BITHALAMIC STROKE DUE TO OCCLUSION OF PERCHERON'S ARTERY

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Introduction: Paramedian thalamus is usually supplied from each side of thalamoperforating arteries. In 1973 Gerard Percheron, a French neurologist, identified a rare anatomical variant which supplies bilateral sides of paramedian thalamus (fig. 1). This variant artery was later named as the "artery of Percheron" (AOP). Occlusion of AOP is presumed to be the main cause of bilateral paramedian thalamic infarction, which may combine with or without ischemic interpeduncular infarction. While normally only one paramedian artery arises from a single P1 segment, in some, both paramedian arteries arise from a common P1 trunk [1]. Typical clinical features of bilateral paramedian thalamic infarction include the triad of altered mental status, vertical gaze paresis and memory impairement. In some cases convulsion are reported [2]. Thalamic stroke account for 11% of vertebrobasilar infarct. Stroke limited to paramedian territories account for about 22-35% of all



Fig. 1: Schematic representation of variation of the paramedian thalamicmesencephalic arterial supply according to Percheron. (A) In the most common variation, many small perforating arteries arise from the P1 segment of the PCA. (B) The AOP is a single perforating blood vessel arising from a P1 segment. (C) In the third variant, an arcade of perforating branches arises from an artery bridging the P1 segments of both PCAs. PCoA, posterior communicating artery; PCA, posterior cerebral artery; BA, basilar artery; SCA, superior cerebellar artery; AOP artery of Percheron.

thalamic infarcts, usually due to cardioembolism [3].

Clinical Case: We report the case of a 55 years old man who presented to emergency department for coma with a Glasgow Coma Scale (GCS) score of 8/15. Pt underwent a CT scan which was unremarkable and a brain MRI with evidence of ischemic infarction in the mesial part of both the thalami and in the upper part of the mesencephalon (fig. 2). The patient soon was admitted to the stroke unit where a neurological examination performed four days after revealed an alert patient with temporo-spatial disorientation, vertical gaze paralysis, convergent strabismus, slight anisocoria (sn>dx), dysarthria, left-side hemiparesis. No sensitive anomalies. NIHSS: 4. Ecocolordoppler revealed a bilateral stenosis of the internal carotids at the bifurcation (50% right; 60% left). A selective arteriography showed the presence of the anatomical variant of Percheron artery, starting from left-side P1 segment. Patient was transferred to neurorehabilitation 16 days after the stroke. Neurological examination at the admittance to our clinic revealed a GCS score of 14/15, vertical gaze palsy and left side inclination of the head because of disallineation of the eyes, bilateral pseudosixth, slight anisochoria, torpid bilateral pupillary reflex. Absence of focal motor and sensory deficits; diffuse brisk reflexes without Babinski sign. Extrapiramidal signs were evident with hypofonia, bradykinesia, diffuse plastic hypertony. Patient was very sleepy and difficult to awake. An EEG revealed a left temporal focus (fig. 3).

Levetiracetam (1000 mg/die) and Levodopa (400 mg/die) were started and during the following two months the pt underwent physical therapic (12/weeks) and logopedic training (6/week) with improvement in alertness and reacquisition of independent deambulation with persistence of memory impairement. We performed memory test that revealed severe impairement of short-term memory, attention ad temporo-spatial orientation. MMSE: 12.9/30.





Conclusion: First described in 1973, the artery of Percheron is an unusual anatomical variant where a single thalamic perforating artery arises from the proximal posterior cerebral artery between the basilar artery and posterior cerebral artery. When occluded, bilateral thalamic stroke, often with upper mesencephalon involvement, occurs. In literature are reported only few cases and nobody focus on follow-up. Symptoms at onset are well described with alteration of consciousness, often with prolonged coma, complex ocular movement disorders, impairment of consciousness. There is a single report of stroke-induced parkinsonism.

Even if bithalamic infarction is a very rare condition it should be suspected in a patient with coma and ocular movement disorder; in this cases obtaining a brain MRI is essential and allow clinician to reveal the typical bithalamic stroke that strongly supports the hypothesis of an anatomical variant of Percheron. Prognosis quoad vitam is good once the pt survive the acute phase, but severe impairement of memory and temporo-spatial orientantion are causes of a total loss of independence.

Our case is typical for the presentation but is very interesting because of the presence of parkinsonism. Furthermore, even in absence of epileptic seizures an EEG revealed abundant focal paroxysmal activity. At six months - follow up our patient is able to walk without assistance and although affected by only slight motor deficits he lost his autonomy and now he is living in a retirement home.



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