Gastroenteric-induced Bilateral Paroxysmal Hemicrania

Corresponding Author:
Dr. Daniel Jacome,
Adjunct Associate Professor of Neurology, Dartmouth Hitchcock Medical Center Department of Neurology, One Burnham Street, Suite 2, Turners Falls, MA 01376, 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

Article ID: WMC00925
Article Type: Case Report
Submitted on: 09-Oct-2010, 01:58:28 AM GMT    Published on: 09-Oct-2010, 06:08:52 AM GMT
Article URL: http://www.webmedcentral.com/article_view/925
Subject Categories: NEUROLOGY
Keywords: Migrane, Paroxysmal hemicrania, Cyclic Vomiting, Dyspepsia, Irritable bowel Syndrome
How to cite the article: Jacome D. Gastroenteric-induced Bilateral Paroxysmal Hemicrania. WebmedCentral NEUROLOGY 2010;1(10):WMC00925

Source(s) of Funding:
No funding

Competing Interests:
No conflict of Interest
Abstract

BACKGROUND: Functional gastrointestinal disorders; i.e., irritable bowel syndrome, cyclic vomiting, gastro-esophageal reflux and dyspepsia, are more common in individuals with migraine. Autonomic manifestations of migraine, and less commonly of paroxysmal hemicranias (PH), include those of gastrointestinal (GI) tract origin.

METHODS: Repeated clinical neurological examinations, long term follow up and ancillary medical tests including imaging procedures.

CASE REPORT: An 81 year old female patient had recurrent episodes of bilateral paroxysmal hemicrania with limited cranial autonomic symptoms, consistently triggered a few minutes after beginning to eat, in the absence of concurrent GI or other autonomic symptoms. There was no specific food trigger and drinking fluids (i.e., cold water) did not cause headache. Repeated clinical examinations and ancillary testing were normal. The patient symptoms were terminated by a course of corticosteroids.

CONCLUSION: Exceptionally, bilateral paroxysmal hemicrania is precipitated by eating, in the absence of underlying structural brain lesions.

Introduction

Paroxysmal hemicrania (PH) constitutes a trigemino-autonomic cephalalgia characterized by the abrupt onset and termination of intense recurrent head pain, with a mean maximal attack duration of 58 minutes [1]. Pain is accompanied by homolateral autonomic features and the average patient experiences from 1 to 3 attacks a day. PH is by definition unilateral, although the involved side by the hemicrania may shift on occasion to the opposite side, or alternate sides [1]. PH is classified as episodic (EPC) and chronic (CPH). In CPH there are no prolonged remissions between symptomatic periods. EPH may evolve into CPH but it does not become a reflex type of headache. Pareja, et al, have advanced the concepts of “hemicrania vera” and “hemicrania generis incerti” to define its response or not to indomethacin, a highly effective non steroidal analgesic successfully employed in a majority of patients with PH [2]. An exceptional patient with bilateral PH and restricted cranial dysautonomia was recently reported [3]. He had no GI triggers for his headache. To extend the spectrum of PH, a patient with PH and visual aura was described by Brazilian authors [4].

There are numerous well known orally ingested triggers of migraine including alcoholic drinks (i.e., red wine), food and food additives. The actual act of eating or swallowing, in contradistinction to epilepsy and syncope, is not a recognized precipitant of migraine, at least as a reflex or immediate response. Of interest, an overlapping exists between migraine and eating disorders, dyspepsia, irritable bowel syndrome and gastro-esophageal reflux disease [5, 6]. A patient is herein described with bilateral PH consistently precipitated by the act of eating. No vomiting occurred but the meals had to be interrupted.

