Vertical one-and-a-half syndrome in artery of Percheron infarct

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A 68-year-old lady with a 5-year history of hypertension was traveling in a bus at night and she slept off. When relatives tried to wake her up at destination, she was found unresponsive. She was taken to a hospital, where she recovered her consciousness in 30 min. On regaining consciousness, she was complaining of binocular vertical diplopia. There was no history of headache, vomiting, dysarthria, oscillopsia, weakness, ataxia, or sensory symptoms. There was no history of any memory impairment. On examination, she was alert and oriented. Her blood pressure was 150/90 mm of Hg and pulse was 80/min and was regular. There was no ptosis. There was bilateral up gaze palsy and impaired infraduction in the right eye. There was upbeat nystagmus on up gaze. Horizontal eye movements were normal. Bells phenomenon and convergence were preserved. Pupils were equal and reacting to light and accommodation. Motor, sensory and cerebellar system examination was normal. Plantars were flexor.

Her computerized tomography head was normal. Magnetic resonance imaging of the brain showed acute bilateral paramedian thalamic and right paramedian midbrain infarcts (Figs. 1-3). Magnetic resonance angiography was normal. Biochemical evaluation showed hypercholesterolemia. Bilateral medial thalamic infarcts with the right rostral paramedian mesencephalic infarct suggested right artery of Percheron (AOP) infarct. In view of up gaze palsy, right infraduction impairment and upbeat nystagmus on up gaze suggested the involvement of the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) and interstitial nucleus of Cajal (INC) with or without the involvement of posterior commissure.

Gérard Percheron first described the AOP in 1973. It is a single arterial trunk originating from either proximal posterior cerebral artery and nourishes the bilateral paramedian thalamus and rostral midbrain. This variation is found in between 4% and 12% of the population. AOP infarct usually presents with a triad of symptoms, including altered consciousness, memory deficits, and supranuclear vertical gaze palsies [1]. Altered mental status can present anywhere on the spectrum from drowsiness or confusion to hypersomnolence or coma. An ischemic stroke due to AOP occlusion can cause stupor, agitation, change in behavior, aphasia (dominant side), hemineglect (non-dominant side), and diplopia due to the involvement of the midbrain and bilateral thalamic nuclei.

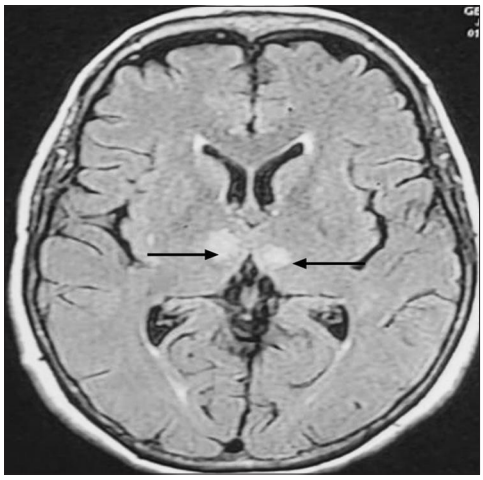

Figure 1: Axial magnetic resonance imaging brain fluid-attenuated inversion recovery image showing bilateral paramedian thalamic infarcts

Vertical one-and-a-half syndrome (VOHS) is a very rare vertical gaze disorder that is caused by damage to the thalamomesencephalic junction [2]. It is characterized either by bilateral upward gaze palsy and ipsilateral limitation of infraduction (classical VOHS, Type I) or bilateral down gaze palsy and impaired monocular elevation (Type II). Classical VOHS is due to unilateral involvement of riMLF and posterior commissure and type II VOHS is usually due to bilateral mesodiencephalic region lesions.

