Kim et al., J Clin Case Rep 2018, 8:2
DOI: 10.4172/2165-7920.10001087

Journal of Clinical Case Reports

Case Report Open Access

Hypertensive Brainstem Encephalopathy Mimicking Peritumoral Edema after Gamma Knife Radiosurgery for Cerebellopontine Angle Meningioma: A Case Report

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Abstract

An 83-year-old man presented with hypertensive brainstem encephalopathy manifesting as general weakness, dysarthria, gait disturbance, urinary incontinence, and decreased cognitive function. The patient had a history of gamma knife radiosurgery (GKR) for cerebellopontine angle (CPA) meningioma 10 years prior. Compared with previous magnetic resonance (MR) imaging results, a recent MR image revealed massive brainstem hyperintensity in T2-weighted images, not in the parieto-occipital lobes. The lesions were nearly completely resolved several days after normalization of the blood pressure. Isolated hypertensive brainstem encephalopathy without concomitant typical parieto-occipital abnormalities are unusual and the diagnosis is challenging especially after gamma knife radiosurgery for cerebellopontine angle meningioma. We describe a patient with hypertensive brainstem encephalopathy that was initially confused with peritumoral edema.

Keywords: Radiosurgery; Hypertensive encephalopathy; Brainstem

Introduction

Hypertensive encephalopathy (HE) is an acute neurological disorder caused by a sudden increase in systemic blood pressure [1,2]. HE is characterized by headache, mental deterioration, visual disturbance, and seizures. Imaging abnormalities are predominant in the parieto-occipital subcortical white matter, and the clinical and neuroradiological abnormalities are reversible after blood pressure normalization. Therefore, the term "posterior reversible leukoencephalopathy" has been used to describe this condition [3-5]. According to a previous studies [6], the brainstem is involved in 58% of HE cases, but isolated brainstem involvement in HE has rarely been reported [7-9]. We describe a patient with hypertensive brainstem encephalopathy that could be confused with peritumoral edema (PTE) after gamma knife radiosurgery (GKR) for cerebellopontine angle (CPA) meningioma.

Case Report

An 83-year-old man presented at the neurosurgery clinic with general weakness, dysarthria, gait disturbance, and mildly decreased cognitive function that lasted for 1 month. The patient had a history of GKR 10 years prior, for right CPA meningioma. The CT scan indicated diffuse decreased attenuation of the pons with extension into the midbrain and also revealed a minimal pontine hemorrhage (Figure 1). Using T2-weighted and fluid-attenuated inversion recovery (FLAIR) images, a massive hyperintensity was identified in the same location identified in the CT scan. Results of the diffusion weighted image (DWI) were normal, indicating the absence of acute infarction, and the apparent diffusion coefficient (ADC) maps showed slightly increased diffusion in the brainstem, consistent with vasogenic edema (Figure 2). At the time of the presenting visit, the patient's blood pressure was systolic blood pressure more than 200 mmHg. Serum laboratory findings were within normal limits, however, renal function parameters suggested decreased renal function. Additional tests were performed at the cardiology and nephrology outpatient departments, in order to determine the cause of hypertensive crisis. Cardiologic evaluations were within normal age-related limits. Kidney ultrasonography and doppler ultrasonography did not indicate renal artery stenosis, but a small left kidney with cortical thinning was observed. These findings demonstrate that this patient had a history of chronic renal insufficiency, and that renal function was recently aggravated. The patient was taking antihypertensive drug to control his blood pressure, and was prescribed oral steroid, due to brainstem swelling. After a month of treatment, systolic blood pressure fluctuated between 90 and 130 mmHg. General weakness, dysarthria, gait disturbance, urinary incontinence, and cognitive dysfunction improved, but right-hand grip weakness remained, presumably due to the pontine hemorrhage. A follow-up CT scan 1 month later showed improvement of the low attenuation in the brainstem and periventricular areas, and resolution of the pontine

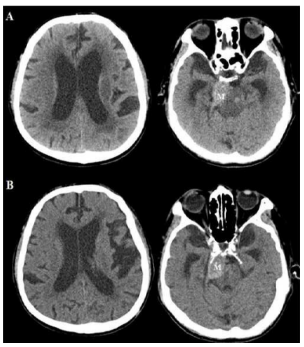

Figure 1: Computerized tomography axial imagesof brainstem. A. Axial computerized tomography (CT) image of the cerebellum without contrast showed diffuse decreased attenuation of the pons with extension into the midbrain, a periventricular lucency, and an area of focal high density in the left pons, which probably represents apontine hemorrhage. B. Follow-up CT scan 1 month later showed improvement of low attenuation in the brainstem and periventricular areas, and resolution of the pontine hemorrhage. M: Meningioma.

