Bilateral Abducens Nerve Palsy due to Pseudotumor Cerebri in a Patient Taking Valproic Acid and Quetiapine: Report of a Case

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Abstract
Purpose: To present a case of bilateral abducens nerve palsy due to pseudotumor cerebri in a patient taking valproic acid and quetiapine.

Methods: Single case report.

Results: A 32-year-old overweight woman taking valproic acid and quetiapine because of bipolar disorder, admitted to the outpatient clinic with diplopia. She had not any other ophthalmic history. Ophthalmological examinations demonstrated esotropia in the left eye and papilledema in the both eyes. The abduction of both eyes was insufficient. CSF analysis revealed an extremely high pressure (28 mmHg) with normal CSF indices. These findings indicated that the patient had pseudotumor cerebri (PTC) that caused bilateral incomplete abducens nerve palsy with papilledema. The patient was discharged with oral acetazolamide therapy and three months after papilledema and abducens palsy were regressed.

Discussion: PTC is associated with abducens nerve palsy; but there was a few report of bilateral abducens nerve palsy due to PTC. So, we aim to report this case because of a rare condition that bilateral abducens nerve palsy due to PTC due to valproic acid and quetiapine.

Keywords
Abducens nerve palsy, Pseudotumor cerebri, Quetiapine

Introduction
Pseudotumor cerebri (PTC) is a condition characterized by increased intracranial pressure without intracranial space occupying lesions or hydrocephaly. It is characterized by headaches and bilateral optic disc edema in classical cases. Temporary vision loss and diplopia can be seen. Neurological symptoms are rare apart from the sixth nerve palsy. However, rare neurological cases have been reported [1-6]. PTC is usually seen in young to middle-aged women and often idiopathic. We report a case of PTC with bilateral abducens nerve palsy in a patient taking valproic acid and quetiapine for bipolar disorder.

Case Presentation
A 32-year-old overweight woman admitted to the outpatient clinic with diplopia for 3 months. She had not any other ophthalmic disorders, ocular surgery or trauma. On examination her vision was 20/20 in both eyes. Esotropia was present the left eye in primary gaze. On neurological examination, the abduction of both eyes was insufficient (left 3/5, right 3/5). Her pupils were symmetric and reactive to light. She had no other cranial neuropathy, no motor or sensory deficits, and no pathological reflexes. Ophthalmological examinations demonstrated bilateral papilledema. The patient’s color discrimination, visual fields were normal. Amplitude and latency of visual evoked potential (Figure 1) were normal.

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was reduced to 300 mg, on the grounds that increase the edema, with psychiatric clinic of the proposal. The patient was discharged with oral acetozalamide therapy. Three months after, her examination, papilledema was regressed and both eyes were 1/5 insufficient. The last examination, after one year, on examination 20/20 in both eyes. Papilledema was regressed and both eyes were 1/5 insufficient, too. She was taking oral acetozalamide 250 mg 6 hourly and she was taking valproic acid and quetiapine for bipolar disorder.

Discussion

PTC was defined by the modified Dandy criteria: 1-Symptoms and signs that may be related to increased intracranial pressure (headache, papilledema, etc), 2-Although the structure of CSF is normal, opening pressure of LP higher than 25 cm H2O, 3-Normal neurological findings except 6th nerve palsy and 4-The absence of space occupying lesion neuroradiologically 5-No other cause of increased intracranial pressure (ICP) identified [7]. The pathophysiology of PTC is not clear. Several risk factors have been reported such as obesity, arterial hypertension, anemia, Addison’s disease, hypothyroidism, hyperthyroidism, uremia, including excessive dosages of some exogenously administered medications (e.g., vitamin A, tetracycline, minocycline, nalidixic acid, corticosteroids), blood dyscrasias, and chronic respiratory insufficiency [8-9].

Abducens nerve palsy is the most common cause of the isolated ocular motor nerve palsies. Patients usually present with binocular horizontal diplopia worse in the distance, and esotropia in primary gaze. Patients also may present with a head-turn to maintain binocularity and binocular fusion and to minimize diplopia. Acquired abducens nerve palsies can be due to trauma, neoplasm, inflammation, and idiopathic etiologies. The sixth nerve is affected frequently in head trauma due to its course of close to the orbital lateral wall and placement in the cavernous sinus. In advanced age, among the reasons for the sixth nerve palsy it is frequently in the vascular pathology as well as the trauma. Rush and Younge reported that the most common

![Figure 1: Visual evoked potential of the patient.](image-url)
cause of abducens nerve palsy was the vascular pathology and trauma. Rush reported that the most frequency etiologic factors in abducens nerve palsy was vascular pathologies in another study involving 277 patients [10-13]. The sixth cranial nerve palsy is rare in young adults but when the abducens nerve palsy is occurred in younger adults, it must be considered a serious underlying pathology. Moster reported that the etiologies of abducens nerve palsy in the study consisting of 49 patients, aged from 15 to 50 years, were vasculopathy 29%, tumors 16% multiple sclerosis 12% [14]. Peter, et al. reported that the most common cause for a sixth nerve palsy in the young adults was a central nervous system mass lesion, although the most common cause for an isolated sixth nerve palsy in this age group was multiple sclerosis [15]. In our case, we thought that bilateral sixth nerve palsy developed secondary to increased intracranial pressure.

Cranial nerve palsies have been reported in PTC by authors with abducens nerve palsy in 10% to 20% of cases [16,17]. There is lot of reports on PTC with unilateral abducens nerve palsy; but there was unique report bilateral abducens nerve palsy due to PTC as the onset of systemic lupus erythematosus [18].

Uddin reported a case in which a patient on chronic valproic acid therapy for a seizure disorder developed PTC [19]. He claimed to changing his antiepileptic medication from valproic acid to topiramate effected a remission of PTC symptoms while maintaining his seizure-free status. In this case, the patient was obesity. Obese women present a significant cohort of PTC, but she gained approximately 40 pounds during topiramate therapy for about 10 years. For 2 years she was not taking topiramate. She was taking valproic acid and quetiapine for bipolar disorder for about one and a half years. Also she diagnosed with PTC 4 months after valproic acid in usage. There was evidence of that quetiapine may produce ocular toxicity [20]. So, we thought it might be due to the use of valproic acid and quetiapine in these patient caused PTC. We aim to report this case because of a rare condition that bilateral abducens nerve palsy due to PTC with valproic acid and quetiapine.

References