Post Hemorrhagic Hemicrania Continua In A Patient With Orgasmic Paroxysmal Hemicrania

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Abstract

BACKGROUND: In its classic form sentinel headache heralds an underlying intracranial aneurysm near rupture. Headache related to sexual activity is normally benign and does not represent a sentinel headache to ulterior intracranial bleeding. Hemicrania continua is a primary headache only exceptionally precipitated by acute central nervous system (CNS) pathology.

METHODS: Clinical case history, repeated neurological examinations, ancillary imaging procedures and review of literature.

RESULTS: A patient with recurrent orgasmic headache manifested as severe left hemicrania of abrupt onset lasting for several hours, developed in the course of her illness a very severe left side headache while performing physiological valsalva during defecation, associated with an opposite side right anterior capsule intracranial bleed. Subsequently she transitioned into hemicrania continua without dysautonomia, ameliorated by valproic acid and by diet-induced weight loss. Her imaging studies showed no evidence of arteriovenous malformation or intracranial aneurysms.

CONCLUSION: On this unusual patient, orgasmic hemicrania was premonitory of a contralateral hemorrhagic stroke, representing a variant of sentinel headache later evolving into hemicrania continua.

Introduction

Paroxysmal hemicrania (PH) is a variant of trigeminal autonomic cephalalgia (TAC) a family of headache disorders also encompassing cluster headache and short lasting unilateral neuralgiform headache with conjunctival injection and tearing, or SUNCT syndrome [1, 2]. Three types of PH are identified: episodic, chronic and hemicrania continua (HC). In episodic PH the patient reports unilateral, paroxysmal, severe, sharp, throbbing or stabbing pain, with no universal precipitants and no aura [1]. Most attacks have no circadian periodicity. The duration of the attacks fluctuates between minutes to hours with a mean duration of fifty eight minutes [1, 2]. Autonomic features in the form of ipsilateral rhinorrhea, lacrimation and conjunctival irritation are common but around one third of the patients report no associated cranial dysautonomic symptoms. Although PH is a primary headache disorder, exceptionally hemorrhagic stroke may present as PH. Bazan, et al, recently described a patient with PH secondary to a contralateral upper brain stem hemorrhage [3]. In chronic PH the pain is consistently recurrent with a variable frequency, ranging from one to ten attacks a day, to one attack every few days in very rare cases [1, 2]. Hemicrania continua is not as infrequent as first believed. In the overwhelming number of cases it constitutes a primary headache. Exceptional post traumatic cases, and secondary cases due to brain tumor and to HIV infection, have been described [4]. A patient with HC associated with an ipsilateral unruptured internal carotid aneurysm was reported by Vikelis, et al [5]. Hemicrania continua may evolve from episodic PH and falls within the category of chronic daily headache, along with chronic tension type headache, newly daily persistent headache and chronic migraine. Hemicrania continua is characterized by unilateral continuous headache, waxing and waning in intensity. The baseline headache may be accompanied of autonomic signs, stabbing head pain or jabs and jolts, nausea and sound and light intolerance. In the review of HC provided by Peres, et al, on their own cases and those collected from the literature, concurrent autonomic symptoms occurred in between 63% to 73% of the patients [4]. Around 25% of those patients with autonomic symptoms reported only a single autonomic symptom. Normally PH and HC are not triggered by sexual activity, and orgasmic headache does not transition into HC, although one unusual patient previously reported in the literature, developed headaches triggered by emotion, following three episodes of coital cephalalgia [6]. A patient with HC evolving from ipsilateral cluster headache responded to the administration of valproic acid [7].

Case Report

A 48 year old female was referred for neurological consultation because intense left side continuous throbbing headache with superimposed stabbing pain,
interfering with her sleep and not responding to common analgesics. Her headache followed a small right capsular hemorrhage, triggered by straining hard during defecation because of constipation. The initial head pain concurrent with the bleeding was extreme (10/10 in VAS). She gave history of preceding transient episodes of similar left side headaches qualified as 8/10 in the VAS, coinciding with orgasms during sexual intercourse for the last several weeks. Orgasmic headaches lasted for a few hours. During an early emergency department visit while experiencing the original sexual headache, she was found hypertensive. Her neurological examination and her head computerized tomography (CT) were found normal at the time (illustration 1). Her subsequent head CT following her extreme hemicrania with defecation revealed a right anterior capsular bleed on the opposite side to the headache (illustration 2). There was no mass effect, subarachnoid hemorrhage or hydrocephalus. Her head computerized tomography angiography (CTA) showed no aneurysms, beaded or stenotic vessels and no arteriovenous anomalies. There were no signs of proliferating angiopathy (“Moyamoya disease”). When symptomatic, and later on when asymptomatic, there was no signs of ocular ptosis, miosis, weakness of eye movements, ipsilateral rhinorrhea, ocular injection or facial flushing on repeated neurological examinations. The remaining of her repeated general physical and neurological examinations was normal including her blood pressure readings. Her past medical history was significant for hypertension, insulin dependent diabetes mellitus, hyperlipidemia and nephrolithiasis. She had no history of migraine. Medications included simvastatin, lisinopril, metformin, fluoxetine and insulin at variable doses. She had no allergies. She had family history of multiple sclerosis but no family history of intracranial aneurysms, hemorrhagic stroke or migraine. Significant clinical information obtained on her review of symptoms was limited to occasional tingling of the toes, interpreted as early signs of diabetic polyneuropathy. She was prescribed valproic acid at a dose of 500 milligrams a day along with tramadol 50 milligrams four times a day, as needed. Although she was not morbidly obese she also embarked on a formal weight loss program. In six weeks her continuous headache subsided. In follow up visit fifteen months later she reports milder episodes of orgasmic headache; blood pressure readings and neurological examination continue normal.

