Hypertensive Therapy Leading to Atypical Posterior Reversible Encephalopathy Syndrome

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Hypertensive therapy is a standard treatment for delayed cerebral ischemia in the management of vasospasm following aneurysmal subarachnoid hemorrhage.[1] Posterior reversible encephalopathy syndrome (PRES) is a brain imaging finding that demonstrates focal regions of symmetric hemispheric vasogenic edema usually in the parietal and occipital lobes. Atypical PRES affecting bilateral basal ganglia although rare can occur as an isolated phenomenon sparing the cortex.[2] PRES can conflict the management of cerebral vasospasm.

A 69-year-old hypertensive patient had sudden onset of severe headache with loss of consciousness. Clinical workup of the patient revealed subarachnoid hemorrhage, World Federation of Neurological Surgeons Grade 1, modified Fischer grade 3, ruptured anterior communicating artery aneurysm, and unruptured right supraclinoid internal carotid artery aneurysm on computed tomography (CT) angiogram (day 1 ictus). Baseline cerebral artery flow velocities on transcranial Doppler (TCD) were within normal limits.

The patient underwent clipping of both the aneurysms on ictus day 2. Intraoperative hemodynamics were stable. Clipping of aneurysms was uneventful and indocyanine green microscopic confirmation of flow in the major arteries was done. In the postoperative period, the mean arterial pressure (MAP) remained 20% above baseline that was accepted. Since the patient became drowsy in the postoperative day 5, a blood workup was done that ruled out hyponatremia. A CT scan of brain was done that ruled out hydrocephalus and infarct. TCD examination on day 5 revealed higher mean flow velocities (MFV 110 cm/s) in bilateral anterior cerebral arteries (ACA) and normal MFV in bilateral middle cerebral artery (MCA), which was normal previously. The patient underwent angiography that revealed vasospasm of both right and left ACA. Hypertensive therapy was initiated; MAP of 120 to 140 mm Hg was targeted with noradrenaline infusion. As there was no improvement, spasmolysis was done using intraarterial nimodipine and milrinone with good angiographic improvement.

Despite a successful spasmolysis, there was no improvement in the neurological condition. Repeat CT brain done on day 6 did not show any significant changes that would correlate with the deteriorating neurology of the patient; hence, an electroencephalogram was done that ruled out nonconvulsive status epilepticus. Magnetic resonance imaging of the

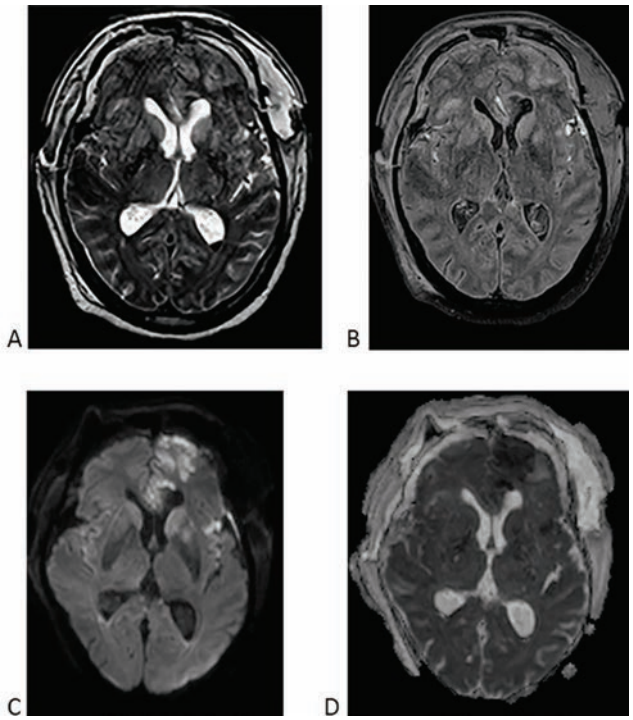

Fig. 1 Axial T2 (**A**) and fluid-attenuated inversion recovery (**B**) images show abnormal hyperintense signal change involving the basal ganglia, mainly the caudate head and anterior lentiform nucleus bilaterally in a slightly asymmetrical pattern. However, there is no diffusion restriction in these areas as seen in images (**C** and **D**) to suggest a hypoxic insult.

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brain was done at this stage as the patient remained deeply comatose that revealed symmetrical bilateral basal ganglia T2 hyperintensity with no diffusion restriction (►**Fig. 1**).

After excluding other causes of basal ganglia hyperintensity, a diagnosis of atypical PRES was made. There was a therapeutic dilemma whether to continue hypertensive therapy for cerebral vasospasm to prevent delayed cerebral ischemia or to reduce the blood pressure for treating PRES.[3] On day 7 as the follow-up TCD showed persistent high velocities in left MCA and bilateral ACA (MFV> 120 cm/s), a decision was made to continue with the hypertensive therapy. Subsequently, patient underwent spasmolysis twice for refractory vasospasm. Despite continuation of hypertensive therapy, cardiac output augmentation with milrinone and intraarterial spasmolysis, the patient developed delayed cerebral ischemia involving both ACA territories and bilateral patchy areas of infarct in the MCA territory and he was discharged with modified Rankin score ⅝.

It is known that hypotension, hypoglycemia, hypoxia, metabolic crisis can cause bilateral basal ganglia hyperintensities.[4] Hypertension could have caused atypical PRES in our case as other causes were ruled out.[5] During therapeutic hypertension in the management of vasospasm, atypical PRES could result in neurological deterioration and should be included in the differential diagnosis.

Conflict of Interest
None declared.

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