

CT diagnosis of hypertensive brainstem encephalopathy (HBE): A diagnostic challenge in the emergency department

Pantelis Kraniotis, MD; Petros Zampakis, MD, PhD; Christina Kalogeropoulou, MD, PhD; Pantelitsa Kalliakmani, MD; and Theodore Petsas, MD, PhD

Hypertensive encephalopathy usually involves the posterior supratentorium, with uncommon involvement of the brainstem. We present a case of acute hypertensive encephalopathy of the brainstem diagnosed by means of CT. The brainstem was markedly hypodense, with no evidence of typical concomitant parieto-occipital involvement. The patient's symptoms and imaging findings improved after hypertension had been controlled.

Case report

A 54-year-old man came to the emergency department complaining of weakness, dizziness, blurred vision, and gait disturbance of three days' duration. He had also had two episodes of vomiting within the last 2 hours. His arterial blood pressure was 280/110 mm Hg, with a pulse rate of 80/min, suggestive of malignant hypertension. Physical examination revealed an unaffected level of consciousness (15/15 GCS) and mild cerebellar signs (broad-based walking). There was no evidence of focal neurologic deficit, and plantar reflexes were normal. Muscular strength was normal (5/5). Visual field examination was unremarkable, but fundoscopic examination showed grade IV hypertensive retinopathy changes. The rest of the physical examination was unremarkable, as was the patient's previous medical history. Specifically, there was no frank history of hypertension according to the patient, who had never been on anti-

hypertensive medication. It was presumed, though, that he was unaware of being hypertensive. This was suggested by his low Mini Mental State Examination (MMES) score of 20 (normal >25), indicative of mild cognitive impairment. In addition, ECG in the emergency setting showed evidence of left ventricular hypertrophy, consistent with longstanding hypertension. Laboratory values from were unremarkable.

The patient was referred for an emergent brain CT (Fig. 1, A-C). The examination showed that the upper pons and midbrain were markedly and diffusely hypodense and that the ambient cistern was obliterated. Low attenuation extended to the superior cerebellar white matter. There was no evidence of obstructive hydrocephalus. The periventricular white matter was mildly hypodense, in keeping with chronic small-vessel disease. The parieto-occipital regions were relatively spared.

The patient was unable to withstand an MRI examination due to claustrophobia. He was subsequently admitted to the nephrology clinic, due to his malignant hypertensive crisis, in order to achieve better blood pressure control. Continuous IV pump infusion of clonidine was administered. The blood pressure reached normal levels after 5 days.

Followup CT after blood pressure normalization showed almost complete reversal of the brainstem hypodensity and ambient cistern obliteration (Fig. 2, A-C). Periventricular hypodensity due to presumed chronic small-vessel disease was unchanged. These findings correlated with alleviation of the patient's symptoms. The patient's overall clinical status was also markedly improved within the next several

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Drs. Kraniotis, Zampakis, Kalogeropoulou, and Petsas are in the Department of Radiology, and Dr. Kalliakmani is in the Department of Nephrology, all at University Hospital of Patras, Rion, Greece. Contact Dr. Kraniotis at pkran@hotmail.com.

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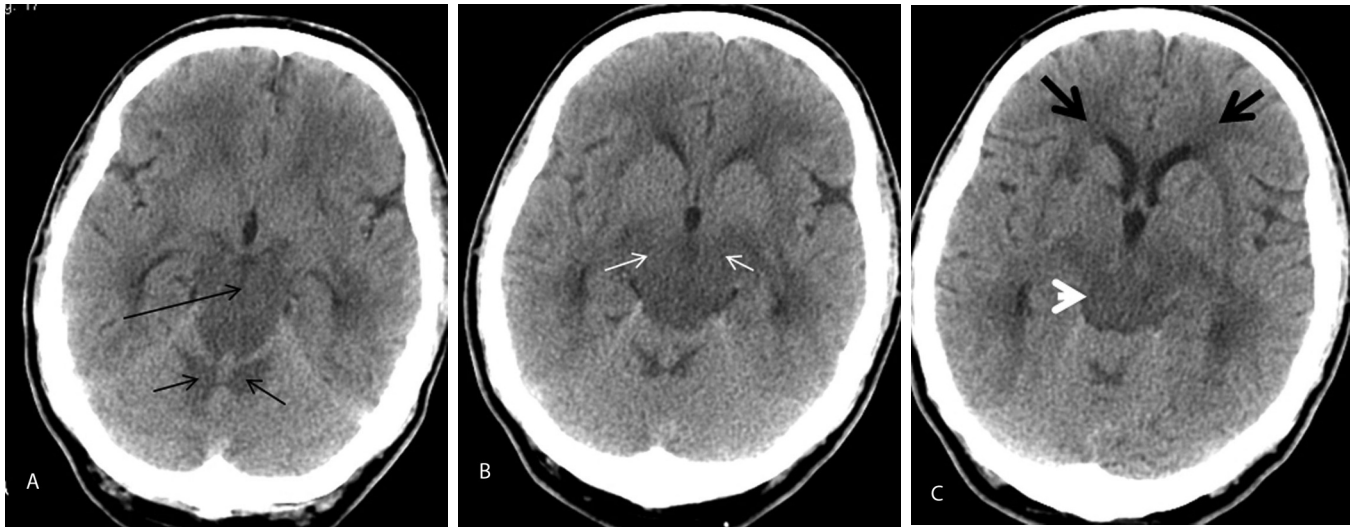


