

# Transient anisocoria following alcohol consumption: a case report



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## HIGHLIGHTS

Transient anisocoria may occur after concurrent alcohol and marijuana use. Pharmacological influences should be considered in the differential diagnosis of anisocoria. Careful clinical evaluation is essential to exclude life-threatening conditions.

## ABSTRACT

We present the case of a 28-year-old woman who developed transient anisocoria following moderate alcohol consumption. This rare presentation highlights the potential exacerbation of physiologic anisocoria or mild Horner syndrome by alcohol. A thorough clinical evaluation, including neurological and ophthalmological assessments, and appropriate diagnostic workup excluded serious underlying conditions. The patient was managed conservatively and advised on follow-up care. This case underscores the importance of recognizing benign causes of anisocoria while ruling out life-threatening etiologies. The concurrent use of marijuana, with its parasympathomimetic effects, may have contributed

to the observed pupillary asymmetry, suggesting a potential synergistic interaction between alcohol and marijuana in inducing transient anisocoria. This case emphasizes the need for further research into the mechanisms of substance-induced anisocoria and its clinical implications, particularly in patients with a history of substance use. Clinicians should consider pharmacological influences in the differential diagnosis of anisocoria to avoid unnecessary interventions and provide appropriate patient education.

**Key words:** anisocoria, alcohol, marijuana, substance use

## INTRODUCTION

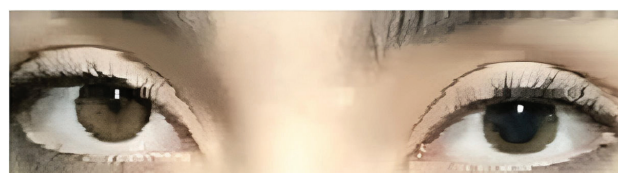
Anisocoria is a condition where the pupils of the eyes are of different sizes, typically with a difference of 0.4 mm or more [1]. It can stem from both benign conditions and severe, vision- or life-threatening emergencies. While it is often associated with life-threatening conditions such as intracranial hemorrhage, traumatic brain injury, or Horner's syndrome, benign and reversible causes of anisocoria are less frequently documented [2]. Alcohol, a widely consumed psychoactive substance, is known to affect the central nervous system and autonomic functions [3, 4]. Also, marijuana, primarily through its active compound  $\delta_9$ -tetrahydrocannabinol (THC), exerts parasympathomimetic effects, typically causing mild pupillary constriction (miosis) due to its action on the autonomic nervous system [5]. The concurrent use of these substances may lead to synergistic effects on autonomic regulation, further complicating pupillary dynamics. While the differential diagnosis of anisocoria is well-documented, cases involving transient anisocoria induced by alcohol and marijuana are rarely reported in the literature. In this case report, we present a unique instance of transient anisocoria observed in a patient with acute alcohol consumption. We aim to highlight the potential mechanisms, diagnostic considerations, and clinical significance of this unusual presentation to enhance awareness among healthcare providers.

## CASE REPORT

A 28-year-old woman presented to the emergency department at 1:00 a.m. with complaints of blurred vision and a headache following a social event where she consumed approximately 4 glasses of alcohol. The patient subsequently revealed that she had also been using marijuana at the same time. However, she was unable to provide specific details regarding the quantity or timing of marijuana consumption due to a lack of precise recollection. She denied any history of head trauma, seizures, nausea, vomiting, photophobia, or previous episodes of anisocoria. Her medical, surgical, and ophthalmologic history was unremarkable, and she was not on any regular medications (fig. 1).

FIGURE 1

Patient with anisocoria.

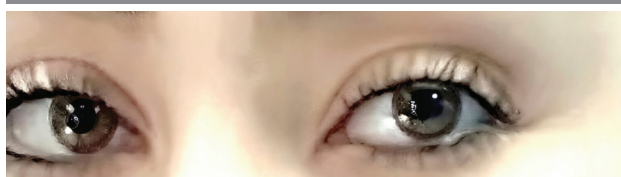


Upon presentation, the patient was hemodynamically stable, with vital signs recorded as follows: blood pressure 110/65 mmHg, heart rate 62/min, respiratory rate 16/min, and oxygen saturation of 98% on room air. The neurological examination revealed a right pupil measuring 5 mm and a left pupil measuring 2 mm, both of which were reactive to light. Marcus Gunn's pupil test was negative. The fundoscopic examination showed no evidence of papilledema, retinal abnormalities, or trauma. Additionally, the slit-lamp examination revealed no corneal abrasions or abnormalities in the anterior chamber. There was no evidence of ptosis, abnormalities in extraocular movements, or focal neurological deficits. The patient was alert, oriented, and exhibited normal speech.

The differential diagnosis for anisocoria in this case included physiological anisocoria, Horner syndrome, third cranial nerve palsy, and pharmacologic or toxicologic effects. Laboratory tests revealed an elevated blood alcohol level, consistent with recent alcohol intake, while the comprehensive metabolic panel and complete blood count were within normal limits. A toxicology screen was negative for other substances. Imaging studies, including a non-contrast head computed tomography (CT), showed no evidence of intracranial hemorrhage, mass effect, or midline shift. For specialized testing, apraclonidine, an  $\alpha_2$ -adrenergic agonist, was chosen for its ability to differentiate between Horner syndrome and physiological anisocoria [6]. The topical administration of apraclonidine 0.5% drops resulted in the resolution of anisocoria (fig. 2). Following the administration of apraclonidine, the patient reported a significant improvement in her blurred vision.

FIGURE 2

Resolution of anisocoria after administration of apraclonidine.



