



Primary auriculotemporal neuralgia. Case report

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Introduction

Neuropathic pain results from a primary nervous system injury or disorder, triggered by local trauma or systemic diseases and affecting peripheral or central nervous structures. Considering the symptom, orofacial neuropathic pain may be classified in two categories: episodic or continuous. Episodic neuropathic pain is characterized by pain paroxysms described as electric shock or twinge, similar to what happens with trigeminal neuralgia (TN). Pain paroxysms are in general followed by remission periods, known as refractory periods. Pain may be classified in primary (classic) or secondary (symptomatic) and the difference between them is important for adequate diagnosis and management. While the vast majority of patients have brief, severe and paroxysmal pain, continuous pain may also be present. Most common neuralgias are trigeminal, post-herpetic, glossopharyngeal and occipital. Less frequent are supraorbital and intermediate nerve neuralgia. These disorders may underlie infectious and/or inflammatory neurologic diseases, in addition to other primary headaches. The auriculotemporal nerve (n.AT) is a sensory branch originating from mandibular nerve posterior trunk. Due to its pathway, there is major risk for compression and irritation. Its nervous roots form a short trunk, which supplies several branches and innervates temporomandibular joint (TMJ), temporal region, pinna, and external acoustic meatus. It conducts sympathetic fibers to the scalp and parasympathetic fibers to the parotid gland. Anatomic relationship between n.AT and masticatory muscles, TMJ and surrounding vessels in the infra temporal fossa region, creates favorable conditions for compressive syndromes. Auriculotemporal neuralgia (NAT) is an uncommon condition. In a tertiary center, reported frequency was just 0.4%. It seems to be more prevalent in middle-aged females. Symptoms are excruciating pain attacks, especially in the temporal region. Pain in TMJ, parotid and ear, with irradiation to temporal region is also described and may be relieved by auriculotemporal nerve anesthetic block. Although not having its own classification, the name epicranial neuralgias has been suggested, including neuralgias of other peripheral branches, such as supraorbital, supratrochlear, nasal, greater and lesser occipital nerves.

Objective

This study aimed at reporting a case of NAT where underlying secondary factors were not found, and which has favorably responded to low dose of carbamazepine.

CaseReport

Male patient, 72 years old, who came for assistance complaining of severe left temporal region pain, described as shock. First crisis had been 5 years ago with spontaneous remission. Two months ago pain reappeared, with very short duration, with several episodes varying from 1 to 2 minutes. These episodes were repeated three to four times a day and did not wake up patient at night. At physical evaluation, no pain trigger-zone was found. Patient had controlled diabetes and referred having been submitted to pros-tatectomy due to cancer. Brain magnetic resonance was normal. Panoramic X-rays of jaws and computerized tomography of tempo-romandibular joints had not shown significant changes. Diagnostic hypothesis was NAT. Carbamazepine (200mg) was prescribed during the first two days, continuing with 400 mg for the following 15 days. In the first week using the drug, patient was reevaluated and reported lower frequency of shocks (2 to 3 per day), which he defined as "pinching". In the following week, patient referred symptoms remission. Maintenance dose of 200mg was then kept and 4 months later there has been total symptoms control.

Conclusion

This case shows that NAT has clinical presentation similar to that of other neuralgias. The diagnosis of this uncommon condition is primarily obtained by evaluating pain characteristics and by excluding possible secondary causes.

Keywords Auriculotemporal Nerve, Auriculotemporal Neuralgia, Neuropathic Pain