Case Report

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Looking Beyond the Mouth–A Rare Case of Acute Glaucoma Presenting with Dental Pain: A Case Report

Urgent Message: Acute angle closure glaucoma can present with a variety of types of head and facial pain. Rapid identification of concern for elevated intraocular pressure (IOP) or confirmation of elevated IOP by tonometry is critical for preserving vision.

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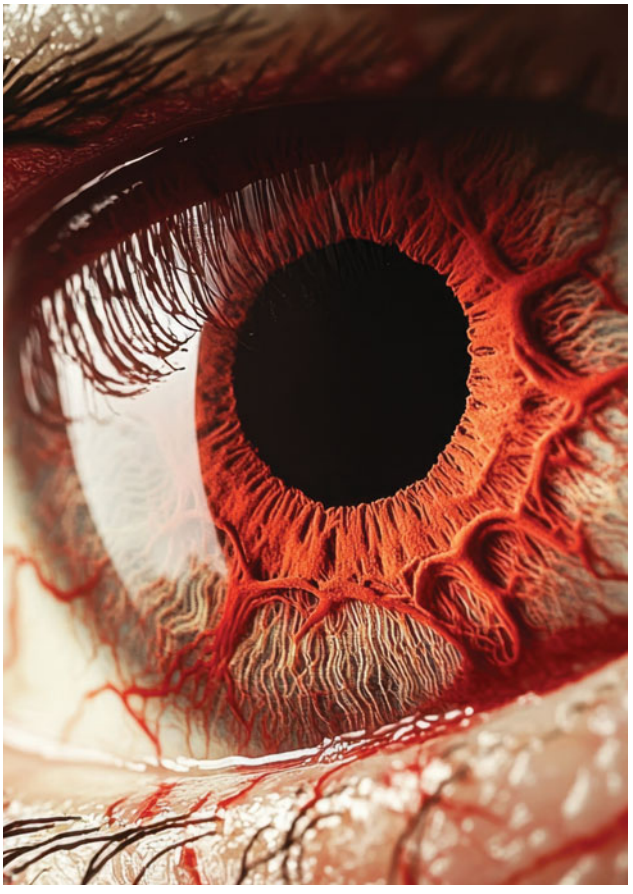
Abstract

Introduction: Acute angle closure glaucoma (AACG) is a rare, serious condition and one of the few true ophthalmologic emergencies. Due to the time sensitive nature of the diagnosis, it is important for urgent care (UC) clinicians to be aware of its various manifestations.

Clinical Presentation: A 60-year-old woman presented to the emergency department (ED) with a chief complaint of acute and sudden-onset dental pain. She also complained of new blurry vision in the ipsilateral eye. Her visual acuity was 20/200 in the affected eye. The triage nurse assigned her an emergency severity index (ESI) level of 4 based on her complaint and vital signs.

Physical examination: After triage, the patient was assessed by a clinician in the waiting room. On exam, the patient was hypertensive, but otherwise her vital signs were normal. In evaluating her face and head, the clinician noted no facial swelling and only chronic



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appearing dental caries that were not in the area of her pain. Ocular exam of her left eye revealed a mid-range fixed pupil, and the globe was firm to the touch with "finger palpation" (tactile assessment over a closed lid). Her right eye was normal in appearance, tactile assessment, and acuity. Her neck was supple, and her ears showed no abnormalities. She was quickly brought back to a room where tonometry was performed revealing a critical intraocular pressure (IOP) of 66 mmHg.

Diagnosis: Acute angle closure glaucoma

Resolution: The patient was aggressively treated with multiple IOP-reducing medications, however, her pressure remained unsafely elevated. Ophthalmology was consulted, and she underwent laser iridotomy by the on-call ophthalmologist. She was discharged the next day. She underwent subsequent laser iridotomy as an outpatient, and her IOP stabilized. With adequate pressure control, normal vision in the affected eye was restored.

