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10 Years of Chronic Intermittent Angle Closure Glaucoma Masquerading as Migraine and Cervicogenic Headaches

Craig J. Helm MD*[1]

[1] Helm Vision Group, Valencia, California

*** Corresponding Author:** Craig J. Helm MD
Address: 27420 Tourney Rd., Suite 100, Valencia California 91355 USA
Email: craighelm@sbcglobal.net

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Abstract

Purpose: To present an unusual case of intermittent angle closure glaucoma masquerading as migraine and cervicogenic headaches.

Methods: A patient with a 10-year history of chronic headaches presented with a 5-day history of pain and blurred vision in the right eye. She was found to have acute angle closure glaucoma. She was treated with intraocular pressure-lowering agents, followed by laser iridotomy. The patient provided copies of her medical visits between 2010 and 2016. These records were carefully reviewed and summarized.

Results: Treatment with laser iridotomy resolved symptoms of ocular pain and headaches that the patient had been suffering from for over a decade. During that period, she was treated with a variety of medications, including anti-migraine drugs, non-steroidal anti-inflammatory medications, muscle relaxants, and narcotic analgesics. She was also treated with chiropractic manipulations, physical therapy, cervical epidural steroid injections, and botulinum toxin injections.

Conclusion: This is a rare case where symptoms presumably caused by intermittent angle closure persisted for over 10 years before progressing to acute angle closure crisis (complete angle closure). The presence of documented cervical spine disease complicated making a definitive diagnosis. The presence of unilateral blurred vision with headaches should prompt investigating possible intermittent angle closure as the cause, even in a patient whose symptoms have been present for as long as ten years.

Introduction

Headache is a common reason for patients to seek medical care. Sometimes the source of pain can be elusive, and the pain can be chronic and debilitating. The following case report shows an unusual 10-year case of intermittent angle closure glaucoma causing headaches masquerading as migraines or cervicogenic headaches.

Case Report

A 50-year-old woman presented on April 25, 2017 with a 5-day history of right eye pain, tearing, nausea, and difficulty sleeping. Her past medical history was significant for chronic, severe, intermittent headaches for the previous 11 years.

She first developed neck pain caused by whiplash from an auto accident on December 25, 2004. She underwent physical therapy, chiropractic manipulations, and two cervical epidural steroid injections in 2005. Her headaches began in 2006, at age 39, and she was diagnosed with migraine. The episodes initially occurred only 2 to 3 times a year, and increased steadily until age 45, when she was experiencing them 2 to 3 times each week. The headaches were commonly triggered by neck flexion while looking down, reading for long periods, or even looking for food in a dark pantry. The pain was severe (9 or 10 out of 10), exclusively right-sided, accompanied by right-eyed blurred vision, photophobia, and always occurred in the evening. She described feeling intense pain behind the right eye, along with a stabbing pain radiating to the right side of her head and down the right side of her neck. Sometimes, she would have neck pain without headaches. The neck pain, exacerbated by head-turning and neck flexion, would radiate to the occipital region of her head, into her shoulders, and, at times, would be accompanied by numbness and tingling in her arms. Frequently, the neck pain would start first and seem to trigger the headaches. At other times, the headache would come first and then trigger the neck pain.

She was referred to a neurologist in 2010 for evaluation and treatment of her headaches. One of the medications tried was topiramate. She used this drug for about a year before discontinuing it because of "cognitive side effects". It neither prevented nor exacerbated the frequency or severity of her headaches. An optometrist saw her in March of 2011, when she was asymptomatic. She was hyperopic, but otherwise normal. Her intraocular pressure was found to be normal at 18 mmHg in each eye.

She tried various oral non-steroidal anti-inflammatory drugs and other pain medications with little to no relief. By 2011, she had undergone further chiropractic manipulations while lying prone, which not only failed to relieve her pain but induced headaches.

Her neck pain began to worsen and seemed to trigger her headaches. A cervical spine MRI done in March of 2011 showed degenerative changes and borderline disc herniation, with significant canal narrowing at the C6-C7 level, leading to a diagnosis of cervicalgia.

A neurosurgeon first evaluated her in May of 2011 and felt that the headaches were cervical spine in origin. He diagnosed her with cervical stenosis and cervical myofascial pain syndrome and felt that the principal migraine trigger was her cervical spine pathology. Although a 2-level cervical microdiscectomy and fusion at C4-C5 and C6-C7 was considered, a lack of radiculopathy prompted conservative management. Physical therapy was prescribed

along with the use of a soft cervical collar, which she used for a year. Struggling with the painful headaches, she used hydrocodone and oxycodone. Physical therapy failed to give her any relief.

