Transient Anisocoria in an Otherwise Asymptomatic Child

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A 10 year-old boy presented to the emergency department with asymmetrical pupils, which were noted eight hours earlier. His past medical history was notable for asthma, for which it was prescribed inhaled ipratropium bromide via a spacer and face mask. That morning, he reported having administered an ipratropium bromide inhalation in the dark, with dubious face mask adaptation. There was no history of trauma. On examination, the child was well, with left mydriasis (Fig. 1). Anisocoria was more pronounced under bright light and there was no ptosis. Vision and eye movements were normal. Near and light pupillary reflexes were present, but slightly slower on the left. The neurological examination by a neurology consultant was unremarkable. Head computed tomography without contrast showed no lesions. Ophthalmology consultation was not immediately available at our hospital and, therefore, pharmacological tests were not performed. Pharmacological mydriasis was suspected, and the child was admitted for overnight observation. By the next morning, anisocoria had dramatically improved, which supported our hypothesis. He was discharged with a scheduled appointment for the following day. Two days after the initial examination, anisocoria had completely resolved (Fig. 2), and there were no new episodes in the following year.

The possible etiologies for anisocoria pose a diagnostic challenge. It is an alarming sign on physical examination, often leading to extensive investigation to exclude Horner syndrome and oculomotor nerve palsy.[1] It can be a sign of both life-threatening diseases (brain herniation, neoplasms, or aneurysms) as well as benign conditions, such as physiological and pharmacological anisocoria.

Observation under bright and dim light is important to identify the abnormal pupil. Since, in this case, anisocoria was more pronounced in bright light, Horner syndrome was excluded. Pharmacological tests with pilocarpine eye drops may differentiate third nerve palsy from pharmacological mydriasis, potentially avoiding further investigation.

Being a direct antagonist at muscarinic cholinergic receptors, ipratropium bromide paralyzes the parasympathetic fibers of the oculomotor nerve,[2] leading to mydriasis if applied to the eye. While there have been reported cases of ipratropium bromide induced anisocoria in children,[3-5] usually due to difficulties in face mask adaptation, it is still an infrequent condition. Although pharmacological tests were not performed in our case, acute onset of anisocoria after ipratropium bromide inhalation, unremarkable neurological examination, and improvement over time pointed toward an iatrogenic etiology.

Figure 1. Left mydriasis noted at first examination.

Figure 2. Symmetrical pupils 48 hours after initial examination.

Keywords: Anisocoria/diagnosis; Anisocoria/etiology; Child; Ipratropium/adverse effects; Mydriasis/chemically induced; Mydriasis/etiology

WHAT THIS REPORT ADDS

- Anisocoria is an alarming sign on physical examination, often leading to extensive investigation to exclude life threatening etiologies.
- A thorough anamnesis and physical examination, along with pharmacological tests, are essential in the differential diagnosis and may avoid unnecessary further testing.
- It is important to consider ipratropium bromide as a cause for pharmacological anisocoria in children with a compatible history and absent alarming signs at physical and neurological examination.

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