Conclusion: This patient's pain was experienced predominantly as dental pain, which led to some delay in focused ocular assessment. However, thanks to aggressive treatment once AACG was identified, the patient was able regain normal vision in the affected eye.

Introduction

Glaucoma is one of the leading causes of blindness in the world.[1] Glaucoma refers to a pathologic increase in IOP, and while the exact mechanism is poorly understood, the increased pressure eventually results in retinal cell death if not adequately treated.[1] The open angle glaucoma subtype tends to develop slowly and is usually asymptomatic—slowly progressive until vision loss occurs.[1] Conversely, AACG, as the name implies, involves rapid increases in IOP and constitutes a medical emergency; vision loss can occur if not treated rapidly, which may be irreversible.[1] Normal IOP ranges from 8-21 mmHg, however in cases of AACG, typical pressures will be 40-80mmHg.[1-6] The formal diagnosis of AACG is clinical, based on identification of an acutely elevated IOP, corneal edema, symptoms suggestive of AACG, shallow anterior chamber, and a close angle on gonioscopy.[1,7,8,9] Acute angle closure glaucoma can be differentiated from other versions of glaucoma which generally don't have acute symptoms, pain, or sudden vision changes.[7,9] Gonioscopy and chamber measurement is almost exclusively restricted to clinics of eye specialists, therefore, such data is likely not available for UC or ED assessment and medical decision making.

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IOP is regulated through the balance of production and drainage of the aqueous humor. Under normal conditions, aqueous fluid is formed in the ciliary body and drained through the trabecular meshwork.[1] In the setting of AACG, this process is disrupted, and the drainage of aqueous fluid is compromised.[1,9] Any degree of increased IOP can lead to excessive pressure on the optic nerve, which subsequently can lead to ischemia of retinal ganglion cells. In the setting of AACG, there is a rapid rise of IOP, causing rapid onset of retinal ischemia.[9] AACG can occur under multiple circumstances with pupil dilation. As the iris relaxes it impedes the outflow of aqueous humor, resulting in a rapid increase in IOP.[10,11] This phenomenon, termed "pupillary block," can be precipitated by various classes of pharmacologic agents that contribute to dilation of the pupil, most notably alpha-1 and beta-2 receptor agonists, anticholinergic agents, antihistamines, sulfonamides, and serotonergic agents; non-pharmacologic causes include an anteriorly displaced or enlarged lens.[1,3,7,11] Risk factors for AACG include Asian ancestry, female sex, narrow globe shape, farsightedness, older age, and history of AACG in the contralateral eye.[3,7,11] In North America, the incidence of AACG is 1.75 cases per 100,000 person years among those aged >50 years.[12] Comparatively, the incidence in Hong Kong and Singapore is 12.2 per 100,000 person years.[13]

Common symptoms of AACG include periocular pain (83% of cases), visual changes (eg, vision loss/halos; 82% of cases), nausea/vomiting (44% of cases), and headache (34% of cases).[3]

The diagnosis of AACG involves suggestive symptoms and an elevated IOP in the affected eye. On exam, AACG almost universally will present with the findings of a mid-range, fixed pupil in addition to elevated IOP.[3] Other less common findings on exam include corneal edema (swelling of the cornea; 80% of cases) and conjunctival injection (75% of cases).[3] In the absence of tonometry,

a firm globe with "finger palpation" has been shown to be correlated with elevated IOP/glaucoma.[3,7]

In one study of patients with AACG, one-third of patients had not had any contact with an ophthalmologist previously, therefore it is vital for emergency medicine and UC clinicians to have familiarity with diagnostic criteria for AACG.[3] As the vast majority of UC centers do not have access to tonometry, tactile assessment of the firmness of the globes with finger palpation may be the only means of assessing IOP. While relatively few studies have assessed the accuracy of palpation to estimate IOP, existing studies do suggest that this exam technique has some clinical utility. While certainly (and expectedly) less accurate than tonometry measurements, comparing the firmness of the affected globe to the patient's contralateral eye and to a control eye (eg, that of the clinician) can offer a useful point of reference.[3,10,14]