Discussion

Orgasmic, coital or sexual headache (SH) refers to a type of exertional headache precipitated by coital or masturbatory sexual activity [8, 9]. Pre-orgasmic- and orgasmic headache are recognized by the International Classification of Headache disorders, but according to Nick and Bakouche, there is an additional form of post coital headache lasting from hours to days, as a sequel of orgasmic headache [9]. The orgasmic “explosive” type of headache is acute in onset and very intense, to the extreme that the patient feels compelled to seek emergency care, fearing a bleeding into the brain. Most SH are benign; exceptionally subarachnoid hemorrhage or embolic strokes are complications of SH. Of specific relevance to this case, Finelli reported a patient with an intracranial basal ganglia hemorrhage triggered by sexual intercourse, secondary to the rupture of a vascular malformation [10]. Finnelli’s clinical example underscores the indication for performing imaging procedures including CT angiogram (CTA), in patients with SH, especially since other intracranial vascular pathologies may coexist, such as basilar artery dissection and basilar artery stenosis [11]. Although post stroke pain may complicate up to two thirds of patients suffering strokes, usually the pain takes the form of lateralized complex regional pain syndrome to the involved (paretic) side, instead than presenting as PH or HC, as in the current patient. Valsalva maneuver precipitates intracranial bleed and cluster headache in exceptional individuals [12, 13]. Valsalva is one of the precipitating mechanisms adduced in the genesis of exertional, cough, laughing and crying headache in general, and of SH in particular. Valsalva mediated headache seemingly results from transient intracranial hypertension and pulsing maximal distortion of the intracranial vessels, on those individuals with a migraine diathesis, perhaps facilitated by incremental central pain sensitization. Central sensitization can explain why the persistent headache in some patients with exertional migraine, i.e, brought by weight lifting, on whom valsalva is only the initial trigger. As in the specific examples of SH discussed above, imaging of the brain is justified on patients with exertional headache, because the occasional occurrence of secondary valsalva-induced headache, on individuals with occult Arnold Chiari malformation and cerebellar tonsillar ectopia, originating from abnormal cerebrospinal fluid hydrodynamics [14]. Additional pathophysiological explanations advanced for the specific appearance of SH are systemic hypertension during sexual activity, loss of normal cerebral vascular autoregulation, and painless, segmental or diffuse, persistent cerebral arterial
vasospasm [15]. When vasospasm is present in a patient with SH however, it needs to be distinguished from Call Fleming syndrome or benign cerebral vasospasm, documented in some patients on whom spontaneous thunderclap headache is the initial presentation, and on whom, in rare occasions, a non-aneurysmal subarachnoid bleed is diagnosed [16]. Underlying stenosis of the intracranial venous sinus, migraine predisposition and central sensitization with repeated intercourse also are potential triggers of SH, as a variant of exertional headache [17].

According to Goadsby and Lipton prevailing hypothesis, the pathophysiology of PH and HC as a subclass of TAC, is based on the over activation of the brain stem trigeminoautonomic reflex, in which pain is mediated by the trigeminal ganglion afferents to the meninges, and its autonomic manifestations are mediated by the facial nerve parasympathetic pathways [18].

Sentinel headache, albeit a controversial diagnostic category, refers to acute headaches, usually spontaneous and taking the form of thunderclap headache, developing on individual harboring an intracranial aneurysm near full rupture, days or weeks before the actual major bleed [19]. Although an aneurysm micro-bleed or “leak” is commonly suspected as its cause, because the initial head computerized tomography (CT), and the subsequent lumbar puncture are often negative for subarachnoid hemorrhage, the diagnosis is established in retrospect, when is maybe too late. It is obvious than identifying these patient are critical to proceed to coiling or clipping of the aneurysm before a potentially catastrophic intracranial bleed occurs. Uterga, et al, described a patient with recurrent SH of bilateral temporal localization and an un-ruptured right carotid saccular aneurysm, on whom the headaches disappeared following intracranial aneurysm embolization. Uterga, et al, case highlights the fact that the discovery of an “asymptomatic” or un-ruptured aneurysm in similar patients, is not necessary an incidental finding [20]. Other than CSF subarachnoid micro-bleed, alternative hypothesis offered for the development of sentinel headache include bleeding within the wall of the aneurysm, sudden expansion of its arterial wall brought by sudden increased in blood flow and pulsation transmission, and peri-aneurysmal focal meningitis [5,19] The patient herein described represents an atypical example of sentinel orgasmic headaches for an ulceror contralateral capsular hemorrhage, ultimately precipitated by physiological valsalva, in the absence of an underlying cerebral aneurysm, arterial dissection, or arteriovenous malformation.

References

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Illustrations

Illustration 1

PRE-HEMORRHAGIC POST-ORGASMIC NORMAL HEAD CT
Illustration 2

HEAD CT SHOWS ACUTE RIGHT ANTERIOR INTERNAL CAPSULE HEMORRHAGE
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