A cervical epidural steroid injection in November of 2011 gave her relief from the neck pain, as well as the headaches, for about 2 months. A nerve conduction study performed in November 2011 was normal.

In April of 2012, the neurosurgeon found her range of cervical motion was limited and painful in all directions. Still not having radicular symptoms, he recommended against surgery. She was prescribed rizatriptan for migraine, a muscle relaxant, and hydrocodone for pain. A repeat cervical MRI in June of 2012 appeared to show progression of the cervical stenosis. She was referred to Stanford University Medical Center for a second opinion.

In June of 2012, a pain management specialist diagnosed her with cervical pain syndrome and occipital neuralgia. On August 1, 2012, another cervical epidural steroid injection was performed, which temporarily improved her neck pain and decreased the frequency of her headaches.

While asymptomatic, she was seen again by her optometrist in October 2012. Her intraocular pressure was found to be normal.

She underwent another epidural steroid injection into the cervical spine at the C3-C4 level on November 19, 2012, which temporarily relieved her neck pain but not her headaches this time.

The neurosurgeon at Stanford University Medical Center documented her complaints of headaches, accompanied by vision changes, occurring 2 to 3 times a week. She continued to note that flexing her head forward to read for long periods triggered the headaches and that relief only came with sleep. The headaches were exclusively right-sided and only occurred in the evening. He diagnosed her with cervicalgia and recommended non-surgical management, including physical therapy, lifestyle modification, chiropractic manipulation, massage, and stretching. A physiatry consultation was recommended to explore the options of injections, including epidural steroids, trigger point blocks, and facet blocks. Lastly, he recommended meditation.

In December 2012, she was started on morphine for pain relief. This helped her by relieving enough pain to allow her to sleep. The pain was always gone when she awoke. She was also prescribed zolpidem (Ambien®) and referred back to the neurosurgeon for cervical decompression, feeling that she had failed conservative management.

Her regular neurosurgeon found her still complaining of headaches accompanied by right-sided blurred vision and remained unconvinced that surgery was necessary. He then referred her to another neurologist to explore other medications for migraine prophylaxis. At different times throughout 2013, she was prescribed tramadol, memantine, gabapentin, and carisoprodol to alleviate her pain. She was also seen by a therapist for stress management.

In May of 2013, she was seen by another neurologist for migraine treatment. She was treated with a Botox® protocol, which included chemo denervation of 13 sites of muscles innervated by the facial nerve, 10 sites around the head, and 4 motor points in the neck and shoulders. At a follow-up visit 2 weeks later, she reported that the botulinum toxin injections had reduced the frequency and severity of her headaches. By mid-August of 2013, she reported that she hadn't had a migraine for over two months, although she did have

headaches associated with a sinus infection. She continued to take memantine and sumatriptan. The headaches did return, however, and the botulinum toxin injections using the same protocol were repeated three months later at the end of August 2013, and again on November 22, 2013. She reported that the severity of the headaches was decreased by about 50%.

She referred herself to an otolaryngologist in October of 2013 for help with chronic bilateral nasal obstruction and snoring. She had suffered a traumatic injury to the nose at age 15, which was never treated. A maxillofacial CT scan found moderate sinus fluid and nasal septum deviation. She had a long history of allergic rhinitis. She was informed that surgery might improve her migraines, and she underwent septorhinoplasty on December 23, 2013. Unfortunately, beginning a week after surgery, she suffered approximately 65 days of daily headaches, like what she had previously experienced.

She continued to take hydrocodone for pain relief throughout 2013. She felt that she was developing a tolerance to hydrocodone between 2011 and 2013, but nothing else had provided her with pain relief. She then substituted opioids for drinking alcohol more regularly at night to help her fall asleep. At different times, she was also prescribed tramadol, memantine, gabapentin, and carisoprodol to try to alleviate her pain. Her previous neurologist retired, and she saw a different neurologist on February 19, 2014, complaining of moderate to severe, throbbing unilateral headaches with nausea and light sensitivity. The headaches lasted for hours and happened several times a week. She reported right-sided blurry vision and right eye pain. On February 24, 2014, she underwent a botulinum treatment protocol using injections into 31 sites of the face, head, and neck. Six weeks later, her symptoms had significantly improved. She continued to use oral diclofenac for pain, but reported that she had tried all triptans on the market and that none of them had helped.

She saw her optometrist again in June of 2014, while asymptomatic. She was again found to have normal intraocular pressure and noted to have deep anterior chambers.

