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Partial Weber Syndrome Presenting with Isolated Ptosis and Mild Contralateral Hemiparesis: A Case Report

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ABSTRACT

Introduction: Weber syndrome is one of the rare crossed paralysis syndromes, in which clinical findings provide important clues for lesion localization. It presents with ipsilateral oculomotor nerve palsy and contralateral hemiparesis, suggesting a ventromedial midbrain lesion, most commonly of ischemic origin. ***Case Report:*** A 62-year-old hypertensive woman presented with a sudden onset of left-sided weakness, difficulty opening the right eyelid, and slurred speech for 2 days. Neurological examination revealed right eye ptosis, left facial and hypoglossal nerve supranuclear palsies, left flaccid hemiparesis (MRC 4/5), and a positive left Babinski sign. Eyeball position, extraocular movements, and pupillary function were normal bilaterally. Neither hemorrhage nor infarction was visible on non-contrast head CT, while MRI was unavailable. The patient showed improvement after 5 days of treatment with citicoline, aspirin, clopidogrel, folic acid, amlodipine, candesartan, simvastatin, allopurinol, and physiotherapy. ***Discussion:*** Patient's presentation was consistent with Weber syndrome, characterized by right oculomotor nerve palsy and left hemiparesis, allowing localization to the right ventromedial midbrain. Isolated ptosis with mild contralateral weakness suggested a partial Weber syndrome, likely caused by a small lesion affecting selective oculomotor fascicles and adjacent corticospinal and corticobulbar tracts. Non-contrast head CT is largely insensitive for detecting brainstem infarcts, especially small lesions. In the absence of hemorrhage, ischemic stroke treatment was initiated, resulting in clinical improvement. ***Conclusion:*** Weber syndrome may present as a partial form with isolated ptosis and mild contralateral hemiparesis. Careful clinical localization remains essential for diagnosis, particularly when brainstem infarcts are not typically visualized on CT imaging, and MRI is inaccessible.

Keywords: partial Weber syndrome; isolated ptosis; midbrain infarction; crossed paralysis

INTRODUCTION

Weber syndrome was first introduced in 1863 by a German-born English physician, Hermann Weber [1,2]. Weber described a distinctive presentation in a 52-year-old male patient with left oculomotor nerve palsy and right-sided hemiplegia, which was caused by left cerebral peduncle hemorrhage [2,3]. This syndrome is one of the rare clinical entities classified as crossed paralysis or alternating hemiplegia, in which clinical manifestations are important keys to localize the lesion to a defined brainstem region [1,4]. Weber syndrome typically presents with ipsilateral oculomotor nerve palsy and contralateral hemiparesis or hemiplegia, consistent with a ventromedial midbrain lesion, affecting the oculomotor nerve nucleus as well as the corticospinal and corticobulbar tracts in the cerebral peduncle [5,6]. The most common cause is vascular origin, such as infarction, hemorrhage, and aneurysms, with other possible causes like tumors and demyelination. Ischemic stroke with Weber syndrome presentation may be associated with cardiac embolism, in situ thrombosis, artery-to-artery embolism, or small vessel disease [7,8]. Affected vascularization includes the peduncular perforating branches of the posterior cerebral artery (PCA) or paramedian mesencephalic branches of the basilar artery [5,9]. A limited number of Weber syndrome have been documented worldwide, particularly in low- and middle-income countries (LMICs) [10]. In patients with posterior circulation infarction, it was found that only 0.7% experienced isolated midbrain infarction [2].

In accordance with oculomotor nerve involvement, patients usually complain of double vision and drooping of one eyelid [11,12]. Examination may reveal ipsilateral ptosis, inferolateral eye deviation with impaired extraocular movements, and dilated pupil with absent pupillary light reflex [1,6]. The oculomotor nucleus can be partially damaged, usually with pupillary sparing [1,8]. This condition is based on the oculomotor nerve fascicular arrangement in the midbrain, in which the fibers

controlling pupillary function, extraocular movements, and eyelid elevation are organized in a rostrocaudal sequence [1,2].

Besides Weber syndrome, several other brainstem syndromes can also affect the oculomotor nerve and should be considered during diagnostic evaluation. Benedikt syndrome presents with contralateral tremor, hemichorea, or hemiathetosis. Nothnagel syndrome manifests with contralateral dysmetria and limb ataxia. In addition, Claude syndrome exhibits contralateral cerebellar ataxia and tremor [13]. A comprehensive neurological examination plays a crucial role in determining the right diagnosis and clinically localizing the lesion.

