Unilateral Oculomotor Nerve Palsy as an Initial Presentation of Bilateral Chronic Subdural Hematoma: Case Report

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Abstract

Isolated oculomotor nerve palsy is well known as a symptom of microvascular infarction and intracranial aneurysm, but unilateral oculomotor nerve palsy as an initial manifestation of chronic subdural hematoma (CSDH) is a rare clinical condition. We report a rare case of an 84-year-old woman with bilateral CSDH who presented with unilateral oculomotor nerve palsy as the initial symptom. The patient, who had a medical history of minor head injury 3 weeks prior, presented with left ptosis, diplopia, and vomiting. She had taken an antiplatelet drug for lacunar cerebral infarction. Computed tomography (CT) of the head showed bilateral CSDH with a slight midline shift to the left side. She underwent an urgent evacuation through bilateral frontal burr holes. Magnetic resonance angiography (MRA) after evacuation revealed no intracranial aneurysms, but constructive interference in steady-state (CISS) magnetic resonance imaging (MRI) revealed that the left posterior cerebral artery (PCA) ran much more anteriorly and inferiorly compared with the right PCA and the left oculomotor nerve passed very closely between the left PCA and the left superior cerebellar artery (SCA). There is the possibility that the strong compression to the left uncus, the left PCA, and the left SCA due to the bilateral CSDH resulted in left oculomotor nerve palsy with an initial manifestation without unconsciousness. Unilateral oculomotor nerve palsy as an initial presentation caused by bilateral CSDH without unconsciousness is a rare clinical condition, but this situation is very important as a differential diagnosis of unilateral oculomotor nerve palsy.

Key words: oculomotor nerve palsy, bilateral chronic subdural hematoma, posterior cerebral artery

Introduction

Chronic subdural hematoma (CSDH) normally occurs in the elderly after a mild head injury.¹⁾ CSDH is a common condition, especially among patients who take antiplatelet and anticoagulant drugs after coronary heart disease and cerebral infarction.¹⁾ Most patients with CSDH present with gait disturbance, hemiparesis, headache, dementia, urinary incontinence, and consciousness disturbance.⁵⁾

On the other hand, the most common cause of isolated oculomotor nerve palsy is microvascular infarction which is caused as a result of diseases, such as diabetes mellitus, hypertension, atherosclerosis, and collagen vascular disease.⁸⁾ Another common cause of isolated oculomotor nerve palsy is direct compression by an intracranial aneurysm, usually an internal carotid-posterior communicating artery aneurysm that sometimes presents a pupil-sparing oculomotor nerve palsy as an early symptom of enlarging aneurysm.⁹⁾

To date, only 3 cases of CSDH presenting with unilateral oculomotor nerve palsy as an initial manifestation have been reported in conscious patients in the English literature.^{2,4,8)} We review these cases and discuss the mechanism of unilateral oculomotor nerve palsy associated with CSDH.

Case Report

An 84-year-old woman with a history of lacunar infarction, who was undergoing treatment with an antiplatelet drug, suffered from occipital head injury and went to the local hospital. Computed tomography (CT) of the head revealed a left thin acute subdural hematoma in the left frontal convex with no cranial fractures (Fig. 1). She had no neurological deficits and went home on that day. Three weeks later, she returned to the local hospital presenting with the left ptosis, diplopia, and vomiting. CT of the head revealed bilateral CSDH that was predominant on the right side (Fig. 2). She was eventually transferred to our hospital for further treatment. Her Glasgow Coma

Fig. 1 Computed tomography (CT) showing a left thin acute subdural hematoma in the frontal convex (arrows) and a calcification in the basilar artery and left posterior cerebral artery (arrow heads).

Fig. 2 Computed tomography (CT) showing the bilateral chronic subdural hematoma with a slight midline shift to the left side 3 weeks later after the initial mild head injury.

Scale was 15. Ophthalmologic examination showed left complete oculomotor nerve palsy with left ptosis, diplopia, and dilated pupil. She underwent an urgent evacuation through bilateral frontal burr holes under local anesthesia. After bilateral burr hole surgery, we initially opened the outer membrane of the subdural hematoma on the right side, and immediately opened the outer membrane on the left side to avoid worsening the uncus herniation. The pressures of the hematoma were very high bilaterally. We inserted the drains bilaterally into the cavity of the hematoma.

The drains were removed on the day after the surgery. Her ptosis and diplopia resolved immediately after the surgery, and her dilated left pupil was improving gradually but still remained at the time of discharge. Magnetic resonance angiography (MRA) revealed mild stenosis of the left internal carotid artery in the cavernous portion, but no intracranial aneurysms (Fig. 3). Magnetic resonance imaging (MRI) revealed no abnormal findings surrounding the brainstem. However, constructive interference in steady-state (CISS) MRI revealed that the P1 and P2 portions of the left posterior cerebral artery (PCA) ran much more

Fig. 3 Postoperative magnetic resonance angiography (MRA) showing mild stenosis of the left internal carotid artery in the cavernous portion, but no intracranial aneurysms (left), and demonstrating that the left posterior cerebral artery runs much more anteriorly (right).

Fig. 4 Postoperative constructive interference in steady-state (CISS) magnetic resonance (MR) imaging revealing that the P1 and P2 portions of the left posterior cerebral artery (PCA) ran much more anteriorly above the left oculomotor nerve (ON) (left). Oblique-sagittal CISS MR imaging revealed that the left ON was very near to the PCA and superior cerebellar artery (SCA), inset: scout view (center). Coronal CISS MR imaging revealing the wide space surrounding the right ON, but the tight space surrounding the left ON (right).

anteriorly compared to the right PCA. Moreover, it is suspected that the strong compression to the left uncus, the left PCA and the left superior cerebellar artery (SCA) due to bilateral CSDH would result in the left complete oculomotor nerve palsy (Fig. 4). The patient subsequently resumed her normal life without any neurological deficits, except for the anisocoria. The patient has been observed for 6 months and though she recovered from the ptosis and diplopia her anisocoria remains. Repeated CT revealed no recurrence of CSDH.

