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Is ultrasound guided pulsed radiofrequency of proximal greater occipital nerve a game-changer for the treatment of pure menstrual migraine?

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To the Editor,

Menstrual migraine (MM) is a condition in which 18% to 25% of women develop migraine attacks during their menstrual cycle.[1] MM is classified into two categories by the International Classification of Headache Disorders (ICHD-3): pure menstrual migraine (PMM) and migraine associated to menstruation (MRM).[2]

Headache experts face therapeutic challenges when dealing with MM. Compared to non-menstrual migraine, it is less responsive to pharmaceutical treatment and is typified by longer-lasting and more intense attacks.[3] The patient's migraine pattern and the occurrence of migraines outside of the menstrual cycle will determine the best course of action, which may include acute treatment, short-term preventive treatment, or daily preventive treatment.[3] Recent research on the pathogenesis of MM suggests that oxytocin agonists and calcitonin gene-related peptide (CGRP) antagonists may be effective treatment options.[4] There is research on the efficacy of blockage and pulsed radiofrequency (PRF) of the greater occipital nerve (GON) in individuals with refractory migraine, but no research on MM was found.[5] Presented here is a case study of a 37-year-old female patient with PMM who underwent ultrasound (US) guided PRF treatment along the proximal GON (PGON) and who has since been free of attacks for six months.

A 37-year-old woman who had been suffering from menstrual migraines for 15 years had been admitted to the Headache Outpatient Clinic seven months ago. During the admission, she was pain-free. However, her left-sided throbbing headaches began 2–3 days before her menstruation and persisted 3–4 days after. She denied having an aura, but she did experience photophobia, osmophobia, nausea, and vomiting during the attacks. For at least two days, she typically required bed rest and intravenous hydration treatment. She worked as a nurse in a busy hospital inpatient department. Her medications included oral contraceptives, nonsteroid drugs, and triptans. Throughout the episodes, only eletriptan worked, but she still required bed rest and hydration. However, for each cycle, she needed to utilize more than six pills of eletriptan. Furthermore, five months before her admission, she had been diagnosed with lower extremity peripheral vascular disease. Therefore, she had to quit the oral contraceptive pills, but she was frightened of more severe attacks. She was referred to the headache outpatient clinic by the gynecologist. She did not have any other primary headaches or comorbidities. She was opposed to the use of prophylactic medications such as topiramate due to their known adverse effects. The US-guided PGONB was scheduled for three days before her subsequent menstrual cycle.

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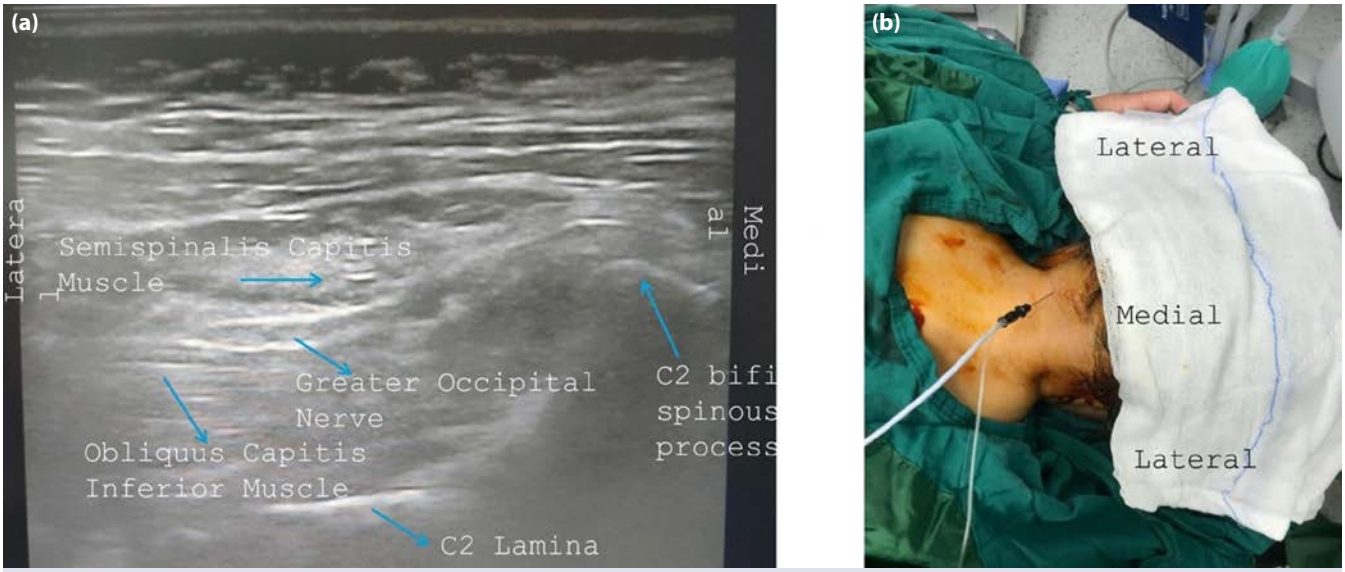