Non-contrast head computed tomography (CT) is the most widely used imaging modality for stroke assessment, owing to its relatively high accessibility, fast performance, and great sensitivity for hemorrhage [14,15]. Further evaluations using CT angiography (CTA) are sometimes used to identify vertebral artery disease or large vessel occlusions [16]. On the other side, magnetic resonance imaging (MRI) remains a more favorable imaging tool with higher sensitivity for detecting infarcts [12,17]. In Weber syndrome, hyperintensity in the T2-weighted image MRI, suggesting an ischemic lesion, was found in the ventromedial midbrain on the same side as the affected eye [12]. MRI becomes the gold standard emergency imaging and modality of choice for suspected brainstem strokes, since CT is relatively insensitive [1]. However, in limited settings, head CT is still commonly used for initial investigation and to decide the plan of treatment, preferably with the addition of CTA [18]. Other basic work-ups, which are usually performed in acute stroke cases, are an electrocardiogram and laboratory tests such as complete blood count, biochemistry panels, coagulation tests, and troponin level [19,20].

Treatment of acute ischemic stroke, including Weber syndrome, depends on the onset and the National Institutes of Health Stroke Scale (NIHSS) [15]. Intravenous thrombolysis with alteplase becomes the main choice of treatment for acute ischemic stroke within 4.5 hours of onset [21]. NIHSS of ≤ 4 indicates minor stroke [22]. For secondary treatment, dual antiplatelet therapy (DAPT) with aspirin and clopidogrel for 21 days is recommended in minor strokes [23]. Other supportive treatments and medications may also be given, such as oxygenation, antihypertensive agents, statins, and rehabilitation [14]. Sometimes, ptotic eyelid may lead to amblyopia; therefore, a physiotherapy program remains important. Amblyopia can be prevented by patching the unaffected eye for about 50% of waking hours [24]. Practicing continuous eye movements in all directions may also help regain extraocular muscle strength [25].

CASE REPORT

A 62-year-old conscious woman, accompanied by her family, presented to the emergency room with a sudden onset of left-sided body weakness and slurred speech of two days duration. The patient could still walk, but with a limp. The patient also had difficulty opening her right eyelid, without double or narrow vision. These complaints occurred for the first time when the patient woke up in the morning. Other symptoms, such as unconsciousness, seizure, headache, dizziness, fever, numbness, tingling sensation, dysphagia, nausea, vomiting, chest pain, and palpitations, were denied. Food and drink intake, urination, and defecation were within normal limits. The patient had a history of uncontrolled hypertension, while other illnesses and medication histories were denied.

The patient was mentally alert with Glasgow Coma Scale (GCS) of eye (E) 4, verbal (V) 5, and motor (M) 6, blood pressure of 191/91mmHg, regular pulse rate of 76 times/minute, respiratory rate of 20 times/minute, oxygen saturation of 99% on room air, and axillary temperature of 36.2°C. Neurological examination showed right eye ptosis (Figure 1), suggesting right oculomotor nerve palsy, with symmetrical eyeballs position, normal bilateral extraocular movements to all directions, isochoric pupils sized 3mm/3mm, and positive bilateral pupillary light reflexes. Other neurological findings included left facial and hypoglossal nerve supranuclear palsies, left flaccid hemiparesis with Medical Research Council (MRC) scale of 4/5, normal deep tendon reflexes (2+), and positive left Babinski sign. No ataxia, tremors, dyscoordination, nystagmus, or other cerebellar signs were observed. There were also no meningeal signs and sensory function disturbance.

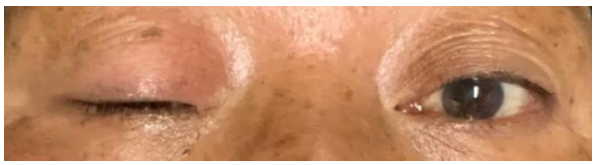

FIGURE 1: Right eye ptosis.

Laboratory tests, an electrocardiogram (ECG), and a non-contrast head CT were performed. Complete blood count (CBC), random and fasting blood glucose, urea, creatinine, and triglyceride (104mg/dL) were within normal limits. In contrast, low-density lipoprotein (LDL) cholesterol (156mg/dL) and uric acid (6.1mg/dL) were increased. ECG and non-contrast head CT showed no abnormalities, with neither hemorrhage nor infarction visible on the head CT. More advanced modalities, such as MRI, were unavailable.

