**An Unusual presentation of Chronic Subdural hematoma as Isolated Oculomotor palsy**

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**CONFLICT OF INTEREST**

None of the authors have potential conflicts of interest to be disclosed.

**RUNNING TITLE**

Chronic subdural hematoma as isolated oculomotor palsy

**Abstract:**

Isolated oculomotor palsy (ONP) is an unusual presentation in chronic subdural hematomas (CSDH) and is very rare as an initial manifestation. Most commonly, ONP is caused by ischemic microangiopathy, posterior communicating artery aneurysm, uncal herniation, neoplasia, traumatic and/or any inflammatory conditions. ONP in chronic subdural hematoma is a sign of cerebral herniation that is typically associated with a deterioration of consciousness. We describe a case of chronic subdural hematoma that presented with an isolated ONP.

Keywords: Isolated oculomotor palsy, Ptosis, Chronic subdural hematoma

**Introduction**

Subdural hematoma is a common type of traumatic intracranial mass lesion. CSDH is predominantly a disease of the elderly, with a peak incidence in the sixth and seventh decades of life and is very rarely seen in young adults 1. Chronic subdural hematoma (CSDH) usually presents with headache, fluctuating level of consciousness, falls, seizures, focal or transient neurological deficits, and hemiparesis 2.

Isolated ONP is well known as a symptom of microvascular infarction, intracranial aneurysm, diabetes mellitus, trauma, and/or in chronic lymphocytic meningeal infiltration, in which there is usually partial or complete ONP sparing of the pupil 3.

To date, only 10 cases of CSDH presenting with unilateral oculomotor nerve palsy as an initial manifestation have been reported in the English literature. We herein report a case of isolated oculomotor nerve palsy as presenting symptoms of unilateral CSDH and review all the cases.

**Case Presentation**

A 37-year- old woman presented to the emergency department with the complaint of mild headache and sudden dropping of the left eyelid for 5 days. She also had a brief period of loss of consciousness 5 days back. There is no history of vomiting, fever, slurred speech, weakness or seizure. She recalls an incident of minor fall injury 2 weeks prior in her bathroom sustaining impact on her head. No cardiac morbidities and neither was on any anticoagulants or anti-platelets. There is no other relevant medical or surgical history.

On examination, she was alert, conscious and well-orientated to time, place and person with GCS15. Vitals were stable. The only deficit was complete left ONP. There was severe ptosis and poor levator function in left eye. Ocular movements were affected on left eye with an absence of adduction and elevation. The left pupil was dilated (6mm) and unreactive to light(vs. 3mm and reactive in right eye). Fundus was normal. Other neurological and systemic examination was essentially normal. Computed Tomography (CT) scan of head was done which showed acute on CSDH on left fronto-temporo-parietal region with significant mass effect compressing ipsilateral lateral ventricle and midline shift with left posterior cerebral artery (PCA) infarct (Figure 1). Immediate left parietal single burr hole and evacuation of hematoma was done. About 60-70 ml of blood was evacuated in pressure. Postoperative period was uneventful and repeat CT scan of the head showed complete resolution of hematoma (Figure 2). Her left ONP gradually improved. The patient was discharged in an ambulatory state on 5th POD. At 1 month follow-up, her ptosis had significantly improved. Cerebral angiogram was done which was essentially normal.

**Discussion**

Isolated oculomotor palsy (ONP) is well known as a symptom of microvascular infarction, intracranial aneurysm, diabetes mellitus, trauma, and/or chronic lymphocytic meningeal infiltration. In these conditions, there is usually partial or complete ONP with pupil sparing. 3 Unilateral ONP as an initial manifestation of chronic subdural hematoma (CSDH) is a rare clinical condition. In our case, there was complete unilateral ONP without pupil sparing.

The oculomotor nerve has somatic and visceral components. The somatic fibers consist of a group of large caliber (10 to 16 µm) fibers that innervate the extraocular muscles and levator palpebrae superioris. The visceral fibers consist of a group of small caliber (1 to 5 µm) parasympathetic fibers that connect to the ciliary ganglion4. The pupils are supplied by parasympathetic fibers originating in the Edinger-Westphal nucleus of the third cranial nerve complex. These parasympathetic fibers are located very superficially in the nerve and are nearly always involved in surgical causes of oculomotor nerve palsies, such as compressive lesions or posterior communicating artery aneurysms5. Pupil-sparing third cranial nerve palsies are usually medical in nature, such as due to diabetes, hypertension, arteriosclerosis, or ischemic lesions3.

The oculomotor nerve runs in an anterolateral and slightly inferior direction after exiting the interpeduncular fossa. It then enters the roof of the cavernous sinus, where it acquires its own sheath of dura in the lateral portion of the anterior petroclinoid fold6. In tentorial herniation, the oculomotor nerve is first only flattened, then compressed and bent over the posterior cerebral artery 7.

