

Management of Great Auricular Neuralgia Confirmed by Electrophysiologic Examination: A Case Report

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The great auricular nerve (GAN) is a sensory branch of the cervical plexus originating from the C2 and C3 nerve roots that innervates the external ear, mandibular angle, and parotid gland. Since idiopathic GAN neuralgia is a rare condition and branches of the GAN overlap with other cervical and cranial nerves, its diagnosis is challenging and can be confused with other facial neuralgias. This article describes the case of a 55-year-old woman with intractable unilateral periauricular and lateral head pain. No significant findings were found on cervical and brain imaging. At first, the patient was suspected to be suffering from trigeminal neuralgia or great occipital neuralgia; however, the symptoms persisted despite pharmacotherapy, cervical plexus and medial branch block, and repetitive transcranial magnetic stimulation. On the basis of an electrophysiologic examination, the patient was diagnosed as having GAN lesions. Pain subsided immediately after ultrasound-guided GAN block with local anesthetics and steroids. These findings indicate that electrophysiologic studies are helpful for accurately diagnosing patients with unclear pain in the periauricular and lateral head. *J Oral Facial Pain Headache* 2018;32:e53–e56. doi: 10.11607/ofph.2144

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The great auricular nerve (GAN) is a sensory branch of the cervical plexus that arises from the C2 and C3 spinal roots.¹ It provides sensory innervation to the skin overlying the parotid gland, external ear, and posterior auricular region^{2,3}; has anatomical variability in its course, branching, and terminal distributions; and also has anastomoses with other cervical and cranial nerves.^{4,5} It has been recognized that damage to the GAN can occur from several types of neck surgery, particularly parotidectomy and rhytidectomy, from a tumor, or from prolonged pressure on the neck.^{6–11} However, idiopathic GAN neuralgia is a relatively rare condition that is challenging to diagnose and often confused with other facial neuralgias. In previous studies, the diagnosis of GAN neuralgia has been made only based on clinical history and diagnostic nerve block.^{12–15} This article describes the first case of GAN neuralgia confirmed by electrophysiologic examination that was treated successfully with ultrasound-guided GAN block.

Case Report

A 55-year-old female patient who presented with a 12-month history of intractable pain around her left ear and mandible was referred to the hospital's physical medicine and rehabilitation outpatient clinic. The patient's symptoms included paresthesia and a pulsatile sense in her left periauricular and mandibular areas, accompanied by ipsilateral ear fullness. These symptoms were aggravated when the patient turned her head to the right. The patient had a medical history of hypertension and an occupation of needlework. She had previously undergone an ear-related examination at an otolaryngology clinic, during which no abnormalities were found. She then visited a neurology clinic, where brain magnetic resonance imaging (MRI) was performed. No space-occupying lesions

Table 1 Sensory Nerve Action Potential of the Great Auricular Nerve (GAN) at Initial Assessment and 3 Months Following Nerve Block

	Onset latency (ms)	Peak latency (ms)	Peak-to-peak amplitude (μ V)
Initial			
Right GAN	1.45	2.00	10.4
Left GAN	1.90 ^a	2.65 ^a	10.0
3 mo following GAN block			
Right GAN	1.40	1.95	13.9
Left GAN	1.45	2.05	14.6
Normal reference (mean \pm SD)	1.34 \pm 0.30	1.89 \pm 0.42	22.4 \pm 17.8

^aProlonged latency of sensory nerve action potential compared to normal reference. SD = standard deviation.

Table 2 Latency of Bilateral Blink Reflex

Stimulation site	R1 (ms)	R2, ipsilateral (ms)	R2, contralateral (ms)
Right supraorbital notch	10.70	38.55	38.95
Left supraorbital notch	10.45	39.00	39.05
Normal reference (mean \pm SD)	10.45 \pm 2.52	30.5 \pm 10.2	30.5 \pm 13.2

SD = standard deviation; R1 = early response; R2 = late response.

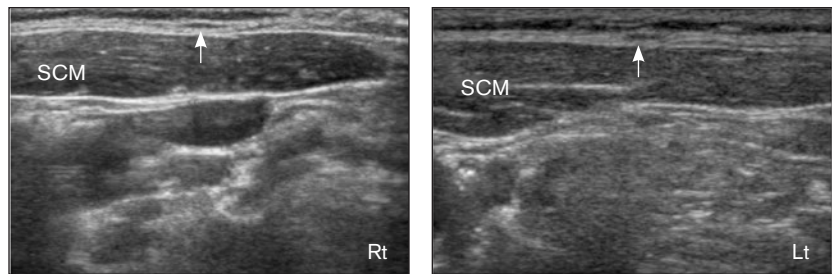
or acute intracranial findings were reported. She was diagnosed with clinical trigeminal neuralgia, and pharmacotherapy was started with ibuprofen, gabapentin, and nortriptyline. She tried several preventive medications over the span of 1 year. Despite using many different medications, including carbamazepine and alprazolam, she experienced no significant improvements; her symptoms fluctuated, but did not improve. The patient was then referred to an anesthesiologist and underwent sternocleidomastoid (SCM) muscle trigger point injection, superficial cervical plexus block, and cervical medial branch block. Despite receiving multidisciplinary medical management, the patient's symptoms did not improve.

