Bilateral Thalamic And Midbrain Infarcts: A Case Of Percheron’s Artery Occlusion.

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Abstract
The variant artery of Percheron could be a rare cause of multiple infarcts in the posterior circulation. An unusual case of multiple ischemic brain infarcts with specific anatomical distribution is presented. A 58-year-old Caucasian man was admitted to the hospital because of confusion, bilateral ptosis and bilateral external and internal ophthalmoplegia since 48 hours. He had history of metallic mitral valve since 2 years treated with warfarin. Brain imaging reveals symmetric bilateral paramedian thalamic and periaqueductal midbrain ischemic lesions. Percheron’s artery occlusion could lead to bilateral paramedian thalamic and midbrain infarcts. The symmetric distribution of infarcts in our patient should be explained by the existence of the variance of Percheron in the blood supply network. The mirror damages in the thalamus and midbrain have to bring suspicion and direct the differential diagnosis which should be taken into consideration in the differential diagnosis especially in cases with cardiogenic emboli.

Key words: Stroke, bilateral thalamic infarcts, artery of Percheron, multiple infarcts, midbrain infarcts, posterior circulation infarcts

Introduction
The arterial supply to the thalamus is provided by perforating branches from the posterior cerebral artery and the posterior communicating artery. (1) The paramedian thalamic arteries arise from the P1 segment of the posterior cerebral artery and supply the medial areas of thalami. It may share a common origin with the superior paramedian mesencephalic arteries that supply the medial areas of the upper brainstem. (2)

Historically Percheron Gerard described four variants of paramedian perforating arteries to the thalami:
-Type I: one paramedian thalamic artery arises on each side from the corresponding P1 segment of posterior cerebral artery
-Type II is asymmetrical: the right and left paramedian thalamic arteries arise from the same P1 separately (type Ia) or by a common trunk which then bifurcates (type Ib, called as artery of Percheron)
-Type III: an arterial arcade connects the two P1 and gives rise to the paramedian arteries. (3)

Multiple cerebral ischemic lesions can be caused by cardioembolic disease, carotid artery disease, occlusion of a small blood vessel, hypercoagulation state, systemic disease (e.g. hypertension, sepsis). Occlusion of the artery of Percheron often results in bilateral infarcts in the middle aspects of thalami with or without midbrain infarcts.

Bilateral paramedian thalamic strokes are typically characterized by fluctuated level of alertness, vertical gaze palsy and memory impairments. These are the commonest presenting features; although the overall clinical picture is highly variable. (4)

Most of the patient with occlusion of the artery of Percheron will have a good prognosis after appropriate treatment. (1) We describe a patient with bilateral brain ischemic lesions most likely explained by the existence artery of Percheron.

Body text
A 58-year-old man was admitted because of somnolence, bilateral ptosis and bilateral external and internal ophthalmoplegia.
He had a metallic mitral valve (under warfarin), type 2 diabetes mellitus, and alcohol and cigarette abuse. The symptoms had begun 48 hours ago. The patient who lives in a rural area woke up with confusion and bilateral ptosis and was admitted urgently to the nearest local hospital where the first medical evaluation reports somnolence, bilateral ptosis, bilateral external and internal ophthalmoplegia (bilateral mydriasis, without pupillary reflex). There was performed a CT brain scan (no contrast) that revealed a hypodense lesion with hyperdense regions at the right frontal lobe and a hypodense lesion at the median region of right thalamus. The patient was admitted to general territory hospital with suspicion of brain tumor for further management, while the warfarin was discontinued.

At the territory hospital the first neurological evaluation revealed fluctuated level of consciousness, verbal and motor response when he was aroused, bilateral ptosis, bilateral external and internal ophthalmoplegia with eyes in abduction, and right side Babinski sign.

A MRI brain scan revealed symmetric bilateral paramedian thalamic ischemic lesions, periaqueductal midbrain and right frontal lesions on a background of chronic ischemic leukoencephalopathy (Fig 1).

Figure 1. Internal and external ophthalmoplegia.

The diagnosis of occlusion of the arP was made. The patient was discharged after 4 weeks of his admission, conscious, oriented, with bilateral ptosis, bilateral internal ophthalmoplegia, abduction nystagmus and vertical gaze palsy.

Result and Discussion

A 58-year-old Caucasian male was admitted to our department with confusion, bilateral ptosis and bilateral external and internal ophthalmoplegia since 48 hours.