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Received January 30, 2018; **Accepted** February 21, 2018; **Published** February 26, 2018

Citation: Kim YG, Chang WS, Jung HH, Chang JW, Park YG (2018) Hypertensive Brainstem Encephalopathy Mimicking Peritumoral Edema after Gamma Knife Radiosurgery for Cerebellopontine Angle Meningioma: A Case Report. J Clin Case Rep 8: 1087. doi: 10.4172/2165-7920.10001087

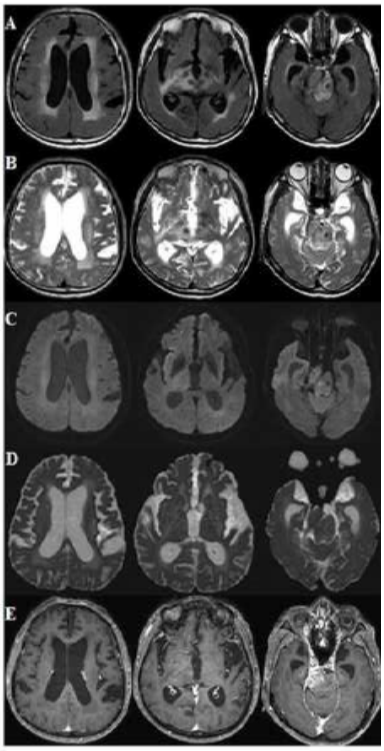

Figure 2: Axial magnetic resonance images of brainstem. Brain magnetic resonance imaging (MRI) 1 month after symptom onset, showing hyperintensity involving the periventricular area, midbrain, and pons on fluid-attenuated inversion recovery (FLAIR) images (A), and T2-weighted images (B). Diffusion-weighted image shows near isointensity (C) and the corresponding apparent diffusion coefficient (ADC) map shows slightly increased diffusion (D) in the brainstem. No evidence of abnormal enhancement was found in the brain parenchyma except in the right cerebellopontine angle (CPA) meningioma (E).

hemorrhage (Figure 1). This study was approved by the Dongguk University Gyeongju Hospital Institutional Review Board.

Discussion

The clinical findings of this patient were initially suggestive of delayed PTE following GKR on right CPA meningioma 10 years earlier. However, post-GKR PTE is known to occur more frequently in 1–23 months following the procedure, with a peak time to edema of 6-8 months [6-10]. Additionally, our patient had a history of a severe hypertensive event, systolic blood pressure more than 200 mmHg, and subsequent improvement after normalization of blood pressure. These finding were strongly suggestive of HE, rather than PTE.

HE is an acute neurological syndrome caused by a sudden increase of systemic blood pressure and is most commonly a secondary event, triggered by paroxysmal hypertension with various etiologies including renal disease, collagen vascular disorders, eclampsia, use of immunosuppressive and cytotoxic drugs, and hematologic disorders [11]. The most widely accepted pathophysiologic mechanism of HE is vasogenic edema that acute and subacute elevation of blood pressure exceeds the upper limit of cerebral vascular autoregulation, causing forced dilation with hyperperfusion of small-caliber arteries, dysfunction of the endothelium, and leakage of fluid and protein into the extracellular space [4,11-13]. The vessels in posterior circulation are more sparsely innervated by sympathetic nerves than are those in the anterior circulation; therefore, they are poorly responsive to initiate protective vasoconstriction in response to an acute increase in arterial blood pressure and are susceptible to the breakdown of autoregulation. Additionally, HE is known to involve the cortex or subcortical white matter of the parietal or occipital lobes rather than the brainstem because the brainstem autoregulations more effectively [14,15]. The upper limit of cerebral autoregulation in deep brain structures such as the thalamus, basal ganglia, or brainstem is higher than that in the cerebral cortex in normotensive and spontaneously hypertensive rat models [16]. The deep brain structures are supplied by direct branches from the middle or posterior cerebral artery or basilar artery, while the cortex and subcortex are supplied by the terminal pial arteries. Therefore, it is likely that severe acceleration of hypertension is required for hypertensive brainstem encephalopathy.

MR image scan is the gold standard of diagnosis and must be performed as soon as possible to rule out HE. MR imaging of hypertensive brainstem encephalopathy typically shows increased T2-weighted and FLAIR signal intensities in the brainstem and cerebellum, without restricted diffusion on diffusion-weighted imaging. These findings indicate vasogenic edema rather than cytotoxic edema such as brainstem infarction [17].

