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Acute Bilateral Ptosis Revealing a Midbrain Infarction

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Abstract | **Case Report**

Acute bilateral ptosis due to brainstem infarction is an exceptionally rare manifestation of ischemic stroke and may indicate involvement of the oculomotor nuclear or fascicular structures in the rostral midbrain. We report the case of a 60-year-old woman who presented with acute diplopia, impaired consciousness, marked gait ataxia, complete bilateral ptosis, mydriasis, and bilateral downward and outward deviation of the eyes. Brain magnetic resonance imaging revealed acute bilateral paramedian infarctions of the rostral mesencephalon, predominantly involving the dorsal mesencephalic tegmentum and periaqueductal gray matter, associated with bilateral paramedian thalamic infarctions. Additional ischemic lesions of different ages involving cortical and cortico-subcortical territories, including a left middle cerebral artery infarction with hemorrhagic transformation, suggested an embolic mechanism. Magnetic resonance angiography demonstrated occlusion of the distal basilar artery extending to the left P1 segment. Extensive investigations for vascular, inflammatory, immunological, infectious, toxic, and metabolic causes were negative. Cardiac evaluation revealed a large patent foramen ovale with spontaneous right-to-left shunt, associated with an atrial septal aneurysm and massive passage of microbubbles during the Valsalva maneuver, supporting a cardioembolic mechanism. The patient was outside the therapeutic window for reperfusion therapy and was treated with anticoagulation and rehabilitation. Despite partial improvement in gait and cognitive disturbances, severe bilateral oculomotor deficits persisted, and the patient died two months later from infectious complications. This case highlights a rare and disabling presentation of distal basilar artery occlusion and emphasizes the importance of accurate clinico-radiological localization and comprehensive etiological evaluation, particularly for identifying potential cardioembolic sources such as patent foramen ovale.

Keywords: Ptosis, Midbrain, Patent foramen ovale.

INTRODUCTION

Acute basilar artery occlusion (BAO) is a rare but potentially catastrophic medical condition, accounting for approximately 1% of all ischemic strokes and 5% of large vessel occlusions (LVO). Despite recent advances in acute stroke management, up to 68% of patients with BAO die or remain severely disabled [1]. Bilateral involvement occurs in only about 10% of third nerve palsy cases, and brainstem stroke accounts for approximately one-fifth of these. Isolated bilateral oculomotor nerve palsy secondary to brainstem infarction therefore remains exceptionally rare [2]. We report a case of severe bilateral oculomotor nerve palsy caused by distal basilar artery occlusion resulting in a cardioembolic midbrain infarction.

CASE REPORT

A 60-year-old African woman presented to the emergency department with the acute onset of diplopia and impaired consciousness. On admission, vital signs were stable: blood pressure was 140/60 mmHg, blood glucose was 1 g/L, respiratory rate was 14 breaths/min, and the patient was afebrile. Cardiovascular risk factors included age and active smoking (20 pack-years). Her medical history was notable for severe depression diagnosed in 2019, treated with amitriptyline and lorazepam. There was no history of diabetes mellitus, hypertension, or dyslipidemia.

Neurological examination revealed marked gait ataxia with difficulty standing. Muscle tone was slightly decreased, while deep tendon reflexes were brisk in all four limbs without Babinski sign. Moderate impairment of deep sensation was noted. The patient also presented with complete bilateral ptosis with inability to open her eyes, mydriasis, and downward and outward deviation of both eyes. These symptoms were constant and did not fluctuate during the day. She was confused and exhibited fluctuating somnolence. History obtained from family

members indicated that symptom onset had occurred approximately 72 hours before admission, placing the patient outside the therapeutic window for reperfusion therapy (intravenous thrombolysis and/or mechanical thrombectomy).

Given this clinical presentation suggestive of a central neurological origin, brain MRI with a vascular protocol was performed. Imaging revealed acute bilateral paramedian infarctions of the rostral mesencephalon, predominantly involving the dorsal mesencephalic tegmentum and the periaqueductal gray matter, with sparing of the ventral midbrain and cerebral peduncles (Fig. 1). These lesions were associated with bilateral paramedian thalamic infarctions. Additionally, an infarction was identified in the territory of the left middle cerebral artery with hemorrhagic transformation, as well as multiple small distal cortical and cortico-subcortical ischemic lesions of different ages in the right occipito-parietal and left temporal regions, suggesting an embolic mechanism (Fig. 2). These findings were associated with occlusion of the distal basilar artery extending to the P1 segment of the left posterior cerebral artery (Fig. 3). The vertebral arteries were unremarkable and showed no evidence of arterial dissection.