The patient was diagnosed with partial Weber syndrome due to suspected ischemic stroke with NIHSS 3 and admitted as an inpatient. Treatment consisted of oxygenation, ringer lactate intravenous fluid drip, intravenous 500mg citicoline twice daily, intraoral 100mg aspirin once daily, intraoral 75mg clopidogrel once daily, intraoral 1mg folic acid once daily, intraoral 20mg simvastatin once daily, and intraoral 100mg allopurinol once daily. During evaluation, the patient was also given intraoral 10mg amlodipine once daily and intraoral 8mg candesartan

once daily. The physiotherapy program was initiated on the fourth day after onset, including step-by-step reclining and out-of-bed mobilization, as well as neuromuscular and muscular electrical stimulation (NMES) of the right levator palpebra superioris muscle. The patient was maintained in a 30° head-up position. During the day, the left eye was covered with gauze pads every 2 hours.

After five days of treatment, the patient showed improvements, with reduced left-sided body weakness and slight recovery of right eyelid opening (NIHSS 2). The patient was discharged with all oral medications continued, whereas intravenous citicoline was switched to intraoral 1200mg piracetam twice daily. The physiotherapy program was also maintained. The patient was then advised to visit the neurology polyclinic for further follow-ups.

DISCUSSION

Patient experienced clinical manifestations which are suitable for crossed paralysis syndrome of brainstem lesions, with ipsilateral cranial nerve palsy and contralateral hemiparesis [1,4]. Presentation of right oculomotor nerve palsy, left facial and hypoglosus nerve supranuclear palsies, left hemiparesis, and positive left Babinski sign raises suspicion towards Weber syndrome. These clinical features allow lesion localization to the right ventromedial midbrain, involving the right oculomotor nerve nucleus and the right cerebral peduncle. The affected adjacent corticospinal and corticobulbar tracts, which have not decussated yet, explain the contralateral weakness below the level of the lesion [5,6]. With a sudden onset and no other chronic symptoms present, the patient was suspected to have an acute stroke, which was the most common cause of midbrain lesion. This is also supported by the patient's history of uncontrolled hypertension, high blood pressure on examination (191/91mmHg), and high LDL cholesterol level (156mg/dL) as stroke risk factors. Involved vascularization may include the PCA or basilar artery [5,9].

Some literatures explain the possibility of partial Weber syndrome presentation, mostly with pupillary sparing [1,8]. This patient presented with isolated ptosis of the right eye, without disturbances in extraocular movements, symmetrical eyeballs position, and normal pupillary functions. These findings raise a suspicion that the oculomotor nerve nucleus is partially damage, possibly affecting only selective fascicles which control eyelid elevation [1,2]. The patient also experienced mild contralateral hemiparesis with MRC scale of 4/5, as well as mild dysarthria and lower facial paralysis (NIHSS 3). With all these subtle manifestations, it is likely that this partial syndrome was caused by a small ventromedial midbrain lesion. In the absence of ataxia, tremors, dyscoordination, nystagmus, and other cerebellar signs, other differential diagnosis such as Benedikt syndrome, Nothnagel syndrome, and Claude syndrome could be excluded [13].

Although MRI remains the gold standard imaging modality for diagnosing brainstem strokes, non-contrast head CT is still widely performed, particularly in limited setting [1,18]. From non-contrast head CT, there was no visible hemorrhage. Therefore, midbrain infarction was considered as the possible cause and ischemic stroke treatment was initiated. The infarction may be undetectable on head CT due to its limited sensitivity and the possible small-sized brainstem lesion. In addition, normal ECG indicates no atrial fibrillation as the risk factor of cardioembolic stroke.

Treatment was given based on the onset and NIHSS [15]. The patient exhibited minor stroke with NIHSS 3 and onset of 48 hours [22]. As secondary treatment DAPT was administered, alongside with other medications such as neuroprotector, statin, antihypertensive agents, and antihyperuricemic drug, as well as rehabilitation [14]. Supportive care for ptosis management included NMES and covering of the normal eye during waking hours, in this case the left eye, to prevent amblyopia [24]. Improvement after five days of treatment with NIHSS 2 supports the suspicion of small midbrain lesion with ischemic origin.

CONCLUSION

Weber syndrome is a rare crossed-paralysis syndrome that may present in an incomplete or partial form, with isolated ipsilateral ptosis and mild contralateral hemiparesis. This case highlights the critical role of meticulous neurological examination in clinically localizing ventromedial midbrain infarction, particularly when neuroimaging findings are inconclusive. MRI is the modality of choice for detecting brainstem strokes. However, in resource-constrained settings non-contrast head CT remains necessary, especially for determining the treatment strategy. Early clinical recognition of partial Weber syndrome allows timely initiation of ischemic stroke treatment and rehabilitation, which may contribute to favorable neurological outcomes.

DISCLOSURE

Both authors contributed equally to the conception of this case report. The authors declare no conflict of interest. This study did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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