There are several case reports of unilateral ONP in unilateral and bilateral CSDH (Table 1). In one such case, constructive interference in steady-state (CISS) magnetic resonance imaging (MRI) showed that the left PCA appeared to be much more anterior and inferior compared to the right PCA. Similarly, the left oculomotor nerve passed in close proximity between the left PCA and the left superior cerebellar artery 8. It was suspected that the left PCA and the left SCA compressed the oculomotor nerve when the left uncus was displaced medially by the left CSDH, along with the compression of the upper brainstem caused by the right subdural hematoma. This provides reasoning for the nerve palsy of the left oculomotor nerve only in spite of the subdural hematoma predominantly being on the right side.

K. Okuchi et al reported a case of right ONP in bilateral CSDH where the MRI 2 days after the evacuation of CSDH showed that the right oculomotor nerve was deflected medially toward the medial aspect of the anterior clinoid process after crossing the posterior cerebral artery. In contrast, the left oculomotor nerve ran normally, straight to the lateral aspect of the anterior clinoid process 9.

Complete oculomotor nerve palsy, along with unilateral decrease in vision and visual hallucinations without altered sensorium, was reported in a case of subacute bilateral subdural hematoma reported by Mulholland et al 10. These symptoms were reported to have been completely resolved following the surgical drainage of the subdural hematoma. Corrivelti et al said that progressive backward shifting of the brain could be produced by bilateral pressure on both cerebral hemispheres, which in turn causes significant posterior displacement of the brainstem resulting in nerve traction throughout this vascular corridor 11.

The cause of the ONP in our case was probably due to the pressure of the herniating uncus of the left temporal lobe, a false localizing sign that is common in raised intracranial pressure due to head injuries and intracranial tumors causing brain shift. Chronic subdural hematomas may present in this way, but usually have other localizing signs, impairment of higher mental functions, and/or deterioration of sensorium. The gradual recovery of the ONP after evacuation of the hematoma suggests that the ONP in our case was due to distortion of the nerve rather than other causes. The cerebral angiogram in our case was normal, without any variation in the course of the oculomotor nerve and PCA.

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| --- | --- | --- | --- | --- | --- | --- | --- |
| **Author** | **Age(Yr)/Sex** | **Comorbidities** | **Symptoms** | **Type of oculomotor nerve palsy** | **Side of hematoma** | **Operation** | **Postoperative recovery of ONP** |
| Crone et al., 12 (1985)  | 66/F  | Diabetes mellitus | Rt. oculomotor nerve palsy  | Pupil-sparing  | Right  | Craniotomy  | Complete recovery 1 month later  |
| Phookan et al.,3 (1994)  | 60/M  | TIA, HTN | Rt. oculomotor nerve palsy | Complete  | Bilateral  | Burr-hole evacuation  | Complete recovery in 6 hrs |
| Okuchi et al.,9 (1999) | 85/M | NA | Rt. oculomotor nerve palsy | Complete | Bilateral | Burr-hole evacuation  | partial |
| Mulholland et al.,10 (2006)  | 73/M | IHD, AF,HTN | Lt. oculomotor nerve palsy | Complete  | Bilateral  | Burr-hole evacuation  | NA |
| Matsuda et al.,8 (2012)  | 84/F  | Lacunar infarction (aspirin) | Lt. oculomotor nerve palsy vomiting  | Complete  | Bilateral  | Burr-hole evacuation  | Partial recovery 6 months later  |
| Mishra, et al.,13 (2013)  | 50/m | NA | Rt. oculomotor nerve palsy | complete | Right  | Burr-hole evacuation  | Complete recovery 1 month later  |
| Abdul Jalil MF, et al.,14 (2013) | 84/F  | HTN, hypercholesterolaemia, Alzheimer’s disease,  | Lt. oculomotor nerve palsy | complete | Lt | Craniotomy  | Oculomotor nerve palsy persisted |
| Corrivetti et al.,11 (2016) | 81/M | Diabetes | Lt. oculomotor nerve palsy | complete | Bilateral | Burr-hole evacuation | Complete |
| Corrivetti et al., 11(2016) | 65/M | HTN | Rt. oculomotor nerve palsy | complete | Bilateral | Burr-hole evacuation | Complete |
| Our Case (2019) | 37/f | Alcoholic | Lt. oculomotor nerve palsy | complete | Lt | Burr-hole evacuation  | Complete recovery 1 month later  |
|  |  |  |  |  |  |  |  |

TIA=Transient ischemic attack,HTN= Hypertension, F=female, M=male, Rt= Right, Lt= Left

Conclusion:

Unilateral ONP as the initial manifestation of CSDH is a rare clinical condition. The etiology may be due to the pressure of the herniating uncus of the temporal lobe, a false localizing sign, or due to distortion of the nerve. In our case, the cause was probably due to the pressure of the herniating uncus of the left temporal lobe. The gradual recovery of the ONP after evacuation of the hematoma suggests that the ONP in our case was due to distortion of the nerve rather than other causes. The importance of considering CSDH in the differential diagnosis of isolated ONP cannot be overemphasized. A high index of suspicion, prompt diagnosis, and timely management are crucial in the management of CSDH presenting with isolated ONP.

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Figure legend

Figure 1

Computed tomography showing acute on Chronic subdural hematoma on left fronto-temporo-parietal region with significant mass effect compressing ipsilateral lateral ventricle and midline shift with left posterior cerebral artery infarct.

Figure 2

Postoperative computed tomography scan of the head showing complete resolution of hematoma.