Bilateral ptosis caused by midbrain hemorrhage: A case report

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Abstract

Ptosis occurring in cases of cerebral ischemic or hemorrhagic stroke, trauma or tumor without involvement of brainstem or oculo-sympathetic mechanism, can be termed as cerebral ptosis. Such eyelid dysfunction can occur with hemispheric involvement of either side. It is associated with a higher frequency of gaze preference to the side of lesion and upgazed limitation compared to patients without cerebral ptosis. Cerebral ptosis is rare. Eliminating an emergency is a real challenge in the face of bilateral ptosis. We report a case of bilateral ptosis occurring after a mesencephalic hematoma. The patient was unable to open her eyes or perform the basic activities. Neurogenic ptosis may improve after treatment of the underlying cause.

Keywords: Cerebral Ptosis; Cerebral Hemorrhage; Computerized Tomography

INTRODUCTION

Blepharoptosis (Ptosis) is defined by a drooping upper eyelid, making it difficult to open the eye and narrowing the upper eyelid margin [1-3]. is a common condition that can be congenital or acquired and varies in its timing of onset, duration, severity, laterality, and underlying etiology [4]. Several etiologies are possible, including supranuclear lesions, oculomotor complex lesions, oculocephalic lesions, neuromuscular junction dysfunction, neuromuscular diseases, and mechanical abnormalities of the eyelid [1]. Ptosis occurring in cases of cerebral ischemic or hemorrhagic stroke, trauma or tumor without involvement of brainstem or oculo-sympathetic mechanism can

be termed as **cerebral ptosis (CP) [6-10]**. Such eyelid dysfunction can occur with hemispheric involvement of either side. It is associated with a higher frequency of gaze preference to the side of lesion and upgazed limitation compared to patients without cerebral ptosis.

The vascular supply of midbrain is complex [1,5]. It is ensured by the posterior cerebral artery (PCA), the basilar artery, the superior cerebellar artery and the anterior choroidal artery [5]. The oculomotor nucleus located in the midbrain at the level of the superior colliculus sends efferent fibers to the medial, the superior and the inferior oblique extraocular muscles. The elevator palpebrae superioris muscle is also innervated by the oculomotor nerve. Its origin is from the central caudal nucleus (CCN) [2]. Midbrain lesions involving the CCN have been reported to cause bilateral ptosis [3]. Besides, pure midbrain infarction involving the anteromedial areas can cause bilateral ptosis, in addition to the median longitudinal fasciculus syndrome, and contralateral cerebellar ataxia [3]. The midbrain is often affected in patients with embolic stroke occurring in the posterior circulation, usually with the concomitant involvement of other structures, such as the pons, thalamus, and the cerebellum. Although midbrain infarcts and particularly hemorrhages are uncommon, their clinical manifestations are diverse mainly because the vertical gaze centers and two of three nuclei of the extraocular muscles lie primarily in the midbrain. Consequently, eye movement disturbances are often the hallmark clinical findings in midbrain stroke or hemorrhage [1]. The reported prevalence of pure midbrain hemorrhage varies from 0.7% to 2.3% [1,2]. Ptosis could be caused by oculomotor nerve palsy in the patients with midbrain infarction [1]. In several cases, bilateral ptosis showed the clinical characteristics of midbrain infarction [1,3,4]. We experienced a case of severe bilateral ptosis that occurred after midbrain hemorrhage in which the patient could not open her eyes and was limited in basic activities and mobility. We herein report the case with a review of relevant literature.

CASE REPORT

A 65 -65-year-old woman with a previous history of hypertension was admitted to the emergency department because, suddenly, she could not open her eyes. She did not complain of headache, nausea, fever, or vomiting. Examination revealed pronounced bilateral ptosis, with symmetrical and intermediate pupils. There was marked limitation of adduction, moderate limitation of depression and elevation in the movement of both eyeballs. Abduction was normal. The patient was respiratory stable. Her SBP was 170 mmHg and DBP 100 mmHg. The rest of the examination was normal, her consciousness was clear, her Glasgow coma scale was 15, no motor or sensory deficits, no localization signs and no evidence of other orbital disease-causing ptosis in an ophthalmologic examination. Blood tests and electrocardiography did not reveal any abnormal findings. Brain computed tomography (CT) on admission showed a small

hemorrhage in the medial side of the left crus cerebri without subarachnoid hemorrhage (Figure 2). The patient was initially admitted to the emergency room, where her blood pressure and neurological status were monitored, antihypertensive treatment was started (nicardipine). The outcome was favorable with stabilization of blood pressure. The next day, she was transferred to the department of rehabilitation medicine for intensive rehabilitation management. Participation in the rehabilitation programs was difficult due to continuing inability to open her eyes. We tried taping her lids with Micropore tape and used lubricants to prevent exposure keratopathy. However, this method was less efficient due to blinking. Fixation of the upper eyelid to the supraorbital structures using Eye-putti eyelash glue proved more effective in conjunction with a rehabilitation program. About two months after the cerebral hemorrhage, the bilateral ptosis improved. The patient was satisfied with the rehabilitation program. She performed activities of daily living and mobility with minimal assistance. The ptosis was gradually resolved after five months.

