

LETTER TO EDITOR

Unusual clinical presentation of hypertensive cerebellar hemorrhage

Sir,

A 60-year-old hypertensive lady presented with acute onset of facial asymmetry, inward deviation of the left eye, and gait imbalance since one day. There were no symptoms of raised intracranial pressure. On examination, she had left lateral rectus palsy, left lower motor neuron facial palsy, and truncal ataxia with left cerebellar signs. Right side gaze, vertical gaze, convergence, and extra ocular movements of the right eye were normal. Computed tomography brain revealed 4×4 cm hematoma in the left cerebellar hemisphere with extension in the left middle cerebellar peduncle causing distortion of the fourth ventricle and brainstem. There was no evidence of bleeding in the pons or in the cerebellopontine angle [Figure 1]. She was conservatively managed with antihypertensive and antiedema medications. On follow-up at six weeks, her left sixth and seventh nerve palsy has resolved with minimal persistence of appendicular ataxia.

Hypertensive cerebellar hemorrhage manifests with a spectrum of clinical presentation ranging from a benign course associated with little or no neurological deficits to a rapidly fatal course with brainstem compression. The swelling around the hematoma may cause acute obstructive hydrocephalus which may necessitate placement of an external ventricular drainage tube. Occurrence of cranial nerve palsy is often due to extension of the bleed in the brainstem or presence of blood in the cisterns through which the nerves course. Involvement of the sixth nerve nucleus or parapontine reticular formation results in gaze palsy towards the side of the lesion.^[1] Involvement of the internal genu of the nerve as it loops around the abducent nucleus results in ipsilateral lower motor neuron facial palsy.^[1] The cisternal segment of the abducent nerve is vulnerable to stretch and compression following raised intracranial pressure due to supratentorial or infratentorial lesions with obstructive hydrocephalus. The resultant downward displacement of the brainstem results in sixth nerve palsy – occurring from distortion of the nerve or its compression against the petrosphenoid ligament in the Dorellos canal or against the ridge of petrous temporal bone.^[2] Dysfunction of the axoplasmic flow is often said to be responsible for this reversible palsy.^[3] Sixth nerve palsy

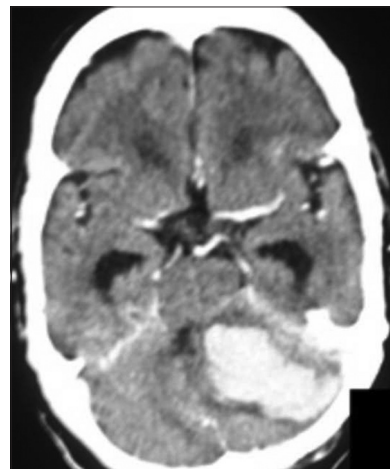


Figure 1: Axial plain CT brain demonstrating evidence of hyperdensity (60 HU suggestive of blood products) involving the left cerebellar hemisphere extending into the left middle cerebellar peduncle. There is no extension into the brainstem or cerebellopontine cistern

is extremely vulnerable to compression against the clivus and is often reported as a false localizing sign in posterior fossa abnormalities in the absence of raised intracranial pressure.^[3-5]

The occurrence of sixth nerve palsy in our case was due to involvement of the cisternal segment of the nerve and is unlikely due to nuclear involvement which would have resulted in gaze palsy. The presence of cerebellar hematoma resulted in distortion of the brainstem causing stretching of the nerve and its compression against the clivus. The facial nerve palsy was probably due to compression of the internal genu of the nerve by the hematoma with sparing of the abducent nucleus. This pattern of concomitant acute sixth and seventh cranial nerve palsy in hypertensive cerebellar hemorrhage, without gaze palsy, is extremely unusual and has not been described previously.

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