

First Episode Psychotic Disorder Possibly Associated with Acetazolamide in a Male Adolescent with Congenital Glaucoma

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ABSTRACT

First episode psychotic disorder possibly associated with acetazolamide in a male adolescent with congenital glaucoma

Primer congenital glaucoma is an uncommon ophthalmological disease manifesting at birth and accounting for 0.01-0.04% of total blindness. Acetazolamide, a carbonic anhydrase inhibitor is one of the options in the medical treatment of glaucoma and acute glaucoma crises. Carbonic anhydrase inhibitors are a class of pharmaceuticals, which are being used as anti-glaucoma and diuretic agent and for their anti-migraine and antiepileptic actions. Carbonic anhydrase inhibitors (topiramate, lacosamide, methazolamide) other than acetazolamide have been associated with psychotic disorders. Psychotic disorder possibly induced by acetazolamide in a male adolescent with blindness due to congenital glaucoma is going to be presented in this case report.

Key words: Acetazolamide, blindness, carbonic anhydrase inhibitors, congenital glaucoma, psychosis

ÖZET

Konjenital glokomu olan bir erkek ergende asetazolamid ile ilişkili olabilecek ilk atak psikotik epizod

Primer konjenital glokom doğumda görülen nadir bir oftalmolojik hastalıktır ve körlüğün %0.01-0.04'den sorumludur. Bir karbonik anhidraz inhibitörü olan asetazolamid glokom ve akut glokom krizlerinde kullanılan tıbbi tedavi alternatiflerinden biridir. Karbonik anhidraz inhibitörleri glokom tedavisi, diüretik etki, migren ve epilepsi tedavisi için kullanılan bir tür ilaç alt sınıfını kapsar. Asetazolamid dışındaki karbonik anhidraz inhibitörleri (topiramate, lakozamid, metazolamid) daha önce psikotik bozukluklarla ilişkilendirilmişlerdir. Bu olgu sunumunda konjenital glokoma bağlı körlüğü olan bir erkek ergende asetazolamidle tetiklenmiş olabilecek bir psikotik bozukluk sunulacaktır.

Anahtar kelimeler: Asetazolamid, körlük, karbonik anhidraz inhibitörleri, konjenital glokoma, psikoz



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INTRODUCTION

Primer congenital glaucoma is an uncommon ophthalmological disorder accounting for 0.01-0.04% of total blindness. The disease is usually manifested at birth (even before birth) or early childhood (before 3 years of age). Surgical treatments are more effective than medical treatments however medical treatment plays a supportive role and may be useful in acute glaucoma crises. Acetazolamide; a carbonic anhydrase inhibitor is one of the options in the medical treatment of glaucoma and acute glaucoma crises with a daily dose of 250mg/day to 1000mg/day (1). Carbonic

anhydrase inhibitors are a class of pharmaceuticals, which are being used as anti-glaucoma and diuretic agent and for their anti-migraine and antiepileptic actions (2). Psychotic disorder possibly induced by acetazolamide in a male adolescent with congenital glaucoma is going to be presented in this case report.

CASE

Sixteen years old male adolescent going to 9th grade in the school for blind children referred to our outpatient clinic with his parents for the complaints of acute socially introverted behavior, talking to himself, refuse

to eat, reference and persecutor delusions, Capgras like findings, disorganized speaking and repetitive aimless body movements. These stated acute psychotic features and symptoms had been evident for 5 weeks. He had total blindness due to congenital glaucoma which had started just after birth. He had experienced countless operations and hospitalizations for that condition. He had no autistic features. Before the onset of psychotic findings, acute glaucoma crisis had occurred and he had taken oral acetazolamide 750mg/day for two weeks long. His psychotic features began after the first week of oral acetazolamide treatment. Acetazolamide was stopped one week after the psychotic features had begun. He had no history of taking corticosteroids and drug abuse. Her mother and father was elementary school graduated and had low socio-economical status and there had been no history of psychosis and bipolarity in the family and probands. Neurological examination, radiological findings, electroencephalogram and Wilson disease parameters had been normal according to previous neurological investigation after the onset of psychotic features. No metabolic-endocrinological-biochemical abnormality was detected which can cause delirium. There was not any detected electrolyte abnormality, a possible side effect of acetazolamide that may cause cognitive alteration. In the psychiatric examination; talking to himself and swaying aimlessly were very prominent. He was not using expressive language for communicating with people. He had restricted thought content, flat affect and unhygienic self-care. He was compulsively touching his eyebrows and eyelashes and continuously perseverating those parents of him had not been actually his real parents. These signs and symptoms had been emerged after the onset of acetazolamide. In the retrospective examination there had not been any clue about autistic features or stereotypical movements that blind people may display. He was diagnosed with schizophreniform disorder. As he had been overweight, aripiprazole with the dose of 5mg/day was initiated. His dose was increased to 15mg/day in the second week and to 30mg/day in the third week. His psychotic features quiet regressed in the first month of the antipsychotic treatment. He began to eat sufficiently.

Negative psychotic features also improved after the positive psychotic features had disappeared. In the third month of follow up he had appropriate affect to the current situation, was quiet social. His thought content and amount and rate of speech were appropriate for his age group. His self-care and hygiene was quiet better. Complete remission was maintained so aripiprazole treatment was reduced to a very low dosage of 5mg/day in the end of the first year follow-up. The patient is still being followed without obvious psychotic symptoms.

DISCUSSION

It is interesting that total blindness had been usually associated with lower risk of psychosis and schizophrenia (3). Sanders et al. (4) claimed that dynamic adaptations of N-Methyl-D-Aspartic (NMDA) receptor channels in the visual cortex may account for cognitive functioning that is insusceptible to psychosis.

As it was known, this is the first case report declaring possible association between acetazolamide and psychotic symptoms. According to the Naranjo classification (5) it was found to be + 3 points which refers to "possible adverse drug reaction". Although psychotic features began just after the acetazolamide usage which brings to mind drug induced psychotic disorder, as psychotic disorders frequently starts at the adolescent period especially in the boys, it is impossible to conclude that there was a certain association with acetazolamide and psychosis in this case report.

It is interesting because acetazolamide has been reported as having antipsychotic properties (6,7) other than antimigraine (7) and antiepileptic action. Acetazolamide induces an intraneuronal alkalinization and resulting shift in the activation curve of hyperpolarization-activated cation current in thalamocortical neurones, which led to a persistent up-regulation of hyperpolarization activated cation. These mechanisms lower the electrical activity in the thalamocortical system and contribute to antiepileptic activity of acetazolamide (2). Antiepileptics with carbonic anhydrase inhibitor activity; lacosamide (8), methazolamide (9) and especially topiramate (10) had

been associated with induction of psychosis in numerous case reports. However topiramate induced psychosis might be related with a different pathway other than the antiepileptics who have also carbonic anhydrase inhibitor activity. The gabaergic and hypoglutamatergic action of topiramate is proposed to cause hyperdopaminergic state, which induces psychosis (10), however it is difficult to predict and determine the underlying mechanism of possibly

psychotic induction due to acetazolamide.

All these causes listed here respectively; having no history of psychosis in the family, abruptly onset of severe psychotic symptoms just after medication, not having prodromal psychotic features, quick improvement with antipsychotic treatment, brings to mind acetazolamide induced psychotic disorder in this case, however it is impossible to exclude coincidental co-occurrence.

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