Figure 1: *Follow-up photographs of the patient's eyelids*

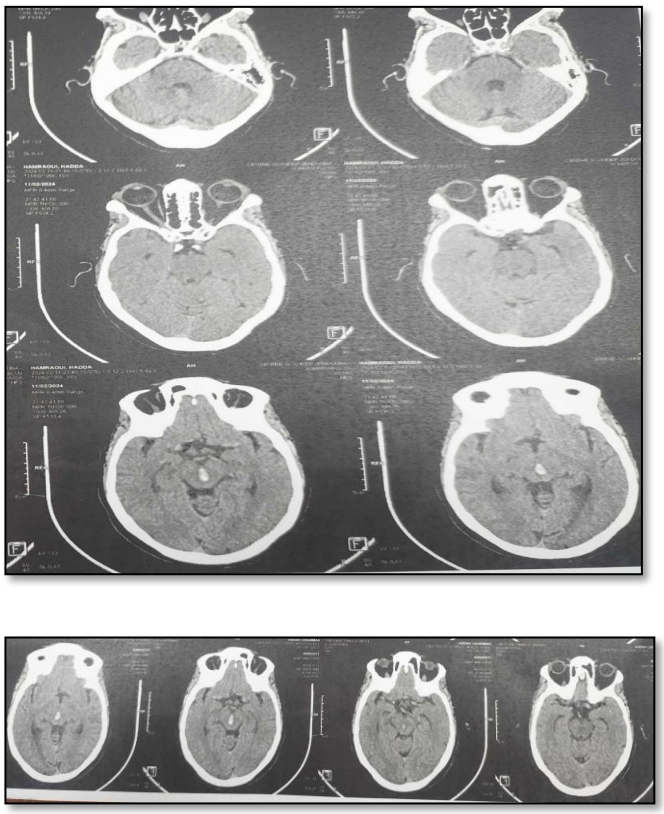

Figure 2: *Computed tomography scan showing hemorrhage in the midbrain tegmentum.*

DISCUSSION

Bilateral ptosis is a rare manifestation and has been reported in 5% of patients with pure midbrain infarction in a previous case series and most of these patients had small lesions in the paramedian or central regions of the midbrain [10-14].

The criteria for the diagnosis of CP have not yet been established. Manconi et al. [10] proposed inclusion and exclusion criteria. The reported inclusion criteria were as follows:

1) sudden bilateral lid drops within 48 hours of stroke or hemorrhage,
2) voluntary, spontaneous, and automatic impairment in eyelid opening,
3) preserved voluntary frontalis muscle

contraction

4) And neuroradiological evidence of supratentorial ischemic or hemorrhagic damage.

The exclusion criteria were presented together to distinguish the following causes of ptosis: apraxia of lid opening (ALO), impaired consciousness, blepharospasm, intrinsic oculomotor dysfunction, neuromuscular disease, brain stem dysfunction, and sub tentorial lesions. In our case, ALO and blepharospasm should be differentially diagnosed [10].

Although the mechanisms and clinical significance of CP remain uncertain, experiments in both animals and humans have shown that opening of both eyelids occurs upon stimulation of

the frontal and occipital lobes [15]. Clinically, it has been hypothesized that eyelid motor control might be lateralized to the right hemisphere in complete CP [9, 10,11]. However, the specific mechanism is not yet fully elucidated, and the above hypothesis is still controversial [5]. The prognosis of CP is variable and depends on the lesion and etiology [5]. Manconi et al. [10] reviewed literature on 75 CP patients, reporting an improvement in ptosis in 70.9%, with an average recovery time of 7.5 days. In most cases of spontaneous midbrain hemorrhage, conservative, supportive treatment is sufficient to ensure a good outcome. Some 25% of patients had no neurologic deficits and 41% of patients had persistent minor neurologic deficits, which related chiefly to cranial nerves III and IV. Only 4% of the patients died [1,10].

In summary, this case demonstrates complete ptosis resulting from a brainstem event. We hypothesize that the patient's oculomotor deficits were secondary to midbrain hemorrhage secondary to hypertension. Additional cases with documented clinical, neuroradiologic, and neuropathologic examination can reinforce our understanding of eye movement disorders and the anatomic organization of the oculomotor nucleus and associated nuclei that mediate conjugate gaze.

Ethical Statement

This research was conducted ethically: Ethics approval was not required by the Ethics Committee because this is a case report. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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