Oculomotor nerve palsy in chronic subdural hematoma

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.

Oculomotor nerve palsy (ONP) usually occurs in chronic subdural hematoma (CSDH) as a common sign of brain herniation that typically is associated with a deterioration of consciousness.

Ninety-eight cases of cSDH were operated over a 6-year period, in which 14 cases were classified as being bilateral. Among these 14 cases, 6 cases showed a rapid and aggressive clinical course. Therefore, complicated risk factors, the initial data on coagulofibrinolytic examination, magnetic resonance imaging appearance, and prognosis were analyzed.

Of the 6 cases, 5 showed a rapid aggravation as they awaited surgery. The period of the aggravation since the initial diagnosis harboring cSDH was 19 to 54 hours. One case was at first neurologically free from any disturbance but 17 hours later experienced a generalized seizure. All 6 cases experienced consciousness disturbance. In addition, 3 of them manifested oculomotor palsy [1].

Case reports

Zavatto et al., reported a bilateral oculomotor palsy after surgical evacuation of chronic subdural hematoma [2].

Corrivetti et al., reported 2 cases of bilateral CSDH who presented with ONP without deterioration of consciousness. An extensive literature review revealed this is an extremely rare finding.

They also investigated all the possible pathogenic mechanisms producing nerve impairment and found a strong association with bilateral subdural hematoma. Vascular compression between posterior circulation arteries and tentorial edge abnormalities also could be involved. Vulnerability of the oculomotor nerve seems to be a necessary condition leading to clinical onset and is caused by predisposing factors to nerve damage, including vascular disease, head trauma, or herpes zoster infection.

Although isolated ONP is a very rare presentation of CSDH, a differential diagnosis is absolutely necessary, because surgical treatment allows good recovery of third nerve palsy in most of the cases [3].

Matsuda et al., reported 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 [4)].

Jalil et al., reported the case of a patient who presented with left oculomotor cranial nerve palsy with an associated large volume left acute on chronic subdural haematoma. Coincidentally, this woman was also found to have a recent history of herpes zoster ophthalmicus [5)].

Moon et al., reported two cases of Kernohan's notch phenomenon secondary to chronic subdural hematoma detected by MRI. In the first case, the patient was drowsy with an oculomotor palsy and a hemiparesis ipsilateral to the chronic subdural hematoma. MRI in the post-operative period showed no abnormal signal or deformity of the crus cerebri. The neurological signs immediately resolved after trephination. In the second case, the patient was admitted with progressive decrease in their level of consciousness and ipsilateral hemiparesis with the chronic subdural hematoma. MRI on admission revealed an abnormal signal in the contralateral crus cerebri against the chronic subdural hematoma. After surgery, the mental state gradually recovered to normal with some degree of residual hemiparesis. In patients with chronic subdural hematoma, a compressive deformity of the crus cerebri, without abnormal signal on MRI, may predict a better neurological recovery in patients with Kernohan's notch phenomenon [6)].

Mishra et al., reported a 50-year old male patient with complaints of drooping of the right upper eyelid, for the past 1 day. He also gave a history of generalized mild headache for the past 1 week. There was no history of any injury, vomiting, fever, seizures, loss of consciousness, slurred speech, numbness, weakness, diplopia or any other major systemic illnesses like hypertension or diabetes. The patient also gave no history of any cardiovascular disorder. Patient was not a known alcoholic and neither was he on any anti coagulant or anti platelet therapy. On examination the patient was conscious and well oriented in time and space. His vitals were all within normal limits. Neurological examination was strictly unremarkable. Blood test revealed a normal blood count, urea, creatinine and electrolytes and was also negative for HIV antibodies. Ocular examination of the right eye revealed a vision of 6/9, improving to 6/6 with pin hole. There was severe ptosis with the marginal reflex distance 1 (MRD1) < −0.5 mm and a poor levator function (<4 mm). The eyeball too was displaced outwards and downwards (infraducted and abducted). The ocular movements were severely affected, with an absence of adduction and elevation; however abduction was full with mild residual depression. Depression was accompanied by intorsion, maximally when the eye was abducted. The

