Constipation-related Migraine Is Linked To The Effect Of The Valsalva Maneuver On The Eye: A Case Report And A Mechanistic Review

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Abstract

Constipation-induced migraine finds rare mention in headache literature. In this self-report, I describe occurrence of pulsating migrainous headache after straining at stools. Surprisingly, reducing rise in intra-thoracic pressure by keeping the mouth open, and, limiting choroidal congestion by forcibly screwing the eyelids together during constipation-related Valsalva maneuver prevented further development of migraine attacks. Constipation is a very common human experience and should be regarded as an important precipitating factor in some migraine patients, particularly those who are found refractory to routine preventive strategies.

Use of both adaptive maneuvers is suggested for migraine patients with severe constipation, whether or not they are aware of this potential pathogenetic link. I also describe my own migrainous positive scotoma and discuss the relevant mechanisms.

Introduction

Migraine is a painful, incapacitating disease with a substantial economic burden on society [1]. Brain dysfunction possibly related to cortical spreading depression (CSD) is currently believed to underlie migraine attacks [2].

Migraine precipitating factors, external or internal, have been well-delineated [3-6]. More than one (generally 4-5) precipitants or triggers operate in the individual migraine patient. Trigger factor are rarely predictable and migraine patients identify such factors in only a minority of attacks [5]. Susceptibility to new precipitating factors can develop several years after onset of migraine; also, patients can become less susceptible to previous migraine triggering factors. Stress, weather changes, missing a meal, perfumes and odours including cigarette smoke, neck pain, and bright sunlight appear to be the commonest precipitating factors. Oversleeping and undersleeping are also common migraine precipitants. Perimenstrual aggravation of the migraine tendency is noted in approximately 50% of women. Food triggers are commonly suspected but difficult to establish. Alcohol is a well-known precipitant of migraine attacks. Crying is an under-recognized but common precipitant of migraine [7-9].

The role of the gastrointestinal tract in migraine is prominent, with nausea or vomiting accepted as a common diagnostic feature [10]. While migrainous vomiting is occasionally associated with diarrhea, constipation may occur as part of the prodrome of migraine [11]. The cause-effect relation between migraine and constipation remains uncertain. Constipation and straining at stools has only rarely been recognized as an immediate precipitant of primary headache [12,13]. Constipation and the attendant straining at stools for evacuation is, however, a common, almost universal experience amongst humans. Straining at stools is effected by raising the intra-thoracic pressure and ultimately the intra-abdominal pressure through the Valsalva maneuver (VM).

This paper provides a first-hand account by a physician of constipation-related throbbing migraine headache, a personal description of migrainous positive scotoma, a brief review of Valsalva-related primary headaches, and a mechanistic discussion.

Case Report

For over two decades, I have experienced migraine without aura headaches (MWA) in accord with the classification of the International Headache Society [10]. Over a period of four days in 2003, at the age of 49 years, I had two episodes of migraine with aura (MA) following severe emotional turmoil and a bout of loud shouting around 12 hours ago. I have been on atenolol, 100 mg daily, for migraine prophylaxis for almost twenty years. I developed Addison’s disease (primary adrenocortical insufficiency) in June 2007 and have been on low doses of prednisolone (7.5 mg/day) since then. I have maintained a rather low systemic blood pressure, both before and after the development of adrenocortical insufficiency.

In the month of August 2004, I suffered five attacks of
left-sided pulsating fronto-temporal headache with mild nausea and dizziness that were precipitated almost immediately (within 5-10 seconds) by straining at stools. Four of these headache attacks were mild to moderate in intensity; only during one episode I required a nonsteroidal anti-inflammatory drug (NSAID) to manage the pain. In the absence of the NSAID, the mild-to-moderate pain lasted for 4-8 hours. The severe constipation arose after starting atorvastatin 20 mg to lower the total cholesterol level and has persisted till today. I kept a dairy of headache and headache-related events for 2 months following the first episode of constipation-associated pulsatile headache.

