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BILATERAL PARAMEDIAN THALAMIC AND MIDBRAIN INFARCTION: A Case Report

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Background: Bilateral paramedian thalamic and midbrain infarct is rare and present with varying symptoms. The occlusion of a rare arterial variant called the artery of Percheron results in bilateral thalamic infarcts, with or without midbrain involvement. To this date, the diagnosis of ischemic infarction due to occlusion of the artery of Percheronhas was uncommon. **Case:** We reports a 50 year-old man with acute ischemic infarcts of both paramedian thalami and midbrain, presenting with complete ophthalmoplegia, arreactive mydriasis pupils, and altered mental status (somnolent and fluctuating hypersomnia).**Conclusions:** Based on clinical and neuroimaging findings, we concluded this case as acute ischemic infarctions due to occlusion of the artery of Percheron.

Keywords: stroke, thalamus, midbrain, artery of Percheron, ophthalmoplegia, hypersomnia

INTRODUCTION

The thalami and the midbrain receive their blood supplies from both the anterior (internal carotid arteries) and posterior (vertebro-basilar system) circulations, and several variations in these supplies are known to exist.[1]One rare variation, named the "artery of Percheron (AOP)," is a solitary arterial trunk that arises from one of the proximal segments of a posterior cerebral artery (PCA) and supplies the paramedian thalami and the rostral midbrain bilaterally.[2] Occlusion of this artery results in bilateral thalamic and mesencephalic infarctions.[3] We describe a patient with acute ischemic infarctions due to occlusion of the AOP.

CASE REPORT

A 50-year-old man, with a history of hypertension, was transferred to the emergency room with a sudden onset of decreased of consciousness that had begun 5 h before admission. He was slightly somnolent and unable to stand or articulate words. No other medical antecedents were recorded. There was no history of injury, fever, or abnormal behavior recently.

Physical examination showed that he opened eyes in response to voice. He was also disorientated to time, place and person, and response to localized painful stimuli. He was rated 12 on the Glasgow Coma Scale (GCS). Fundus was normal. His pupils were 5 mm in diameter and fixed with no response to light. He had bilateral ptosis. His reflexes were symmetrically brisk. Oculocephalic maneuvers could not elicit horizontal or vertical eye movements. No facial droop was noted. Gag reflex was present.Signs of meningeal irritation were absent. Routine laboratory investigations were normal.

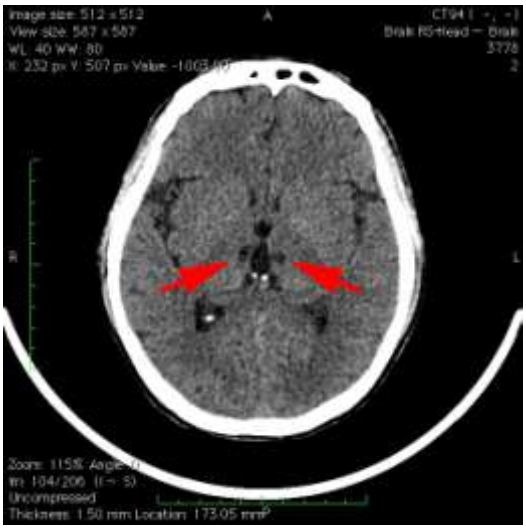

Figure 1. Head CT scan demonstrating a small hypodense lesion in bilateral paramedian thalamic (red arrows)

Acute vertebro-basilar stroke was suspected, so a head computed tomography (CT) scan was performed and demonstrated a small hypodense

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lesion in bilateral paramedian thalami (Fig. 1). A brain magnetic resonance imaging (MRI) was performed 12 days after the stroke. The MRI showed symmetrical areas of low signal intensity on T1-weighted images bilaterally in paramedian thalami and midbrain. It also showed symmetrical areas of high signal intensity on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images bilaterally in paramedian thalami and midbrain (Fig.2). There was no hyperintensity at other levels of the brainstem and no enhancement was seen in post-contrast images. Ten days later brain CT angiography (CTA) was performed. Apart from bilateral thalamic infarcts, the result was normal.