Botulinum toxin injections had proven to be the most effective means of pain relief to date. She reported an overall 50% improvement in migraine frequency and severity since starting botulinum toxin therapy. Zolmitriptan nasal spray was tried, but it did not provide additional relief. Using the same protocol, her neurologist administered botulinum toxin injections in May, August, and October of 2014, using up to 155 units per session. Regular botulinum toxin injections were administered every three months throughout 2015 and 2016.

In 2017, she moved to Southern California. She had been experiencing headaches 2 to 3 times per week since January and had been using oral naproxen to treat them with marginal success. The severity of the headaches was increasing. Previously, the headache pain was gone when she awoke in the morning. Now, she was waking up with residual pain, and she was also beginning to experience a gritty foreign body sensation in her right eye when the headaches occurred.

On April 20, 2017, she awoke with her worst headache to date and, for the first time, had blurry vision in her right eye in the morning. She saw a general practitioner who diagnosed her with conjunctivitis and treated her with gentamicin ointment. She discontinued the ointment after 2 days because the pain had improved. The pain returned 2 days later, and she then presented to our clinic on April 25, 2017. On examination, her visual acuity was counting fingers at 2 feet in the right eye and 20/20 with correction in the left eye. The right

eye was markedly injected. Slit lamp examination showed diffuse microcystic edema of the right cornea. The right anterior chamber angle was shallow. Gonioscopic examination showed the anterior chamber angle of the right eye to be completely closed for 360 degrees, while the left eye showed narrow, but not closed angles. The intraocular pressure measured 66 mmHg in the right eye and 20 mmHg in the left eye.

She was treated medically with pressure-lowering drops and oral acetazolamide. After 2 hours of observation, a paracentesis was performed, which brought her pressure down to 17 mmHg. Her cornea was then clear enough to perform a laser peripheral iridotomy in the right eye. One week later, a prophylactic laser iridotomy was performed in the left eye.

Her headaches resolved immediately after the laser iridotomy was performed on the right eye. She was followed regularly in our office over the next 5 years. She never suffered any recurrence of the headaches. Although her neck pain continued to wax and wane, the headaches were gone. The patient relocated to another state in 2022. In a phone call in February 2026, she reported that she continues to be headache-free. The only medication she uses for neck pain is naproxen.

Discussion

Acute angle closure manifests as a rapid onset of severe unilateral eye pain or as a headache associated with blurred vision. [1, 2] The visual disturbance may include rainbow-colored halos around light sources. Nausea and vomiting are also common.[3] Because of this, many patients are unaware that their eye is the source of the problem. When presenting to an urgent care or emergency department, these symptoms can be misinterpreted as being neurological in origin, leading to unnecessary cranial imaging and neurology consultation before considering an ophthalmology consultation.[4]

Intermittent angle-closure is challenging to diagnose because the intraocular pressure and vision may be normal between episodes. The symptoms of intermittent angle closure are those of acute angle closure but typically remit after a few hours, often resolving with sleep.

Because the symptoms may not be present when the patient is being examined, the physician should inquire about particular symptoms or risk factors that may raise suspicion of intermittent angle closure in headache patients. The occurrence of headaches in low light, such as at dusk, is characteristic of angle closure. Our patient noted that searching for items in her dark pantry was a headache trigger.

Hyperopia is a risk factor for angle closure.[5] Hyperopic eyes are generally shorter than average and associated with a shallower anterior chamber.[6] Our patient was moderately hyperopic and had shallow anterior chambers, as proven with optical biometry. These eyes become more likely to develop angle closure as the crystalline lens thickens with age, narrowing the anterior chamber angle.[7] Pupillary block occurs when aqueous production from the ciliary body, coincident with decreased flow through the trabecular meshwork, pushes the iris forward, blocking the trabecular meshwork.[8] This can lead to a downward spiral of increased forward bowing of the iris (bombe), further blockage of the trabecular meshwork, and increased intraocular pressure. This can lead to the complete closure of the anterior chamber angle, sending the intraocular pressure up into a sight-threatening range.

Interestingly, our patient was treated for about a year with topiramate for migraine prophylaxis. Topiramate is a well-known, but rare, cause of acute angle closure.[9] The mechanism of angle

closure is that topiramate-induced congestion of the ciliary body causes it to rotate anteriorly, pushing the peripheral iris against the trabecular meshwork. Our patient does not recall that it improved or exacerbated her headache frequency. She discontinued topiramate because of non-ocular side effects.

