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Case Report

Self-Ocular Compression Maneuver Immediately Relieves Migraine Headache Attacks: Case Report of Managing 100 Attacks Over 9 Years and a Mechanistic Review

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Abstract

The pathophysiologic basis of migraine is uncertain. Abortive management strategies for acute migraine attacks are, therefore, based on presumptive approaches. Widespread use of triptans or analgesics either alone or in combination with caffeine to abort episodic migraine headaches does not allow for precise knowledge of pharmacologic site or mechanism of action. Ocular compression maneuver (OCM) has been previously reported to manage primary cough headache by immediate reduction of Valsalva-induced corneoscleral envelope distention/undulation and nociceptive ocular ophthalmic (V1) trigeminal neural traffic activation. I report the successful and safe use of OCM induced painless self-ocular tamponade through closed eyelid(s) to abort recurrent episodic migraine headache attacks and post-coital headache attacks. The scientific basis for ocular origin of migraine headache has been previously elucidated. The mechanistic link between ocular autonomic nervous system (ANS) dysfunction, choroidal overperfusion/intraocular pressure (IOP) elevation, and nociceptive corneoscleral distention/undulation in migraine headache and primary headache with sex/post-coital headache (PCH), and, their abortive management with the self-OCM is presented for the first time in migraine literature.

Keywords: Migraine; Primary Headache Associated with Cough; Primary Headache Associated with Sex; Self-ocular Compression Maneuver; Ocular Tamponade; Choroidal Vascular Circulation; Intraocular Pressure; Ocular Autonomic Nervous System; Digital Ocular Massage

Abbreviations

ANS: Autonomic Nervous System; IOP: Intraocular Pressure; BCH: Benign Cough Headache; PCH: Post-Coital Headache; OCM: Ocular Compression Manœuvre; V1: Ophthalmic Division of Trigeminal Nerve; MWA: Migraine Without Aura; CBF: Chroidal Blood Flow; POAG: Primary Open Angle Glaucoma; NTG: Normal Tension Glaucoma; DOM: Digital Ocular Massage

Introduction

Migraine is a primary common recurrent characteristically lateralizing (unilateral, bilateral, side-shifting or side-locked) and self-limited (4 - 72 hours) headache disorder with a poorly-defined genetic predisposition [1,2]. With a substantial economic impact, and, with significant absenteeism from school as well as work, migraine is regarded as a social stigma [2-4]. Currently, the pathophysiologic basis of migraine attacks is poorly understood and, consequently, all pharmacotherapeutic and non-pharmacotherapeutic management strategies are based on suggestive theories, serendipity or empirical principles [5,6]. Certain well-established migraine preventive drugs, such as atenolol and nadolol, do not cross the blood-brain barrier and cannot influence brain neuronal function [5,6]. Beginning with the chance "discovery" of cortical spreading

depression in rodents in the mid-twentieth century, a number of brain/brainstem neuronal/thalamic neurovascular theories have gained ascendency [7-9] without generating a progressively robust biologically-plausible and generalizable overarching matrix [5,6,10] Besides other critical physiologic and pharmacologic limitations of brain/brainstem theories of genesis of migraine [5,6,10], the single most important phenotypic limitation is their inability to rationalize the typical lateralization of headache to ophthalmic division of the trigeminal nerve (V1) [10,11]. Additionally, why such spontaneous brain neuronal oscillations should arise particularly in females, in the first instance, and start at puberty/adolescence or at menstruation or following sleep-related arousal or alcohol imbibition, and, subside with pregnancy (second and third trimester) or sleep or menopause or advancing age remains unknown [5,6,10] Non-lateralizing fMRI brain neuroimaging changes recorded after onset of headache are very unlikely to represent primary pathogenetic changes in migraine [5,6,10] No systemic influence or non-localizing/-lateralizing diffuse brain neuronal influence outside the V1 cortical/subcortical brain region can explain the pathophysiologic basis of migraine [5,6,12,13]. Intriguingly, following enucleation of one or both eyes, typical paracentral horse-shoe-shaped expanding positive scintillating scotoma (SS) and characteristic migraine headache are both not reported [14-17]. Sightlessness markedly attenuates migrainous SS as well as photophobia [18].