He had history of metallic mitral valve since 2 years due to rheumatic fever treated with warfarin, type 2 diabetes mellitus treated with insulin, alcohol and cigarette abuse.

The patient who lives in a rural area woke up with confusion and bilateral ptosis and was admitted urgently to the nearest local hospital where the first medical evaluation reports somnolence, bilateral ptosis, bilateral external and internal ophthalmoplegia (bilateral mydriasis, without pupillary reflex). A CT brain scan (no contrast) revealed a hypodense lesion with hyperdense regions at the right frontal lobe and a hypodense lesion at the median region of right thalamus. The patient was admitted to general territory hospital with suspicion of brain tumor for further management, while the warfarin was discontinued.

At the time of his admission on Emergency Department he had fluctuated level of consciousness, from being alert to being aroused only by painful stimuli, oriented verbal and motor response when he was aroused, bilateral ptosis, bilateral external and internal ophthalmoplegia with eyes in abduction (Figure 1), no other motor or any sensory deficit, deep tendon reflexes were symmetrical, left plantar reflex was upgoing and right plantar reflex was downgoing.

A second CT brain scan revealed bilateral thalamic and median midbrain hypodense lesions and the hypodense lesion at the right frontal lobe with hyperdense regions as referred to the first CT brain scan.

The following laboratory tests were unremarkable: blood cell count, glucose, electrolytes, liver enzymes and creatinine, HbA1c; INR was 1.85 compare to 3.27 at his first admission at the local hospital. Transthoracic echocardiogram revealed a minor mitral valve insufficiency and no presence of any clot in the left atrium. After 2 efforts we could not managed to perform a transesophageal echocardiogram due to the patient’s poor cooperation Doppler ultrasound evaluation of carotids and vertebral arteries was unremarkable.

A brain MRI brain scan revealed hyperintense symmetric bilateral paramedian thalamic, periaqueductal midbrain and right frontal lesions on a background of chronic ischemic leukoencephalopathy. (Figures 2 and 3)

Figure 2. Axial T2 FLAIR brain MRI of our patient. Revealing hyperintense symmetric bilateral paramedian thalamic lesions and a hyperintense right frontal lesion on a background of chronic ischemic leukoencephalopathy.
The diagnosis of occlusion of the vascular variant known as artery of Percheron was made. The patient was treated conservatively as an ischemic stroke with restoration of anticoagulation treatment. Intravenous thiamine supplementary treatment was also initiated early, due to the history of alcohol abuse and the initial differential diagnosis of Wernicke’s encephalopathy.

The patient was discharged from after 4 weeks of his admission. He was conscious and oriented, with bilateral ptosis, bilateral internal ophthalmoplegia, abduction nystagmus and vertical gaze palsy and no other motor or any sensory deficit. We report long-term memory deficits in our patient.

Our patient had the main clinical features of the occlusion of the artery of Percheron with midbrain involvement such as:

a) Fluctuating level of consciousness, explained by the disrupted connections between the thalamus and the anterior, orbitofrontal and medial prefrontal cortices. (2,4,5)

b) Vertical gaze palsy, explained by the thalamic lesions affecting the rostral interstitial nuclei of the medial longitudinal fasciculus. (2,3,4)

c) Bilateral internal ophthalmoplegia, bilateral ptosis and the deficits of horizontal eye movements explained by the bilateral oculomotor nerve insult and the possible bilateral internuclear ophthalmoplegia due to the midbrain lesion. (6)

Brain MRI is the best imaging modality to delineate the damaged areas. Performing conventional angiography may not be indicated, because lack of visualization of the artery of Percheron does not exclude its presence. (10, 11)

The hypodense lesion with hyperdense regions at the right frontal lobe is considered as a former ischemic lesion with hemorrhagic transformation since it is shown in the first CT scan.

In our patient we assume that the occlusion of the artery of Percheron is the cause of the thalamic and midbrain lesions rather than multiple emboli that should affect the left anterior circulation in priority.

In our case the diagnosis of occlusion of the arP was only indirectly diagnosed since the artery is is too small to be visualised by MRA and can possibly be detected on angiograms. In addition lack of visualization of the artery of Percheron does not exclude its presence. (7, 8, 9, 10)

Conclusion

Occlusion of the artery of Percheron is a rare cause of bilateral paramedian thalamic and midbrain infarcts. Clinicians should be aware of this arterial variant in the management of patients with acute symptoms from thalamic and midbrain areas.

References