Figure 1, A-C. 54-year-old man with hypertensive brainstem encephalopathy. Admission computed tomography (CT) scan: Sequential axial CT images at the level of the upper brainstem show diffuse hypodensity of the upper pons (long black arrow), as well as hypodense areas within the superior cerebellar hemispheres (short black arrows) (A). Hypodense areas are also visible at subthalamic nuclei bilaterally (white arrows) (B). The ambient cistern is obliterated (white arrowhead). The periventricular white matter is diffusely hypodense, in keeping with chronic small-vessel disease (black thick arrows) (C).

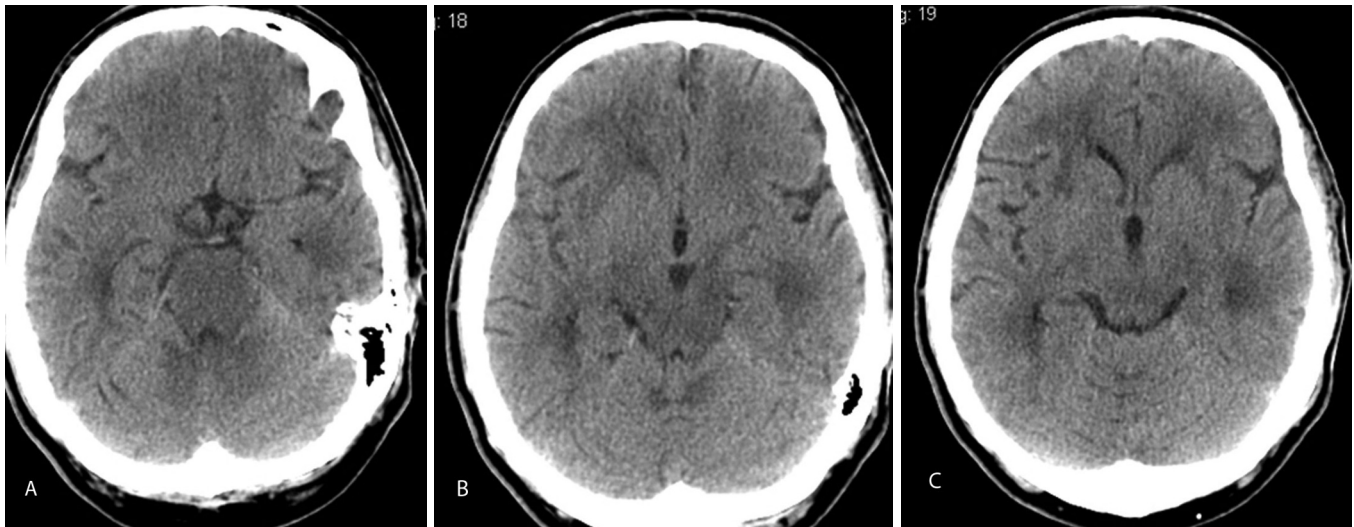


Figure 2, A-C. 54-year-old man with hypertensive brainstem encephalopathy. A and B: Followup computed tomography (CT) scan: Sequential axial CT images at the level of the upper brainstem show almost complete resolution of the hypodense appearance of the upper pons, cerebellum white matter, and subthalamic nuclei bilaterally. C: There is reversal of the obliteration of the ambient cistern. The periventricular white matter hypodensity is unchanged.

days, and he was discharged one week later. The combination of imaging and clinical findings and their subsequent improvement was compatible with a diagnosis of acute hypertensive encephalopathy of the brainstem.

