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Case Report

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An Unusual Catamenial Ailment: Trigeminal Neuralgia and Menstrual Cycle

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Abstract

A 45-year-old woman presented for trigeminal neuralgia with a unique clinical course. Her trigeminal neuralgia was exacerbated with increased severity in pain and frequency in episodes 3 to 4 days prior to, but subsided 2 to 3 days after, the start each of her menses, manifesting as a catamenial syndrome. Her trigeminal neuralgia was well controlled with low dose of carbamazepine. This is the first case report on a woman with primary trigeminal neuralgia with a plausible relationship to her menstrual cycles.

Keywords

Catamenial syndrome, Menstrual Cycle, Pain, Trigeminal neuralgia

Introduction

Trigeminal Neuralgia (TN), known as tic douloureux, is a chronic pain syndrome that affects the trigeminal nerve. Pain is typically localized to mainly the second and/or third, also infrequently the first, divisions. The incidence increases gradually with aging. Most primary (or idiopathic) TN cases begin after age of 50 years.

TN is a clinical diagnosis by history, examination and exclusion. Typically, an individual with TN will have lightening like jabs of excruciating pain which occurs and resolves on its own. These episodic attacks usually last for 20 seconds but may be prolonged up to 2 minutes or longer. The number of attacks varies from less than 1 to hundreds a day. Non-painful sensory stimulation at the trigger zones of the cheek, nose or mouth by touch, cold, wind, tooth brushing, talking, or chewing can trigger pain. Typically, TN does not occur during sleeping.

The diagnostic criteria for a classic TN include at least three attacks of unilateral facial pain occurring in one or more divisions of the trigeminal nerve with no radiation beyond the trigeminal distribution [1]. The pain should have at least three of the four following characteristics: 1) recurring in paroxysmal attacks lasting from a fraction of a second to two minutes; 2) severe intensity, electric shock-like, shooting, stabbing, or sharp quality; 3) at least three attacks and 4) precipitated by innocuous stimuli. There should be no evidence of other neurologic deficit and should not be better accounted by other diagnosis. Most cases of the primary TN are caused by a vascular compression of the trigeminal nerve root, usually within a few millimeters of entry into pons [2].

Case Report

A 45-year-old right-handed woman began to complain in October 2016 of episodic electrical shock-like sharp pain on her right face in the V2 and V3 region lasting up to 90 seconds. The pain would be triggered with cold or putting making-up on the right side of her face. No episode occurred during sleeping. Over the course of time, she noted a clear correlation between her TN episodes and her menstrual periods, namely steadily worsening episodes of her TN symptoms related with her menstrual periods. Her exacerbated TN symptoms occurred approximately 3-4 days prior to, and subsided 2 to 3 days after, the start of each her menses.

Her past medical history included chronic depression, and chronic lumbago and a surgery for her left limb fractures after a motor vehicle accident 30 years ago. She had no recent travel outside of the USA, no complaint of rash, and no use of cigarette, alcohol, or illicit drugs. No family members had TN. Her physical and neurological examinations showed normal findings except for mild residual left leg weakness from her previous motor vehicle accident.

Brain MRI with and without gadolinium initially in December of 2017 and repeated in February of 2018 showed stable findings of periventricular and subcortical white matter intensities without abnormal enhancement, mass effect or volume loss (Figure 1). Cervical spine and MRI and brain MRA with and without gadolinium were unremarkable.

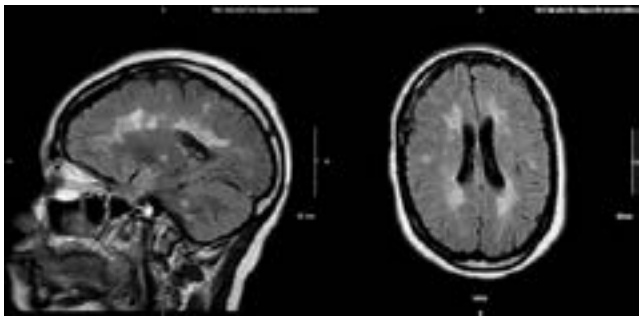

Figure 1: Brain MRI without gadolinium of sagittal (left) and axial (right) imaging show periventricular, subcortical and corpus callosum white matter intensities without mass effect or volume loss.

Laboratory studies showed all normal values including hematology, comprehensive metabolic panel, vitamin B12, folate, C- reactive protein, erythrocyte sedimentation rate, Lyme western blot, rapid plasma regain, antinuclear antibody, double stranded DNA, HIV panel, angiotensin converting enzyme, rheumatoid factor, neuromyelitis optica IgG autoantibody and *Sjögren* antibodies of anti-SS-A/SS-B. She refused lumbar puncture for CSF study.

She was started on carbamazepine 200 mg twice a day and achieved a drastic reduction in severity of symptoms and frequency of attacks. Since carbamazepine was making her sleepy during the daytime, the dose was adjusted to 100 mg twice a day. Her attacks were well controlled for 5 months then she discontinued carbamazepine on her own.

Discussion

The clinical presentation and laboratory findings in this patient were consistent with, and met, those of the diagnostic criteria for idiopathic or primary TN. Interestingly, her TN course correlated with her menstrual periods indicated a causal relationship with the fluctuation in estrogen level during menstrual cycles and suggested a catamenial syndrome involving TN, which was not seen in the literature.

Increased, followed by abruptly declined, plasma estrogen level during the menstrual cycle influences pain threshold in painful disorders including cephalic (migraine headaches, TMJ disorders, periodontal pain) and non-cephalic (fibromyalgia, chronic pain syndrome including back and neck pain) [3]. The mechanism of worsening pain symptoms correlates well with the abruptly declined level of estrogen prior to menses [4, 5]. Occurrence of migraine in reproductive-aged women related to menstrual cycles in corresponding to the withdrawal of estrogen prior to the starting menses has been termed as catamenial migraine [4], which is defined as that headache attacks occur regularly in at least 2 of 3 consecutive menstrual cycles, and occur exclusively on day 1 to 2 of menstruation, but may range from 2 days before to 3 days after [4]. A correlation in their migraine attacks with declining plasma estradiol levels during menstrual cycles has well been studied and documented [4, 5]. Notably, tension type headaches also are more frequent during the menstrual phase [6]. The highest severity of TMJ pain was observed during the late luteal phase and early follicular phases [7] and periodontal pain was found significantly greater during the peri menstrual than in postmenstrual phase after supragingival and subgingival debridement, suggesting a role of estrogen in modulating pain threshold [8]. The underlying mechanism responsible for worsening TN in our patient appeared to share the similar mechanisms through the fluctuation of estrogen level aforementioned.

The abnormal MRI findings in our patient raised a suspicion for multiple sclerosis (MS) as approximately 2% to 14% of MS patients can develop TN, which could be caused by demyelination of the trigeminal nerve root or nuclei in the pons [9]. However, no pontine pathology in MRI, no other waxing-waning symptoms rather than TN and nor relapsing-remitting course were observed in our patient, which made the diagnosis of MS in this patient unlikely.

Summary, this is the first case report on a woman with primary TN with a worsening course related to her menstrual cycles suggestive of a catamenial syndrome. The underlying pathophysiology is likely due to the fluctuation in abruptly declined plasma level of estrogen causing pain threshold changes as seen in the other pain disorders such as migraine.

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