VOHS is an uncommon presentation resulting from a unilateral thalamomesencephalic stroke with involvement of the riMLF and posterior commissure. The burst neurons in the riMLF are known to generate vertical saccades. Excitatory burst neurons in the riMLF project bilaterally to motoneurons contributing to upward saccades (superior rectus and inferior oblique), whereas they project ipsilaterally to motoneurons for downward saccades (inferior rectus and superior oblique) (Fig. 4) [3]. As riMLF projects bilaterally to motoneurons for elevation but only unilaterally for depression, down gaze palsy can be produced only by bilateral lesions. For upward saccades, fibers from one

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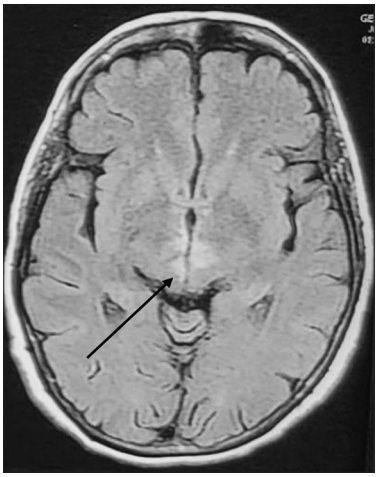

Figure 2: Axial magnetic resonance imaging brain fluid-attenuated inversion recovery image showing right paramedian infarct in midbrain. Bilateral paramedian thalamic infarcts also seen

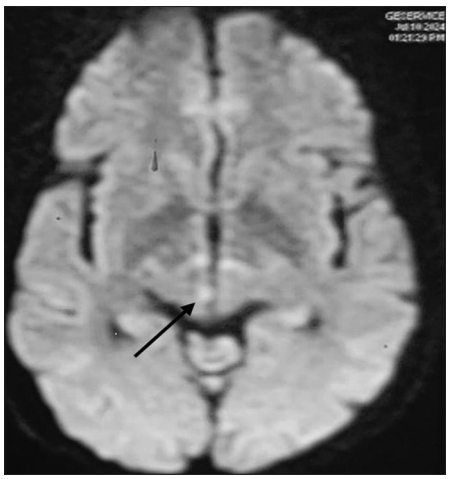

Figure 3: Axial brain diffusion-weighted image showing right paramedian infarct in Midbrain

riMLF course dorsally and cross to the opposite side through the posterior commissure and get connected to both the oculomotor nuclei. The fibers from the other riMLF behave similarly. Hence, a unilateral lesion of the riMLF produces only a transient upward gaze palsy, whereas a lesion in the posterior commissure produces an enduring up gaze palsy. The INC is the neural integrator for vertical eye movement and is involved in vertical gaze holding and lesion involving INC can cause upbeat nystagmus on up gaze [4].

Acute thalamic infarction accounts for approximately 11–14% of acute ischemic stroke in the posterior circulation [5]. Bilateral thalamus infarction accounts for 22–35% of thalamic infarctions [6]. Thalamic infarction caused by AOP occlusion is a special type of bilateral thalamus infarction. There are four ischemic patterns of AOP infarction: (1) Bilateral paramedian thalamic with midbrain involvement (43%), (2) bilateral paramedian thalamic without midbrain involvement (38%), (3) bilateral paramedian thalamic with anterior thalamus and midbrain involvement (14%), and (4) bilateral paramedian thalamic with anterior thalamus without midbrain involvement (5%). The "V" sign on fluid-attenuated inversion recovery and

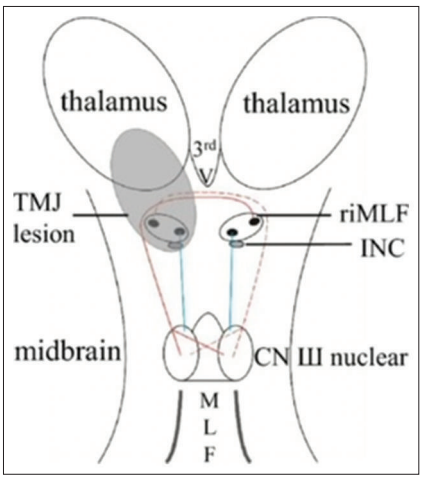

Figure 4: Vertical gaze pathways. Projections from rostral interstitial nucleus of the medial longitudinal fasciculus for down gaze are ipsilateral and for up gaze is bilateral. Fibers for up gaze cross to opposite side through posterior commissure and supply bilateral superior rectus and inferior oblique [3]

diffusion-weighted image sequences was identified in 67% of cases of AOP infarction with midbrain involvement and supports the diagnosis when present. AOP occlusion accounted for 0.4% of the first stroke. Small artery disease (33–38.9%), cardioembolic source (0–22%), large-vessel disease (13.2–22.2%), and idiopathic causes (10%) are the main risk factors for AOP ischemia and thrombosis [7]. VOHS due to AOP infarct is a rarity [8].

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