Reversible hypertensive brainstem oedema

A 60-year-old man presented with a 2-week history of generalised fatigue and bilateral lower limb weakness. He had been found to have hypertension during a health screen performed 1 year previously but was not taking any medications. A physical examination revealed a grade-III hypertensive retinopathy. He had full muscle strength and normal reflexes. His orientation and attention levels were satisfactory but his spontaneous speech was reduced. He did not complain of headaches, vomiting, or visual impairment. His initial blood pressure was 231/157 mm Hg. Blood tests showed an elevated serum urea (7.9 mmol/L; reference range, 3.3-7.0 mmol/L) and creatinine (207 μmol/L; 60-120 μmol/L), suggestive of renal insufficiency. His electrolytes and liver enzymes were normal and his vitamin B12 level was reduced (126 pmol/L; reference range, 133-675 pmol/L). His haemoglobin level was 118 g/L (reference range, 130-170 g/L), the red cells were normochromic and normocytic, and the white cell count was normal. A plain computed tomography (CT) of the brain was done to assess his subtle mental dysfunction, and showed diffuse hypodensities in the pons, midbrain and periventricular white matter, sparing the occipital lobes (Fig 1). A lumbar puncture was then performed with an opening pressure of 140 mm H_2O. Examination of his cerebrospinal fluid (CSF) revealed a mild elevation of total protein (0.96 g/L; reference range, 0.20-0.50 g/L), normal cell counts (2/mm^3) and glucose (3.3 mmol/L). His blood pressure was controlled with antihypentensive therapy, and his speech and general well-being significantly improved over the next few weeks. A follow-up plain CT of the brain performed 6 weeks later showed complete resolution of the brainstem lesions (Fig 2).

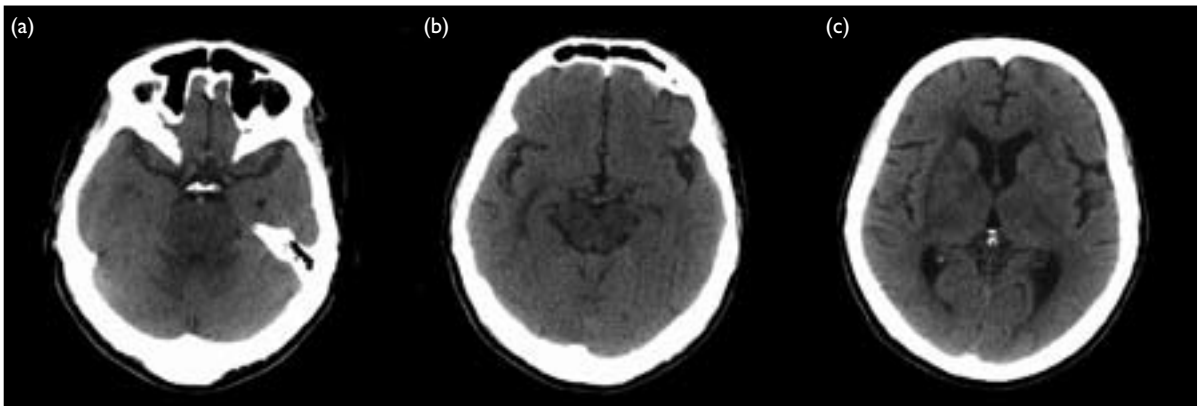

FIG 1. Initial computed tomographic scans of the brain at pons, midbrain and occipital lobe levels show diffuse hypodensity in the pons (a), midbrain (b), and periventricular white matter (c) with sparing of the occipital lobes

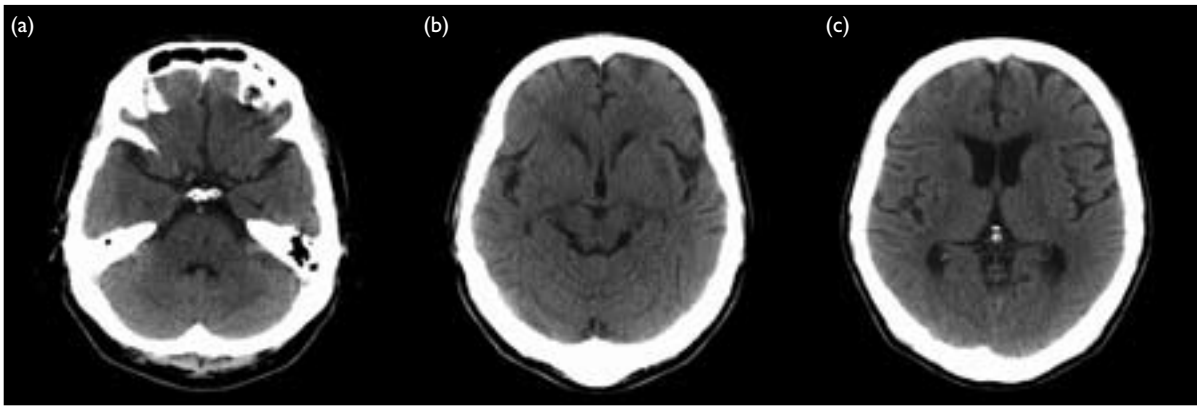

FIG 2. Follow-up computed tomographic scans of the brain at 6 weeks show complete resolution of the pons and midbrain lesions (a, b) with minimal residual changes in the periventricular white matter (c)

The hallmark CT abnormalities seen in hypertensive encephalopathy are bilateral posterior parietal and occipital lobe hypodensities with variable involvement of the brainstem and cerebellum, readily reversible with adequate control of blood pressure.[1] These radiological changes are presumed to be vasogenic oedema from hyperperfusion as a result of disrupted autoregulation in a hypertensive crisis.[2] The predilection for posterior circulation territory involvement in a hypertensive crisis may be explained by the deficient sympathetic innervation of the vertebrobasilar system that predisposes it to plasma extravasation and oedema. This hypertension-induced vasogenic oedema should be distinguished from cytotoxic oedema in acute infarction. While aggressive lowering of the blood pressure is the treatment of choice in the former, the same treatment could be harmful in the latter. A diffusion-weighted magnetic resonance image (DWI) is particularly helpful in this regard. Cytotoxic oedema in acute infarction appears bright on DWI with a low apparent diffusion coefficient (ADC) due to restricted diffusion. Hypertension-induced vasogenic oedema has a high ADC, usually appears normal on DWI, and is bright on the ADC map.[2]

Occasionally, involvement may be confined to the brainstem or cerebellum, sparing the occipital cortex.[3] This variant form of hypertensive encephalopathy may be confused with other serious conditions, including central pontine myelinolysis, brainstem encephalitis, and acute disseminated encephalomyelitis. His normal electrolyte and CSF cell counts made these differential diagnoses unlikely in our patient. The low vitamin B12 level appeared to be just coincidental. Despite the extensive brainstem hypodensity, patients with this syndrome usually do not have prominent focal signs of brainstem dysfunction.[4] This implies that the oedema is relatively benign or reversible. Our patient's symptoms were so mild that it was hardly conceivable that he was experiencing a hypertensive crisis.

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