Nerve block as neuropathic pain treatment for the great auricular nerve neuropathy: A case report

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INTRODUCTION

The great auricular nerve (GAN) is a major sensory branch of the cervical plexus. It emerges through the posterior border of the sternocleidomastoid muscle.1 The relatively superficial location makes it particularly vulnerable to cranial or cervical trauma and iatrogenic procedures.2 As in other terminal branch neuralgias and neuropathies, pain is circumscribed to the territory of the terminal branch, and the pain is temporarily eased after selective anesthetic blockade of the nerve.3 The clinical symptoms are circumscribed to the territory of the GAN, which include the inferior preauricular region, the jaw angle, the ventral pinna, and the mastoid region.4

GAN neuralgia and GAN neuropathic pain are rare and not included in the International Classification of Headache Disorders, 3rd edition (ICHD-3)3 nor in the International Classification of Orofacial Pain.5 A confusion exists within the literature regarding the distinction of these terms. In the ICHD-3, a distinction is made between neuralgia and painful neuropathy in the description of other cranial
neuropathies such as the trigeminal nerve. Neuralgic pain included paroxysmal attacks in the distribution of the nerve lasting from seconds to minutes, whereas neuropathic pain is usually continuous and superimposed brief pain paroxysms may occur, and there is usually hypohesthesia in the territory of the nerve on examination. In addition, neuralgia is usually idiopathic, and neuropathy may be secondary to another process.

To date, 17 cases of GAN neuralgia have been reported. GAN neuropathy is more common; cases related to iatrogenic manipulations or cervical trauma typically resolve within a few months. In 2019, Duvall et al. proposed diagnostic criteria for GAN neuralgia, which included the presence of paroxysmal attacks in the distribution of the GAN, the presence of at least two of the following three characteristics: recurring pattern with attacks lasting from seconds to minutes, severe intensity and/or shooting, stabbing or sharp quality. In addition, the pain must be accompanied by dysesthesia or alodynia during innocuous stimulation of the nerve territory, must be precipitated by neck rotation, or must exhibit prominent tenderness or Tinel’s sign upon palpation. As with other cranial neuralgias, the pain must be eased by selective anesthetic blockade of the nerve, and it could not be better accounted for by another ICHD-3 diagnosis.

We identified 21 cases using a PubMed search for the terms “auricular neuralgia,” “great auricular nerve block,” and “auricular neuropathy,” and we include 17 cases that meet the criteria for GAN neuralgia and 4 cases that meet the criteria of GAN neuropathy.

All the reported cases are unilateral. Out of 21 cases, 12 were idiopathic, and the remaining were related to submandibular gland resection, pacemaker placement, Sjögren syndrome, trauma or localized tumors (low-grade lymphoma). Tables 1 and 2 summarize the main demographic, clinical, and therapeutic characteristics of the GAN neuralgia and GAN neuropathy published cases, respectively.

From a therapeutic point of view, various drugs have been used, with little relief, including gabapentin, carbamazepine, tricyclic antidepressants, opioids, or anti-inflammatory drugs. Out of 21 patients, 12 were treated with GAN block, with local anesthetic agent and steroids with excellent response in 10 out of 12 patients and local anesthetic agent in 2 out of 12 patients, also with successful results. The anesthetic blockade technique usually describes the injection proximal to the bifurcation of the nerve, along the sternocleidomastoid muscle. In addition, in 10 cases, the blockade is guided by ultrasound (US). Because US is not always available and the anesthetic blockade is not only a diagnostic criterion but also the most effective therapy, we present a case with two singularities. First, it is the first case of bilateral GAN neuropathy published so far. Second, we describe a novel technique for the anesthetic blockade, which does not require US to be done.

CASE REPORT

We present a 46 year-old right-handed woman with prior medical history of insomnia, meniscectomy, and lumbar disk herniation. The patient was referred to our outpatient headache clinic due to a 10-year history of facial pain. She describes a stable frequency of at least 20 days of pain per month, following a continuous pattern, and without specific circadian variation. The pain was symmetrical in localization and clinical phenotype and circumscribed to both preauricular and infra-auricular regions, and 2 cm retro-auricular (Figure 1). She denied auricular or intra-auricular pain. She had never experienced pain in any other territory. The quality of the pain was described as shooting and burning, without pulsating, intermittent stabbing, or oppressive pain. The mean intensity was judged to be 9 out of 10 on a 0–10 numeric rating scale. The pain was acute and stabbing paroxysms of severe intensity with constant mild discomfort. She had no photophobia, phonophobia, osmophobia, nausea, or vomiting. The pain was not aggravated by routine physical activity, but it increased, and was precipitated by cervical horizontal rotation and prominent jaw movements. There was no change or precipitation of the headache related to postural changes or to Valsalva maneuvers.