An extensive etiological work-up was conducted, including investigations for vascular causes (arterial dissection or vascular anomalies), inflammatory diseases, immunological disorders (vasculitis and antiphospholipid syndrome), infectious etiologies, and toxic or metabolic causes. All results were negative. However, cardiac evaluation revealed abnormalities. Transthoracic echocardiography followed by transesophageal echocardiography demonstrated a large patent foramen ovale (5 mm) with a spontaneous right-to-left shunt on contrast study, associated with an atrial septal aneurysm (ASA type 2L) and a massive passage of microbubbles (>25) during the Valsalva maneuver. Transcranial Doppler ultrasonography confirmed the presence of microbubbles during the Valsalva maneuver. The remainder of the cardiac evaluation was unremarkable. There was no evidence of atrial enlargement, intracardiac thrombus, valvular disease, or aortic atheroma. Left ventricular function and left atrial appendage flow velocity were normal.

Repeated 48-hour electrocardiographic monitoring detected no significant arrhythmia, and duplex ultrasonography revealed no evidence of deep venous thrombosis.

During the subsequent rehabilitation period after hospital discharge, persistent deficits related to bilateral oculomotor nerve palsy became evident. The most disabling manifestation was complete bilateral ptosis. Examination of ocular motility demonstrated bilateral fixation in abduction. Pupils were bilaterally dilated with absence of the pupillary light reflex on the right side. Convergence paresis and complete vertical gaze palsy were also present.

These oculomotor disturbances, together with severe bilateral ptosis, led to pronounced dizziness and marked gait instability, preventing independent standing and ambulation and rendering the patient bedridden. Gait and postural instability showed mild improvement after rehabilitation therapy.

Throughout the hospital stay, the patient also exhibited fluctuating somnolence, memory impairment, and temporospatial disorientation. These cognitive disturbances showed partial improvement over time.

Anticoagulant therapy was initiated together with comprehensive management of cardiovascular risk factors. Surgical closure of the patent foramen ovale was planned; however, the patient died two months later from infectious complications.

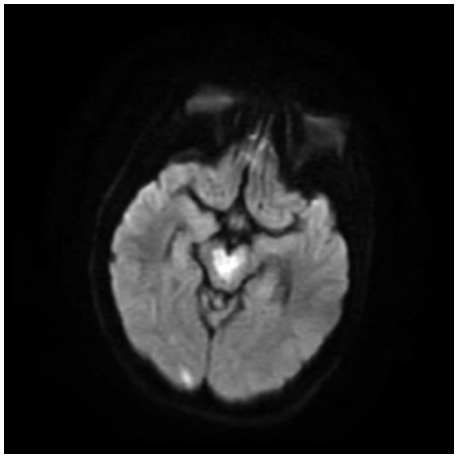

Figure 1: Axial diffusion-weighted MRI showing acute bilateral paramedian infarction of the rostral mesencephalon. Hyperintense lesions are visible within the dorsal midbrain tegmentum, corresponding to ischemic involvement of the oculomotor nuclear complex