He remained slightly somnolent for two days. However, his level of consciousness gradually improved. Over the following days he was alert,yethad fluctuating hypersomnia. His ophthalmoplegia and pupils remained unchanged during the period of hospitalization.

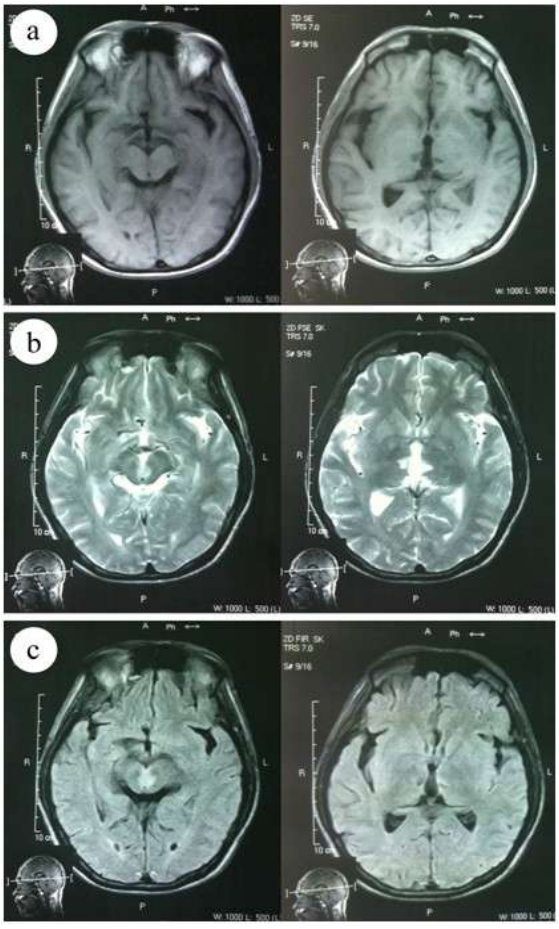

Figure 2. The MRI demonstrating symmetrical areas of low signal intensity on T1-weighted images bilaterally in midbrain and paramedian thalami (a), symmetrical areas of high signal intensity on T2-weighted (b), and FLAIR (c) images bilaterally in midbrain and paramedian thalami

DISCUSSION

The vascular territories of the thalamus are categorized into anterior, inferolateral, posterior, and paramedian territories.[4]Paramedian thalamic-midbrain infarction results from occlusion of the thalamic-midbrain perforating arteries, which arises from the proximal PCAs or from a single artery (the AOP).[5]The AOP, firstdescribed by Percheron in 1973, has been identified as a rare variation of the paramedian thalamic-mesencephalic arterial.[2] Occlusion results in a characteristic pattern of ischemia: bilateral paramedian thalamic infarcts with or without midbrain involvement. The most common underlying causes of an AOP occlusion are small artery disease and cardioembolism.[2]The prevalence of the AOP is unknown, a small study of 15 cadaver brains demonstrated the AOP in one specimen. To date, the diagnosis of ischemic infarction due to occlusion of the artery of Percheron has been uncommon.[6]

Gerard Percheron described three variations involving the paramedian thalamic-midbrain arterial supply: small branches arising from both P1 segments, an asymmetrical common trunk arising from a P1 segment (this variation is called the AOP), or an arterial arcade emanating from an artery bridging the two P1 segments (Fig.3).[11]

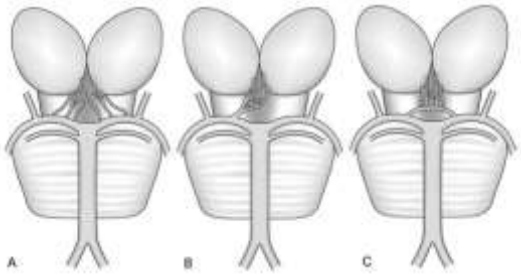

Figure 3. Variations of the paramedian thalamic-midbrain arterial supply according to Percheron. (A) In the most common variation, there are many small perforating arteries arising from the P1 segments of the PCA. (B) The AOP is a single perforating blood vessel arising from one P1 segment. (C) The third type of variation is that of an arcade of perforating branches arising from an artery bridging the P1 segments of both PCAs.[1]

We describe a patient with acute ischemic infarcts involving both paramedian thalami and midbrain, presenting with complete ophthal-moplegia, arreactive mydriasis pupils, and altered mental statusas the neurological abnormality.This patient demonstrated similar symmetric thalamic and mesencephalic lesions typically seen in occlusion of the AOP.

