A 34-year-old man, previously in good health with no past history of hypertension, presented with a 2-day history of bitemporal headaches and a sudden onset of left-sided weakness 4 days after ingesting traditional Chinese medications for non-specific abdominal pain. He was afebrile, drowsy, disoriented, and dysarthric, with a blood pressure of 270/170 mm Hg. Clinical examination revealed mild left facial weakness and strength of Medical Research Council grade 4/5 in the left upper and lower limbs. Deep tendon reflexes were brisk, and he demonstrated bilateral extensor plantar responses. Fundoscopy revealed grade 4 hypertensive retinopathy changes (Figure 1). ECG was consistent with left ventricular hypertrophy. Treatment with intravenous glyceryl trinitrate was commenced to achieve normotension. Blood tests suggested rhabdomyolysis with 127 μmol/L (42–112) creatinine, 11231 U/L (30–350) of creatine kinase, >1000 ng/mL (16–96) of serum myoglobin, and 31.4 U/L (<7.6) of aldolase.

Computed tomography of the brain showed low-attenuation abnormalities in the brain stem and white matter of both cerebral hemispheres, consistent with edema (Figure 2). Likewise, fluid-attenuated inversion recovery sequence of the magnetic resonance brain image revealed asymmetrical hyperintensities within the brain stem, dentate nuclei, inferior cerebellar peduncles, and white matter of the cerebrum (Figure 2). Diffusion-weighted imaging was normal, indicating the absence of infarction. There was no enhancement with the administration of gadolinium contrast. Twenty-four hour urinary total protein was 3.45 g/d (normal, <0.30), and creatinine clearance was measured at 49 mL/min (75–135). Ultrasound and Doppler studies of the kidney were unremarkable. Urinary metanephrine, normetanephrine, and vanillylmandelic acid were within normal ranges, excluding pheochromocytoma as a cause of the patient’s malignant hypertension. Toxicological and microbiological studies of the blood and urine to investigate the cause of rhabdomyolysis were negative. With judicious fluid management, creatine kinase and myoglobin normalized 5 days later. The patient’s neurological deficits and magnetic resonance imaging changes also normalized with achievement of normal blood pressure. He was discharged 10 days later, with residual hyperreflexia in the limbs, but no neurological deficits.

Hypertensive crises are important medical emergencies that can result in acute end-organ injuries such as stroke, pulmonary edema, congestive heart failure, aortic dissection, acute myocardial infarction, unstable angina, acute renal failure, and hypertensive encephalopathy. Up to 16% of patients with hypertensive crisis have hypertensive encephalopathy, which is characterized by severe hypertension, confusion, visual complaints, headache, stupor, and seizures. Brain-stem and cerebellar involvement in hypertensive encephalopathy are rarely reported. These occur more frequently in younger patients (ie, younger than 40 years) with secondary hypertension. Hypertensive encephalopathy is believed to arise from the breakdown of autoregulation, resulting in dilatation of cerebral arterioles and disruption of the blood–brain barrier, causing vasogenic edema. The posterior circulation (vertebrobasilar and posterior cerebral arteries) is sparsely innervated by sympathetic nerves, accounting for the susceptibility of the occipital lobes, brain stem, and cerebellum to this breakdown of autoregulation. Despite the wide availability of antihypertensive therapies, hypertensive crisis remains an important reversible medical emergency that requires immediate reduction of blood pressure.

None.

References
Figure 1. Fundoscopic appearance of grade IV hypertensive retinopathy, with papilloedema (1), arteriovenous nipping (2), flame-shaped hemorrhages (3), and soft (4) and hard (5) exudates.

Figure 2. Computed tomography image of the brain. A, Hypodensities in the brain stem (BS) and cerebral white matter (CWM), consistent with cerebral edema. Axial (B) and coronal (C) fluid-attenuated inversion recovery sequenced magnetic resonance image showing hyperintensities in the brain stem, cerebellar peduncles (CP), and cerebral white matter.
Hypertensive Brainstem Encephalopathy
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