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Review Article

The physiology of orgasmic headache

Emojevwe Victor[1], Oyovwi Mega Obukohwo[2]*, Gbemi Olasunkanmi Korede[1], Naiho Alexander Obidike[1,3], Oyeleke Abiodun Abioye[4]

[1]Department of Physiology, University of Medical Sciences, Ondo City, Ondo State, Nigeria
[2]Department of Physiology, Adeleke University, Ede, Osun State, Nigeria
[3]Department of Physiology, Delta State University, Abraka, Delta State, Nigeria
[4]Department of Anatomy, Federal University Oye-Ekiti, Nigeria

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***Correspondence:**
Oyovwi Mega Obukohwo,
E-mail: megalect@gmail.com

ABSTRACT

Orgasmic headache is a headache caused by sexual activity that emerges as sexual excitement increases (progressive at onset) or as an immediate and powerful headache following orgasm (thunderclap at onset) or combines these two characteristics. The idea that orgasmic headache (OGH) is caused by physiologically inappropriate responses is extremely simplistic. As a result, a complete analysis of the physiological mechanisms is provided here in order to comprehend the complex situation of OGH. The physiology of OGH was studied in humans utilizing peer-reviewed papers from Pubmed, Science direct, EBSCO, Scopus, Cochrane library, Sage Journals, and Google Scholar. Author, year of publication published between 2003 and 2020. OGH can regulate psychophysiological reactions, but it can also cause a rise in blood pressure, persistent pain, intracranial hemorrhage, and cerebral infarction. This review explains two physiological systems: the release of calcitonin gene-related peptide (CGRP), which induces the creation of less serotonin, resulting in an inflammatory response and discomfort. The release of epinephrine and nor-epinephrine can cause cerebral ischemia, which can lead to headaches in headache-prone patients. Fear of an orgasmic headache can lead to lower libido, leading to lower sex pleasure. As a result, the condition may deprive sex of its pleasure and turn it into a 'headache'. We conduct a literature review to study the physiological processes of OGH in connection to its physiological maladaptive responses. A greater understanding of the physiological mechanisms underlying Orgasmic headache will allow practitioners to properly identify and counsel patients without attributing physiological maladaptive reactions to OGH.

Keywords: Orgasm, Headache, CGRP, HSA

INTRODUCTION

Orgasmic headaches (OGH) were the focus of intensive research a few decades ago, but interest has since waned. However, new material has slowly emerged, and the authors believe that it is now acceptable to bring this topic back into focus. Orgasm is commonly acknowledged to be one of the most powerful sensations a human can experience, yet its fundamental mechanics are unclear.[1] There is no accepted definition of orgasm. It is difficult to get an agreement on a definition because each specialty, such as endocrinology or psychology, analyzes this activity from its own perspective. Although the two physiological processes are distinct, orgasm and ejaculation are frequently mistaken.[2] It is usually caused by rhythmic stimulation of bodily areas with high concentrations of sensory receptors, but this is not always the case.[3] This stimulation is typically accomplished through physical manipulation of the genitals via body-to-body contact or, in certain cases, using vibro-tactile mechanical devices. Although most orgasmic actions are focused on the genitals, orgasm can also be reached by stimulating other body parts or even with thought alone.[4] Although many of these systems are shared by other

mammals, vertebrates, and even invertebrates, certain aspects of the orgasmic experience may be unique to humans.[1,5] However, this strong pleasure has been linked to a type of headache, which is mentioned somewhere in this article.

