

CASE REPORT

Orgasmic Headache Treated with Nimodipine

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DOI: 10.1111/jsm.12093

ABSTRACT

Introduction. Orgasmic headache (OH) is a sudden and severe headache that occurs at the time of or shortly after an orgasm.

Aim. We present the case of typical primary headache associated with sexual activity, especially during an orgasmic period.

Methods. A 34-year-old man complained of sudden and severe headache during sexual activity, or orgasmic period, for 2 months. The headache developed abruptly with an orgasm and then decreased shortly over a period of 4–8 hours.

Results. Magnetic resonance angiography revealed severe spasm of the M1 segment of both the middle cerebral arteries. He was treated with oral nimodipine (30 mg every 8 hours), which alleviated the headache and prevented its recurrence.

Conclusions. We postulated a pathophysiological relationship between OH and migraine, especially with respect to vasoconstriction, and believe that in such cases, nimodipine may be an effective therapy. **Lee JW, Ha YS, Park SC, Seo IY, and Lee HS. Orgasmic headache treated with nimodipine. J Sex Med 2013;10:1893–1896.**

Key Words. Post-Orgasm Illness; Headache; Nimodipine

Orgasmic headache (OH) is a sudden and severe (“explosive”) headache that occurs at the time of or shortly after an orgasm [1]. It has been hypothesized that arterial vasospasm that occurs secondary to an impaired myogenic mechanism of cerebral autoregulation plays a role in the pathogenesis [2]. However, the pathophysiology of OH is still uncertain. Although OH has a benign course, repeated attacks may carry a risk of ischemic stroke [3]. Also OH responds poorly to analgesics.

Nimodipine is a calcium-channel blocker that can alleviate vasospasm after subarachnoid hemorrhage [4]. Intravenous nimodipine was effective in two patients with primary thunderclap headache after the development of vasospasm [5]. We describe the case of recurring OH treated with oral nimodipine.

Case

A 34-year-old man presented to the urology outpatient department for treatment of headache that was associated with sexual activity, especially during an orgasm. For 2 months the headache was bilateral, and of both pulsating and throbbing type. The pain originated in the posterior neck and occipital area, and extended to the vertex. It was accompanied by nausea, vomiting, and dizziness. The headache developed abruptly with an orgasm and then decreased shortly over a period of 4–8 hours. He did not report any photophobia, phonophobia, or osmophobia. However, the headache was so severe (8–10 on a 0–10 verbal numerical scale) that he had to stop sexual intercourse immediately. Further, the patient denied any history of similar headaches or

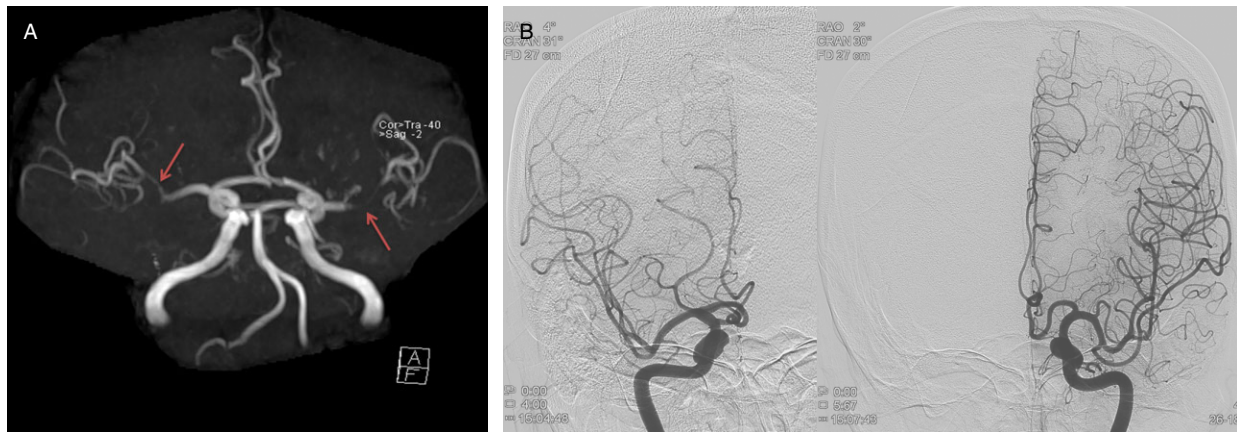


Figure 1 (A) Magnetic resonance angiography of both middle cerebral arteries narrowing 2 hours after orgasmic headache. (B) Digital arteriography 7 days after orgasmic headache.

migraine; in fact, his medical history was unremarkable except for asthma. The findings of physical and neurological examinations were normal. Notably, there was no sign of meningeal irritation such as neck stiffness. We treated analgesics, including aspirin and Tylenol, but they were not effective in controlling the headache. So we consulted to the neurology outpatient department and then checked the hematology test and urinalysis.

All findings on hematology tests and urinalysis were within normal limits, including glucose (fasting), creatinine, urea nitrogen, erythrocytes, leukocytes, platelets, hemoglobin, hematocrit. Both brain computed tomography and brain magnetic resonance angiography (MRA) showed no subarachnoid or intracerebral hemorrhage, aneurysm, vascular malformation, atherosclerosis, infarction, or space-occupying lesion. MRA revealed severe spasm of the M1 segment of both the middle cerebral arteries (Figure 1A). The patient was therefore administered oral nimodipine therapy (30 mg to be taken every 8 hours). Approximately 7 days later, a digital cerebral arteriography was performed to further evaluate and disclose other causes of arterial narrowing. Findings on examination were normal (Figure 1B). One month after the treatment, the headache and all associated symptoms subsided and did not recur.

Discussion

We reported the case of recurrent OH defined by the International Classification of Headache Disorders (2nd edition) [1]. The diagnosis of OH is a diagnosis of exclusion, i.e., all other possible causes of headache must first be ruled out. In our

patient, the episodic OH and normal findings of neurological examination and brain imaging studies, which did not reveal hemorrhage, were not consistent with the diagnosis of subarachnoid hemorrhage.

As treatment for the acute episodes, analgesics given after the onset of headache are of limited or no benefit in nearly all patients [6]. In our case, the patient had recurring OH attacks that responded poorly to analgesics. However, after taking a 1-month oral nimodipine therapy, he remained in a headache-free state, and the same therapy was successful in preventing the recurrence of OH.

Because of its relative rarity, management of OH is based on experience and case series, not on any randomized trials. Usually OH responds poorly to analgesics. There has been a single report of successful short-term prophylaxis with naratriptan in a 54-year-old woman suffering from OH [7]. Also there has been a report of prophylactic treatment with propranolol and indomethacin. Empirically, response rates for both drugs are approximately 80% [5].

Our case is comparable to those previously reported. Oral nimodipine could prevent headache in nine OH patients without any vasospasm [5]. Nimodipine infusion could effectively prevent thunderclap headache in a 58-year-old woman who had vasospasm and posterior ischemic infarct; serial MRA showed a marked improvement in vasospasm within 2 days [3]. Repeated attacks cause disability and carry a risk of ischemic stroke [1,6]. IV nimodipine was used in two patients with primary thunderclap headache after ischemic stroke had developed [1,6], but its efficacy remains

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