Slotty et al. performed a CT-based cerebral perfusion measurement in patients with chronic subdural hematoma and reported significantly up-regulated cerebral blood volume (CBV) and cerebral blood flow (CBF) in chronically compressed cortical area with the mean transit time (MTT) as the set point parameter of cerebral perfusion clearly elevated above normal values. Therefore, the brain appears to maintain normal or approximately normal cerebral perfusion despite increased local pressure [18]. These findings are applicable to our patient. The observation of chronic brainstem compression by CPA meningioma in this patient with HE suggests that up-regulated CBV and CBF of the compressed brainstem could increase its vulnerability to edema caused by paroxysmal hypertension. However, previous reports do not describe this relationship in detail and further experimental studies in animal models of HE is necessary to investigate our hypothesis.

Conclusion

We presented the case of hypertensive brainstem encephalopathy that could be mimicked PTE after GKR for CPA meningioma. Clinicians should be aware of the clinical course of PTE or aggravation after GKR for meningioma, and at the same time, consider the possibility of hypertensive encephalopathy.

References

1. De Seze J, Mastain B, Stojkovic T, Ferriby D, Pruvo JP, et al. (2000) Unusual MR findings of the brain stem in arterial hypertension. Am J Neuroradiol 21: 391-394.
2. Morello F, Marino A, Cigolini M, Cappellari F (2001) Hypertensive brain stem encephalopathy: clinically silent massive edema of the pons. Neurological Sci 22: 317-320.
3. Weingarten K, Barbut D, Filippi C, Zimmerman R (1994) Acute hypertensive encephalopathy: findings on spin-echo and gradient-echo MR imaging. Am J Roentgenol 162: 665-670.
4. Schwartz R, Jones K, Kalina P, Bajakian R, Mantello M, et al. (1992) Hypertensive encephalopathy: findings on CT, MR imaging, and SPECT imaging in 14 cases. Am J Roentgenol 159: 379-383.
5. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, et al. (1996) A reversible posterior leukoencephalopathy syndrome. New Eng J Med 334: 494-500.
6. Bhagavati S, Chum F, Choi J (2008) Hypertensive encephalopathy presenting with isolated brain stem and cerebellar edema. J Neuroimag 18: 454-456.
7. Chang GY, Keane JR (1999) Hypertensive brainstem encephalopathy: three cases presenting with severe brainstem edema. Neurology 53: 652.

8. Thambisetty M, Biousse V, Newman NJ (2003) Hypertensive brainstem encephalopathy: clinical and radiographic features. J Neurological Sci 208: 93-99.
9. Gamanagatti S, Subramanian S (2006) Hypertensive encephalopathy: isolated pons involvement mimicking central pontine myelinolysis. Korean J Radiol 7: 218-219.
10. Cai R, Barnett GH, Novak E, Chao ST, Suh JH (2010) Principal risk of peritumoral edema after stereotactic radiosurgery for intracranial meningioma is tumor-brain contact interface area. Neurosurg 66: 513-22.
11. Garg RK (2001) Posterior leukoencephalopathy syndrome. Postgraduate Med J 77:24.
12. Schwartz RB (2002) Hyperperfusion encephalopathies: hypertensive encephalopathy and related conditions. Neurologist 8: 22-34.
13. Schwartz RB, Mulkern RV, Gudbjartsson H, Jolesz F (1998) Diffusion-weighted MR imaging in hypertensive encephalopathy: clues to pathogenesis. Am J Neuroradiol 19: 859-862.
14. Baumbach GL, Heistad DD (1985) Regional, segmental, and temporal heterogeneity of cerebral vascular autoregulation. Ann Biomed Eng 13: 303-310.
15. Verrees M, Fernandes Filho JA, Suarez JI, Ratcheson RA (2003) Primary hypertension-induced cerebellar encephalopathy causing obstructive hydrocephalus. Case report. J Neurosurg 98: 1307-1311.
16. Sadoshima S, Fujii K, Yao H, Kusuda K, Ibayashi S, et al. (1986) Regional cerebral blood flow autoregulation in normotensive and spontaneously hypertensive rats-effects of sympathetic denervation. Stroke 17: 981-984.
17. Karabay N, Emin L, Ada E (2013) Brainstem swelling and noncommunicating hydrocephalus caused by hypertensive brainstem encephalopathy. Emergency Radiol 20: 579-582.
18. Slotty PJ, Kamp MA, Steiger SHJ, Cornelius JF, Macht S, et al. (2013) Cerebral perfusion changes in chronic subdural hematoma. J Neurotrauma 30: 347-351.