The pain was not alleviated by paracetamol/acetaminophen 1 g, metamizole 575 mg, or ibuprofen 600 mg. As prophylactic therapies, she had used amitriptyline (50 mg/24 h), gabapentin (300 mg/8 h), tizanidine (12 mg/24 h), pregabalin (75 mg/8 h), duloxetine (60 mg/24 h) during at least 3 months each, with no clinical relief. She had a normal cranial magnetic resonance imaging (MRI), a normal cervical MRI, and a normal temporomandibular joint MRI. The MRI did not include thin cuts thorough the trajectory of GAN. She had been evaluated by otolaryngology and maxillofacial surgery, with no evidence of underlying cause. Ancillary laboratory exams showed a normal basic autoimmune workup, absence of serological evidence of recent infection, and normal acute phase reactants.

The clinical examination of the patient showed a very subtle hypohesthesia circumscribed to the territory of both GANs (Figure 1, obtained with the patient’s approval and after signing the informed consent form) and focal hypersensitivity to the jaw angles and mastoid processes palpation. We performed an anesthetic block, using 2% lidocaine, 0.75 cc in two points. First, we infiltrated the ventral branch of the GAN, over the jaw angle, locating the nerve by palpation, where the patient described the pain as maximal, and directed the needle in both craniocaudal and ventrodorsal directions. Second, we infiltrated the dorsal branch, using as reference the mastoid process. The nerve was located by manual palpation, where the patient described the maximal pain, and we directed the needle in the craniocaudal and ventrodorsal directions (Figure 2). Following infiltration, the patient lay down for 5 minutes. The patient described complete disappearance of the pain for 2 weeks, and significant relief that lasted for 2 months. Thereafter, the pain reappeared, and we repeated the blockade, four times so far, with identical therapeutic responses. The patient experienced no adverse effects.

DISCUSSION

Painful GAN neuropathy is a relatively rare cause of facial pain; however, it might be underdiagnosed. Herein, we present the first
TABLE 1 The main demographic, clinical, and therapeutic characteristics of the GAN neuralgia published cases

| Patient no. | Author | Year | Age at onset | Sex | Time of evolution | Quality | Intensity | Paroxysms | Associated symptoms | Symptomatic treatment | Preventive treatment | Nerve block | Time of response after nerve block |
|-------------|--------|------|--------------|-----|------------------|---------|-----------|-----------|-------------------|---------------------|---------------------|-------------|----------------------------------|
| 1 | Fukushima et al. 2017 | 2017 | 60 | M | 6 months | Tingling | Mild | Yes | No | No | Electroacupuncture therapy | No | – |
| 2 | Maimone-Baronello et al. 2003 | 2003 | 75 | M | 7 months | Electric | Severe | Yes | No | No | Gabapentin | No | – |
| 3 | Eghtesadi et al. 2017 | 2017 | 41 | F | 1 year | Sharp and tingling | Severe | Yes | Allodynia with chewing | Naproxen, acetaminophen, muscle relaxant, tapentadol, opioids, topical analgesics | Gabapentin, tricyclic antidepressant, carbamazepine, bupropion | Yes | 1 month |
| 4 | Jeon et al. 2017 | 2017 | 25 | M | 10 days | Burning, sharp, lancinating | Severe | Yes | No | Tramadol | Pregabalin, amitriptyline | Yes | 6 months |
| 5 | Duvall et al. 2020 | 2020 | 40 | F | UNK | Stabbing | Severe | Yes | No | Acetaminophen | Pregabalin, gabapentin, carbamazepine, baclofen | Yes | 4 months |
| 6 | Duvall et al. 2020 | 2020 | 54 | M | UNK | Stabbing | Severe | Yes | No | Hydrocodone, cyclobenzaprine | Carbamazepine | Yes | 3 months |
| 7 | Duvall et al. 2020 | 2020 | 34 | F | UNK | Sharp, stabbing, pressure | Severe | Yes | No | No | No | Yes | 2 years |
| 8 | Duvall et al. 2020 | 2020 | 11 | F | UNK | Stabbing | Severe | No | No | Prednisone | Pregabalin, gabapentin, amitriptyline, duloxetine, oxcarbazepine | Yes | Weeks |
| 9 | Duvall et al. 2020 | 2020 | 36 | F | UNK | Stabbing, dull aching | Severe | No | No | No | No | Yes | 5 months |
| 10 | Duvall et al. 2020 | 2020 | 55 | F | UNK | Stabbing and burning | Severe | Yes | No | No | Pregabalin, gabapentin | Yes | Months |
| 11 | Duvall et al. 2020 | 2020 | 56 | F | UNK | Sharp, stabbing | Severe | No | No | No | No | No | – |
| 12 | Duvall et al. 2020 | 2020 | 46 | F | UNK | Sharp, shooting | Severe | Yes | No | No | Gabapentin | Yes | 7 weeks |
| 13 | Duvall et al. 2020 | 2020 | 51 | M | UNK | Sharp, shooting | Severe | Yes | No | No | Gabapentin | No | – |
| 14 | Duvall et al. 2020 | 2020 | 59 | F | UNK | Sharp, shooting | Severe | Yes | No | No | Gabapentin, carbamazepine | No | – |
| 15 | Duvall et al. 2020 | 2020 | 49 | F | UNK | Shooting, Sharp | Severe | Yes | No | No | Pregabalin, gabapentin, cervical facet blocks | No | – |
| 16 | Duvall et al. 2020 | 2020 | 54 | F | UNK | Stabbing, pressure | Severe | Yes | No | No | No | No | – |
| 17 | Duvall et al. 2020 | 2020 | 36 | F | UNK | Sharp, stabbing | Severe | Yes | No | No | Pregabalin | No | – |

