Unilateral Ophthalmoplegia in Delirium Tremens Patient Due to Subdural Hematoma

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Abstract

INTRODUCTION

Ophthalmoplegia or the paralysis of nerves supplying extraocular muscles can be due to numerous etiologies. In alcohol users, ophthalmoplegia might be due to Wernicke's encephalopathy. A high dose of thiamine should be used with a high index of suspicion in such cases, but at the same time, other life-threatening conditions should not be missed. Careful history taking, clinical features and neuro imaging can guide in identifying the likely aetiology. Here, we present a case of isolated unilateral oculomotor nerve palsy due to subdural hematoma in a patient with delirium tremens.

KEYWORDS

Alcohol dependence, Delirium tremens, Oculomotor Nerve Palsy, Ophthalmoplegia, Sub dural hematoma

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INTRODUCTION

Ophthalmoplegia is the paralysis of one or more extraocular muscles responsible for eye movement. Any pathology resulting in palsies of cranial nerve (III, IV & VI) results in ophthalmoplegia or paresis that can lead to impairment of eye movements, according to which muscles are involved, ocular misalignment and ptosis.[1] In alcohol-dependent patients, Wernicke's encephalopathy due to thiamine deficiency is one of the causes of ophthalmoplegia along with other symptoms of ataxia and global confusion.[2] However, other aetiology such as Subdural hematoma, can have ophthalmoplegia as presenting symptom, which when not identified and managed early can result in life-threatening consequences. We describe a case of ophthalmoplegia in an alcohol-dependent patient presenting with delirium tremens.

CASE REPORT

A 37-year- old male patient farmer by occupation presented in Emergency with complaints of consumption of alcohol for 27 years, restlessness and sleep disturbance since 1-day before his presentation. The patient had a history of chronic alcohol use with progressively increasing amounts for 27 years. Craving, tolerance, withdrawal features of alcohol, loss of control and progressive neglect of alternative pleasures as more time was spent consuming and procuring alcohol. His usual amount was 4-5 litres of locally fermented alcohol (40-50 units of standard drink) with occasional locally distilled alcohol daily. The patient developed a fever 5 days before the presentation after which he gradually decreased his alcohol consumption. His last intake was 3 days before his presentation to the ER. The patient developed tremors,

tremulousness, and sweating after 12 hours of his last alcohol intake and agitation and sleep disturbance after around 72 hours of his last intake. Visual hallucinations and hallucinatory behaviour (such as seeing insects crawling in the ceiling, and walls and the patient trying to chase) were frequently reported. There was a fluctuation in his sensorium and frequent misrecognition of time, place and person was reported. Possible history of withdrawal seizure was present but it could not be confirmed. As he was difficult to manage at home, he presented to ER.

On examination, there was flushing, sweating and intense tremors and tremulousness. His vitals include BP: 140/90 mm Hg, Pulse: 92 beats/min, T: 98 degrees F, Respiratory rate: 22 cycles/ min. Systemic examination was normal but a detailed neurological examination could not be performed as he was uncooperative. On mental status examination, the patient appeared anxious, had increased psychomotor activity with frequent muttering to self, and impaired attention and concentration. He was disoriented to time and place but oriented to person. He was admitted to the Substance dependence treatment ward.

The patient had 2 discrete episodes of alcohol-induced psychotic symptoms in the past but no history of alcohol-related or other physical complications.

The patient was admitted with the diagnosis of Alcohol dependence syndrome presented in complicated withdrawal Delirium tremens with history of alcohol-induced psychotic disorder as per ICD-10. After admission, fluid supplementation was done, and parenteral thiamine along with multivitamins was given. Detoxification with intravenous lorazepam was done. On the third day of admission, the patient was out of delirium. But he started to complain of swelling and blurring of vision in his right eye. He also complained of a generalized severe headache on and off. The aggravating factor of headache was lying down in bed which would gradually improve in standing or sitting position. On ocular examination, there was drooping of the right eyelid. The patient's right eye was displaced laterally. He had difficulty moving his right eye inward and upward but could move his eye laterally. The pupil of the right eye was dilated (5mm) compared to the left eye. Reactivity to light was present. Fundus examination showed blurred disc margin nasally, minimal tortuous vein. His left eye was intact. Ophthalmology consultation was done and right oculomotor nerve palsy was confirmed. With suspicion of eye signs of Wernicke's encephalopathy, parenteral thiamine up to 1500 mg/ day in a divided dose was given for 3 days. However, there was no improvement in the patient's condition and his headache was persistent which was severe and progressive. Blood pressure was persistently raised (Systolic BP 180-190 mm of Hg and Diastolic 80-90 mm of Hg) and bradycardia (60 beats/ min) were noticed. No presence of vomiting, confusion or other signs of focal neurological deficits. The patient's history was reviewed which revealed a history of a fall injury. MRI brain showed Crescentic extra-axial areas of altered signal intensity in right front-tempo- parietal occipital lobe convexity –likely to be late-stage sub-acute subdural hematoma with mass effect and midline shift. The patient was then transferred to the neurosurgery unit for burr hole evacuation. After surgery, his oculomotor nerve palsy resolved and there were no complaints of a headache. The patient was discharged and advised to follow up.

