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Case Report

Artery of Percheron infarction presented with isolated downgaze paralysis: A case report ☆,☆☆

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ABSTRACT

Isolated downgaze paralysis is the most infrequent expression of vertical gaze abnormalities. Vertical eye movements are controlled by nuclei and circuits located in the thalamic-mesencephalon region, and more particularly the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF). The Artery of Percheron (AP) is a rare vascular anatomic variation that supplies the paramedian region of the thalami and the rostral portion of the mesencephalon. We present a unique case of isolated downgaze paralysis caused by AP ischemia.

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Introduction

Vertical gaze paralysis could be specific, affecting only up or down gaze, or total with disturbance of up and down vertical eye movements. Isolated downgaze palsy is the rarest presentation of the vertical gaze disorders [1]. The nuclei and circuits in the mesencephalon and thalamus regulate vertical eye motions. The interstitial nucleus of Cajal (iNC), the posterior commissure, and the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) have all been linked to particular impairments in vertical gaze [2].

The artery of Percheron (AP) was first mentioned by Gérard Percheron in 1973. It is a solitary branch that emerges from the proximal posterior cerebral artery (PCA) and supplies blood to the upper mesencephalon and both paramedian thalami [3]. The most common signs of AP infarction are altered awareness, memory impairments, and supranuclear vertical gaze palsies [4]. In this report, we describe an unusual case of isolated downgaze palsy secondary to AP infarction.

Case report

A 37-year-old man was hospitalized 24 hours after experiencing intermittent diplopia and dizziness. He had no particular medical history or long-term medication use. Neurologic ex-

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Fig.1 – Extraocular eye movements in all cardinal points. Note the downgaze paralysis.

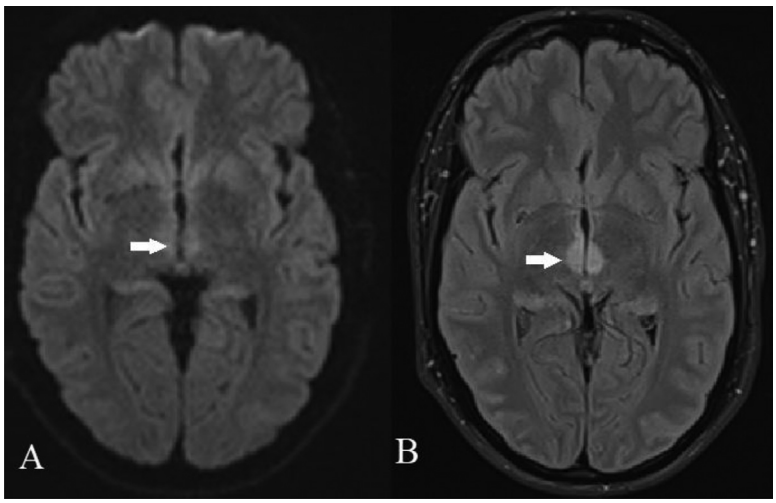

Fig. 2 – Cranial MRI on axial DWI (A) and FLAIR (B) sequences demonstrated high signal intensity on bilateral paramedian thalami (arrows).

amination showed a conscious, well-oriented, and cooperative patient with no motor, coordination, or sensory deficit.

Neuro-ophthalmologic evaluation found that visual acuity, color vision, and pupillary reflex were intact. Slit-lamp and fundus exams found normal results. Confrontation test showed no visual field anomalies, with orthophoria at the primary position in both eyes. Conjugate downgaze voluntary saccades and pursuit movements were absent. The globes could not move below the horizontal meridian, and the patient had to rotate his head to observe the lower quadrants (Fig. 1). Paradoxically, the oculocephalic reflexes were preserved, suggesting supranuclear downgaze paralysis.

Cerebral magnetic resonance imaging (MRI) on fluid attenuated inversion recovery (FLAIR) and diffusion-weighted imaging (DWI) sequences showed bilateral paramedian thalamic high signal intensity (Fig. 2). Flair and T2 weighted sequences demonstrated hyperintensity in the rostral midbrain (Fig. 3). Magnetic resonance angiography (MRA) demonstrated an aberrant small artery emerging from the P1 portion of the right PCA, compatible with AP (Fig. 4).

Large paraclinical tests were normal (complete blood count, prothrombotic tests, hepatic and renal tests, hemoglobin electrophoresis, glycosylated hemoglobin, autoantibody tests, syphilis serology, hepatitis and HIV tests). Cerebrospinal fluid analysis was normal. Electrocardiogram (ECG), Doppler ultrasonography of the cervical arteries, transesophageal echocardiography, and Holter ECG were unremarkable.