Figure 1. (a) Sonoanatomy of The Proximal GON. GON: Greater occipital nerve. **(b)** Radiofrequency needle placement.

We performed the interventions in a sterile operating room without anesthesia using an ultrasound machine with a 13–5 MHz linear probe (GE Healthcare, VolusonTM E6, Türkiye). No sedation was required. A 3-mL injection of bupivacaine 0.025% (Buvasin %0.5, Vem] was given during the diagnostic block. After the block, she did not experience any migraine attacks in the subsequent cycle. Consequently, a week later, PRF of PGON was performed not exceeding 42 degrees for 360s at 45V. The sonoanatomy of PGON and RF needle replacement were shown in Figure 1.

The patient reported no complications following the procedure. Following in the six months, her periods were as regular as they had been previously, but without migraines. She had not used any triptans or oral contraceptive pills since the first visit.

The potential of PRF of PGON as a viable alternative treatment for PMM without adverse effects is illustrated by this case. The underlying etiology of MM and how it relates to the effectiveness of treatment will be covered by the literature.

The high prevalence of migraine among women and its correlation with the early follicular phase of the menstrual cycle indicates that the decrease in estrogen was suggested as the cause of MM.[6] Falling estrogen levels enhance the sensitivity of blood vessels to prostaglandins, linked to neurogenic inflammation. In the trigeminovascular system, CGRP is modulated by changes in ovarian steroid hormone levels. The neuropeptide CGRP plays a significant role in the pathophysiology of migraines, and its signaling may be a target of hormones that affect migraine occurrence. A migraine attack is fundamentally characterized by an elevated discharge of CGRP from the trigeminal ganglia.[7] Antibodies against CGRP or receptor are highly successful in migraine treatment, and the fact that they do not pass the blood-brain barrier suggests that therapeutic intervention within the peripheral trigeminal pathway is sufficient to stop or prevent migraine attacks. An observational case study of women with MM examined the effects of erenumab in the premenstrual, menstrual, and non-menstrual periods. The results indicated that erenumab decreased the number of headache days in all phases of the cycle.[8] Further research is needed to determine the effects of CGRP antibodies on migraine attacks caused by hormonal fluctuations.

The GON is the branch of the second cervical root and the primary sensory nerve of the occipital area of the skull. Considering the convergence in the trigeminal nucleus in the upper cervical segments, the inhibition of the transmission of nociceptive information from GON can control headaches.[5] In an observational study of 40 migrenous patients, the authors showed the beneficial effect of US-guided GONB in chronic migraines that was associated with lowering interictal CGRP levels, implying a potential role for CGRP in the mechanism of action of GONB.[9] Consequently, it was theorized that the patient

did not experience an MM attack following the diagnostic block as a result of this role. Furthermore, as previously documented, PRF can affect the descending noradrenergic and serotonergic pain inhibitory pathways.[10] Therefore, the PRF of the PGON developed the six-month efficacy.

In conclusion, the GON may serve as an innovative therapeutic target for MM. Further prospective randomized controlled trials with a larger patient population are needed to conclusively demonstrate our findings whether to be as a placebo effect or not.

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