Case Report
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A case of dorsal midbrain syndrome in 2nd trimester of pregnancy

Abstract

The incidence rates for ischemic strokes associated with pregnancy or puerperium vary in the literature from 5 to 210 per 100000 deliveries.[1–4] Wiebers[5] estimated that pregnancy increases the likelihood of cerebral infarction to about 13 times the rate expected outside of pregnancy; most of them were diagnosed as having cerebral ischemia and thought to be due to cerebral venous thrombosis. Cross et al.[6] identified 31 cases of ischemic carotid territory stroke in pregnant women in Glasgow during the 10-year period of 1956 through 1967. The authors suggested that the frequency of cerebral infarction was approximately 5 cases per 100000 deliveries. Syndrome consists of impaired vertical gaze, retraction nystagmus, pupillary abnormalities, convergence nystagmus, convergence spasm, vertical nystagmus and extra-ocular palsies. The first three signs are most commonly present.[12] We present a case of a pregnant patient presented to us within minutes of acute onset of nystagmus and vertical binocular diplopia following syncope for a few seconds. The initial examination at scene of the incident revealed signs of dorsal midbrain syndrome. Although MRI and MRV were normal, we insisted on a subsequent MRI with contrast to be done which had revealed the stroke site being in the midbrain region.

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Case report

A 28 years old female patient, who was 27 weeks pregnant, had a syncope followed by noticing vertical binocular diplopia. On examination, the patient had impaired elevation which was asymmetrical being more on obvious on the right side, retraction nystagmus, and pupillary light-near dissociation in both eyes. Otherwise no other focal neurological deficits were detected. MRI and MRV showed no abnormalities. While an MRI with contrast revealed a focus measuring 4X8mm of altered MRI signal at the superior para-sagittal region in the right half of the midbrain. (tegmentum) just below the thalamus with no perifocal edema or mass effect, denoting being ischemic in nature. All blood tests were within normal limits, and her coagulation profile was within normal limits (Table 1). The cause of that ischemic lesion couldn't be identified, but a planned CTA will be carried out after her delivery (as an elective c-section) to identify any vascular abnormalities. Until then, the patient is kept on oral aspirin (Figures 1 & 2).

Table 1 Published causes of ischemic stroke during pregnancy[7–11]

Etiologies of ischemic stroke	Preeclampsia/ eclampsia (%)	CSN vasculopathy (%)	Arterial dissection (%)	TTP/DIC (%)	Thrombophilia (%)	Amniotic fluid embolism (%)	Cardioembolic (%)	Atherosclerosis (lacune) (%)
Kittner et al.[7] n(%) (total n= 17)	4 (24)	3 (18)	1 (6)	1 (6)	–	–	–	–
Sharshar et al.[8] n(%) (total n= 15)	7 (47)	1 (7)	1 (7)	–	1 (7)	1 (7)	–	–
Jaijobin et al.[9] n(%) (total n= 21)	6 (29)	-	1 (5)	-	5(24)	-	4(19)	-
Jeng et al.[11] n (%) (total n= 27)	1(4)	–	–	–	10(37)	–	9(33)	-
Liang et al.[10] n (%) (total n= 1)	2(8)	–	–	–	–	1(9)	4(36)	1(9)

DIC, disseminated intravascular coagulation; TIP, thrombotic thrombocytopenic purpura

Discussion

The control of vertical eye movements is thought to be located in the dorsal midbrain in the region of the posterior commissure and mesencephalic tegmentum.[13–15] The vertical gaze center lies in close vicinity to the superior colliculus, with some of the main nuclei being the interstitial nucleus of Cajal and the rostral interstitial nucleus of the MLF. Interestingly, downward gaze is often preserved. This is in distinction to progressive supranuclear palsy, which also presents with vertical gaze palsy, but one which preferentially affects downward gaze. The reasons for this difference are not entirely clear, but it has

been suggested that the pathways for downward gaze are directly medially out of the rostral interstitial nucleus of the MLF, while those for upward gaze are directed laterally and decussate in the posterior commisure, making them more susceptible to external mass effect. With Parinaud's syndrome, patients may have a downgaze at rest, known as the "setting sun" sign. Patients may also exhibit a pseudo Argyl Robertson pupil, where the pupil is poorly reactive to light but constricts with convergence.