Drummond reported manual compression of the common carotid artery (CCA) as an effective, albeit risky, maneuver to "identify" and to "manage" migraine headache [19]. Drummond also reported on manual compression of the superficial temporal artery to occlude its blood flow for migraine headache relief [19]. Yamada., *et al*. reported self-compression of the CCA to attempt management of throbbing temporal headache due to carotid-cavernous fistula [20]. Zanchin., *et al*. reported momentary relief with self-administered pain-relieving maneuvers mainly on the temple and forehead by 46% of 400 patients [21]. Sprouse-Blum., *et al*. found that targeting the carotid arteries at the neck with the application of frozen wrap at onset of migraine headache significantly reduced pain of headache [22]. Vascular cooling as well as a triptan-like vasoconstrictive effect based on a different mechanism might be involved [22]. Cranial vessels, however, do not seem to be a primary source of pain in migraine [19].

The theoretical basis for ocular origin of primary headache with cough/benign cough-induced headache (BCH) has been presented earlier [23]. Use of the ocular compression maneuver (OCM) to abort BCH by decongesting manual ocular tamponade was also described more than a decade previously [24]. The role of gut-generated aberrant ocular hemodynamics in the origin of migraine attacks has been detailed [25]. The precise link in physiological imbalance(s) between BCH, gut-originating oculo-cranial aberrant hemodynamics through the Valsalva manœuvre, PCH, and migraine is, however, unknown.

In this case report, I describe the effect of recurrent brief manual-ocular self-compression induced tamponade in a male migraineur to rapidly abort 100 attacks of acute migraine without aura (MWA) headache over a period of 3½ years from May 2012 to November 2015. Self-OCM technique can involve a few cycles of externally applied pressure to the ocular globe for up to 10-20 seconds without generating or worsening ocular pain, followed by brief (up to 20-30 seconds) intervals without pressure application. I also present a unifying hypothesis for several variants of primary headache currently classified as distinct entities.

Case Report

I present the case of a migraineur, male, 58 years old when seen first. As defined by the International Headache Society (IHS) [1], the patient had been experiencing recurrent moderate-to-severe right-sided MWA headache attacks since over last two decades. The patient would develop 2 - 4 attacks of MWA headache every month in response to a variety of known triggers, despite using different preventive medications. Triptans were not effective for his migraine attacks, and, he had to use proprietary analgesics to get pain relief. Propranolol, amitriptyline, and their combination was not effective in preventing MWA attacks. In 2005, I had described the OCM to abort BCH [24]. Since this patient also experienced BCH besides MWA headache, in May 2012, I decided to instruct him to use OCM through closed eyelids on himself whenever he had migraine attacks. He was advised to be careful with each OCM, not to induce ocular pain, not to apply OCM to both eyes simultaneously, not to apply pressure for more than 20 seconds, and to do mental counting for duration in seconds.

In June 2012, on awakening from sleep one morning, the patient developed a severe (9/10 on Visual Acuity Scale) throbbing right-sided non-pulsatile MWA headache attack. Fifteen minutes into the headache, he took a proprietary combination of ibuprofen