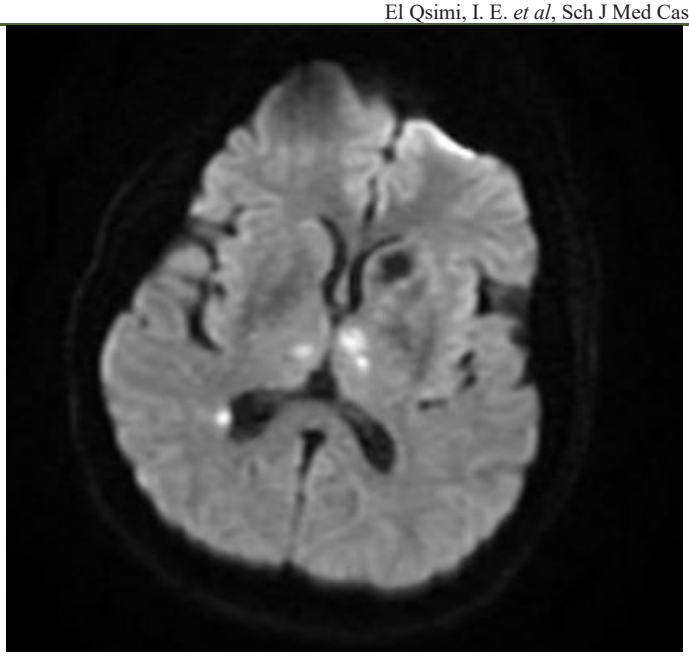

Figure 2: Axial diffusion-weighted MRI demonstrating multiple acute ischemic lesions involving the deep territory of the left middle cerebral artery (MCA) and bilateral thalamic territories supplied by the posterior cerebral arteries (PCA), consistent with an embolic stroke pattern affecting both anterior and posterior circulation

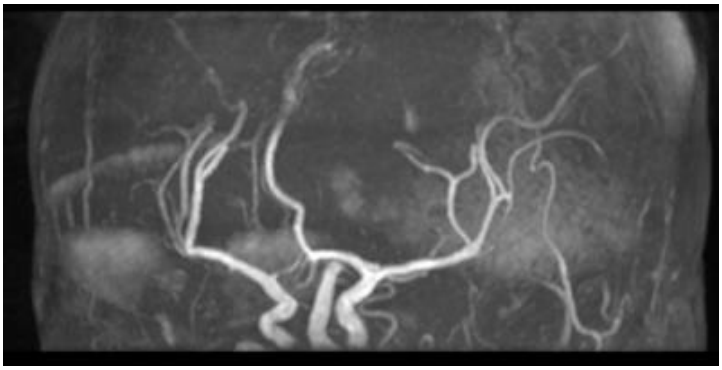

Figure 3: Time-of-flight magnetic resonance angiography demonstrating occlusion of the distal basilar artery (top-of-the-basilar occlusion) with absence of flow signal at the basilar apex

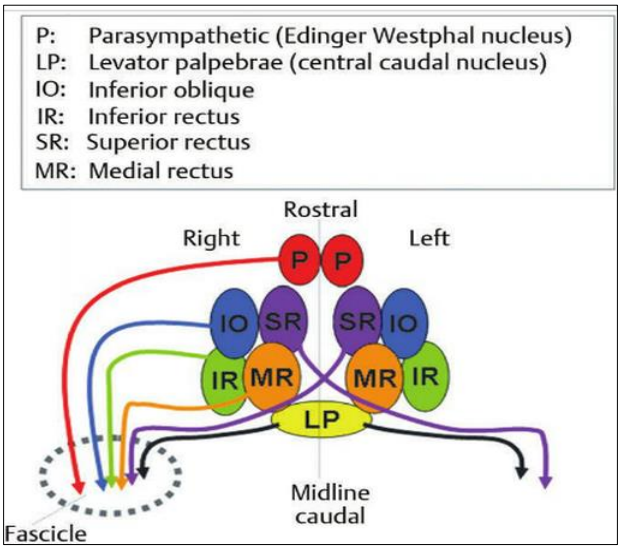

Figure 4: Schematic representation of the oculomotor nuclear complex and its subnuclei illustrating the central caudal nucleus responsible for bilateral levator palpebrae innervation, explaining bilateral ptosis in nuclear lesions

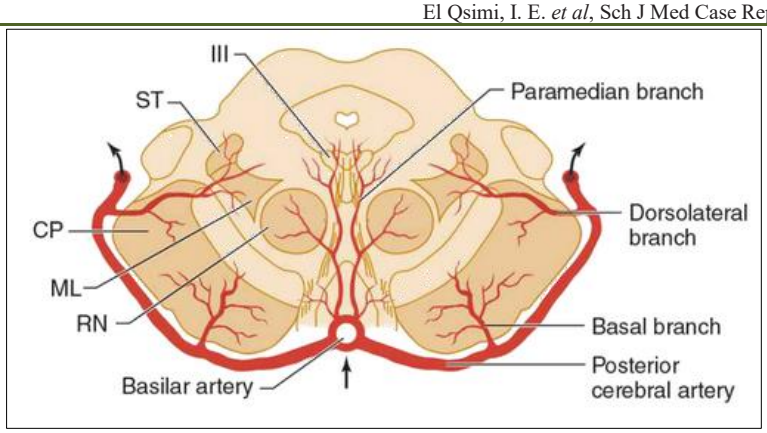

Figure 5: Vascular supply of the rostral midbrain showing the paramedian perforating branches arising from the basilar and posterior cerebral arteries that supply the oculomotor nuclear complex