pupil was dilated (6 mm) and un-reactive to light (vs. 3 mm and reactive in the left eye). Fundus was essentially normal. The left eye was uninvolved. A provisional diagnosis of isolated unilateral oculomotor nerve palsy, right eye, was made and the suspected site of involvement of the nerve was clinically deduced to be around the fascicular subarachnoid portion. This is because the fascicles of the third cranial nerve exit the mid brain through the medial aspect of the cerebral peduncles and are not near any other cranial nerves at this point. So isolated third cranial nerve palsy occurs from lesions in this location. Aneurysm is the most common lesion to affect the third cranial nerve in the subarachnoid space. The fact that the pupil too was involved pointed towards a posterior communicating artery aneurysm. A provisional diagnosis of a posterior communicating artery aneurysm with or without overt subarachnoid haemorrhage was made and the patient was sent for an urgent computed tomography (CT scan) of the brain and orbits, which revealed a CSDH in the right fronto-temporo-parietal lobe, causing mass effect in the form of compression of the right lateral ventricle and a midline shift of 16.5 mm. The patient was immediately transferred to a higher neurological centre where he underwent evacuation of the haematoma via a right frontal burr hole surgery. Post operative period was uneventful and the patient was put on anti epileptics (tablet dilantin 300 mg once daily), observed for 2 months and then sent on 04 weeks sick leave. His oculomotor nerve palsy gradually recovered completely and CT scan brain repeated on his return from sick leave showed a complete resolution of the haematoma. He was finally discharged back to his unit with no residual adverse effects whatsoever [7)].

Cortes-Franco et al.,published in 2006 a Isolated IIIrd nerve palsy as the only sign of chronic subdural haematoma [8)].

Ortega-Martínez et al., reported a patient with a chronic subdural hematoma that presented with a complete third nerve palsy and normal consciousness. Complete recovery was achieved after surgical evacuation. Rebleeding within the hematoma cavity, most possibly favored by antiaggregating agents, was considered responsible for this rare presentation. In these cases expeditious surgical evacuation is indicated [9)].

A case of a 41-year-old man with a 1-month history of postural headache due to spontaneous intracranial hypotension (SIH). His MRI revealed bilateral chronic subdural hematoma (CSH) and diffuse dural enhancement after gadolinium infusion. Indium-111 radionuclide cisternography revealed a CSF leak from the cervico-thoracic junction and rapid accumulation of radioisotope in the bladder. Postural headache failed to resolve with prolonged bed rest. The patient became restless and suffered recent memory disturbance. We therefore decided to treat the CSF leak with an epidural blood patch. After the procedure, the patient's headache resolved completely. However one day later, left oculomotor nerve palsy developed. MRI revealed enlargement of the left CSH with mass effect and midline shift. After hematoma drainage, the patient became alert and oculomotor palsy recovered gradually. To treat cases of CSH with SIH, the best method is to repair the CSF leakage and treat subdural hematoma at the same time. If the patient shows depressed consciousness, we recommend initial drainage of the subdural hematoma, because, following the repair of CSF leakage, mass effect such as uncal herniation may occur [10)].

An 85-year-old male presented with bilateral chronic subdural hematomas (CSDHs) resulting in unilateral oculomotor nerve paresis and brainstem symptoms immediately after removal of both hematomas in a single operation. Initial computed tomography on admission demonstrated marked thick bilateral hematomas buckling the brain parenchyma with a minimal midline shift. Almost simultaneous removal of the hematomas was performed with the left side was decompressed first with a time difference of at most 2 minutes. However, the patient developed right oculomotor nerve paresis, left hemiparesis, and consciousness disturbance after the operation. The relatively marked increase in pressure on the right side may have caused transient unilateral brain stem compression and herniation of unilateral medial temporal lobe during the short time between the right and left procedures. Another factor was the vulnerability of the oculomotor nerve resulting from posterior replacement of the brain stem and stretching of the oculomotor nerves as seen on sagittal magnetic resonance (MR) images. Axial MR images obtained at the same time demonstrated medial deflection of the distal oculomotor nerve after crossing the posterior cerebral artery, which indicates previous transient compression of the nerve and the brain stem. Gradual and symmetrical decompression without time lag is recommended for the treatment of huge bilateral CSDHs [11].

In 1994 Phookan and Cameron published a bilateral chronic subdural haematoma with isolated oculomotor nerve palsy [12].

Crone et al published in 1985 a patient with adult-onset diabetes mellitus who developed an oculomotor palsy with pupillary sparing. Five days after her initial evaluation, she presented in a confused state with a complete oculomotor palsy. Computed cranial tomography revealed a chronic subdural hematoma. They recommend that noninvasive radiographic intracranial investigation be considered in elderly patients with adult-onset diabetes mellitus who present with headache and pupil-sparing oculomotor palsy [13].

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