During straining at stools, I noticed that I generally tended to keep my eyes open and my mouth shut. On the occasions that I did develop headache while straining at stools, I had kept my eyes open and my mouth shut. With my medical background, I speculated that the Valsalva maneuver (VM) during straining at stools was responsible for my pulsatile headaches. After the fifth attack of headache, I started to keep my mouth open during straining at stools and to shut my eyes tightly by forcibly screwing the eyelids together. Surprisingly, I have since then never developed a migraineous headache after straining at stools. On the other hand, despite atenolol prophylaxis, I continue, uncommonly, to experience MWA due to my own eyes, as described previously for benign migraine [17]. VM (coughing, sneezing, or straining) is itself a form of VM; emesis can rarely induce subperiorbital (subperiosteal) hemorrhage during a migraine attack or during influenza [21-23]. VM during parturition can also result in an orbital subperiosteal hematoma [24]. VM (coughing, sneezing, or straining) can precipitate cluster headaches [25]. Despite the Valsalva-like effect of emesis, the latter commonly ameliorates or aborts migraine headaches. Emesis is consistently associated with considerable increase in arginine-vasopressin release, a likely mechanism for attenuation or elimination of migraine headaches [26]. Shouting also involves the VM; the effect of shouting on migraine is, however, largely unknown.

Defecation, urination, and childbirth are common events involving the VM. Clearing the eustachian tubes in an airplane or multiple orgasms are less well-known activities involving the VM. Heavy weight lifting also involves the VM to keep chest and shoulders rigid for better support for the arms. Headache during power-lifting may involve the VM [17]. Valsalva maneuver commonly aggravates ongoing headache of migraine [11]. Nevertheless, VM is only rarely known to precipitate migraine; a close temporal association of the onset of aura and the VM suggests that opening of right-to-left shunt acted as a trigger for migraine [18]. A cohort of migraine with aura patients, however, identified at least one Valsalva-provoking activity as headache trigger in 45.8% [19]. Sneezing, laughing, and stooping are other Valsalva-like activities that can precipitate cough- or other effort-induced migraine-like headache [13,20]. Emesis is a form of VM; emesis can rarely induce subperiorbital (subperiosteal) hemorrhage during a migraine attack or during influenza [21-23]. VM during parturition can also result in an orbital subperiosteal hematoma [24]. VM (coughing, sneezing, or straining) can precipitate cluster headaches [25]. Despite the Valsalva-like effect of emesis, the latter commonly ameliorates or aborts migraine headaches. Emesis is consistently associated with considerable increase in arginine-vasopressin release, a likely mechanism for attenuation or elimination of migraine headaches [26]. Shouting also involves the VM; the effect of shouting on migraine is, however, largely unknown.

VM augments intra-thoracic pressure that transmits to epidural veins and raises intra-cranial pressure [27]. Functional venous valves are frequently present in jugular veins, adjacent to the jugular-subclavian junction; direct transmission of raised intra-thoracic pressure likely does not occur through the jugular veins [28]. VM-related headaches respond inconsistently or occasionally to indomethacin,
propranolol or methysergide [27,29]. Lateraling (or side-locked) headaches, such as cluster headache or migraine, however, cannot be explained on the basis of diffuse or bilateral increases in intracranial pressure. Conversely, choroidal congestion due to raised intra-thoracic pressure has been proposed to underlie bilateral benign cough-induced headache [30].

Increase in jugular venous pressure through the VM is transmitted to the orbit by valveless veins. While the VM raises the intraocular pressure (IOP) by 4-5 mm, forcible expiration against a counter-pressure can elevate the ocular tension by 60%; this a complex reaction unrelated to the systemic arterial pressure, that may show reduction in these circumstances [31]. Sneezing, laughing, and parturition also give rise to brief increases in the IOP. Venous congestion of the head during VM involves elevation of the episcleral venous pressure and congestion of the ocular choroidal vascular bed. The trigeminal axon reflex (antidromic discharge) is the key feature in the effector (or efferent limb) of headaches, including migraine [32]. Acute experimental IOP elevations discharge impulses in iris, corneo-scleral, and whole nerve ocular trigeminal fibres [31,33] that likely generate headache related to VM-like activities, including straining at stools [32].

The IOP system has an important tamponade function that limits choroidal congestion. Forcible squeezing together of the eyelids -- eyes tight-shut -- can raise the IOP by 60-70 mm and prevent choroidal congestion [31]. Ocular compression can also limit choroidal venous surges and limit rises of IOP in response to VM-like activities [34,35]. Ocular self-compression can prevent development of benign cough-induced headache [14]. Over the last 5 years, squeezing eyes shut tightly or manual compression of both eyes along with preventing or limiting rise in intra-thoracic pressure by keeping the mouth open has repeatedly prevented development of constipation-induced MWA in this case.