For 3 weeks, the patient received acetylsalicylic acid and clopidogrel, followed by aspirin with orthoptic treatment. Three months after his admission, the clinical evaluation noticed partial recovery of the downgaze palsy. Cranial MRI control showed lacunar ischemic lesions involving the thalami and the rostral mesencephalon (Fig. 5).

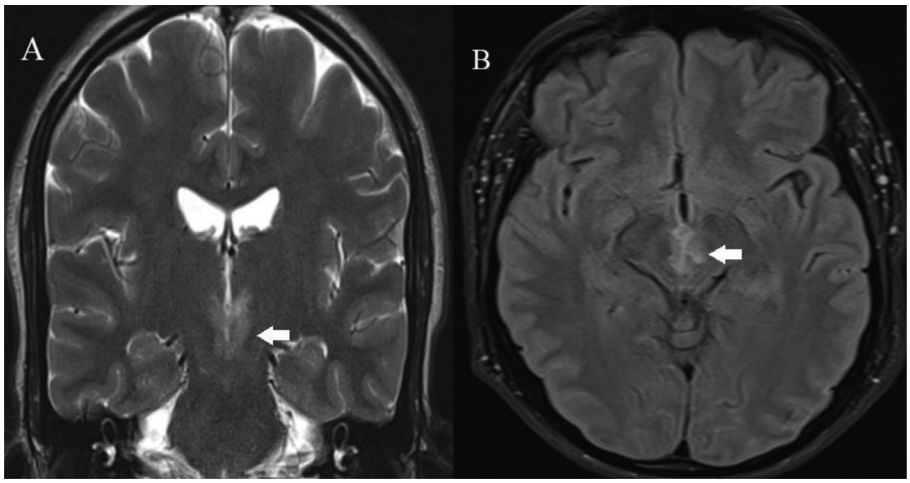

Fig. 3 – Cerebral MRI on coronal T2 (A) and axial FLAIR (B) sequences showed hyperintensity on the rostral midbrain (arrows).

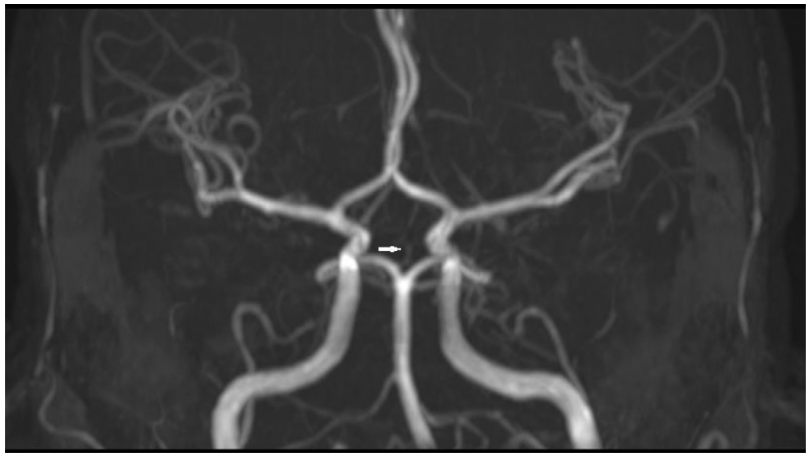

Fig. 4 – MRA demonstrated an abnormal small vessel emerging from the P1 segment of the right PCA (arrow).