Lying prone can increase the risk of angle closure by causing the lens to move forward, pushing the peripheral iris into contact with the trabecular meshwork. Our patient reported that chiropractic manipulations, when she was positioned prone, exacerbated her headaches. She also noted early on that prolonged reading, while flexing her head forward, brought on the headaches. Reading can also pose a risk because the crystalline lens moves anteriorly when accommodation is activated for near-vision tasks. This can push the iris forward and potentially close a shallow anterior chamber angle.

The time course for progressing from symptomatic intermittent angle closure to complete angle closure is not known but thought to be weeks to months. A large prospective study of asymptomatic primary angle closure suspects (those having narrow angles) showed them to have a less than 1% per year risk of developing angle closure glaucoma over the 6-year study period.[10] Our case appears unusual in that the symptoms persisted for so long. Her records show that for at least 7 years before presenting to us with angle closure, she was consistently complaining of evening-only headaches with exclusively right-sided blurred vision. It wasn't until 2017 that the headaches began to linger into the following morning. At no time was she referred to an ophthalmologist. It is reasonable to assume that angle closure would have been diagnosed had she been seen when she was symptomatic. In such a patient, current ophthalmology practice guidelines recommend prophylactic peripheral laser iridotomies when patients have anatomically narrow anterior chamber angles, along with symptoms of intermittent headaches and blurred vision.[11]

The diagnosis in our patient was particularly challenging in the presence of well-documented cervical spine disease. Cervicogenic headaches are common and can be chronic and recurrent.[12]

Headaches are a common reason for patients being referred to an ophthalmologist. Among the most common ocular causes for headaches are uncorrected refractive error, accommodative insufficiency, accommodative spasm, eye muscle imbalance, and intermittent angle closure glaucoma.[13] The headache associated with angle closure is well-documented and listed in the International Classification of Headache Disorders as one of 4 ocular causes of headaches.[14]

In headache sufferers, the ophthalmologist should inquire about symptoms of intermittent angle closure. In addition to measuring intraocular pressure, gonioscopy should be performed to examine the anterior chamber angle for narrowness and the presence of peripheral iris anterior synechiae, which would suggest previous angle closure. Ocular coherence tomography (OCT) imaging is very useful since the software allows the measurement of the chamber angle to be quantified and followed for progression.

When intermittent angle closure is suspected, a provocative dilation test or prone positioning test may be performed in the office to try to elicit an angle closure attack and make a definitive diagnosis. Placing the patient in a prone position in a dark room for 30 minutes may cause symptomatic angle closure in susceptible individuals.

In approximately 25% to 30% of patients with migraine, the migraine attacks are preceded by

an aura.[15] Over 90% of migraine auras are visual. The visual aura of migraine is characteristically bilateral. This is different from a retinal migraine, a rare condition in which the transient visual symptoms are unilateral.[16] A retinal or ocular "migraine" is now thought to be a misnomer because the pathophysiology is believed to be related to vasospasm. In contrast, the visual symptoms in migraines are no longer believed to be secondary to vasospasm, but to a complex series of events triggered in the hypothalamus.[17, 18] These events lead to cortical spreading depression beginning in the occipital cortex. Our patient complained of unilateral blurred vision accompanying her headache, a condition that should prompt an ophthalmologic consultation. An optometric evaluation is generally insufficient to rule out the diagnosis.

This report discusses a rare case where intermittent angle closure produced chronic recurrent symptoms of headache, eye pain, and blurred vision, over a period of at least 10 years before progression to complete angle closure led to definitive diagnosis and treatment. Although angle closure may have become the main source of her headaches sometime after she began to experience them, the medical records document unilateral right-sided blurred vision accompanied by right-sided headaches for at least seven years. Her symptoms, headache triggers, narrow angles, and complete resolution of headaches for almost 6 years after undergoing treatment with a laser iridotomy, strongly support the disease timeline in this case. In addition, the patient is very familiar with the nature of her headaches, which greatly impacted her life, and states that she suffered the same symptoms from the outset. The duration of her symptoms, lack of redness in the right eye, and co-existing severe neck pathology likely contributed to the delayed diagnosis.

The presence of unilateral blurred vision coincident with headache should prompt an investigation for intermittent angle closure glaucoma as the possible cause, even when symptoms have been present for many years.

Declarations/Disclosures

Ethics approval: The study adhered to the ethical principles outlined in the Declaration of Helsinki.

Consent: Consent to publish this case report has been obtained from the patient in writing.

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Other relationships: Author have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

Authors' contributions: All research and writing were performed by the author (CJH).

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