Abbreviations: F, female; M, male; UNK, unknown.
case of bilateral GAN neuropathy, and we describe a modified anesthetic blockade that may help in the diagnosis and treatment of this condition.

In evaluating the published cases, the mean age of those with GAN neuralgia was found to be 46 years, with a female:male ratio of 2.4:1. The quality of the pain is usually described as shooting or lancinating, with paroxysms of pain in 82% of the cases. Besides the fact that the pain is exquisitely circumscribed to the territory of the GAN, some criteria that may help in the diagnosis are the aggravation of the pain by cervical movements and the complete relief after the anesthetic blockade. The presence of persistent pain or sensory deficit possibly raises suspicions of a secondary etiology, and it would suggest a GAN neuropathy rather than a GAN neuralgia. Its absence in the ICHD-3 and the relatively low number of reported cases potentially contributes to its under-recognition. In addition, some patients may consult orofacial and/or otorhinolaryngology clinics rather than pain clinics.

The GAN is a branch of the upper cervical plexus, which arises from the C2 and C3 spinal cervical nerves. It comes from the back edge of the sternocleidomastoid muscle and extends up to the pinna, behind the external jugular vein to later divide itself near the jaw angle in two principal branches. The ventral branch innervates the skin of the preauricular region, the jaw angle, and the parotid. The dorsal branch innervates the skin of the ventrocaudal pinna and the mastoid region. The provocation by cervical movements may be caused by the nerve entrapment within the sternocleidomastoid heads.

As with other terminal branch neuralgias and neuropathies, the anesthetic blockade is helpful both for diagnosis and treatment. The anatomy of the nerve complicates the anesthetic blockade. Contrary to other nerves, part of the nerve goes over soft tissue, which may be harmed by the blockade. We took advantage of the nerve trajectory toward the jaw and the mastoid process. Injecting above a bone surface may minimize the diffusion of the anesthetic and the possibility of complications. In the case of the jaw, the specific direction of the needle aims to avoid the parotid gland and the masseter muscle (Figure 2). Some authors have suggested the use of US for the nerve location; however, the availability of, and experience with, the technique may result in variability in the results. We used the Tinel’s sign as guidance, expressed by tenderness over the affected nerve branch, similar to other terminal branch neuralgias. Given the lack of anatomical foramen or notch, as occurs with the mental nerve, supraorbital nerve, or infraorbital nerve, the patient lie down to avoid the caudal spread of the anesthetic due to gravity.

Anesthetic blocks are relatively safe procedures with adverse effects being mild and temporary in most cases. The most frequent adverse events are vasovagal syncope, allergy to local anesthesia, and local bleeding. In the case of GAN, due to the anatomical structure of the nerve, patients may experience local hematomas, circumscribed pain, subcutaneous infection, or oral cavity bleeding if salivary glands are punctured.
In conclusion, GAN neuropathy is a rare cause of localized facial pain. Being the first bilateral occurrence reported to date, our case report should be considered in the differential diagnosis of localized facial pain. It is characterized by a shooting or lancinating and paroxysmal pain along the distribution of the GAN. As with any anesthetic nerve block, GAN block has a diagnostic and therapeutic role, offering long-lasting relief for weeks or months without adverse systemic effects. The blockade can be done over the jaw angle and the mastoid process.

**CONFLICT OF INTEREST**

The authors declare no conflicts of interest.

**AUTHOR CONTRIBUTIONS**

*Study concept and design*: Silvia Moreno Pulido, David García-Azorín.

*Acquisition of data*: Silvia Moreno Pulido, David García-Azorín.

*Analysis and interpretation of data*: Silvia Moreno Pulido, Ángel Luis Guerrero Peral, David García-Azorín.

*Drafting of the manuscript*: Silvia Moreno Pulido. *Revising it for intellectual content*: Silvia Moreno Pulido, Ángel Luis Guerrero Peral, David García-Azorín. *Final*
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PATIENT CONSENT

Fully informed, voluntary and written consent to publish was obtained from the patient.

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