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LETTERS TO THE EDITOR

Refractory neuralgia of the superior laryngeal nerve: A case study^{☆,☆☆}



Neuralgia del nervio laríngeo superior: a propósito de un caso de difícil control

Dear Editor:

Superior laryngeal neuralgia is a rare entity first described in 1900 by Avellis.¹ It is characterised by severe pain in the lateral aspect of the throat, the submaxillary region, and/or under the ear.

From an anatomical point of view, the superior laryngeal nerve branches off the vagus nerve and descends to the larynx along with the carotid artery. At some point in its trajectory, the superior laryngeal nerve divides into an external and an internal branch.² The external branch of the superior laryngeal nerve is essentially motor, while the internal branch provides sensory innervation. The internal branch penetrates the thyrohyoid membrane and innervates the base of the tongue, the epiglottis, and the mucous membrane of the larynx.³

Here we present the case of a patient with idiopathic superior laryngeal neuralgia whose symptoms were especially difficult to manage.

The patient was a 61-year-old man with no relevant medical history who presented at the headache unit due to a 6-month history of pain, characterised by paroxysms of severe stabbing pain (9 out of 10 on the visual analogue scale [VAS]; 0 = no pain, and 10 = worst pain imaginable) irradiating from the right side of the neck to the submandibular area and below the ipsilateral ear. Paroxysms lasted between 30 seconds and 1 minute and were triggered by swallowing and straining his voice. During the neurological examination, pain upon palpation of the right thyrohyoid membrane was observed. The otorhinolaryngological examination and

the imaging tests (cranial MRI and cervical CT) revealed no alterations.

Pharmacological treatment with carbamazepine at 500 mg/day was initiated but the patient showed no response. He was subsequently treated with lamotrigine (100 mg/day) and pregabalin (150 mg/day), but no improvement was seen. Ultimately, lacosamide at a dose of 150 mg/day achieved almost complete pain control.

The causes of superior laryngeal neuralgia can be classified as either central or peripheral. Pain worsened by swallowing is thought to be due to central causes. Peripheral causes include scarring from carotid artery surgery,⁴ deviation of the hyoid bone,⁵ amygdalectomy,⁶ lateral pharyngeal diverticulum,⁷ or trauma⁸. None of these findings or relevant history were present in our patient.

Patients must respond to a local anaesthetic block to be diagnosed with superior laryngeal neuralgia according to the criteria of the second edition of the International Classification of Headache Disorders (ICHD-2) (code 13.4).⁹ However, we did not perform this procedure in our patient due to its technical complexity and the large amount of anaesthetic it requires. Other authors indicate anaesthetic block only for cases refractory to medical treatment. Besides this, the high dose of local anaesthetics involved may cause wallerian degeneration with Schwann cell damage and axonal dystrophy.¹⁰ Despite the lack of an analysis of our patient's response to anaesthetic block, his clinical symptoms and the identification of a trigger in the thyrohyoid membrane supported the diagnosis of superior laryngeal neuralgia.

The differential diagnosis of superior laryngeal neuralgia includes such underlying structural conditions as inflammatory processes of the larynx and laryngeal neoplasms.¹¹ In our case, these conditions were ruled out after an otorhinolaryngological examination. Neuralgia of other peripheral nerves differs from superior laryngeal neuralgia in the location of pain and trigger points. Carotidynia resembles superior laryngeal neuralgia in that it is characterised by throbbing pain in the anterior cervical region which may be accompanied by migraine-like headache.¹² Palpating over the carotid bifurcation induces pain and helps distinguish between the 2 entities.

Superior laryngeal neuralgia is an uncommon condition that requires prompt treatment; early identification and management may avoid unnecessary procedures.¹³ Control of neuropathic pain can be difficult on occasions. Among antiepileptics, also called neuromodulators, lacosamide has a novel action mechanism which enhances slow inactivation of voltage-gated sodium channels without affecting fast

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inactivation. This, combined with its high oral bioavailability, low plasma protein binding, and minor pharmacological interactions,¹⁴ makes lacosamide an option to consider in the management of neuralgia.¹⁵ In our patient, the use of this drug was decisive.

In summary, a diagnosis of superior laryngeal neuralgia should be considered in patients with unilateral paroxysmal pain in the lateral aspect of the neck and the ipsilateral ear. Lacosamide may be a good option in refractory patients before resorting to more invasive techniques such as anaesthetic block.

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C. Rodríguez-Martín, C. de la Cruz Rodríguez,
M. Ruiz Piñero, Á.L. Guerrero Peral*

Servicio de Neurología, Hospital Clínico Universitario de Valladolid, Valladolid, Spain

* Corresponding author.

E-mail address: gueneurol@gmail.com

(Á.L. Guerrero Peral).

Pontine and extrapontine myelinolysis secondary to glycemic fluctuation[☆]



Mielinólisis pontina y extrapontina secundaria a fluctuaciones en la glucemia

Dear Editor:

Central pontine myelinolysis is an osmotic demyelination syndrome which, in the majority of cases, occurs after rapid correction of hyponatraemia.¹ Alcoholism, malnutrition, the prolonged use of diuretics, and liver transplantation are associated with increased risk for developing this syndrome.² Hypernatraemia and, to a lesser extent, hypokalaemia and treatment with magnesium or lithium have also been suggested as possible trigger factors together with hyponatraemia.³ We present the case of a patient who developed central pontine and extrapontine myelinolysis secondary to glycaemic fluctuations.

The patient was an 84-year-old woman who was found in her home with a low level of consciousness and hypoglycaemia (60 mg/dL). Her medical history included arterial hypertension, dyslipidaemia, and type-2 diabetes mellitus; levels of glycated haemoglobin (11.5% 6 months before) suggested poor glycaemic control. The patient had presented at the emergency department 2 days previously with dizziness and nausea. Nonketotic hyperglycaemia (600 mg/dL) was observed at that point and, as a consequence, the patient's normal insulin dose was increased. She lived alone and was independent, although in the preceding few months some self-neglect had become apparent, as the patient did not adhere to treatment and had poor hygiene habits. During the first few days after admission, our patient's level of consciousness improved. Three days later, she displayed no metabolic alterations and underwent a neurological examination where she was found to be alert but showed practically no initiative or voluntary activity. Extensor plantar response was observed bilaterally. The results from the examination of the sensory system and cranial nerves were normal. Spoken language, while infrequent, was normal and showed no signs of aphasia. Likewise, the patient displayed adequate verbal comprehension. Our patient's symptoms were therefore compatible with akinetic mutism; she also had signs of bilateral corticospinal dysfunction. A brain MRI revealed hyperintensities in the pons and both middle cerebellar peduncles in T2-weighted sequences (Fig. 1). These

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