Case Report

An 81 year old female was seen in neurological consultation because of extremely severe stabbing-like headache of abrupt onset and rapid termination, experienced over both sides of her head, consistently triggered within few minutes of ingesting her meals. Along with her headache she had binocular lacrimation but no nasal congestion and only minimal conjunctiva injection. She had no facial flushing. Symptoms had begun a few months earlier and were of progressive intensity and frequency that led her to seek help. She reported “mini clusters” of several days of pain alternating with three to four headache free days. At her worst, at least 80% of her meals precipitated headaches. Her difficulty was greater with eating lunch, which represented her main meal. She could not identify a food trigger and she did not drink wine with her meals. Furthermore, drinking fluids including cold water was tolerated. Her headache lasted no more than one hour. The patient denied having any warnings and she had no associated vomiting or abdominal pain. She needed to interrupt her meals, what often spoiled what otherwise promised to be a nice leisure time with her husband, when going out to eat at a restaurant. She recalled having similar but milder unilateral headaches alternating sides, in the recent past. Past medical
history included migraine with aura that went away with menopause, and of polymyalgia rheumatica (PMR) in remission for several years. During her bout of PMR she had a normal temporal artery biopsy, despite her sedimentation rate was elevated. PMR resolved with oral steroids prescribed for several months. She had hypertension, gastroesophageal reflux and history of recurrent intestinal obstruction from old post-surgical adhesions, following initial abdominal surgery for acute diverticulitis. She was allergic to penicillin and sulfa drugs but reported no food allergies. Her mother was treated for an intracranial aneurysm. There was no family history of migraine or cluster headache. She was medicated with verapamil slow release 240 mg once a day, metoprolol 50 mg once a day, omeprazole 20 mg once a day and trazodone 50 mg at night as needed for insomnia. She complained of occasional forgetfulness. On examination her blood pressure was 140/75 mm Hg, heart rate 76 per minute and respirations 20 per minute. She was afebrile. Her temples were neither erythematous nor tender at palpation. Mental status, language, cranial nerves, muscle strength, muscle tone, deep tendon reflexes, sensory examination, gait and station, and cerebellar testing were normal. She exhibited no pathological reflexes. She had no ptosis or anisocoria. She had some mild tenderness of the deltotoid muscles on palpation. No muscle atrophy or fasciculations were observed. No involuntary movements were observed. There was no discoloration of the skin or rash. Ancillary normal tests included brain MRI, MRA of the brain and neck, CT angiogram and electroencephalogram. Her CBC, chemistries and serum protein electrophoresis were normal. Her CRP titer was normal and her ANA test was negative. Her CK levels were normal and her sedimentation rate was onemillimeter in one hour. Her headache did not respond to the sequential administration of increasing doses of lamotrigine, topiramate, indomethacin or larger doses of verapamil. Headache improved, and eventually subsided, after several months of treatment with oral prednisone, at a daily dose of twenty mg. In follow up after two and a half years she has remained free from hemicrania.

Discussion

Breweron and George brought to light the statistical correlation between eating disorders and migraine [7]. Applying two different types of clinical inventories they found common psychological profiles between these two populations of patients, which included high scores for body dissatisfaction, perfectionism, interpersonal distrust and ineffectiveness. Many of the participants in the migraine group reported diet behaviors, self induced vomiting, laxative abuse and binge eating, but had no meals induced migraine [7]. The GI accompaniments of migraine, even as persistent postdrome, are well recognized and almost universal. A puzzling migraine equivalent is cyclic vomiting, more prevalent in children, and probably under recognized in adults. A review of 45 cases of cyclic vomiting in adults was recently published [8]. Half of the patients reported dyspepsia in between attacks. Listed triggering factors were menses in women, noxious stress, pleasant excitement, fatigue and intercurrent infection, but no meal or food precipitants.

“Ice–cream headache” (ICH) could be classify as secondary “gastrointestinal migraine” (GIM) (see below). The latter condition resembles idiopathic stabbing headache but is experienced normally in the forehead or over the vertex rather than over the occipital region [9] ICH is believed to be precipitated by stimulation of palatal pain receptors. A condition akin to ICH is “cold water drinking headache” [10]. In the exceptional example of gustatory migraine, headache follows a few hours after tasting a sugary compound that triggers an immediate reflex facial pain that functions as a painful aura [11]. The differential diagnosis in any patient with eating induced migraine should also include aspartamate-induced chewing gum migraine, hypoglycemic migraine (“yom kippur headache”) and the rare case of hypoglycemia rebound migraine, generated by the normalization of glycemia following profound fasting, or insulin-induced hypoglycemia [12,13]. None of the above clinical examples however, are specifically reproduced by the act of eating

