J Stomatol Oral Maxillofac Surg xxx (2017) xxx–xxx

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53rd SFSCMFCO Congress

Trigeminal neuralgia (TN): A descriptive literature analysis on the diagnosis and management modalities

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ARTICLE INFO

Article history:
Received 31 May 2017
Accepted 19 June 2017

Keywords:
Trigeminal neuralgia
Retrosigmoid approach
Micro-vascular decompression

ABSTRACT

Objective: The primary objective of this review is to explore the different aspects of the diagnosis and management of Trigeminal neuralgia (TN). We look at the role of radiological imaging in the work-up of this condition, and based on the findings in the literature, we report data on the medical and surgical management of TN.
Materials and methods: A literature review was conducted using PubMed and Cochrane search engines in order to explore the data available on the diagnosis and management of TN. Clinical features and various treatment modalities were analyzed by the authors. The identified studies were evaluated and data was reported on the different aspects of the condition in order to provide an evidence-based update on the topic.
Discussion: The diagnosis of TN is based on the patient's clinical history and radiological imaging. The commonest cause of TN is a micro-vascular compression by a looping blood vessel. Radiological evaluation is critical in the work-up of the disorder and in order to eliminate other possible causes. Management of the disorder can be medical or surgical, with micro-vascular decompression having the highest remission rate.
Conclusion: Patients with TN present paroxysmal pain attacks in the territories innervated by the trigeminal nerve. Diagnostic investigations must allow precise anatomical evaluation of the CPA, and MR imaging is the gold-standard radiological investigation for this purpose. Management of TN can be medical or surgical, with micro-vascular decompression having the highest success rate.

1. Introduction

Trigeminal neuralgia (TN) is one of the commonest cranial neuralgias. It is a condition that causes severe paroxysmal pain in one or several territories innervated by the trigeminal nerve. Although the symptomatology seems fairly straightforward, clinical diagnosis of this disorder is very complex, and the symptoms are frequently mistaken for dental or jaw pain, leading to unnecessary radiological investigations and, often, unrequired surgical interventions [1].

The reported prevalence of actual TN in the literature is that of 0.07% in the general population, which makes only a small percentage of the prevalence of general facial pain, which is reported at approximately 2% [2].

2. Definition and classification of TN

In order to avoid confusion with regards to the diagnosis of TN, researchers and many international medico-surgical associations have proposed strict definitions and classification systems. TN is defined by the International Association for the Study of Pain (IASP) as a 'sudden, usually unilateral, severe, brief, stabbing, recurrent episodes of pain in the distribution of one or more branches of the trigeminal nerve' [3]. This definition highlights the importance of paying close attention to the description of the pain and its characteristics as outlined by the patient. The pain is classically paroxysmal, usually unilateral (except in certain etiologies), and is described by patients as 'stabbing' or similar to an 'electric shock'. Although these pain paroxysms are sometimes described as arriving 'spontaneously', most patients can identify triggering factors. This trigger, or stimulus, can be as simple as a touch or even a hint of air. Even simple facial movements can be triggers for pain paroxysms. Furthermore, the location of the pain may be different from the zone where a stimulus is applied, and can therefore be felt

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http://dx.doi.org/10.1016/j.jormas.2017.06.010
2468-7855/

as a 'radiating pain' [2]. Understandably, this condition can cause severe disability in pursuing simple daily activities such as eating and shaving, and can, equally, lead to severe mental illness and even clinical depression [4].

The use of different terms to classify different sub-types of TN has often led to confusion in recent years. These terms include *classic, idiopathic, secondary and symptomatic*. These terms have been used to guide physicians towards the alleged etiology of the symptoms. These causes include several neurological disorders, such as multiple sclerosis, neoplasms of the cerebellopontine angle (CPA), and vascular compression of the trigeminal nerve [2,4]. To demystify the classification dilemma surrounding TN, the disorder has been classified under three categories, further detailed by the International Headache Society (IHS) [2,5,6]:

- idiopathic TN: this category is used to identify patients presenting signs and symptoms compatible with TN, except that no identifiable cause is found after detailed clinical examination and radiological investigations;
- classic TN: this term is used to describe TN caused by vascular compression of the trigeminal nerve root;
- secondary TN: the term secondary is used when the TN is a clinical manifestation of an underlying disease, such as multiple sclerosis, or when it is caused by a space occupying lesion or a tumor of the CPA.

Of all of the above categories, classic TN caused by arterial or venous compression of the trigeminal nerve at the root entry zone (REZ) is by far the commonest cause, accounting for 80–90% of all TN cases (Fig. 1) [7–9].

One of the established pathophysiological theories in both classic and secondary TN is focal demyelination of the trigeminal nerve at its entry into the pons [10]. It is believed that this zone, also known as a *locus minoris resistentiae*, represents a site of neural vulnerability and higher susceptibility to damage by compression because it is the site of Schwann cell substitution by oligodendroglia in the formation of the myelin sheath [2]. Three other major theories exist on the pathophysiology of neuro-vascular compression in TN. In 1984, Nielsen [11] proposed the concept of ephaptic transmission, where the damaged axons in the area of focal demyelination, caused by the vascular compression, become a source of ectopic generation of high frequency discharges. This process of hyper excitability and ectopic high frequency impulses is believed to continue in a vicious cycle, known as the cascade effect, as long as the compression lasts, and would therefore only be relieved by vascular decompression.