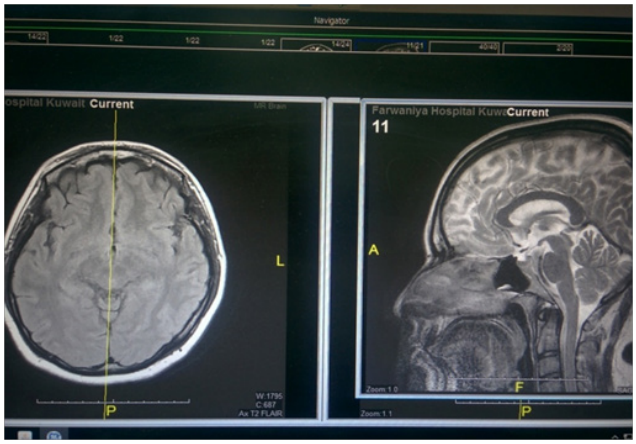

Figure 1 MRI T2 axial and sgittal sections showed no anomalies in the midbrain region.

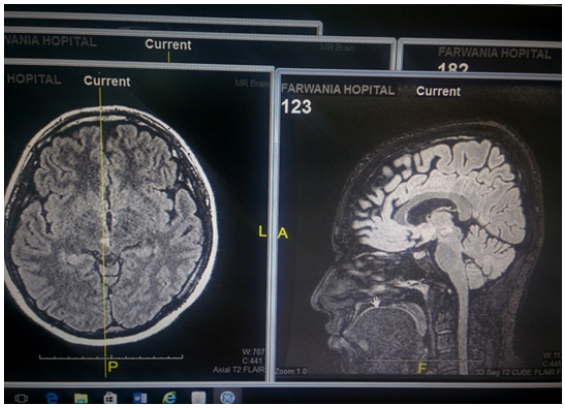

Figure 2 MRI T2 axial and sagittal sections with contrast showed the lesion in the midbrain.

This is because the impulses from the optic tract synapse in the pretectal area and then travel via the posterior commisure to both Edinger-Westphal nuclei in the posterior commisure, once again making them susceptible to external compression. Patients may also exhibit lid retraction and convergence-retraction nystagmus, which is described respectively as intermittent quick jerking movements of the eyes toward each other and retraction of the globes, are believed to be closely related.[16,17] The mechanism of convergence-retraction nystagmus is not clear, but it may represent a release phenomenon of the supranuclear cortical inhibitory fibres which results in a loss of the normal pattern of reciprocal innervation and an anomalous co-firing of the rectus muscles. Upgaze paralysis and convergence-retraction nystagmus are both associated with lesions of the dorsal midbrain and, therefore, often occur together clinically[18] A number of conditions have been reported in association with a clinical dorsal midbrain syndrome, including pineal region neoplasms, obstructive hydrocephalus, aneurysms and dorsal midbrain infarction, infection or demyelination (Figure 3).

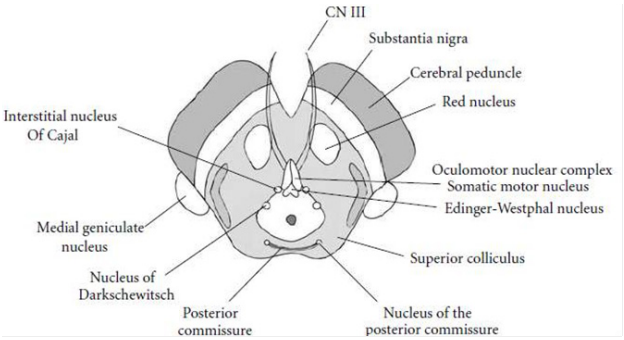

Figure 3 Midbrain anatomy at the level of the Superior Colliculus. The red nucleus and oculomotor nuclear complex are noted at this level in addition to other essential nuclear groups.[19]

The clinical signs and symptoms of the dorsal midbrain syndrome have specific localizing value, and careful directed neuroimaging studies usually will disclose an etiology. In patients without a definite lesion on initial studies, repeat neuroimaging of the dorsal midbrain may be indicated. The close anatomic relationship of multiple important structures within a relatively compact dorsal midbrain may account for the presence of pronounced clinical signs even in patients with a small lesion. In addition, a small lesion in the dorsal midbrain maybe missed or overlooked on an initial neuroimaging study.[20]

Acknowledgements

None.

Conflicts of interest

The authors declare there are no conflicts of interest.

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