The patient was referred to the physical medicine and rehabilitation clinic for further evaluation of her persistent intractable pain. The patient rated her pain as an intensity of 7 on a numeric rating scale (NRS) ranging from 0 to 10. Given the distribution of her pain, the patient's symptoms were suspected to reflect not only trigeminal neuralgia but also other pathologies, including great auricular neuralgia. However, the diagnosis was not clear due to the patient's complex symptoms. Due to the diagnosis of intractable trigeminal neuralgia, repetitive transcranial magnetic stimulation (rTMS) was performed. rTMS was conducted twice, but resulted in no improvement in symptoms, indicating the low possibility of trigeminal neuralgia.

In order to make an accurate diagnosis, an electrophysiologic examination of the GAN was performed. Recording electrodes 2 cm apart were placed on the back of the patient's earlobe. Stimulation was applied along the lateral border of the SCM muscle 8 cm from the active electrode.^{16,17} The examination re-

vealed a prolonged onset and peak latency (1.90 and 2.65 ms, respectively) of the left GAN sensory nerve action potential compared to the normal reference (mean \pm standard deviation [SD] 1.34 \pm 0.30 and 1.89 \pm 0.42 ms, respectively) (Table 1).¹⁶ In addition, a blink reflex test was performed to assess trigeminal and facial nerve function. The results were in the normal range (Table 2).¹⁸ Eventually, the patient was diagnosed with incomplete lesion of the left GAN. Following the diagnosis of great auricular neuralgia, ultrasound-guided left GAN block was conducted using a local anesthetic agent and steroid (Fig 1). The ultrasound machine (Accuvix V20; Samsung Medison) equipped with a 38.4-mm, 8-MHz linear array transducer was used to identify the nerve structure. With the patient in the prone position and the neck slightly rotated to the right, the GAN was identified at its emergence from the dorsal border of the SCM muscle at the level of the cricoid cartilage. The left GAN was slightly enlarged compared to the asymptomatic side. The left GAN was blocked at the point at which it winds around the posterior border of the SCM muscle. Using an in-plane approach, a 25-G, 38-mm needle was introduced, and 2 mL of 0.1% ropivacaine and 1 mL of dexamethasone (2 mg) were injected around the nerve.^{12,19} The patient reported immediate pain relief with a total absence of periauricular and lateral headache, which lasted several days. A second GAN block was performed 2 weeks later in the same manner. After two rounds of GAN block, the symptoms subsided, and the patient rated her pain as a 1 on the NRS. The symptoms did not recur. A follow-up electrophysiologic examination performed 3 months later showed that the latency of the sensory action potential of the left GAN had been restored (Table 1).

Fig 1 Transverse view on ultrasound depicting the right (*rt*) and left (*lt*) great auricular nerve (arrows) above the sternocleidomastoid muscle, slightly enlarged on the symptomatic left side.



Discussion

Idiopathic GAN neuralgia is a very rare condition. The GAN passes vertically upward over the SCM muscle and then divides into anterior and posterior branches. The anterior branch of the GAN distributes across the skin over the preauricular, parotid, and jaw angle and overlaps with the distribution of the auriculotemporal branch of the trigeminal nerve.⁵ In addition, the GAN has an anastomosis with the posterior auricular branch of the facial nerve, lesser occipital nerve, and the auriculotemporal branch of the trigeminal nerve.^{5,20,21} As a result, diagnosis of greater auricular neuropathy is challenging.

To date, evaluation of the GAN has been restricted to a diagnostic nerve block. While GAN block is an established procedure, little is known about the electrophysiologic appearance of pathologic conditions involving the GAN.¹²⁻¹⁵ In the present case of idiopathic GAN neuralgia mimicking trigeminal neuralgia, the diagnosis was strongly supported by the electrophysiologic examination. In the GAN nerve conduction study, the symptomatic GAN showed a prolonged latency of the sensory nerve-evoked potential with normal peak-to-peak amplitude. As a result of a more accurate diagnosis, the intractable pain that was resistant to multidisciplinary medical management for 1 year was treated successfully with ultrasound-guided GAN block.

Recently, high-resolution ultrasonography for diagnostic assessment of the GAN has been considered.²² In that study, two cases classified as idiopathic GAN neuralgia were evaluated. The first case showed slight swelling of the GAN over most of its course. However, in the second case, the GAN did not show any morphologic alterations on ultrasonography. In the present case, the GAN was slightly enlarged on the symptomatic left side compared to the contralateral side, implying nerve swelling due to compression or neuritis. This finding is consistent with the prolonged latency of the GAN in the electrophysiologic study.

This is the first report of a case in which electrophysiologic examination was used to confirm

idiopathic GAN neuralgia, suggesting that electrophysiology may be used for the diagnosis of chronic refractory periauricular and lateral headache in patients. A thorough work-up comprising a complete history, physical examination, and brain imaging studies is crucial. Electrophysiologic studies should also be considered in order to avoid diagnostic errors in cases of periauricular orofacial pain and unnecessary treatment. However, it remains a limitation of this case report that this useful diagnostic approach has not been reproduced in other cases due to the lack of such condition.

Conclusions

GAN neuralgia is an uncommon cause of facial pain that can mimic trigeminal or occipital neuralgia. An electrophysiologic examination may be helpful for accurate diagnosis in patients with unclear pain in the periauricular and lateral head and to quantify the effect of interventions on GAN lesions, but further investigations are needed to ensure the reliability of the electrophysiologic diagnosis of GAN neuralgia.

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