Discussion

Hypertensive crisis is a medical emergency, potentially resulting in major complications such as stroke, pulmonary edema, congestive heart failure, aortic dissection, myocardial infarction, angina, renal failure, and hypertensive en-

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cephalopathy (1). Hypertensive encephalopathy (HE) is caused by severe hypertension and has a relatively acute onset (2). This sudden increase in systemic blood pressure often occurs in patients with no history of chronic hypertension (3). HE rates are up to 16% in patients presenting with a hypertensive episode (4). Symptoms are nonspecific and include headache, confusion, stupor, visual disturbances, nausea, vomiting, and seizures (5). Because of the nonspecific nature of the symptoms, the diagnosis is not commonly made by imaging (3). HE is a subset of posterior reversible encephalopathy syndrome (PRES), which also includes conditions such as pre-eclampsia/eclampsia and cyclosporine- and tacrolimus-related encephalopathy (6, 7). This syndrome has also been associated with renal insufficiency (6).

The most characteristic feature of PRES is its predominant involvement of the posterior supratentorial areas. Brainstem involvement is not infrequent but is commonly associated with the more typical supratentorial lesions (7, 8), which were absent in our patient. Isolated involvement of the brainstem and cerebellum is rare, with a few cases in the literature. Although MRI has greatly increased the recognition of hypertensive encephalopathy, the brainstem variant has been rarely reported (1-3, 9-13). This occurs more often in patients less than 40 years of age and is associated with secondary hypertension (4).

Vasogenic edema caused by failure of cerebral autoregulation and endothelial dysfunction is considered to be the underlying mechanism of hypertensive encephalopathy (14). When systemic blood pressure rises over the autoregulatory threshold of the cerebral vasculature, it results in brain hyperperfusion, due to dilatation of cerebral arterioles. This causes blood-brain barrier breakdown, with subsequent transudation of fluid and protein material (vasogenic edema) (6, 8, 15). This pathophysiologic mechanism is supported by the increased ADC values reported in these patients (16). (This of course could not be demonstrated in our case due to the patient's claustrophobia.) Another proposed mechanism is endothelial damage or dysfunction, which may trigger, via increased production of nitric oxide, increased capillary permeability and loss of autoregulation (3). Responsive vasoconstriction-causing ischemia to the affected territory may play a role in some cases (6, 15). This tends not to predominate, though, given the reversible nature of the clinical and radiological findings. In cases resolving after control of the hypertensive episode, the lesions visualized on imaging are most consistent with vasogenic edema.

As mentioned, hypertensive encephalopathy lesions occur mainly posteriorly, which may be due to relatively decreased sympathetic innervation of the posterior circulation (vertebrobasilar and posterior cerebral arteries) compared to the anterior circulation (2). This accounts for the increased susceptibility of the parieto-occipital regions, brainstem, and cerebellum when autoregulation breakdown occurs (17). It has been suggested by Kumai et al that differences in the arterial pressure level are sufficient to cause the development of vasogenic edema in cortical and subcorti-

cal regions and deep structures, such as the basal ganglia and brainstem (18). Cortical and subcortical regions are less tolerant to hypertension compared to deep structures. For this reason, vasogenic edema is thought to involve the deep structures when the systemic blood pressure rises at a highly accelerated rate. Differences in the sympathetic innervation may also exist between the posterior supratentorial and infratentorial circulation, which could explain infratentorial predominance (2).

Newer evidence with diffusion-weighted MRI and anisotropy diffusion studies also suggests that MRI signal change is caused by transient vasogenic edema (19). MRI characteristically shows a posterior leukoencephalopathy, affecting predominantly the white matter of the parieto-occipital regions (20). On the other hand, HBE affects predominantly the brainstem and cerebellum, while parieto-occipital regions are spared (12). However, brainstem and deep-white-matter involvement seems to have less reversibility than cortical and subcortical areas (19). DWI is reported to be helpful in HBE cases because lack of restricted diffusion can rule out infarct.