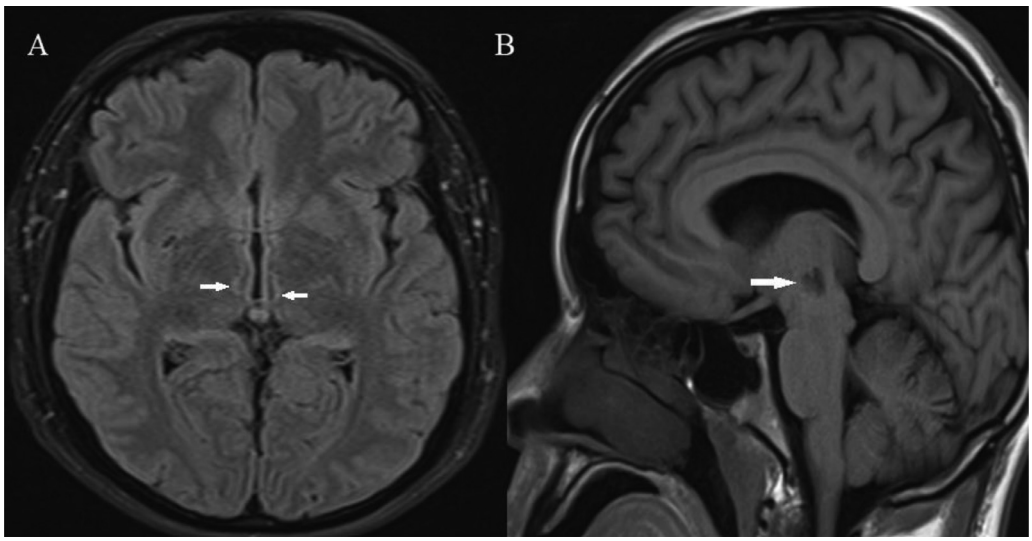

Fig. 5 – MRI control on axial FLAIR sequence (A) showed lacunar infarct of bilateral paramedian thalami (arrows). Sagittal T1 sequence (B) found low signal intensity in the rostral mesencephalon (arrow).

Discussion

Selective horizontal and vertical gaze paralysis can be noted clinically or produced experimentally because the neuroanatomical structures regulating the horizontal and vertical eye movements are situated at various levels in the neural axis. The thalamic-mesencephalon junction and the rostral midbrain area contain the regions that regulate vertical eye movements [5].

Although the iNC, superior colliculus, and posterior commissure have all been implicated in the regulation of vertical gaze, recent research indicated that riMLF is likely to be responsible [6]. The medial portion of the riMLF is the crucial region for downgaze, according to Pierrot-Deseilligny et al. [7]. riMLF lesions can impair either the up or down gaze, or both, but isolated downgaze paralysis is uncommon.

Neuropathological investigations are limited because the lesions that are mostly infarctions that cause vertical gaze palsy are typically small and resolve spontaneously. This highlights the significance of neuroradiological imaging techniques in comprehending the supranuclear regulation of vertical gaze [8]. Based on MRI study findings, bilateral synchronous electrical activation of riMLF produces downgaze, and bilateral riMLF lesions cause downgaze disturbances [1]. The riMLF located in the rostral mesencephalon and the thalamic-midbrain junction are nourished by the posterior thalamic paramedian artery, also known as AP.

AP is an unusual vascular branch variant emerging from the P1 section of PCA and represents the source of bilateral perforating thalamic arteries. The exact prevalence of the AP is not well known, due to its tiny diameter and the possibility of remaining unrecognized. The obstruction of the AP is a unique variant that may cause bilateral paramedian thalamic and midbrain infarction [9].

Thalamic infarcts have been documented for a long time, but they still constitute an uncommon form of stroke, accounting for only 11% of all vertebrobasilar infarcts [10]. A large study on the prevalence of AP infarction among all ischemic strokes concluded that it occurs at an estimated prevalence of 0.1% [11]. Individuals with bilateral paramedian thalamic infarctions compatible with an AP occlusion generally manifests with 3 major symptoms, including coma, memory loss, and vertical gaze palsy [9].

The diagnosis of AP infarction is essential for determining the proper treatment and avoiding needless procedures [9]. The exams of priority for detecting AP ischemia are DWI and FLAIR MRI sequences. AP shows on cerebral MRA as a unique vessel emerging from the P1 section of 1 PCA and divided into 2 arteries nourishing bilateral thalami [12].

Radiological differentials in AP infarction are essentially vascular with the basilar syndrome and venous thrombosis of internal cerebral veins. Nonvascular differentials in bilateral paramedian thalamic lesions include, bacterial or viral infection, demyelinating diseases, hypoxic injury, thiamine deficiency, and tumors [13]. The usual causes of bilateral thalamic ischemia are cardio-embolism and small vessel occlusion. The risk factors are diabetes mellitus, hypertension, atrial fibrillation, and coagulopathy.

Conclusion

Despite being an uncommon anatomical variation, AP should be considered once paramedian thalamic lesions appear on cerebral imaging. Downgaze palsy could be a unique clinical manifestation of AP infarction. A solid awareness of the distinctive pattern of AP thrombosis, as well as the diversity of its clinical signs, will aid in rapid diagnosis, appropriate treatment, and good prognosis.

Patient consent

I qualify as the corresponding author to this manuscript warrant that I have informed the patient of this scientific manuscript, and I confirm that I obtained his written and informed consent for the publication of this article.

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