Finger palpation of the globe involves asking the patient to close their eyes and gaze down. Over their closed lids, the examiner then uses both index fingers to palpate the eye with gentle pressure on the upper eyelid using alternating fingers.[10,14] The exam is then repeated on the other eye. If one eye has a markedly increased IOP, it will feel comparatively firm to the touch. [10,14] Finger palpation was evaluated in a 2019 study where first-year ophthalmology residents and attendings were asked to evaluate the IOP of 58 participants by tactile assessment only, which was then compared with actual IOP.[10] In this study, the first-year residents' estimates were only about 50% accurate for the first 15 assessments, but accuracy (ie, within 5mmhg +/- the actual measured IOP value) improved with subsequent testing to 80-100%.[10] This study suggests greater accuracy than prior investigators who have evaluated the palpation technique. However, prior studies do suggest that with experience and at significantly elevated IOP values (ie, >30mmHg), finger palpation estimates can be reasonably accurate. [15,16] Regardless, the best way to measure IOP is with a tonometry device. In cases of reasonable suspicion for AACG, patients should be immediately referred to a clinic or ED with tonometry capabilities.

Case Presentation

A 60-year-old woman presented to the ED with a chief complaint of dental pain. She reported severe left upper dental pain that radiated to her left eye, which began when she awoke 12 hours earlier. Her past medical history included open angle glaucoma, peripheral arterial disease, and prior surgical removal of cataracts. Additionally, she reported nausea and vomiting as well as blurry vision in the left eye since the pain began. She had previously been prescribed several eye drops for glaucoma and admitted occasionally not remembering to use them as directed.

Figure 1.

"This case highlights the importance of considering AACG in the differential diagnosis for undifferentiated headache and facial pain, especially in patients with risk factors, such as a history of glaucoma and/or ocular surgery."

Physical Exam Findings

At triage in the ED, her initial vitals were: blood pressure of 179/92 mmHg; heart rate of 78 beats per minute; respiratory rate of 18 per minute; temperature of 36.5°C. Her visual acuity was 20/200 in the left eye (OS), and 20/30 in the right eye (OD). On clinical exam by the triage clinician, she appeared to be in pain. Her head and neck exam revealed chronic dental caries, but no trismus, facial swelling, or other apparent acute findings. Her neck was supple with normal range of motion. Her ocular exam revealed a round, 3mm, pupil that was briskly reactive on the right. The pupil of her OS was mid-range and unreactive to light. A screening visual field exam revealed no deficits. The left cornea appeared somewhat opaque (ie, "steamy"), and there was con-

Table 1: Medications For Acute Angle Closure Glaucoma Treatment[21-23]

Class	Names	Mechanism	Potential Side Effects
Beta-Blockers	timolol, metipranolol, carteolol (topical)	Lower aqueous humor production	Chronic obstructive pulmonary disease exacerbation, bronchospasm, bradycardia, heart block
Carbonic Anhydrase Inhibitor	acetazolamide, methazolamide, ethoxzolamide (systemic)dorzolamide and brinzolamide (topical)	Lower aqueous humor production	Pain and burning (topical)
Prostaglandins	latanoprost, travoprost, bimatoprost, urnoprostone, tafluprost (topical)	Enhance outflow of aqueous humor	Ocular discomfort, hyperpigmentation of eyelids, uveitis
Alpha-Agonists	brimonidine, apraclonidine (topical)	Lower aqueous humor production	Systemic hypotension with apraclonidine, allergic blepharoconjunctivitis, dry mouth and nose with brimonidine
Hyperosmotic Agents	mannitol, isosorbide (systemic)	Lower aqueous fluid volume by osmotic effect	Electrolyte and fluid imbalance, diuresis, peripheral edema, dehydration, hypotension, tachycardia