400 mg and paracetamol 500 mg. Half-hour later, with the headache worsening, he applied the OCM by applying pressure with the thenar eminence to his right eye through closed eyelids. To his surprise, his MWA headache disappeared almost instantaneously with the first application of self-OCM for 10 seconds. He was more surprised when the MWA headache did not reappear after cessation of self-OCM, in effect completely aborting the severe attack. Since then, he has applied self-OCM regularly to his MWA headache attacks. Using his own Smartphone-app, he kept record of the first 100 moderate-to-severe right-sided MWA headache attacks (with severity 7-10/10) between June 2012 and November 2015. While he could completely abort 77 MWA headache attacks (15 bilateral and 62 right-sided) in few seconds (usually, less than 10s), migraine attacks that were well-developed but non-throbbing upon awakening from sleep (12 in number) or had a more subtle onset at any other time (11 in number) did not respond or responded only partially to self-OCM. Pulsatile or throbbing migrainous headache attacks (20 attacks out of 100) invariably responded to self-OCM in 2 - 5 seconds. Before and after every self-OCM, he was advised to check his vision for distant objects and close reading. He was warned never to apply the OCM to his own eye continuously for more than 10-20 seconds by mental count of time or to generate or worsen ocular/neck pain.

At the age of 60 years, in May 2014, he developed his first typical attack of primary headache associated with sexual activity/post-coital headache (PCH), as detailed by the IHS [1]. He would abruptly develop sharp bilateral headache just before or after orgasm, diagnostic criteria, C.2. The headache would last for 20-30 minutes. Triptans and analgesics, alone or in combination, taken 45-60 minutes before sexual intercourse were of no avail. Allowing his partner to mount pre-orgasm did not offer relief, either. After suffering 8 more attacks of post-coital headache over the next six-months, he gave up sexual intercourse for a year. In early January 2015, with severe burning non-pulsatile bilateral frontal head pain after orgasm, without my counselling, he desperately applied the self-OCM to both of his eyes while lying prone over his partner slipping both of his elbows onto her supine body. The PCH was immediately decreased by >50% with complete relief in a few minutes. Similar or better relief has been observed in subsequent attacks of PCH as he learnt to position himself better in the prone position in order to apply the self-OCM more effectively.

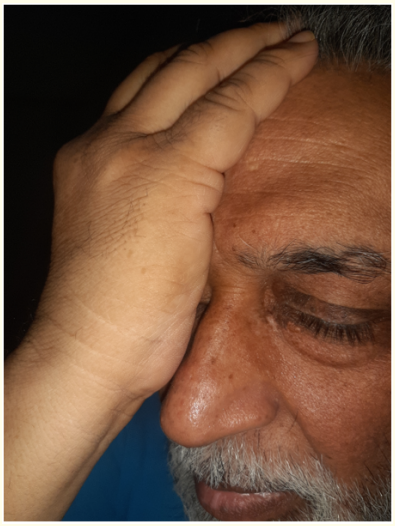

Figure: Self-ocular compression maneuver for immediate cessation of migraine attacks

Discussion

In 2006, a biologically-plausible and logically-robust hypothesis for pathogenesis of migraine with a single central assumption of idiopathic primary autonomic nervous system (ANS) ocular insufficiency was put forward, presenting the role of the eye(s) as the key target organ with selective nociceptive activation of V1 [6]. Fibres from the V1 alone or predominantly are believed to also reach the spinal trigeminal centre. Recently, selective involvement of the V1 division of the trigeminal nerve in migraine headache attacks has been further elucidated [6,10,11]. The pathophysiology of migraine is uncertain, and, its therapy (both abortive as well as preventive) is multimodal and uncertain, both interlinked key features being devoid of established scientific principles. The pathogenesis of PCH is also unknown, and, its management is equally unsatisfactory. Involvement of intraocular hemodynamics was suggested in BCH in 2004 [23].

The eye receives its arterial blood supply from the ophthalmic branch of the external carotid artery (and central retinal artery branch of the ophthalmic artery). Ophthalmic veins (vortex veins) and a central retinal vein drain blood from the eye, emptying into the external jugular vein. Per unit weight, the ocular choroid is the most vascular structure of the body, with a vascular circula-