DISCUSSION

Mesencephalic infarction is rare, accounting for approximately 1% of cases in major stroke registries, of which only about 10% are bilateral[3]. Approximately one-third of mesencephalic strokes involve dysfunction of the third cranial nerve [4]. The oculomotor nerve originates in the midbrain. Its nucleus is composed of a cluster of subnuclei located in the dorsal midbrain. Axons from the oculomotor nuclear complex, including both motor and parasympathetic neurons, course anteriorly as oculomotor nerve fascicles and exit the midbrain at the medial aspect of the cerebral peduncle. Each extraocular muscle innervated by the third nerve is supplied by a specific subnucleus. Eyelid elevation is controlled by the unpaired central caudal nucleus located medially and dorsally to the main nucleus. The pupillary sphincter is innervated by the Edinger–Westphal nucleus, which lies posterior to the oculomotor nucleus or by more dorsally located neurons[5] (Fig. 4).

Because of the anatomical organization of the oculomotor nuclear complex, selective involvement of individual nuclei may occur. This explains rare cases of partial central third nerve palsy in which not all extraocular muscles are affected. For example, unilateral nuclear lesions may produce bilateral ptosis and bilateral superior rectus weakness in approximately 50% of cases, while the other contralateral ocular muscles remain spared [5].

The paramedian region of the rostral midbrain is mainly supplied by the superior medial mesencephalic branches of the posterior cerebral artery, with additional contribution from direct basilar artery branches supplying the superficial ventral paramedian tegmentum. These branches form the short interpeduncular arteries arising from vessels within the interpeduncular fossa and supply the cerebral peduncles and oculomotor nerves (III) [6].

When occlusion occurs at the apex of the basilar artery, ischemic lesions in the dorsal paramedian midbrain tegmentum may lead to bilateral involvement of the oculomotor nuclei [7] (Fig. 5).

Bilateral third nerve palsy has been reported in association with several neurological conditions. Vascular causes include midbrain hematoma [8] and compression of the bilateral nerve exits by a dolichoectatic basilar artery [9]. Infectious causes such as melioidosis have also been reported [10]. Other etiologies include systemic diseases such as neurosarcoidosis, inflammatory disorders such as temporal arteritis [11], neuromuscular disorders such as myasthenia gravis, and toxic causes including chemotherapy.

The long-term consequences of bilateral oculomotor nerve palsy can be profoundly disabling and require comprehensive neuro-rehabilitative management, including prisms, occlusion therapy, visual scanning exercises, refractive correction, and, in selected cases, surgical intervention aimed at improving functional autonomy [12].

Recent studies have further refined the neuroimaging characterization of patent foramen ovale (PFO)–associated cryptogenic stroke. In a large contemporary cohort, PFO-related strokes exhibited distinct infarction patterns compared with non-PFO cryptogenic strokes, including a higher prevalence of small, often multiple lesions with predominantly cortical or cortico-subcortical distribution, supporting an embolic mechanism rather than small-vessel disease [13].

Subsequent reviews have confirmed these findings and highlighted a relative predilection for posterior circulation involvement in PFO-associated stroke across different age groups and clinical subtypes [14]. This posterior distribution is thought to result from paradoxical embolism through right-to-left shunting, potentially influenced by vertebrobasilar flow dynamics, gravitational factors, and embolus size [15].

CONCLUSION

This case illustrates a rare presentation of acute ischemic stroke manifested by severe bilateral fascicular oculomotor nerve palsy caused by distal basilar artery occlusion leading to bilateral paramedian rostral mesencephalic infarction.

Beyond its neuroanatomical significance, this observation reinforces the growing body of evidence linking patent foramen ovale to embolic stroke patterns preferentially affecting the posterior circulation. The multiplicity of infarcts of different ages, their cortical and posterior distribution, and the presence of a large patent foramen ovale associated with an atrial septal aneurysm strongly support a cardioembolic mechanism.

Recognition of this rare brainstem stroke phenotype, together with accurate clinico-radiological localization and systematic etiological evaluation, is essential for appropriate management and prognostic assessment.

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