Figure 1: Right eye oculomotor nerve palsy resulting in right eye displacement laterally

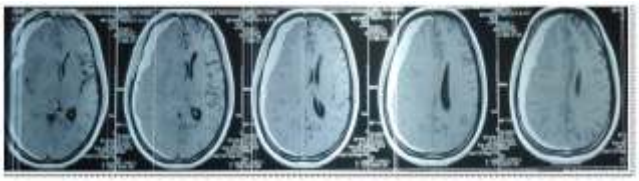

Figure 2: MRI Brain showing Crescentic extra-axial areas of altered signal intensity in right front-tempo- parietal occipital lobe convexity with mass effect and midline shift.

DISCUSSION

ere our patient who presented with the complicated withdrawal of alcohol- delirium tremens, had ophthalmoplegia which was noted later after resolving of his delirium. Ophthalmoplegia refers to the paralysis of extraocular muscles due to palsies in any cranial nerves supplying them. The third, fourth, and sixth cranial nerves innervate the extraocular muscles that position the globes in the orbits. Extraocular muscle paralysis resulting from lesions in one or all of these cranial nerves fails one or both eye movements in different directions. Palsies of these nerves are commonly used as clinical indicators of neurological injuries.[3]

In our case, the patient had difficulty moving his right eye inward and upwardly but could move his eye laterally. There was also ptosis of the right eye, all of which indicate involvement of the right oculomotor nerve. The oculomotor nerve (Cranial nerve III) is a somatic and visceral motor nerve which innervates the levatorpalpebraesuperioris and most of the ocular muscles (except the lateral rectus and the superior oblique muscles). The exact clinical presentation of IIIrd nerve palsy depends on the location of lesions which can extend from the fascicle of the nerve in the midbrain to its eventual termination in the orbit, however, in general, clinically it is observed that the ipsilateral eye is turned out and slightly depressed by the intact lateral rectus and superior oblique muscle respectively. The eye may only be moved laterally. As the parasympathetic fibres of this nerve innervate ciliary muscles, there can be a dilated fixed pupil, absent accommodation, and ptosis of the upper lid, often severe enough to cover the pupil.[1]

Isolated oculomotor nerve palsy as observed in our patient can result due to various etiologies ranging from infectious, vascular, traumatic, toxic and metabolic disorders.[4] In

ischaemic lesions pupil responses are usually spared whereas, in compression, including aneurysmal, the pupil is involved, initially loss of reactivity and then also dilation.[1] Indirect causes of IIIrd nerve palsy may include raised ICP or local compression and displacement of the nerve along its path as the result of aneurysms, traumatic hematomas, and other lesions.[5] In our patient, there was a mass effect due to subdural hematoma, so the right eye pupil was also involved. On reviewing the literature, the presence of isolated oculomotor nerve palsy due to subdural hematoma is in itself, a rare finding.[6] In addition, most of the cases described earlier were hematomas in bilateral regions, unlike our case which is unilateral. Subdural hematoma with mass effect can cause downward displacement of the cerebral hemispheres with herniation of the uncal portion of the temporal lobe. This in turn is likely to have led to compression and paresis of the oculomotor nerve.[3] Subdural hematoma is a potentially life-threatening consequence of head trauma that requires surgery, mortality estimates range from 40% to 65%, and these rates are higher still in certain high-risk populations.[7 8] Nature and character of headache as complained by the patient and the presence of bradycardia indicated raised Intracranial pressure due to expanding subdural hematoma. There may be other accompanying general symptoms such as vomiting, ataxia, and deterioration of consciousness, the last of which may occur as a sign of worsened ICP and cerebral herniation.[8] Thus, early recognition and intervention are essential for preventing mortality or long-lasting functional deficits.

As our patient presented with delirium tremens and the history of fall injury could not be confirmed initially led to the dilemma in identifying and determining the likely aetiology for his ophthalmoplegia. Furthermore, Wernicke's encephalopathy is a frequently described phenomenon in chronic alcohol users who can present with a triad- eye signs such as ophthalmoplegia, cognitive problems and cerebellar signs such as ataxia, nystagmus etc. However, studies have shown that only 16% of Wernicke's patients present with typical clinical symptoms, and 19 % of the patients had none of the triad symptoms.[10] Wernicke's when not treated acutely can result in irreversible Korsak off syndrome or even death. For these reasons, initially, after evidence of acute ophthalmoplegia, a high dose of thiamine was supplemented. However, it should be noted that in Wernicke's disease, ocular motor abnormalities include horizontal nystagmus on lateral gaze, lateral rectus palsy (usually bilateral), conjugate gaze palsies, and rarely ptosis. Pupils are usually spared in Wernicke's disease. Furthermore, those Wernicke's patients who recover may show improvement in ocular palsies within hours after the administration of thiamine[2] which was not observed in our patient. MRI was preferred as the choice of neuro imaging compared to CECT, as it may not only help in identifying life-threatening brain lesions but may also detect lesions in regions such as thalami, mammillary bodies, the periaqueductal area which are indicative of acute Wernicke's encephalopathy.

CONCLUSION

Ophthalmoplegiain the form of oculomotor palsy is not a usual presentation in alcohol-dependent patients and such cases may pose a diagnostic dilemma in the acute phase. Further, when not identified and treated early can be fatal. However, one should bear in mind that oculomotor palsy is very unlikely in Wernicke's disease and other etiologies should be sought. Symptoms like headache and bradycardia may be helpful and indicative of raised ICP with mass effect. In such a dilemma, review of history and careful examination, prompt evaluation with MRI along with ongoing treatment with high dose thiamine may be beneficial.

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