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Review

Midbrain Hemorrhage Presenting with Bilateral Oculomotor Nerve Palsy and Visual Hallucinations: Report of Two Cases and Literature Review

Hongpeng Chen[1], Shiyu Luo[1], Lanqing Meng[2*]

[1] *Graduate School, Youjiang Medical University For Nationalities, Baise 533000, Guangxi Zhuang Autonomous Region, China.*

[2] *Department of Neurology, Affiliated Hospital of Youjiang Medical University for Nationalities, Baise 533000, Guangxi Zhuang Autonomous Region, China.*

Abstract

Objective: To explore the clinical features, anatomical location, and pathogenesis of bilateral oculomotor nerve palsy (ONP) with visual hallucinations (VH) caused by midbrain hemorrhage.

Methods: We report two cases of midbrain hemorrhage presenting with bilateral ONP, one of whom also experienced VH.Combining our cases with a literature review, we analyzed the relationship between clinical manifestations, hemorrhage location, midbrain anatomy, vascular supply, and the mechanism of VH.

Results: Both patients presented with bilateral ptosis and impaired supraduction, infraduction, and adduction of the eyes, with no other significant neurological deficits. A cranial CT scan confirmed a small hemorrhage located paramedianly in the midbrain tegmentum. Case 1 reported experiencing vivid visual hallucinations in the evening. A literature review suggests that small lesions near the midline can simultaneously affect adjacent oculomotor nuclei or their fibers bilaterally, leading to bilateral ONP. The pathogenesis of visual hallucinations is complex and may be related to impaired visual processing in the superior colliculus, reticular activating system, and abnormalities in the basal ganglia-cortical circuit.

Conclusion: Small hemorrhages in the paramedian midbrain can cause rare bilateral ONP, which is of great significance for understanding the midbrain anatomy and vascular supply of the midbrain. Concurrent VH is another rare manifestation, the mechanism of which involves complex dysfunction of the midbrain visual pathways, arousal regulation system, and cortical connectivity. A detailed history, physical examination, and imaging studies are crucial for a definitive diagnosis.

Keywords: Midbrain; Cerebrovascular disease; Oculomotor nerve palsy; Peduncular hallucinosis

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Introduction

The midbrain is a complex and delicate structure, and even minor lesions can produce diverse neurological deficits. Isolated bilateral ONP from midbrain hemorrhage is rare, and midbrain lesions causing VH, also known as peduncular hallucinosis, are infrequently reported. We describe two patients with midbrain hemorrhage who presented with bilateral ONP; one of these patients also experienced VH. Both were admitted to the Department of Neurology, Affiliated Hospital of Youjiang Medical University for Nationalities. Detailed case analysis and literature review aim to elucidate the anatomical localization and mechanisms of these symptoms, enhancing understanding of midbrain hemorrhage complications.

Medical Records

Case 1: A 55-year-old male patient presented with acute

*Correspondence: Lanqing Meng, Department of Neurology, Affiliated Hospital of Youjiang Medical University for Nationalities, Baise 533000, Guangxi Zhuang Autonomous Region, China..Email:menglanqingsj@qq.com.

dizziness for 8 hours, persistent head heaviness upon waking, diplopia, and difficulty opening his eyes. He denied disturbance of consciousness, limb numbness, or weakness. He developed vivid and detailed visual hallucinations in the evening of his admission, including scenes of rice paddies, his family fishing, and peanut picking. No history of hypertension. Physical examination revealed that the patient was clear consciousness, mildly dysfluent and slow speech, and appropriate responses. Attention and comprehension were mildly impaired. Both pupils were equal in size and round (approximately 3.0 mm in diameter) and sensitive to light. Ptosis was noted, with impaired adduction, supraduction and infraduction, but normal abduction. Convergence reflex was absent (both eyes could not adduct). Other neurological examinations were unremarkable. Lab tests revealed no abnormalities. A cranial CT scan revealed a small paramedian hemorrhage in the midbrain tegmentum (approximately 1.1 ml) (Figure 1).

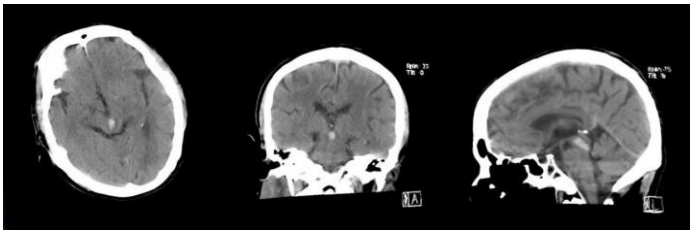

Figure 1. Case 1 head CT showing a high-density lesion near the midline of the midbrain tegmentum

Case 2: A 56-year-old male presented with 7 days of difficulty opening his eyes (ptosis), accompanied by dizziness and vomiting (non-projectile). He denied headaches, diplopia, impaired consciousness, speech disturbances, limb numbness, or weakness. He had a history of uncontrolled hypertension with a maximum 200/110 mmHg. On admission, his BP was 165/107 mmHg. Examination showed clear consciousness, fluent speech, appropriate responses, anisocoria (L: 4.5 mm, R: 3.5 mm, sluggish light reflex), bilateral ptosis, impaired supraduction, infraduction and adduction. Other neurological examinations were unremarkable. Lab tests were normal. A cranial CT scan revealed a paramedian hyperattenuating shadow in the midbrain tegmentum (approximately 1.3 ml) (Figure 2). DSA showed no vascular stenosis and malformation.