Discussion

To date, only 3 cases of CSDH initially presenting with unilateral oculomotor nerve palsy without coma have been reported in the English literature. A summary of CSDH with initial presentation of oculomotor nerve palsy

Table 1 Summary of chronic subdural hematoma with an initial presentation of oculomotor nerve palsy without unconsciousness

Author (years)	Age/sex	Complication (drugs)	Symptom	Type of oculomotor nerve palsy	Side of hematoma	Type of operation	Postoperative results of oculomotor nerve palsy
Crone (1985)	66/F	Diabetes mellitus	Rt. oculomotor nerve palsy H/A	Pupil-sparing	Rt	Craniotomy	Complete recovery 1 month later
Phookan (1994)	60/M	TIA, HT (warfarin)	Rt. oculomotor nerve palsy	Complete	Bilateral	Burr-hole evacuation	Complete recovery in 6 hrs
Kurokawa (2005)	NA	NA	Rt. oculomotor nerve palsy	Complete	Bilateral	Burr-hole evacuation	NA
Our case (2012)	84/F	Lacunar infarction (aspirin)	Lt. oculomotor nerve palsy vomiting	Complete	Bilateral	Burr-hole evacuation	Partial recovery 6 months later

F: female, H/A: headache, HT: hypertension, Lt.: left, M: male, NA: not available, RT: right, TIA: transient ischemic attack.

without coma is shown in Table 1.^{2,4,8)} Among these 4 cases, including our case, 2 patients were women and 1 was a man and the other was not known. The mean age was 70 years old (range: 60–84 years). Two patients took anti-coagulants; one was warfarin, the other was aspirin. One patient had a history of diabetes mellitus. Preoperative radiological findings showed bilateral CSDH in 3 cases and unilateral CSDH in 1 case. One patient presented a pupil-sparing oculomotor nerve palsy and the others presented complete oculomotor nerve palsy. In this series, the oculomotor nerve palsy improved between 1 week and 3 months after surgery. There was no mention of the relationship between the oculomotor nerve and the PCA.

Etiology of oculomotor nerve palsy

In general, unilateral oculomotor nerve palsy with unconsciousness is one of the most important symptoms of cerebral herniation. In particular, if the patients present with unconsciousness, ipsilateral oculomotor nerve palsy, and contralateral hemiparesis, after severe traumatic head injury and cerebral infarction, then urgent treatment of decompressive surgery should be considered.^{3,11)} On the other hand, the most common cause of isolated oculomotor nerve palsy in a conscious subject is microvascular infarction caused by diseases, such as diabetes mellitus, hypertension, atherosclerosis, or collagen vascular disease.⁸⁾ Another common cause of isolated oculomotor nerve palsy is direct compression by an intracranial aneurysm; usually an internal carotid-posterior communicating artery aneurysm. Especially, a pupil-sparing oculomotor nerve palsy is known as an early symptom of enlarging aneurysm, because anatomically parasympathetic fibers supplying pupillary functions pass along the dorsal medial surface of oculomotor nerve, so are preserved to compress by internal carotid-posterior communicating artery aneurysms projecting laterally from the carotid artery.⁹⁾ While it is a rare condition, some case reports have revealed that direct compression of the oculomotor nerve by the PCA induced complete oculomotor nerve

palsy and microvascular decompression were useful in such cases.^{6,10)}

Mechanism of oculomotor nerve palsy due to CSDH

In this case, although the subdural hematoma was predominant on the right side, left complete oculomotor nerve palsy occurred. This discrepancy could be explained as follows: A slight midline shift to the left side and subsequent compression of the upper brainstem were induced gradually by the right subdural hematoma. On the other hand, postoperative CISS MRI demonstrated that the P1 and P2 portions of the left PCA ran much more anteriorly and inferiorly over the left oculomotor nerve compared to the right PCA and that the left oculomotor nerve was very close to both the left PCA and the SCA in the cisternal segment. We suspected that the left oculomotor nerve was compressed by the left PCA and the left SCA when the left uncus was dislocated medially due to the left CSDH combined with the co-existing compression to the upper brainstem from the right subdural hematoma. This mechanism could explain why the left oculomotor nerve palsy occurred alone. After evacuation of the bilateral hematoma, the symptom of left oculomotor nerve palsy started to improve immediately. We considered that this immediate recovery from the oculomotor nerve palsy except pupil dysfunction after the surgery further supported this mechanism.

As mentioned in the previous paragraph, bilateral CSDH takes an important role in the oculomotor nerve palsy. In the literature, bilateral CSDH may present rare central nerve disorders including divergence palsy as the result of subsequent compression of the upper brainstem.⁷⁾ In fact, 3 of the 4 cases of CSDH with isolated oculomotor nerve palsy presented bilateral CSDH.

CSDH with unilateral oculomotor nerve palsy without unconsciousness is a rare condition but an important differential diagnosis. In such cases, we considered that CT of the head is very useful to diagnose CSDH. If the patient presented the isolated oculomotor nerve palsy due to CSDH, we recommended an urgent evacuation of

the CSDH to avoid progressive aggravation and to recover from the oculomotor nerve palsy.

Conflicts of Interest Disclosure

We have no conflicts about this case report.

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