junctival injection (**Figure 1**). Finger palpation of both eyes was performed, which demonstrated a relatively firm left globe compared to the right. Topical anesthetic drops were applied without pain relief, and the IOP was measured with a Tono-Pen tonometer bilaterally. The IOP of OD was normal (16mmHg), however, the IOP of OS was markedly elevated at 66mmHg. Fluorescein staining revealed no corneal defects bilaterally. A screening neurologic exam, including extraocular movements (EOM), was unremarkable.

Differential Diagnosis and Medical Decision Making

The differential diagnosis for the patient's presentation considered included: temporal/giant cell arteritis, migraine, odontogenic infection, maxillary sinusitis, cavernous sinus thrombosis, ocular infection/endophthalmitis, invasive fungal sinus infection, ocular trauma, uveitis, scleritis or episcleritis, keratitis, dental pain, corneal abrasion, or ulcer. However, given the constellation of symptoms, exam findings, and markedly elevated IOP, the leading diagnosis of concern was AACG. Additionally, while the patient complained of dental pain, there were no exam findings to suggest the pain was odontogenic in nature. Based on this initial clinician assessment from triage, the patient was quickly moved from the waiting room to a bed in the ED.

Case Continuation and Resolution

The patient was treated symptomatically with intravenous analgesia and antiemetics. To mitigate her presumed acutely elevated IOP, she was treated with acetazolamide, dorzolamide-timolol, latanoprost, and brimonidine ophthalmic drops. The treating clinician consulted with the on-call ophthalmologist who recommended hourly administration of the ophthalmic drops until the pressure had normalized and symptoms were controlled. Over the subsequent 4 hours, all of the above ophthalmic medications were administered, and the ophthalmologist came to the ED to assist at the bedside. Despite these measures, the patient achieved minimal reduction in her OS IOP. Given recalcitrant elevation in pressure, the consulting ophthalmologist elected to proceed with laser iridotomy. After the iridotomy, the patient's IOP improved to 37 mmHg with corresponding improvement in her facial pain and nausea symptoms. She was discharged after the procedure with plans for repeat evaluation and pressure check in the ophthalmology clinic the next morning.

When the patient was seen the following day, her IOP had continued to decline to 8 mmHg. She underwent repeat laser iridotomy at that time and was then referred back to her glaucoma specialist for follow-up. Her visual acuity had returned to baseline at her last known ophthalmology follow-up visit.

Table 2: Medications That Can Precipitate Acute Angle Closure Glaucoma[2]

Class	Medication Examples	Mechanism
Alpha-1 Adrenergic Agents	phenylephrine, ephedrine	Pupil dilation/pupillary block
Beta-2 Adrenergic Agents	albuterol, salbutamol	Pupil dilation/pupillary block
Anticholinergics	atropine, glycopyrrolate, scopolamine	Pupil dilation/pupillary block
Antihistamines	cetirizine	Pupil dilation/pupillary block
Cholinergic Agents	pilocarpine	Anterior lens displacement
Sulfonamides	topiramate, acetazolamide	Anterior lens displacement
Serotonergic Agents	venlafaxine, escitalopram, triptans, aripiprazole	Pupil dilation/pupillary block