In 1987, Moller et al. [12] proposed the nuclear theory. Here, the authors believe that the compression caused by the offending blood vessel causes a nuclear hyperactivity in the injured axons along with a chronic, self-sustaining neural discharge in the trigeminal ganglion – a process here-by called 'trigeminal ganglion ignition'. A few years later, Roth et al. [13] proposed a pathophysiological explanation that unifies the two previous theories proposed by Nielson and Moller. This 'unified' theory reports that there is actually a combined process involving ephaptic, ectopic transmission of axonal discharges and nuclear hyperactivity in the pathophysiology of TN.

3. Diagnosing TN

The diagnosis of TN is based on clinical grounds, and therefore, the vital step in making the diagnosis is a comprehensive clinical history with particular attention to the patient's description of the symptoms. Similarly, the patient's description must fulfill at least two criteria in order to identify a possible TN: the pain must be characteristically paroxysmal, and it must affect only the territories innervated by the trigeminal nerve. The presence of those two criteria must raise high clinical suspicion of TN in the physician's clinical work-up [2].

Furthermore, in order to confirm this clinical suspicion, detailed, high-resolution radiological examinations are fundamental. The imaging technique chosen must be capable of eliminating all possible differential diagnoses, including neoplasms and neurological diseases such as multiple sclerosis which could cause TN. Moreover, this radiological examination must enable detailed visualization of any vascular compression along the trajectory of the trigeminal nerve and/or other cranial nerves.

Based on our local experience in the department of otology and oto-neurological surgery in the hospital of la Conception in Marseille, Magnetic Resonance Imaging (MRI) is the gold standard radiological imaging in the diagnosis of TN, especially in outlining any vascular compression, which is the commonest cause, accounting for more than 85% of cases (Fig. 2) [7–9]. This local practice is supported by the literature and evidence-based medicine worldwide. In their article on the neuro-vascular relationship at the trigeminal REZ, Lang et al. [14] have equally affirmed that MRI with high-resolution T2-weighted images is the imaging tool of choice in visualizing any vascular compression on cranial nerves. Similarly, Elaini et al. [15] have declared MRI in T2 sequences as the key assessment of neuro-vascular compression.

There is no doubt that recent advances in MRI techniques allow precise evaluation of the vascular intra-cerebral anatomy and,

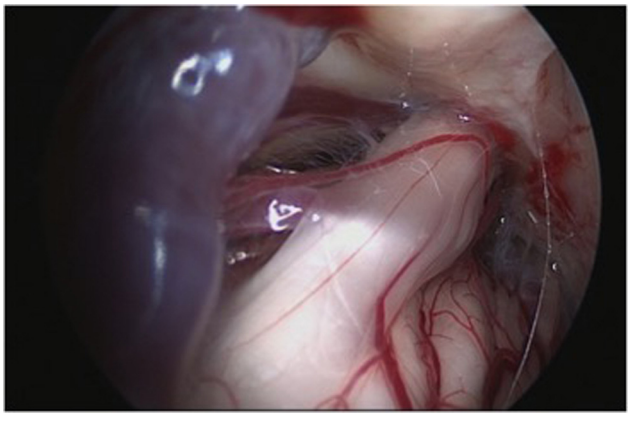

Fig. 1. Intra-operative, endoscopic view of a neuro-vascular compression at the root of the right trigeminal nerve.

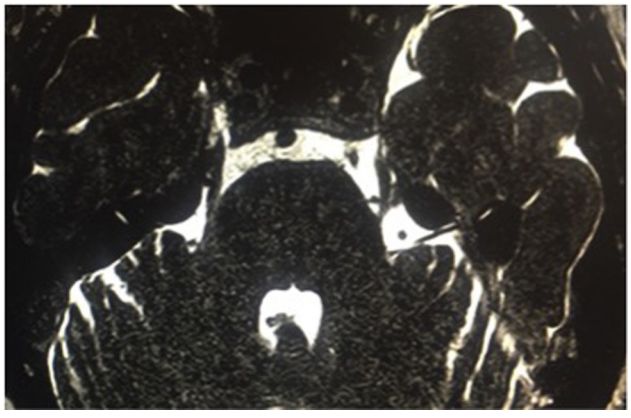

Fig. 2. MRI imaging (T2-CISS) showing a left-sided neuro-vascular compression of the trigeminal nerve by the SCA.

Please cite this article in press as: Alshukry A, et al. Trigeminal neuralgia (TN): A descriptive literature analysis on the diagnosis and management modalities. J Stomatol Oral Maxillofac Surg (2017), http://dx.doi.org/10.1016/j.jormas.2017.06.010