

mmHg). She subsequently developed seizures and became progressively obtunded. A history of recurrent pharyngitis was elicited. Renal function on presentation was extremely deranged (urea 65 mmol/l and creatinine 1 095 umol/l). ASOT (antistreptolysin O titre) was > 2 560 IU (normal < 200) and antiDNAse B 1 960 (titre). Renal biopsy showed rapidly progressive post-infective nephritis with crescents. Peritoneal dialysis was required for 7 days and she responded well to steroids and cyclophosphamide. Creatinine at discharge from hospital was 56 umol/l. In view of her seizures, a CT scan was performed. CT showed low-density changes within the white matter of the high occipital and posterior parietal regions on either side of the midline, with focal areas of peripheral enhancement (Fig. 2).

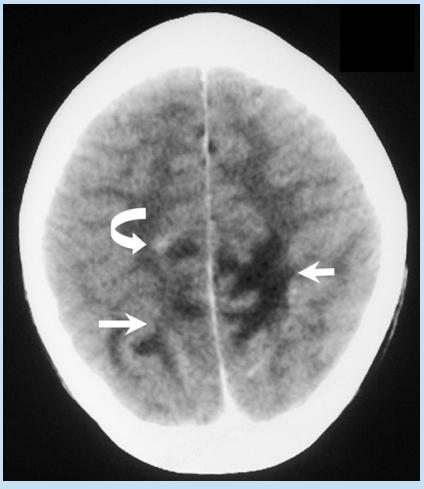

Fig. 2. Post-contrast CT scan of the brain demonstrates bilateral parafalcine areas of low density in the posterior parietal and occipital white matter (straight arrows). There is focal enhancement at the periphery (curved arrow).

Case 3

A 10-year-old boy presented with a 2-day history of generalised body swelling, dark urine, vomiting and headaches and also a past history of skin infections. Clinically, there were widespread impetiginous lesions together with a scabies infestation. At presentation he had a generalised tonic-clonic seizure and was poorly responsive with a BP of 164/107 mmHg (95th percentile for his height – 113/74 mmHg). His renal function deteriorated rapidly and peaked at a urea of 14.5 mmol/l and a creatinine of 114 umol/l, but settled to a urea of 6.2 mmol/l and creatinine of 55 umol/l on discharge 5 days later. AntiDNAseB was greater than 2 560 (titre) and ASOT was 320 IU, and therefore a diagnosis of post-streptococcal glomerulonephritis was made. At CT there were symmetrical white matter hypodensities in the high parietal parasagittal regions (Fig. 3).

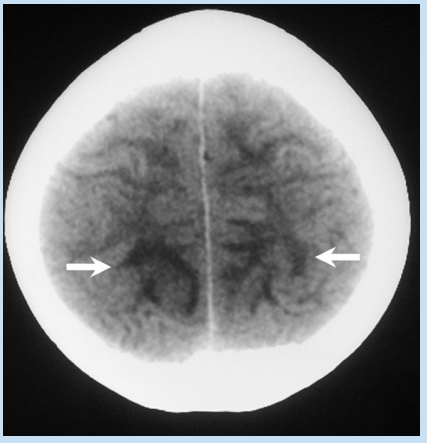

Fig. 3. Axial post-contrast CT scan of the brain shows subtle parasagittal white matter low densities in the high parietal regions (arrows).

Case 4

An 8-year-old boy with acute renal failure as the result of tumour lysis after chemotherapy for Burkitt's lymphoma, presented with a seizure. The BP at the time was 133/102 mmHg (95th percentile for his height - 113/76 mmHg). He also had pulmonary oedema and was clinically fluid overloaded. Urea was 21 mmol/l, creatinine 205 umol/l, uric acid 0.61 mmol/l and lactate dehydrogenase (LDH) 1 300 U/l. He was placed on haemodialysis for a week and recovered his renal function (urea 2.4 mmol/l and creatinine 35 umol/l). CT scan showed bilateral, assymetrical cortical and subcortical hypodensities in the cerebellar hemispheres, as well as in the posterior parietal regions. No associated enhancement was noted (Fig. 4). A repeat CT scan after 2 months demonstrated complete resolution.

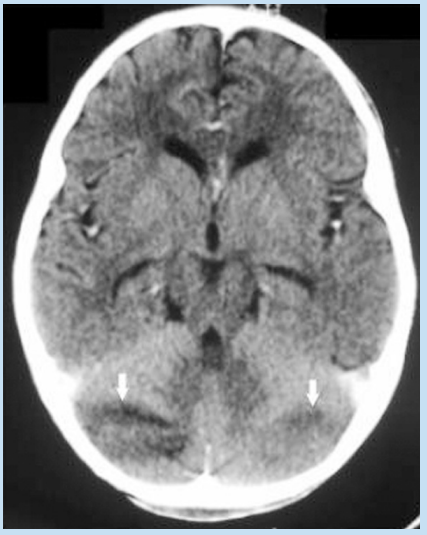

Fig. 4. Axial post-contrast CT scan of the brain demonstrates bilateral assymetrical hypodensities in the cortical and subcortical regions of the cerebellar hemispheres with no associated enhancement (arrows).

Discussion

Although clinical hypertension occurs less frequently in children than in adults, it still has significant consequences. New data for normal BP ranges are available for children and adolescents, and depend on height

percentiles, age and gender.[3] Each of the children in our cases was plotted on these charts and had elevated BPs on presentation. Clinically, they all had features of hypertensive encephalopathy, presenting with seizures.

Typically in HE, cerebral imaging indicates oedema in the posterior temporal, parietal and occipital regions, predominantly in the paramedian subcortical white matter.[4] The oedema appears hypodense on CT and may enhance post contrast. On T2-weighted magnetic resonance imaging (MRI), these areas show increased signal intensity and on diffusion-weighted imaging, there are no features of ischaemia or infarction.[1,2] Abnormalities have also been reported in the parieto-occipital areas, frontal lobes, basal ganglia, brainstem and cerebellum, but occur less often.[2]

Further confirmation of the diagnosis is obtained with resolution of the imaging abnormalities after normalisation of the blood pressure. If the BP is not normalised, irreversible cerebral ischaemia and infarction or haemorrhage may result. It is reasonable to assume that the precise range of cerebral blood flow autoregulation is lower in children than adults. Children may develop HE at a lower absolute BP than adults.

Although several paediatric cases of HE have been reported, a limited number have been documented or illustrated with CT or MRI findings.[1,5-10] The 4 cases reviewed showed characteristic imaging abnormalities of HE on CT. At follow-up all 4 patients were found to have neither neurological symptoms nor any clinical sequelae. However, confirmation was not obtained with a repeat radiological investigation in each case.

In conclusion, HE is often underdiagnosed in children and early recognition of characteristic radiographical findings on CT or MRI allows for the implementation of appropriate antihypertensive therapy with the potential for complete neurological recovery.

References

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