tion 10-20 times that of the cerebral cortex. Blood enters the thin erectile choroid through the short posterior ciliary arteries and is distributed in three layers: Haller's, Sattler's, and choriocapillaris [26]. Arterial blood flows consecutively through each of these layers and is then collected by venules. The venous choroidal blood drains through the superior orbital vein into the intracranial cavernous sinus. The human choroid is thickest (up to 1 mm) at the far extreme rear of the eye, while in the outlying areas it narrows to 0.1 mm. The choroid provides oxygen and nourishment to the outer layers of the retina. Along with the ciliary body and iris, the choroid forms the uveal tract. Branches of the ciliary nerve, that supply both elements of the ANS to the iris and the ciliary body, also supply the choroid [26-28]. Free nerve bundles and perivascular fibres around all choroidal blood vessels are seen, both originating from the ciliary nerves. In humans, the largest number of variables, including migraine triggering and remitting factors, are known to affect ocular hemodynamics, including blood flow and IOP [6,26-29].

Unilateral or bilateral self-applied OCM for a few (< than 20) seconds in migraine/PCH/BCH is designed to suddenly limit choroidal blood flow (CBF) and/or rise in IOP along with reduction of generation of corneoscleral nociceptive traffic by physical or mechanical tamponade [24] The thenar eminence of the hand is both protuberant and moldable across the ocular globe, and is best suited for self-use in a controlled fashion to generate painless tamponade to limit sudden increase(s) in CBF/IOP. Such a transient painless self-maneuver in the awake state limits or reduces: (i) overall volume of the ocular globe, CBF, and IOP; (ii) corneoscleral distention/undulation; (iv) and nociceptive V1 neural traffic. Conversely, post-operative digital ocular massage (DOM) or compression used by glaucoma surgeons after trabeculectomy definitively elevates IOP, mechanically forces passage of aqueous humour through the surgical wound, breaks weak adhesions in the outflow pathway, and visually extends the filtration bleb through the surgical wound and/or thereby substantially reduces IOP [30,31]. The postoperative technique also involves cycles of externally applied pressure to the eye, followed by intervals without ocular pressure application. DOM is often taught to patients for self-administration at home but the force required and its endpoints are unregulated. The average IOP induced by typical DOM ranges between 95 - 104 mm Hg [31]. I never recommend use of the self-applied OCM beyond the point of discomfort and advise not to generate ocular or neck pain, unilateral or bilateral. Under these circumstances for managing primary headaches, IOP levels in self-induced OCM are likely to remain much lower than that induced by DOM used by ophthalmic surgeons. The likelihood of the bradycardia induced by the trigemino-vagal oculo-cardiac reflex as during strabismus surgery [32] is also low.

The patient's analgesic consumption due to migraine or PCH has reduced dramatically to occasional use. He continues to apply OCM early in the pain phase of an impending migraine attack without any complication, now 10 years since the surprise discovery of its efficacy. With the efficacy of OCM for migraine, for PCH, and for BCH, the long process of "lumping" of both a very large number of purely narrative-related primary headache "variants" or "entities" as well as the elucidation of physiologic processes underling the complete spectrum of primary headache(s) has begun. I also predict that OCM will prove beneficial in both cluster headache management as well as other entities under trigeminal autonomic cephalalgias (TAC). Use of the self-applied OCM in these clinical circumstances is expected to be safe and uncomplicated. Nevertheless, the first few applications of OCM should always be gentle and undertaken only after a full understanding of the underlying principles.

An interface appears to be surfacing between migraine, primary open angle glaucoma (POAG), and normal tension glaucoma (NTG) [33-35]. The cause-effect relationship between these clinical entities and its physiologic basis can now be better defined. Such a relation between migraine and glaucoma was suspected as early as over three decades ago [36]. Home tonometry was advised earlier in migraine patients to pick up subtle variations in the IOP, and the possible link between migraine, POAG, and NTG [37-39].

Conclusion

The role of self-OCM to manage migraine attacks and primary headache associated with sexual activity/PCH is presented. A central common pathogenesis at the level of the eye probably underlies the many "variants" of primary headache, including migraine, PCH, BCH, gut-induced Valsalva manœuvre-linked migraine, cluster headache, and TAC.

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Volume 4 Issue 8 August 2021