Headache is the most common neurological condition and continues to be a prominent cause of emergency department use.[6,7] It is a painful disorder of the head that is occasionally combined with discomfort in the face and/or neck. It is a nearly universal human experience and one of the most commonly reported symptoms in medicine and neurology.[8,9] People of various races, backgrounds, and nationalities suffer from headache issues. More than half of the adult human population is thought to have experienced headaches associated with various headache diseases at least once in their lives.[7] Headaches are not only medically significant, but also have a significant socioeconomic impact, and can be divided into two types: primary headaches and secondary headaches.[10,11]

When it is caused by sexual activity, it is known as orgasmic headache, a disorder that was the focus of intense investigation a few decades ago. In light of the foregoing, orgasmic headache is described as an acute headache that occurs during or after orgasm or is associated to sexual intercourse. It has been observed in both genders and typically begins as a dull, bilateral ache as sexual excitement grows, then becomes acute during orgasm.[12,13] It is well understood that headache and sex do not travel in the same direction, and that dread of orgasmic headache may become a potential explanation for decreased frequency of sexual intercourse because it dampens sexual drive and libido.[14] Despite its frequency, there is a lack of understanding of the underlying pathophysiological mechanisms that generate headaches.[15] The impact of headaches on daily activities is related to the patient's age, marital status, socioeconomic status, headache quality, headache site, pulsatile/non-pulsatile characteristic, headache frequency, headache severity, and nausea.[7]

HISTORY AND PREVALENCE OF ORGASMIC HEADACHE

Headaches caused by sexual activity have been documented since Hippocrates' time; nevertheless, it wasn't until 1970 that the primary or benign variety of these headaches was described in conventional literature.[14,16] As a result, the disease was included in the first version of the International Classification of Headache Disorders (ICHDs) published in 1988. Orgasmic headache was once thought to be a harmless condition that didn't require treatment, but this opinion has shifted as more research has been published on the subject. When it was revealed that this condition could have hazardous and life-threatening underlying causes, such as aneurysmal subarachnoid haemorrhage, interest in it grew. It is worth noting that coitus has long been recognized to be the immediately preceding activity in some cases of burst aneurysmal subarachnoid haemorrhage, and the entire purpose of studying OG today is to rule out such a potentially fatal disease.[17,18]

The global reported prevalence of sexual headache is 1.1%.[12,19] Patients, particularly in conservative communities, find it difficult to convey their issues to doctors. It can be terrifying and distressing, but it can also significantly limit their sexual activities. Orgasmic headache has been observed in both genders, but it is more common in men, with the mean age of onset ranging between 22 and 50 years.[20,21]

Apart from pharmaceutical prevention if needed, psychotherapy and reassurance play a key role in management.[20] The duration of pain ranges from a few minutes to 24 hours. Severe pain usually lasts for less than four hours.[21] Subarachnoid hemorrhage, vascular thrombosis, hemispherical infarction, reversible sensory disturbances, and homonymous hemianopia are all symptoms of headache. Thus, they can be confused with thunderclap headaches that occur during coitus and signal a sudden intracranial event, such as subarachnoid hemorrhage.[20]

CLASSIFICATION OF ORGASMIC HEADACHE

There are two types of orgasmic headache. The initial (type-I) headache is characterized by a dull discomfort in the head and neck during sexual activity. The pain may worsen as a result of sexual excitement. The second (type II) is sudden and strong, similar to a thunderclap headache experienced during orgasm. This is the most common and concerning t.[22]

Physiology of orgasmic headache

What causes OGH and what causes its pathophysiology are still unknown. Although there is a major muscular component, the fundamental mechanism of OGH is trigeminal vascular activity.[23] Muscular contraction is important, especially in lesser headaches that worsen as sexual arousal grows. A study found that persons with type II headaches may have impaired cerebrovascular autoregulation. When compared to a typical healthy control, the cerebral vasculature of these patients may respond to low pH by dilating in unanticipated ways.