Discussion

While pain is a common feature in AACG, reporting pain associated with this condition as jaw or dental pain has not been previously described in the literature. This case highlights the importance of considering AACG in the differential diagnosis for undifferentiated headache and facial pain, especially in patients with risk factors, such as a history of glaucoma and/or ocular surgery. One possible explanation could be that the shared somatic innervation of the facial structures through the trigeminal nerve (ie, the fifth cranial nerve) may be responsible for this sort of atypical presentation. As headache is a highly non-specific symptom and AACG is a highly time-sensitive, vision threatening diagnosis, it is critical to consider AACG in other forms of cephalgia beyond eye pain.[17] In the absence of immediately available tonometry, risk stratification after consideration of AACG is the most crucial task for the UC clinician. As there are currently no clinical decision rules for AACG risk stratification, this assessment of pretest (ie, tonometry) probability relies on clinician gestalt. This gestalt, furthermore, relies on clinician understanding of signs, symptoms, and risk factors of AACG. As previously noted, patients of Asian ethnic descent, women, and those aged >50 years are at the greatest risk. Additionally, patients with history of hyperopia (farsightedness), and/or glaucoma are also at increased risk.[3,7,11,17] Certainly, medication non-adherence was a factor in this case, however, it is important to consider pharmacologic precipitants specifically in cases of suspected AACG. Medications that can trigger AACG include agents that cause pupillary dilation and lens position AACG (**Table 2**).[2]

In UC settings without access to tonometry, finger palpation can be used to offer additional data as to the likelihood of clinically significant IOP elevation.[10] Studies on the accuracy of finger palpation estimates of IOP are limited and have been conducted exclusively among patients cared for by eye specialists, therefore, it is not advisable for UC clinicians to rely on palpation as a surrogate for tonometry, especially in cases with suggestive symptoms and/or concerning findings on ocular exam.[15,16] In cases with moderate-high clinical suspicion for AACG where tonometry is not available to the UC clinician, immediate ED or eye clinic evaluation is indicated.

"Prompt lowering of IOP is essential to mitigate the risk and degree of permanent vision loss."

Emergency management in settings with access to the pharmacologic agents in **Table 1** involves ophthalmologist consultation to guide medicine administration until pressures are controlled, ideally within the range of 22-24 mmHg and symptoms resolve.[7,8,11,13] Prompt lowering of IOP is essential to mitigate the risk and degree of permanent vision loss.[6,7] If this can be achieved, urgent/next day follow-up is reasonable. However, medical management may not be definitive in the acute setting in many cases, and emergent surgical therapy may be required (as was the case with the patient presented).[7] Hyperosmotic agents like mannitol can also be used for refractory cases, although cardiovascular side effects may occur.[13]

Conceptually, it is important to understand that medications serve only as a bridge until definitive surgical management. Iridotomy is the most efficacious inter-

vention for AACG. The combination of medical therapy and laser iridotomy has 86-99% efficacy in reducing IOP and preserving retinal perfusion; laser iridotomy also has the advantage of being safe and generally well tolerated.[1,13,19,20] Additional procedures that may be implemented include needle aspiration (ie, anterior chamber paracentesis), incisional iridectomy, phacoemulsification, and cyclophotocoagulation.[20] Ophthalmologists may also recommend other procedures to prevent future attacks including performing a prophylactic iridotomy in the contralateral eye.[20] After adequate reduction of IOP, patients require regular follow-up at an appropriate ocular specialist to ensure the IOP remains in a safe range and vision remains unaffected.[20]

Takeaway Points

- AACG is an ocular emergency, and prompt recognition and treatment of elevated IOP is critical to prevent permanent vision loss.
- Consider AACG in patients presenting with headache or other forms of cephalgia, especially if accompanied by vision changes, nausea, and vomiting.
- Characteristic physical exam findings suggestive of AACG include mid-range, non-reactive pupil and conjunctival injection.
- In the absence of tonometry, finger palpation of both globes can provide further objective data of elevated IOP. However, the accuracy of finger palpation estimations of IOP has not been assessed in UC, and clinicians should not rely on apparently normal globe palpation to obviate concerns for elevated IOP in cases of moderate-high clinical suspicion.
- In cases of confirmed or suspected AACG, immediate eye specialist or ED evaluation is indicated.
- Medical management of elevated IOP is generally not definitive, and many patients require urgent, if not emergent, surgical intervention. ■

Ethics Statement

The patient provided verbal consent for the case and image to be published.

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