Vertical Gaze Palsy with Skew Deviation
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ABSTRACT
With the advancement of neuroradiology, clinical localization followed by radiology, had made neurology more interesting. Vertical gaze palsy as presentation cerebrovascular disease is not so common. Vertical gaze palsy usually localizes the lesion to dorsal mid brain. A 56 years male patient presented with sudden onset vertigo, diplopia, transient loss of consciousness and sways toward right side while walking. Clinical examination showed vertical gaze palsy with skew deviation along with swaying towards rt. during walk. MRI brain showed – infarct involving dorsal midbrain at superior colliculus level and medial thalamus

KEY WORDS
Mid brain, skew deviation, thalamus, vertical gaze palsy

INTRODUCTION
An eye movement abnormality in which the two eyes move conjugately but have limited movement in one direction is called gaze palsy. It is due to malfunction of one of the “gaze centers” (cortical and brainstem regions responsible for conjugate gaze) or to interruption of the pathways leading from them. Vertical gaze palsies usually result from midbrain damage due either to tumours’ or vascular lesions.¹ Less frequent causes include progressive supranuclear palsy’ and Niemann-Pick disease, type C.² These gaze palsies have been attributed to associated lesions of vertical eye movement control centers in the rostral midbrain rather than the thalamic injury. The frequent coexistence of both midbrain and paramedian thalamic infarction is related to their vascular supply; a single vessel arising near the top of the basilar may branch to supply both the paramedian region of the thalamus and the rostral medial mesencephalon.³ In patients with infarcts of the midbrain/thalamic junction, clinical features can be correlated with lesion location with the use of MRI.

CASE-REPORT
DISCUSSION

A prominent clinical finding in this patient is vertical gaze palsy (b) and (c) of above picture. However horizontal gaze is normal (a) and (d) picture. (Fig 1) Acute vascular events presenting as vertical gaze palsy and skew deviation is not so common. These patients typically present with upward and downward gaze palsies may be associated with confusion and a decreased level of consciousness. The gaze palsies have been attributed to coexisting lesions of the rostral midbrain.4

The neural structures known to be involved in the mediation of vertical gaze lie in the mesencephalic reticular formation. These include the nucleus of Darkschewitsch, the interstitial nucleus of Cajal, and the posterior commissure. Isolated paralysis of downward gaze can also be produced by bilateral lesions of the rostral mesencephalic reticular formation, which includes the interstitial nucleus of the medial longitudinal fasciculus.5

The rostral interstitial medial longitudinal fasciculus contains burst neurons for vertical saccades, and in most pathological studies cases of upward and downward gaze paralysis have been attributed to bilateral infarction in the rostral interstitial medial longitudinal fasciculus.6

An important clinical feature in our patient was the skew deviation, which has been reported with thalamic infarctions.7

Our case presented with prominent vertical gaze palsy along with skew deviation without any sensory features. On evaluation MRI revealed infarction involving the mid brain dorsal area at the superior colliculus level along with medial thalamus infarct. (Fig 2) This is probably due to the occlusion of the artery arising from the basilar which bifurcated to supply the dorsal mid brain and the medial thalamus. The clinical features of vertical gaze palsy is being explained by mid brain lesion while the skew deviation by medial thalamus. Ischemic stroke presenting as vertical gaze palsy is not so common.

REFERENCES