In cases when no typical parieto-occipital lesions coexist, the differential diagnosis for brainstem lesions includes acute infarction, tumor, encephalitis, and vasculitis. Our patient's clinical status of mild cerebellar syndrome that subsequently resolved was not consistent with acute infarction. Consciousness level was normal, and there was no focal neurologic deficit. Mild symptomatology, like headache and confusion, with lack of cranial nerve findings and focal neurologic deficits despite brainstem involvement (referred to as clinical-radiological dissociation) (4) suggests hypertensive encephalopathy (2). The improvement would also not be compatible with tumor. Laboratory tests during his hospitalization were negative, including inflammatory and collagen-vascular-disease markers. These findings, along with the rapid clinical evolution and resolution of both symptoms and brainstem lesions with correction of hypertension, established the clinical diagnosis of hypertensive encephalopathy (12).

Recognition of the brainstem variant of hypertensive encephalopathy is important so that prompt treatment can be initiated. Radiologists may be the first to notice this alarming appearance of the brainstem. Along with the imaging findings, the presence of "clinical-radiological dissociation" should alert radiologists to suggest the diagnosis of this rare variant of hypertensive encephalopathy.

References

1. Seet RC, Lim EC. Images in cardiovascular medicine. Hypertensive brainstem encephalopathy. *Circulation* 2007;115:e310-311 [PubMed]
2. Fujiwara H, Momoshima S, Kuribayashi S, Sasamura H. Hypertensive encephalopathy of brain stem with minimal supratentorial involvement: a rare manifestation of hypertensive encephalopathy. *Radiat Med* 2005;23:504-507 [PubMed]

CT diagnosis of hypertensive brainstem encephalopathy (HBE): A diagnostic challenge

3. McCarron MO, McKinstry CS. Vanishing brainstem edema. *J Stroke Cerebrovasc Dis* 2008;17:156-157 [[PubMed](#)]
4. Cruz-Flores S, de Assis Aquino Gondim F, Leira EC. Brainstem involvement in hypertensive encephalopathy: clinical and radiological findings. *Neurology* 2004;62:1417-1419 [[PubMed](#)]
5. Chester EM, Agamanolis DP, Banker BQ, Victor M. Hypertensive encephalopathy: a clinicopathologic study of 20 cases. *Neurology* 1978;28:928-939 [[PubMed](#)]
6. Hinchey J, Chaves C, Appignani B, et al. A reversible posterior leukoencephalopathy syndrome. *N Engl J Med* 1996;334:494-500 [[PubMed](#)]
7. Casey SO, Sampaio RC, Michel E, Truwit CL. Posterior reversible encephalopathy syndrome: utility of fluid-attenuated inversion recovery MR imaging in the detection of cortical and subcortical lesions. *AJNR Am J Neuroradiol* 2000;21:1199-1206 [[PubMed](#)]
8. Weingarten K, Barbut D, Filippi C, Zimmerman RD. Acute hypertensive encephalopathy: findings on spin-echo and gradient-echo MR imaging. *AJR Am J Roentgenol* 1994;162:665-670 [[PubMed](#)]
9. Kitaguchi H, Tomimoto H, Miki Y, et al. A brainstem variant of reversible posterior leukoencephalopathy syndrome. *Neuroradiology* 2005;47:652-656 [[PubMed](#)]
10. Yasuda Y, Akiguchi I, Imai T, Sonobe M, Kage M. Hypertensive brainstem encephalopathy. *Intern Med* 2003;42:1131-1134 [[PubMed](#)]
11. Fong CS. Hypertensive encephalopathy involving the brainstem and deep structures: a case report. *Acta Neurol Taiwan* 2005;14:191-194 [[PubMed](#)]
12. Albin TA, Lakhanpal RR, Foroozan R, Lopez GA, McPherson AR. Retinopathy and choroidopathy as the initial signs of hypertensive brainstem encephalopathy. *Arch Ophthalmol* 2006;124:1784-1786 [[PubMed](#)]
13. Nagata M, Maeda M, Tsukahara H, Maier SE, Takeda K. Brain stem hypertensive encephalopathy evaluated by line scan diffusion-weighted imaging. *AJNR Am J Neuroradiol* 2004;25:803-806 [[PubMed](#)]
14. Lamy C, Oppenheim C, Meder JF, Mas JL. Neuroimaging in posterior reversible encephalopathy syndrome. *J Neuroimaging* 2004;14:89-96 [[PubMed](#)]
15. Skinhoj E, Strandgaard S. Pathogenesis of hypertensive encephalopathy. *Lancet* 1973;1:461-462 [[PubMed](#)]
16. Schwartz RB, Mulkern RV, Gudbjartsson H, Jolesz F. Diffusion-weighted MR imaging in hypertensive encephalopathy: clues to pathogenesis. *AJNR Am J Neuroradiol* 1998;19:859-862 [[PubMed](#)]
17. de Seze J, Mastain B, Stojkovic T, et al. Unusual MR findings of the brain stem in arterial hypertension. *AJNR Am J Neuroradiol* 2000;21:391-394 [[PubMed](#)]
18. Kumai Y, Toyoda K, Fujii K, Ibayashi S. Hypertensive encephalopathy extending into the whole brainstem and deep structures. *Hypertens Res* 2002;25:797-800 [[PubMed](#)]
19. Pande AR, Ando K, Ishikura R, et al. Clinicoradiological factors influencing the reversibility of posterior reversible encephalopathy syndrome: a multicenter study. *Radiat Med* 2006;24:659-668 [[PubMed](#)]
20. Vaughan CJ, Delanty N. Hypertensive emergencies. *Lancet* 2000;356:411-417 [[PubMed](#)]