Complete ophthalmoplegia, the combination of bilateral ptosis with loss of all extra-ocular movements, is rarely encountered in clinical practice.[5]Bilateral infarction usually produce

socular motor abnormalities. Complete ophthalmoplegia in our patient could potentially result from bilateral pontine and midbrain lesions, at the level of the Edinger-Westphal nucleus. However no pontine abnormality was present on imaging in our patient. Although we could not be sure that there was no pontine lesion without histopathologic confirmation, we believe that our patient most likely had bilateral pseudo-abducens palsies. The pupils are often abnormal and can be unreactive, as in our patient. Pupil size varies depending on involvement of the Edinger-Westphal nucleus or oculomotor fascicles (large pupils), descending sympathetic fibers (small pupils), or some combination of these structures (midrange pupils).[5]

Alteredmental status can present anywhere on the spectrum from drowsiness or confusion to hypersomnolence or coma. Our patient was slightly somnolent for two days and had fluctuating hypersomnia over the following days. Hypersomnia is a well-known manifestation of bilateral thalamic ischemic stroke.[7,8] Hypersomnia is attributed to interruption of arousal pathways which relayed from ascending reticular activating system (ARAS) through the thalamus and diffusely to the cortex.[7]

Appropriate imaging is a mandatory to make an exact etiological classification of any thalamic stroke. Besides the initial need to exclude a hemorrhage by means of conventional head CT scan, CT scanning further allows to perform a CTA immediately thereafter. An accurate diagnosis may also be done by MRI.[9] The AOP is rarely visualized with angiography because it is too small to be seen radio-graphically on conventional-angiography, CTA, and MR angiography.[2,6] However, this can be utilized to roll out the involvement of larger vessels as the etiological origins of bithalamic infarction.[2] CTA provides a means to rapidly and noninvasively evaluate the intracranial and extracranial vasculature in acute, subacute, and chronic stroke settings and thus to provide potentially important information about the presence of vessel occlusions. The accuracy of CTA for evaluation of large-vessel intracranial occlusions is very high.[10]In our case, head CT scan demonstrating bilateral paramedian thalamic infarction, MRI showing the involvement of the rostral midbrain, and CTA role out the involvement of larger vessels.

Intra-arterial thrombolytic therapy requires intervention within the first few hours after the onset of stroke. Thrombolytic therapy that is administered many hours or days after the onset of stroke can even be dangerous to the patient due to hemorrhagic effect.[10] Intra-arterial thrombolysis and anticoagulation have been proven to be good treatment options for AOP infarction.[11] Our patient was transferred to the emergency room with a sudden onset of decreased of consciousness that had begun 5 h before admission. We did not follow the procedure of intra-arterial thrombolytic therapy; furthermore, the facilities in the hospital were insufficient enough to do so, then we give general management and secondary prevention.

Prognosis after thalamic infarction is generally regarded as being rather good compared with lesions of the cerebral cortex or other sub-cortical structures, but this generally applies to the low incidence of mortality and the good recovery from motor deficit.[8]Our patient slightly somnolent for first two days. However, his level of consciousness gradually improved. Over the following days, he was alert but had fluctuating hypersomnia. His ophthalmoplegia and pupils remained unchanged up to the present moment.

CONCLUSION

In conclusion, we describe the patient with acute ischemic infarctions of both paramedian thalami and midbrain, presenting with complete ophthalmoplegia, arreactive mydriasis pupils, and altered mental status (somnolent and fluctuating hypersomnia) as the neurological abnormality. This patient demonstrated similar symmetric thalamic and mesencephalic lesions typically seen in occlusion of the AOP, even in the absence of angiographic evidence.

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