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Case report

Midbrain and bilateral paramedian thalamic stroke due to artery of Percheron occlusion

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ABSTRACT

Introduction: Bilateral thalamic strokes are rare manifestations of posterior circulation infarcts. Usually the etiology is cardioembolic or small vessel disease combined with individual anatomical predisposition. The symptoms include a variety of neurological deficits depending on thalamic structure involvement, such as paresthesias or numbness, hemiparesis with increased reflexes and Babinski sign, third cranial nerve palsy, speech and cognition disturbance, memory impairment and stupor. Neuroimaging usually reveals ischemic loci in adequate thalamic nuclei.

Case presentation: We report a case of 61-year-old man, active smoker (25/per day, 50 pack-years) with untreated hypertension who presented at admission consciousness impairment (Glasgow Coma Scale score 9 points), left pupil dilatation without reaction to light, left eye deviation downwards and outwards, vertical gaze paralysis and left-sided hemiplegia. Initial brain computed tomography (CT) was normal. Brain magnetic resonance with diffusion weighted imaging and fluid attenuation inversion recovery sequences (MR DWI/FLAIR) performed on admission showed ischemic changes in bilateral thalami, which were confirmed in routine MRI. Thrombosis of basilar artery and cerebral venous was excluded in CT angiography. Further diagnostic assessment revealed hyperlipidemia, paroxysmal atrial fibrillation and renal cancer with hepatic metastases.

Conclusion: Bilateral thalamic stroke due to artery of Percheron occlusion is a rare presentation of stroke, which can be overlooked in routine CT scan. If diagnosed, it requires further evaluation for stroke risk factors, especially cardiovascular disorders associated with increased embolic risk.

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1. Introduction

The thalamus, a part of diencephalon, is a structure located between the cerebral cortex and the midbrain, which is involved in receiving, modifying and transferring sensory and motor signals and which regulates consciousness, sleep and alertness by relaying limbic system information [1]. The thalamus is supplied with blood by several arteries: the polar artery which derives from posterior communicating artery, paramedian thalamic-subthalamic arteries (arising from segment P1 of posterior cerebral artery), inferolateral (or thalamogeniculate) artery and choroidal arteries all arising from segment P2 of posterior cerebral artery [2]. In some individuals both thalami are supplied by artery of Percheron, which is a single artery that derives from posterior cerebral artery and its occlusion may result in bilateral thalamic infarction [2].

Ischemic or hemorrhagic lesions in different vascular territories of thalamus result in various neurological syndromes. Polar artery occlusion usually causes infarction of anterior group of thalamic nuclei with neuropsychological deficits (abulia and apathy), personal changes, memory impairment (especially in left-sided infarcts) and consciousness disturbances [3–6].

Paramedian thalamic-subthalamic arteries supply centromedian part of the thalamus and their occlusions result in decreased level of consciousness (arousal impairment, lethargy and hypersomnolence), neuropsychological disturbances and vertical gaze impairment, loss of convergence or pupils abnormalities [6–9]. In case of inferolateral (or thalamogeniculate) artery infarctions, dorsal and posterolateral regions of the thalamus are involved, which clinically results in Dejerine–Roussy syndrome with sensory loss, hemiparesis and sometimes poststroke thalamic pain [6,10].

Characteristic clinical features for posterior choroidal arteries infarcts are visual deficits (mainly quadrantanopia) with visual hallucinations, mild sensory or motor impairment and hyperkinetic syndromes (e.g. ataxia, tremor, dystonia, chorea) [7,11,12].

Thalamic strokes represent 3% of all ischemic strokes and only 11% of all vertebrobasilar infarcts [13,14]. Bilateral infarcts are rare and have been reported in several case presentations [15–17]. Most thalamic strokes involve median part of thalamus (25%) and 72% of them are ischemic [18]. The incidence of bilateral thalamic stroke is unknown.

The main risk factors for thalamic strokes are atrial fibrillation, hypertension and atherosclerosis secondary to cigarette smoking, diabetes mellitus, hypertension or dyslipidemia. These conditions are usually associated with multiple lesions [18]. Other conditions, which are associated with increased embolic risk, such as congenital atrial septal defect [15] or other inherited cardiac disease (e.g. hypertrophic or dilated cardiomyopathy) can be also a cause as well as other conditions associated with increased embolic risk (such as neoplasms, hypercoagulable states). As a rather rare causes of thalamic stroke are listed migraine and vasculitis secondary to chronic central nervous system (CNS) infections [6,18].

Brain MRI is a reference imaging modality for thalamic stroke and should be performed as primary or at least confirmative imaging method with routine FLAIR/DWI and T1 and T2 sequences [19]. Brain CT is useful for hemorrhagic lesion exclusion. As far as artery of Percheron imaging is concern MR/CT angiography or conventional angiography should be considered, but probability for occlusion detection is small [19].

2. Case report

61-year-old man, active smoker (25/per day, 50 pack-years) with untreated hypertension was admitted due to *consciousness disturbance and left-sided paresis*. The exact time of beginning of the symptoms was unknown – the patient was found unconscious early in the morning on the day of admission (wake-up stroke).

In Emergency Room his Glasgow Coma Scale (GCS) score was 9, in neurological examination the left pupil was dilated, without reaction to light, and left eye was deviated downwards and outwards. Moreover, patient presented with vertical gaze paralysis and left-sided hemiplegia with bilateral Babinski sign.

An urgent brain CT scan was performed, but it did not revealed any acute ischemic changes in thalamic region (Fig. 1), although there were signs of previously silent ischemic lesions localized mainly in deep structures of the brain. Brain MRI DWI/FLAIR performed on admission showed ischemic changes consistent with acute infarction in bilateral thalami (Fig. 2). Symmetrical areas of low signal intensity were shown on ADC maps bilaterally in paramedian thalami, symmetrical areas of high signal intensity were presented on diffusion weighted images (DWI), in FLAIR only subtle hyperintense signals were seen in corresponding areas (which suggested acute lesions).

Carotid and transcranial Doppler ultrasound revealed hemodynamically irrelevant 40% stenosis of the left internal carotid artery and 30% stenosis of the right internal carotid artery. Blood flow in basilar, vertebral and visible intracranial arteries was normal.

The routine MRI (Fig. 3) performed 3 days after admission confirmed subacute infarction in bilateral thalami and anteromedial midbrain suggesting occlusion of the artery of Percheron (AOP). The hyperintense signal intensity along the pial surface of the midbrain interpeduncular fossa representing the ''V sign'' [19].

Computed tomography angiography (both arterial and venous phases) did not revealed direct signs of AoP occlusion, although there was secondary evidence for it.

During hospitalization, the patient was newly diagnosed with hyperlipidemia and paroxysmal atrial fibrillation. He remained slightly somnolent for several days. Initially his level of consciousness gradually improved, but his oculomotor symptoms and pupils remained stable. After 10 days of hospitalization level of consciousness had begun worse, without evidence of epilepsy or new vascular lesions. Additional diagnostic work up performed because of increased levels of D-dimers showed renal cancer with hepatic metastases. The functional as well as neuropsychological outcome was pure, partially probably because of severe comorbidity. The long-term outcome was also unfavorable, 3 months after stroke, the patient was fully dependent on relatives with persistent somnolence and apathy.