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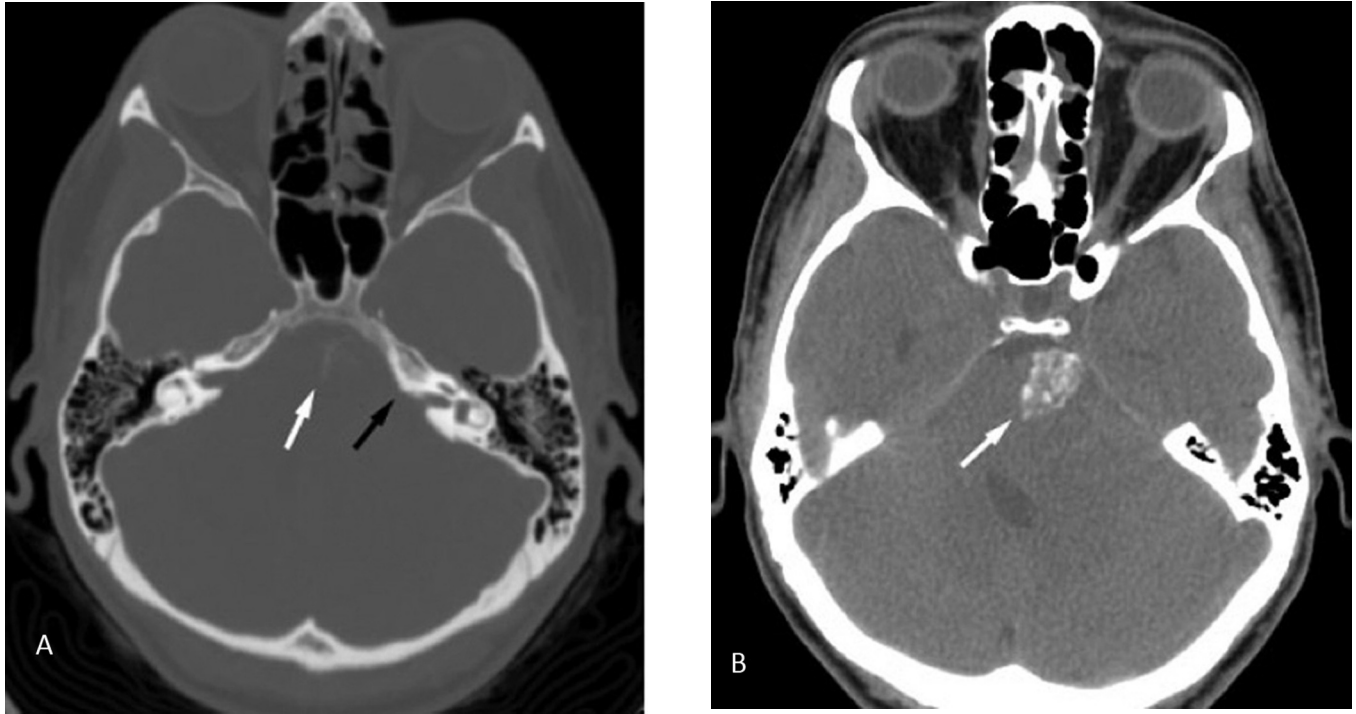


Figure 6: 49-year-old woman with meningioma. Axial CT in (A) bone window and (B) soft-tissue window shows subtle hyperostosis of the left petrous ridge at the porus acusticus (black arrow) and coarse calcification (white arrows) anteriorly within the mass.