Periodicity in headache is well documented. Catamenial migraine is the most common example. The circadian variability in pain sensitivity in pericranial muscles is common knowledge. This author for instance, reported a patient with stereotyped recurrent nightly hypnagogic painful “exploding head syndrome” occurring at the same time, simulating thunderclap headache [14]. This patient periodic headache could not be strictly classified as either EPH or CPH, since it was consistently reproducible by meal ingestion and it was normally bilateral, both atypical characteristics

The pathogenesis of this patient recurrent headache is unclear. Her history of recurrent mechanical ileus and intercurrent infection, but no meal or food precipitants.
dyspeptic migraineurs, when compared to healthy controls or dyspeptic subjects without migraine [15]. These investigators measured gastric tone, gastric volume, gastric accommodation and discomfort threshold, in fasting and following the administration of a liquid meal by barostat. Discomfort threshold was lower in cases of migraine and dyspepsia but not in the group of dyspepsia without migraine or healthy volunteers. How could gastric hypersensitivity could ultimately produce PH is unknown. The study of gastric physiology in relation to its possible role in the genesis of migraine/PH is complex, and the information available on the subject is fragmentary, given the diverse background discipline and interest of the investigators. That there is a direct relation between migraine and the GI tract was demonstrated in an elegant study conducted by Aurora, et al [16]. These researchers showed by means of gastric scintigraphy the existence of gastric stasis both interictally and during spontaneous or visually triggered migraine. 

I believe it is permissible to speculate that meal induced gastric stimulation results normally in mucosal afferent vagus nerve sensory activation of the various brain stem nuclei implicated in the genesis of migraine. In this scenario, headache will only develop on individuals with a migraine diathesis. Reducing gastric mucosal hyperemia with a calcium channel blocking agent may indirectly oppose detrimental over excitation of the myenteric neuron following a meal. This patient was already on a calcium channel blocking agent when she experienced PH, negating this hypothesis. The participation of vasointestinal polypeptide (VIP), a parasympathetic neurotransmitter secreted by the myenteric plexus neurons, in synergism with secretin to cause gastric dilatation, will be appealing to investigate as a biochemical link between the stomach and the brain pain generators. The VIP role could be demonstrated experimentally by measuring postprandial plasma and salivary concentrations of this gastro-enteric hormone in normal controls and in migraine sufferers [17]. Of relevance to the topic been discussed here, indomethacin, a prostaglandin synthesis inhibitor and a highly specific agent in the therapeutic armamentarium against PH, is endowed with a protecting effect over the gastric mucosa, when not administered in excessive doses [19]. The role of serotoninergic pathways and the effects of tryptans on the myenteric neuron-vagus-trigeminal triangle needs to be explored. The above, if well disparate and at times contradictory elements of information on drugs employed in the management of headache, should entice scholars to attempt to define a coherent theory on the GI (peripheral) actions of anti-migraine pharmaceuticals, in lieu of their potential significant indirect CNS effects. Furthermore, it is conceivable, at least in some examples, that migraine represents referred cephalic pain of gastric origin, similar to the referred head pain experienced by individuals with cardiac cephalalgia. 

References

4. Peres MFP, Masruha MR, Young WB Side-shifting hemicrania continua with aura (migraine with aura with autonomic symptoms responsive to indomethacin ?). Cephalalgia 2006; 26: 917-919.
14. Jacome DE Exploding head syndrome and...
idiopathic stabbing headache relieved by nifedipine. Cephalalgia 2001; 21: 617-618
Disclaimer

This article has been downloaded from WebmedCentral. With our unique author driven post publication peer review, contents posted on this web portal do not undergo any prepublication peer or editorial review. It is completely the responsibility of the authors to ensure not only scientific and ethical standards of the manuscript but also its grammatical accuracy. Authors must ensure that they obtain all the necessary permissions before submitting any information that requires obtaining a consent or approval from a third party. Authors should also ensure not to submit any information which they do not have the copyright of or of which they have transferred the copyrights to a third party.

Contents on WebmedCentral are purely for biomedical researchers and scientists. They are not meant to cater to the needs of an individual patient. The web portal or any content(s) therein is neither designed to support, nor replace, the relationship that exists between a patient/site visitor and his/her physician. Your use of the WebmedCentral site and its contents is entirely at your own risk. We do not take any responsibility for any harm that you may suffer or inflict on a third person by following the contents of this website.