Occlusion of the artery of percheron: Clinical and neuroimaging correlation

A 72-year-old woman was admitted to the emergency department with acute altered levels of consciousness, respiratory difficulties and bradycardia. She had a history of hypertension and ischemic cardiopathy. On admission, she had a Glasgow Coma Scale score (GCS) of 8. Physical examination showed executive dysfunction, initiative loss, hypersomnia, a right Babinski sign and bilateral cranial nerve-III palsies (ptosis and mydriasis). For several hours, the patient exhibited fluctuating levels of consciousness (GCS score varying from 8 to 12) and improvement in her respiratory state. The CT-scan performed within three hours was normal. The electrocardiogram showed auricular fibrillation alternating with auricular flutter. Transthoracic echocardiography indicated grade-II mitral regurgitation with no intracavitary thrombi. Brain MRI (Fig. 1), including fluid-attenuated inversion recovery (Flair), showed thalamic and midbrain hypersignals related to infarction. Non-contrast T1- and T2*-weighted MR sequences showed a hemorrhagic component in the infarction and postcontrast T1-weighted images demonstrated a blood–brain barrier rupture. The electroencephalogram performed 15 days after the onset of symptoms was normal. During hospitalization, the patient recovered partially, but the bilateral ptosis persisted.

Percheron studied thalamic vascularization and described three anatomical variations. When the so-called 'artery of Percheron' is occluded, the bithalamic infarcts are paramedian, relatively symmetrical and associated to a mesencephalic involvement [1]. Clinically, the main symptoms are vigilance disturbances in the early stages and lasting for hours to days, pseudohypersomnia, which persists even longer and vertical gaze disorder [2,3]. Such signs could be associated with cognitive impairment, especially amnesia and executive dysfunction, and mood changes [4]. The vertical gaze palsy reported in cases

Figure 1  A: sagittal T1-weighted image shows a hyperintense signal in the midbrain; B: axial T2*-weighted image shows a hypointense signal in the midbrain secondary to a microscopic haemorrhage; C: axial Flair images show infarcts in the medial inferior thalamus and medial superior midbrain; D: post-contrast axial T1-weighted image shows contrast enhancement in the infarct.

Figure 1  A : coupe sagittale en pondération T1 montrant un hypersignal du mésencéphale ; B : coupe axiale en pondération T2*: hyposignal punctiforme du mésencéphale témoin d’un saignement microscopique ; C : IRM en coupe axiale Flair : infarctus bilateral de la partie médiale et inférieure du thalamus et de la partie médiale et supérieure du mésencéphalique ; D : coupe axiale en pondération T1 après injection de gadolinium : prise de contraste au niveau de l’infarctus.
of paramedian thalamic infarcts is due to an associated involvement of the rostral midbrain tegmentum, including the interstitial nucleus of Cajal [5]. The ptosis and mydriasis observed in our patient were related to the mesencephalic lesion, resulting in nuclear involvement of the third cranial nerve bilaterally. Conventional MRI and diffusion/perfusion-weighted imaging confirmed the presence of the infarcts [6]. Small-artery disease and cardioembolism were the most frequent stroke mechanisms in paramedian infarcts [4]. In our case, cardiac rhythm disturbances (auricular fibrillation and auricular flutter) were incriminated.

The prognosis differs from one patient to another, depending on the underlying etiology and associated diseases. In general, the consciousness-related problems resolve favorably within a matter of hours to days, although the hypersomnia can last considerably longer.

References


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