The patient was managed conservatively. Symptomatic treatment included the administration of acetaminophen for her headache. She was reassured about the benign nature of her condition and provided with discharge instructions, which included warning signs such as persistent anisocoria, severe headache, or focal neurological deficits. Lifestyle modification was advised, with specific recommendations to avoid excessive alcohol consumption. A follow-up appointment was scheduled with an ophthalmologist for further evaluation, including consideration of pharmacologic testing to definitively exclude Horner syndrome.

At the follow-up visit, the patient reported no recurrence of anisocoria. Further testing confirmed the absence of any underlying pathological cause, supporting the initial diagnosis of a benign condition.

## DISCUSSION

Physiological anisocoria, which affects up to 20% of the population, is often exacerbated by factors such as stress, fatigue, or pharmacological agents [7, 8]. The differential diagnosis for sudden anisocoria encompasses a wide spectrum of potential causes, ranging from benign pharmacological exposures to urgent, life-threatening conditions [9]. While the differential diagnosis of anisocoria is well-documented, cases involving transient anisocoria induced by alcohol consumption are rarely reported in the literature.

This case report presented a unique instance of transient anisocoria in a 28-year-old woman following acute alcohol and marijuana use and highlighted the diagnostic challenges and clinical implications of such presentations. The patient's presentation was notable for the absence of focal neurological deficits, normal imaging findings, and resolution of anisocoria following the administration of apraclonidine 0.5% drops. The resolution of anisocoria with apraclonidine strongly supported a benign etiology, as this medication reverses anisocoria in Horner syndrome by enhancing sympathetic tone [6]. In this patient, the absence of ptosis, extraocular movement abnormalities, or other neurological deficits, combined with normal imaging studies, made emergent causes such as third cranial nerve palsy or intracranial pathology unlikely.

Alcohol is known to affect the autonomic nervous system, which regulates pupillary size and reactivity [10]. The transient unmasking of physiologic anisocoria, in this case, may be attributed to alcohol's depressant effects on the central nervous system, leading to temporary dysregulation of sympathetic and parasympathetic tone. Additionally, the patient's concurrent use of marijuana, its active compound THC exerts parasympathomimetic effects, typically causing mild pupillary constriction (miosis) due to its action on the autonomic nervous system which also has psychoactive properties, may have contributed to the observed pupillary asymmetry [11].

In the context of this case, the patient's concurrent use of marijuana and alcohol raises the possibility of a synergistic effect on pupillary asymmetry. Alcohol, a central nervous system depressant, affects the balance between sympathetic and parasympathetic tone, which can transiently unmask physiological anisocoria [4, 12]. Marijuana, on the other hand, has been shown to cause mild pupillary constriction (miosis) due to its parasympathomimetic effects [5]. The combination of these substances may exacerbate autonomic dysregulation, leading to transient anisocoria. This hypothesis is supported by studies demonstrating that the combined use of alcohol and marijuana can produce additive or synergistic effects on neurological and autonomic functions [13]. This interaction may explain the transient anisocoria observed in this patient, as the combined pharmacological effects of both substances could disrupt normal pupillary reflexes. However, further research is needed to elucidate the precise mechanisms by which marijuana and alcohol interact to influence pupillary size and symmetry.

The diagnostic approach in this case underscores the importance of a systematic evaluation to rule out emergent conditions. This case report emphasizes the need for a thorough clinical assessment and targeted diagnostic workup in patients presenting with anisocoria, especially in the context of alcohol consumption. While transient anisocoria induced by alcohol is rare, it should be considered in the differential diagnosis to avoid unnecessary interventions and alleviate patient concerns. Further research is needed to elucidate the mechanisms by which alcohol and other substances may influence pupillary asymmetry, as well as their potential clinical significance.

The management of this case highlights the value of conservative treatment and patient education. The patient was reassured about the benign nature of her condition and advised to avoid excessive alcohol consumption. Follow-up with an ophthalmologist confirmed the absence of underlying pathology, reinforcing the initial diagnosis. This case illustrates the importance of considering pharmacological and lifestyle factors in the evaluation of anisocoria, particularly in patients with a history of substance use.

This case report has several limitations. First, the patient could not provide specific details about the quantity or timing of marijuana use, limiting our ability to definitively link her anisocoria to marijuana consumption. Second, while apraclonidine supported a benign etiology, the lack of definitive pharmacological testing for Horner syndrome (e.g., cocaine or hydroxyamphetamine drops) leaves some diagnostic uncertainty. Third, the rarity of reported cases involving marijuana-induced anisocoria makes it difficult to draw firm conclusions about its role. Future studies should investigate the pharmacological effects of marijuana on pupillary dynamics, particularly in combination with alcohol.

## CONCLUSION

This case report highlights the diagnostic challenges of transient anisocoria following alcohol and marijuana use,

emphasizing the importance of considering pharmacological influences in the differential diagnosis. Alcohol's depressant effects on the autonomic nervous system, combined with marijuana's parasympathomimetic properties, likely contributed to the transient pupillary asymmetry observed in this patient. The absence of neurological deficits and normal imaging findings supported a benign etiology, further confirmed by the resolution of anisocoria with apraclonidine. This case underscores the need for a thorough clinical evaluation to rule out emergent causes while recognizing the potential synergistic effects of alcohol and marijuana on pupillary reflexes. Further research is warranted to elucidate the mechanisms underlying substance-induced anisocoria and its clinical implications.

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**Conflict of interest:**

None.

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**Ethics:**

The content presented in the article complies with the principles of the Helsinki Declaration, EU directives and harmonized requirements for biomedical journals.