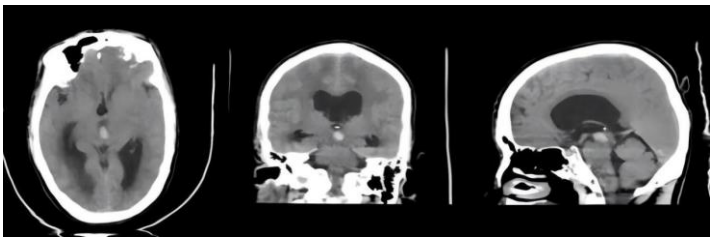

Figure 2. CT scan of the head of case 2 shows a high-density lesion near the midline of the midbrain tegmentum

Discussion

The typical manifestations of ONP are ptosis, impaired supraduction, infraduction, and adduction, often accompanied by pupil dilation, weakened or absent light and accommodation reflexes[1]. Lesions may affect the entire oculomotor nerve pathway[2]. Midbrain lesions such as hemorrhage, infarction, tumor, inflammation, etc. are common causes[3]. The accompanying symptoms vary depending on the location of the lesion, such as contralateral hemiplegia (Weber syndrome) or contralateral tremor and ataxia (Benedikt syndrome). Small midbrain lesions usually lead to unilateral ONP, while bilateral ONP without involving adjacent conduction bundles is rare. Both our patients exhibited bilateral incomplete ONP due to small midbrain hemorrhages.

Bilateral ONP caused by small midbrain hemorrhage is extremely rare, with only one prior case reported by Chia-Yi Lee[4]. The transverse diameter of the upper brainstem, including the midbrain, is relatively wide, and small lesions usually affect only one side. The oculomotor

nucleus is located in the ventral part of the gray matter around the horizontal aqueduct of the superior colliculus in the midbrain, and is distributed closely on both sides of the midline. Therefore, small lesions near the midline can simultaneously affect both nuclei or their fibers, resulting in bilateral symptoms. The hemorrhage in our patients were both located in the paramedian area of the midbrain (Figures 1, 2). This phenomenon has important implications for understanding the functional division of the midbrain and its vascular supply. The paramedian branches of the basilar artery supply the central midbrain, including the oculomotor nuclei [5]. Lesions here are most likely to cause bilateral ONP. This is because the central midbrain area supplied by these branches includes the oculomotor nucleus and related structures, which are located near the midline. Lesions are likely to affect the oculomotor nuclei on both sides. This is consistent with a case of bilateral ONP caused by a small central infarction of the midbrain tegmentum reported by IN Y S and SHIN S Y [6]. Therefore, midbrain lesions located near or across the midline can simultaneously damage bilateral oculomotor nuclei or their crossing fibers, causing bilateral paralysis.

The VH in Case 1 (peduncular hallucinosis) is another salient feature. First described by Lhermitte in 1922[7] and named by Van Bogaert in 1927[8], it often involves vivid, colorful visual scenes. Cerebrovascular disease is the most common cause[9]. Proposed mechanisms include:

Visual Pathway Disruption: Lesions may involve the tectum (superior colliculus),present in both cases. The superior colliculus is a key structure of the visual system and the control of eye movements[11]. It is at the same level as the ventral oculomotor nucleus. In the cases reported by Thomas Benke, all patients had eye movement disorders[10]. The superior colliculus receives signals from the retina and visual cortex and plays a central role in integrating visual information and guiding eye movements. Donaldson and Long's research suggests that these signals might inhibit internal visual images via the superior colliculus or visual pathways [12]. Reduced retina and extraocular proprioceptive input (due to ophthalmoplegia) may diminish inhibitory control over internal imagery, thereby triggering VH. This effect may be exacerbated in low evening light [13].

Arousal-Consciousness Dysregulation: Midbrain reticular formation damage, part of the ascending reticular activating system (ARAS), can disrupt sleep-wake cycles and lower arousal, potentially inducing dream-like rapid eye movement (REM) sleep related hallucinations [9, 14]. Reticular formation dysfunction is considered to be one of the important mechanisms of peduncular hallucinosis.

Cortical Network Dysfunction: There is an extensive connection between the midbrain and the cerebral cortex, especially the visual and auditory cortices. Research by Middleton and Strick revealed that the temporal lobe and the basal ganglia are interconnected [15]. The substantia nigra receives input from the inferior temporal gyrus via the visual striatum and projects to the inferior temporal gyrus via the thalamus, forming a basal ganglia circuit involved in higher-level visual processing. Brainstem lesions may disrupt this circuit, leading to abnormally increased thalamic input to the temporal lobe, disrupting normal perceptual processing and causing visual hallucinations.

Although rare, Bilateral ONP caused by small hemorrhage in the paramedian midbrain region highlights the importance of the anatomy and vascular supply. Detailed clinical assessment and prompt neuroimaging (CT/MRI)

are essential for diagnosis.The associated visual hallucinations reveal the complex role of the midbrain in visual processing, arousal regulation, and cortical network connectivity, providing important insights into the clinical understanding of this unique neuropsychiatric condition.

Acknowledgments

None

Conflict of Interests

None

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