According to some authors, there may be a link between type II headaches and migraines, and catecholamines, neurokinins, and serotonin may be released during OG. Orgasmic headache is well known in adults, but it has also been recorded in teenagers. A 12-year-old kid was the youngest recorded patient with sexual headache.[21] The trigeminal nerve, which has a large sensory root and a smaller motor base, is the largest cranial nerve.[24] The trigeminal ganglion cells have both peripheral and central projections. The peripheral processes are the three sensory branches of the trigeminal nerve: ophthalamic, maxillary, and mandibular. These three branches are responsible for the distribution of pain and temperature sensors on the

face, forehead, eyelids, nose, pinna, tongue, teeth, cerebral blood vessels (trigeminovascular system), dura mater (membrane that covers the brain), and the posterior portion of the head and neck. When the trigeminovascular system is activated, several vasodilators are released, most notably calcitonin gene-related peptide (CGRP), which dilates cerebral blood vessels and promotes an inflammatory response that causes pain. The creation of CGRP reduces the neurotransmitter serotonin, which increases during attacks.[25,26]

Excess catecholamines, such as epinephrine (Ep) and norepinephrine (NEp), as well as the neuroendocrine hormone cortisol, may be directly or indirectly associated to the development of OGH. Palpitations, increased blood pressure, heat, and perspiration, which can occur during sexual actions, have been associated to abnormally high Ep and NEp levels. Overproduction of these catecholamines leads to potentially excessive dopamine consumption, which may be the major source of the syndrome's physiological symptoms.[27] Prolactin levels may increase significantly and remain high for an extended period. This further modifies the dopaminergic neurons in the nigrostriatal and mesolimbocoritcal systems and the medial preoptic area of the hypothalamus of the brain to alter sexual behavior in psychic pattern.[28]

The anterior pituitary gland secretes adrenocorticotropic hormone (ACTH), which increases Ep and NEp synthesis. As a result, ACTH increases the activity of tyrosine hydroxylase and dopamine hydroxylase. ACTH may be produced at extremely high levels if the anterior lobe of the pituitary gland overproduces prolactin. Increased ACTH levels may also cause the adrenal cortex to release cortisol, which enhances the production of phenylethanolamine N-methytransferase (PNMT), which in turn raises epinephrine synthesis in a vicious cycle.[29]

Dopamine is transformed into norepinephrine at a faster rate when ACTH is present, potentially depleting this neurotransmitter. Furthermore, the activities of prolactin during the post-orgasm stage are still largely understood, suggesting that prolactin could directly limit dopamine secretion in the same way that dopamine inhibits prolactin secretion. When high, epinephrine had negative effects on cerebral microvascular blood flow via its alpha1-agonist action, such as increasing the degree of cerebral ischemia during sexual activities. Cerebral hemodynamic or neurochemical alterations that occur during sexual engagement and would not ordinarily induce pain might then easily trigger headaches in headache-prone people.[21] A pressor response to exercise has been suggested as a mechanism in some patients, and for others, the pain appears to be specifically activated by sexual excitement and contraction of facial and neck muscles.[30]

TREATMENT

NSAIDs have been shown to be beneficial in the treatment of this illness over the years.[19,31] Indomethacin (25-50 mg/day) or propranolol (40-200 mg/day) were found to be effective therapies in a study of various forms of headaches.

Patients are also urged to engage in more frequent but less vigorous sexual intercourse. Some approaches that may be beneficial in the management of the disorder include regular exercise, avoiding excessive alcohol consumption, and maintaining a healthy weight.

CONCLUSION

It is well understood that sex and headache do not travel in the same direction, and that the fear of orgasmic headache may become a potential reason for decreased frequency of sexual intercourse. It is supposed to depress sexual drive and libido by depriving sex of its joy and turning it into a ‘headache’. This review explained two fundamental mechanisms. The first is due to the release of calcitonin gene-related peptide, which induces the creation of less serotonin than normal, resulting in an inflammatory reaction and discomfort. The second method involves the release of epinephrine and nor-epinephrine. When adrenaline levels rise, its alpha1-agonist activity alters cerebral micro-vascular blood flow, creating cerebral ischemia and discomfort, which can easily provoke headaches in headache-prone patients.

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