The mechanistic basis of anomalous or exaggerated choroidal venous surge during VM or VM-like activities remains unknown. It can be speculated that a regional intraocular vascular autonomic insufficiency contributes to abnormal choroidal venous congestion during VM or VM-like activities. Normally, three homeostatic mechanisms limit such surges in choroidal blood volume [31]. There is a subtle but important link between systemic BP and IOP; higher levels of BP are likely to be associated with higher levels of IOP that offer significant ocular tamponade [36]. Most patients with migraine maintain a low systemic BP [11], and, might be, therefore, more susceptible to VM or VM-like activities due to a normal or low-normal IOP. Also, lowering of the systemic BP as well as the IOP through migraine prophylactic drugs such as propranolol, atenolol, or verapamil can accentuate the tendency to develop VM-related migraine attacks.

CSD is widely accepted since over half-a-century as the pathophysiologic aberration underlying migraine – an intuitive extrapolation from experimental physiology in animals that has little, if any, clinical relevance to human neuropathology, particularly to migraine [37,38]. CSD has generated much confusion in neuroscience research and waylaid the migraine research process. Sacrificing animals in research laboratories without a logically-defensible theoretical background is also an unethical disregard for life itself without advancing the human cause. The clinical equivalent of occipital or visual cortex CSD would be the simultaneous onset of bilateral, homonymously distributed migrainous scintillating scotoma with gradual spread from one macula to the temporal visual field and from the other macula to the nasal visual field – a feature that has never been recorded in the migraine literature [37,38]. A single description of a visual spreading phenomenon by Lashley has effectively blunted the critical thinking of a very large body of neuroscientists who are otherwise highly logical and intellectually formidable [37-39]. While neuroscience researchers continue to cheer-on CSD with all possible (and impossible or even incredible) assumptions, migraine research has itself got lost hopelessly in the woods [37,38]. My own visual aura – scintillating scotoma (SS) –experienced on 2 occasions in 2003, unrelated to constipation or the VM, appeared suddenly without any spreading, as a fully-developed colourless oval-shaped mass of shimmering lines or waves, blending into each other, while being confined to the para-macular temporal half of the left eye (as determined by closure of both eyes alternately, by shift of the image by digital indentation, and by disappearance by closure of the left eye) for approximately 10 minutes. This description of the migrainous SS clearly indicates that the site of origin is the retina and not the visual cortex. In contrast to the negative homonymous hemianopia experienced by some migraineurs that clearly indicates cerebral ischemia [11,40], the migrainous SS does not have (or have to have) a sharply distinguished edge that reflects involvement of one complete half of the visual field. The topography of the retina, unlike that of the cerebral cortex, does not support sharply divided halves of the visual field. Also, since topiramate suppresses CSD but cannot prevent migraine aura [37], migrainous SS is unlikely to be of cortical origin.
Finally, drugs that can relieve the SS, such as nitroglycerin, nifedipine, or isoprenaline, have no influence on the CSD [37]. A pressure-mediated transient and localized distortion / dimpling at the posterior pole of the eye with mechanical rather than electrical "retinal chaos" probably underlies migrainous SS. Nevertheless, VM or VM-like activities and their consequences (such as constipation-related migraine) can have no possible link to CSD – an unqualified clinical and research absolute.

The limitations of this study include a self-diagnosis based on the IHS criteria without recourse to diagnostic neuroimaging or other "hard" data, and, a personal bias in the interpretation of first-person phenomenon. Neuro-imaging is not a diagnostic requirement for migraine or migraine-like headaches [10]. Migraine commonly affects medical personnel, including doctors (including physicians and neurologists) and nursing personnel. Most lay migraine patients cannot understand the complexities of VM-induced cranio- or oculo-vascular aberrations. Both limitations of this study will certainly be overcome by more case reports from the medical fraternity of similar observations regarding this otherwise occult pathophysiologic phenomenon. Even Lashley described and interpreted his own scotoma [39], thereby setting a precedent that has been followed by phenomenological descriptions of their own migraines by several other scientists. Likewise, correct descriptions of the migrainous SS have to come from the unbiased personal descriptions of other migraine sufferers with appropriate medical background. This key missing piece of information – that can begin to unravel the gigantic Gordian puzzle of migraine – cannot, however, come from observational studies of lay cohorts (whether retrospective or prospective) or from sub-analyses in randomized clinical trials or meta-analyses.

Conclusion

Straining at stools can precipitate migraine headaches. Screwing the eyeballs tight-shut or keeping the mouth open or manual self-compression of the eyeballs (or any combination of these maneuvers) can prevent development of such headaches. Such pressure-related intraocular aberrations have no conceivable link to CSD. Migrainous SS is also likely linked to a pressure-related retinal aberration. As the use of statins becomes widespread, the link between constipation and migraine will become more apparent.

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