Anorgasmic Sexual Headache

**Corresponding Author:**
Dr. Daniel E Jacome,  
MD, Dartmouth Hitchcock Medical Center Department of Neurology, One Burnham Street, Suite 2, 01376 - United States of America

**Submitting Author:**
Dr. Daniel E Jacome,  
MD, Dartmouth Hitchcock Medical Center Department of Neurology, One Burnham Street, Suite 2, 01376 - United States of America

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Author(s): Jacome DE

Abstract

A forty six year old female with history of migraine without aura and isolated visual auras recently increasing in frequency, was prescribed topiramate as a preventative drug against migraine. Her repeated neurological examinations and her imaging procedures were normal. Topiramate at a dose of 50 mg b.i.d. was effective in controlling her spontaneous headaches but resulted in the appearance of recurrent headache only with sexual activity in attempting to reach orgasm (i.e., anorgasmic headache) while reporting no headaches with other modalities of physical activity, cough or sneezing. A slight reduction on the dose of topiramate was sufficient to suppress her sexual anorgasmic headaches by allowing her achieving orgasms once more. After a few months, restoration to the originally intended dose was accomplished without causing a recurrence on her anorgasmic headaches, or interference with sexual climax. Anorgasmic sexual headache may occur in isolation in cases of dose dependant topiramate-induced anorgasmia on a temporary basis, permitting the continuation of its use if efficacious otherwise in controlling spontaneous migraine, and causing no other significant adverse reactions.

Introduction

Sexual dysfunction in women with migraine is prevalent. A very recent study carried by a group of investigators from the University of Pavia, Italy, in 100 woman with primary episodic and chronic headache, both migraine and tension-type headache, both migraine and tension-type headache, found that 29% of their study group reported sexual distress (1). The Female Sexual Function Index and Female Sexual Distress Scale-Revised were administered to their patients (1). Higher distress occurred in women with chronic tension-type headache and was associated with diminished sexual desire. Depression was positively correlated with sexual distress. No medication was single out as a particular offending agent although patients with chronic headache employed greater numbers of analgesics per month.

Case Report

A forty six years old bank officer was seen in neurological consultation because of headaches of increased frequency and intensity. Headaches began twenty five years earlier. They were described at the time of her original visit as weekly episodes of right side sharp steady head pain lasting for hours. Headaches were not preceded by auras and were associated to nausea and light sensitivity. Yet, she reported rare visual auras without headache. She reported no associated right eye ptosis or tearing of the eye and no congestion of the right nostril. Pain was not alleviated by over the counter analgesics. Barometric pressure changes and strong odors triggered headaches sometimes while they had the tendency to cluster on week-ends when she was not at work. She had experience many years earlier two episodes of mild short-lived headache with orgasm. Her past medical history was positive for currently asymptomatic Wolff Parkinson White (WPW) syndrome, and for uncomplicated lumbar laminectomy for disc herniation. She had history of anxiety and depression but never incapacitating. There was migraine on her family and she was non-smoker. She was allergic to penicillin and sulfa drugs. She took citalopram 20 mg q.d., bupropion XR 75 mg q.d. and betaxolol HCL 5 mg q.d. On initial evaluation her BP was 129/88 mm Hg, and her heart rate was regular at 65 beats a minute. She was afebrile. Her neurological examination was normal, including normal appearing optic nerves. Imaging procedures included a normal MRI of the brain except minimal amount of white matter non-specific small size T2 hyper-intensities. There was no hydrocephalus, evidence of tumor, multiple sclerosis or vascular malformation. Magnetic resonance venogram (MRV) with contrast was negative for cerebral venous sinus stenosis or thrombosis and the carotid ultrasound revealed no dissection or stenosis. She was prescribed topiramate as preventative medication as a dose of 50 mg b.i.d. On her return visit few weeks later she reported that although her spontaneous weekly headaches were not any longer present, she was having now similar intense headaches, only in the context of prolonged sexual intercourse, since she was unable to reach orgasm. She had no other medication side effects. Headaches were not precipitated by physical activity, even if vigorous, except when having intercourse, and were not precipitated by coughing, sneezing, or...
involuntary Valsalva, for example, when straining for any reason. The dose of topiramate was reduced to 25 mg in a.m. and 50 mg in p.m. Her headaches associated with sexual activity dissipated while she was able to reach orgasm again. After few months of follow-up, the initially recommended dose was reinstated with no adverse reactions (anorgasmia or sexual headache).

Discussion

Orgasmic headache (OH) is the classic form of sexual headache (SH). It is normally benign, self-limited, often recurrent but not predictable or periodic, and typically lasting only for a few hours. The headache is usually very intense (“explosive or thunderclap”) and of rapid development during the climax of sexual activity. It can be precipitated by either intercourse or masturbation (2, 3). Sexual headache may be understood as a Valsalva-induced or exertion type of benign headache; however, several examples of ominous significance have been described in the literature. They include cases of embolic stroke, subarachnoid hemorrhage, brain parenchyma hematoma, basilar artery stenosis and basilar artery dissection (4,5).

Pre and post-orgasmic headaches are recognized as variants of sexual headache where the headache develops before or after the actual orgasmic climax (6). Although it is often believed that orgasmic headache is the one of greater significance in regards to intracranial vascular events, a patient with pre-orgasmic headache complicated by carotid artery dissection was reported by Edvarsson and Persson in a 37 year old male with mild hypertension (7). Their patient had none of the classic symptoms of acute carotid dissection and her carotid ultrasound was normal.

Topiramate use rarely results in anorgasmia. Sun, et al described 7 patients (5 women and 2 men) with topiramate induced anorgasmia (10). Six of the 7 patients had migraine without aura and one migraine with aura. Anorgasmia was dose related and promptly reversible with symptom resolution within 1 week of discontinuation. The authors postulated the possible reduction of central excitatory neurotransmission and the simultaneous enhancing of inhibitory mechanisms to explain anorgasmia in their patients. Sun, et al, implicated serotonin mediated dopamine mesolimbic release inhibition and topiramate's well know action as carbonic anhydrase inhibitor, to account for the occurrence of sexual dysfunction on their study cases (10). The subject of this case report not only developed anorgasmia with topiramate; she exhibited anorgasmic headache as a specific variant of pre-orgasmic headache.

In contrast to the above adverse sexual effects, topiramate may abolish pre-orgasmic headache as documented by Bandini, et al, in a 55 year old woman (11). The patient had negative head CT, MRI, MRA and spinal fluid analysis. She responded to a small dose of topiramate (50 mg a day). The authors concur with the opinion that pre-orgasmic headache is a sub-type of tension-type headache, while orgasmic headache is true migraine (11). Earlier investigators equally found topiramate efficacious in suppressing orgasmic headache (12).

It is of interest than on this patient the spontaneous migraine episodes were suppressed by topiramate, while favoring, albeit transiently, the development of exertion-specific headaches, only in the context of sexual arousal. This seems to indicate that the pathophysiology of SH is different to that of primary exertion headache, since her headaches did not occur with other kinds of exercise or physical activity. In support of the latter assumption is the study conducted by Donnet, et al (13). These investigators studied 10 patients with primary cough headache, 11 patients with primary exertion headache and 20 with primary sexual headache, and compared their results with a
control group of 16 headache-free subjects. Donnet, et al, encountered that a significant proportion of individuals with primary cough and sexual headache had cerebral venous sinus stenosis on their MRV, in contradistinction to individuals with (only) primary exertion headache (13). If a hormonal component or post-orgasm illness syndrome contribute to the generation of SH in contrast to primary exertion headache, it is unknown at this time (14).

References

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