Post-Traumatic Anisocoria Caused by Iris Sphincter Rupture in a Child: An Unusual Case Report

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Abstract

Anisocoria is a rare and unusual post-traumatic condition among eye pathologies. After a traumatic eye injury, the problem can often be masked by complaints of bleeding in the eye and loss of vision. Therefore, anisocoria, which may occur as a result of iris sphincter rupture, should be considered and investigated in the examination, along with other eye examination findings in patients with complaints of traumatic injury to the eye. In this case report, we present a case of a 14-year-old male patient who was investigated for the differential diagnosis of anisocoria in the eye after head trauma and was later diagnosed as iris sphincter rupture.

Keywords: anisocoria, trauma, iris sphincter rupture, family practice, primary care

Introduction

Anisocoria is a condition characterized by unequal pupils with a size difference of 0.4 mm or more (1). It may occur in 20% of the population (1), and may be completely harmless, but its sudden onset can be a sign of a serious medical problem, such as neurological emergencies (2). In this report, a patient who applied with the complaint of post-traumatic anisocoria will be presented.

Case

A 14-year-old male patient with no pre-existing disease had applied to the emergency service with the complaints of redness in the eye and loss of vision due to trauma by slippers on the right frontal region of his friend 20 days ago. In emergency service it was said that there was slight bleeding in the eye, and an eye drop medicine to be used for 3 days was prescribed and he was discharged. The patient, who stated that he could not see clearly 10 days later, applied to the eye outpatient clinic of state hospital. There he was told that he had 90% vision loss in the right eye and he was referred for further differential diagnosis for anisocoria. Due to his head trauma history, the patient was firstly consulted with neurosurgery department, underwent brain computed tomography, which was found to be normal, and then he was referred to pediatric neurology because the current clinic could not be attributed to brain trauma.

The patient was evaluated by us as Family Medicine before the pediatric neurology evaluation. In his physical examination, his general condition was good, he was conscious, oriented, cooperative, and his vitals were stable. Eye movements were normal in all directions, and not painful. However, anisocoria was observed. Direct and indirect light reflexes were found bilaterally positive. There was no ptosis and ophthalmoplegia. Visual acuity in the right eye was 1/10.

During pediatric neurology examination, deep tendon reflexes were found to be normoactive, clomus and Babinski were negative, muscle strength was 5/5 in all extremities, walking and speaking were normal. There was no sensory defect or sphincter defect. Motion induced blindness (MIB) and relative afferent pupillary defect (RAPD) were negative. Cranial nerves were found to be intact and cerebellar tests were normal. Routine laboratory tests revealed unremarkable. The patient underwent with craniocervical and orbital contrast MRI, and MRI angiography. Visually evoked potential (VEP) was reported as normal. In orbital evaluation; bulbus oculi, extraorbital muscles, retrobulbar adipose tissue and optic nerves were evaluated as normal, likewise in the evaluation of brain tissues, posterior fossa formations, bulbus, pons, 4th ventricle were evaluated as normal. No abnormality was observed in the bilateral cerebellopontine corner.

The patient, who was evaluated as normal by neurosurgeon and pediatric neurologist, was further re-evaluated by ophthalmologist, and slit-

lamp examination revealed nasal and temporal iris sphincter rupture. After diagnosing the cause of anisocoria, the patient was transferred and followed up by the ophthalmology department.

Discussion

There are several causes of anisocoria: The most common one was the physiological one. Physiological anisocoria is defined as pupillary disparity, usually 0.4 mm, rarely greater than 0.8 mm, not due to a secondary cause (1). If anisocoria is physiological, the difference in pupil sizes should remain equal in dim and bright light (3).

Another well-known anisocoria reason, Horner's syndrome, occurs as a result of lesions in the brainstem and cervical cord, along the oculo-sympathetic pathway. Clinical features include ipsilateral miosis, ptosis, anhidrosis, enophthalmos, and ciliospinal problems (4).

In the Adie's tonic pupil, caused by damage to the nerve of the iris sphincter muscle, the larger pupil cannot become as small as the other pupil. However, as the eye adapts, the pupil constricts more. This cause of anisocoria can be diagnosed with dilute pilocarpine, which causes significant constriction of the larger pupil. Other causes of a dilated pupil typically do not respond to dilute pilocarpine and help confirm the diagnosis of Adie's tonic pupil (5).

Neurological emergencies, such as stroke, intracranial aneurysm (6), demyelinating diseases, head trauma and brain tumors are the most common causes of oculomotor nerve palsy in adults. In ischemic lesions of the oculomotor nerve, pupillary function is usually preserved, while in compressive lesions the pupil is involved.

Pharmacological Anisocoria

Pharmacological agents with anticholinergic or sympathomimetic properties cause anisocoria, especially if administered to one eye. Some examples of pharmacological agents that may affect pupils include pilocarpine, opioids, scopolamine patches, inhaled ipratropium, nasal vasoconstrictors, and glycopyrrolate antiperspirants. Alkaloids found in plants, such as Jimson weed, Angel's trumpet, and blue nightshade, can also cause anisocoria (6).

An interesting pharmacological anisocoria has been reported after topical treatment of axillary hyperhidrosis with glycopyrronium, an anticholinergic resulting from accidental ocular exposure (7). Another ocular exposure was reported in an asthmatic patient who developed ipratropium bromide-related anisocoria during nebulizer therapy (8).

Traumatic new-onset anisocoria is mostly seen as a result of acute brain injuries (9). Aneurysmal or intracranial hemorrhage and stroke may accompany anisocoria after blunt brain trauma and may even reflect the severity of brain damage (10). However, anisocoria after traumatic eye injury is a rare clinical condition.

In our case, eye bleeding, vision loss and anisocoria were detected in a child secondary to eye trauma. To the best of our knowledge, there was no study in the literature on iris sphincter rupture as the cause of anisocoria as reported in our case. In traumatic iris sphincter rupture, anteroposterior compression of the eyeball leads to equatorial dilation that will actively pull along the corneoscleral junction, resulting in a sphincter tear (11). Although this sphincter tear is unusual, it should be investigated in a patient with anisocoria, especially after brain injury and other neurological causes have been excluded. As occurred in our case, somehow traumatic bleeding in an eye may overlap the anisocoria and may be neglected. Therefore, anisocoria should be kept in mind in the post–traumatic eye injury.

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