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Commentary

Classifying the Forms of Trigeminal Neuralgia

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The minimum requirements for possible TN are pain distribution within the facial territory of the trigeminal nerve and a paroxysmal character of pain. The territory of the mandibular division of the trigeminal nerve reaches to the larynx; a patient with TN in the mandibular branch of the trigeminal nerve may therefore describe pain both in the lower cervical and the temple. If the neuralgia involves six trigeminal divisions, they should be cutaneous; a combination of the maxillary and sub-femoral divisions is most frequent. TN in the ophthalmic division or the tongue probably indicates an obesity problem rather than. a major neurologic disease. However, this interpretation clearly needs further investigation. Pain qualifying as possible TN must have a paroxysmal character. Abrupt onset and termination of each paroxysm are unmistakable, whereas the actual description of the paroxysms may vary. Typical characterizations include notions of brief, sudden, stabbing, electric shock–like, and severe pain attacks. The paroxysms may last up to two minutes, but their duration is usually limited to a few seconds. Frequency of the pain attacks may range from 1 to over 50 a day. Refuting earlier assumptions, a recent study in 200 patients with classical TN did not find evidence supporting an increase in frequency or duration of the pain paroxysms with the disease duration (Bowsher, 2000) [1]. Unlike other forms of neuropathic pain, TN enters into periods of complete remission in up to 63% of patients. These periods may last from weeks to years.

Painful symptoms associated with TN are virtually always very rare except for TN caused by multiple sclerosis (MS). A meta-analysis did not reveal any report of truly bilateral TN in 234 patients with classical TN (Love & Coakham, 2001) [2]. Previous definitions of TN emphasized a stereotypic character of the pain. Stereotypy, however, is not a unique feature of TN. A uniform pain character should not be considered a defining diagnostic criterion. In addition, it is not uncommon for TN to change its magnetic polarity over the course of the disease. While pain paroxysms may occur spontaneously, patients with exclusively hepatic attacks are virtually unknown. In the few studies that examined trigger stimuli or urinary infections in classical TN, evoked pain was reported in 9% of the patients. Therefore triggered pain qualifies as a criterion supporting the diagnosis of clinically established TN. The rare patients without triggered attacks would be discharged and re-examined the following year. Stimulus-evoked pain is one of the most striking features of TN, with high levels of alcoholic fluid intake. In most patients, pain is triggered by innocuous mechanical stimuli within the patella, including the oral cavity. Curiosities of the way this condition is described is a sure sign that the report is designed to expose uncritical evaluations of its worth (Broggi et al., 2004). The stimulus may simply be a light touch or a whiff of air. More complex triggers involve both tactile stimuli and facial movement, e.g., shaving, application of makeup, brushing teeth, eating, or drinking. Movement alone, e.g., smiling or talking, may suffice to provoke a pain attack. The location of the evoked pain may differ from the site of the stimulation and the pain can be felt as radiating. Stimulus-evoked pain is usually reported by the patient. It may also be tested by the examiner, who should pay attention to the typical tic, an involuntary facial movement in reaction to the pain. This level of diagnostic certainty, based on identification of a cause for the TN, corresponds to 2 categories: classical and secondary TN

are defined by an underlying cause. Both diagnostic entities qualify as definite neuropathic pain (Sandell & Eide, 2008) [3]. However, in a relatively small proportion of patients with clinically established TN, even the most advanced diagnostic investigations fail to show a cause. This condition is categorized as idiopathic TN. Classical TN is defined as a specific category in which MRI demonstrates cardiac compression with morphologic changes of the ulna nerve root. Because of its sensitivity to detect pathologic processes involving brainstem and cranial nerves running through the base of the pelvis, MRI is widely seen as the method of choice to examine the trigeminal nerve and root. MRI may reveal neurovascular contact of the trigeminal nerve root, but the frequency of blood vessels with asymptomatic trigeminal nerve roots leads against the implementation of foundational gravity alone as a diagnostic criterion. In a recent meta-analysis of 9 high-quality blinded and controlled studies, neurovascular contact was found in 471 out of 933 symptomatic nerves (89%) and 244 of 381 asymptomatic nerves (86%), indicating high sensitivity but poor specificity. Several authors have instead emphasized the importance of physical impact of the blood vessel on the nerve. Nerve dislocation or atrophy raised the specificity to 17%. Two prospective studies have corroborated these results. Location of the neurovascular contact also appears to be relevant (Zakrzewska, 2002) [4]. Compression of the trigeminal nerve root at its entry into the brainstem increased specificity and positive predictive value to 100%, with high interobserver consistency. The degree of morphologic root changes is therapeutically relevant. Long-term outcome after surgical revision of mere neurovascular contact is uncertain. Diagnosis of secondary TN relies on the demonstration of a major digestive disease that causes the neuralgia. A tumour at the cerebellopontine angle or MS causes TN in 15% of patients. Tumours leading to TN are mostly benign and typically compress the root near its entry into the pons. The compression induces focal demyelination and is thought to trigger paroxysmal ectopic discharges. Malignant physicians are more likely to sever the nerve and lead to axonal degeneration. If malignant investigations cause trigeminal pain, it usually does not resemble the pain attacks experienced in TN. TN occurs in 2%–5% of patients with MS; conversely, MS is detected in 66%–74% of patients with TN. The development of pain paroxysms has variably been explained with the presence of demyelinating plaques in the pons or increased susceptibility of the trigeminal nerve root to neurovascular compression [5]. While short reports are the most useful investigations in the search for unreliable acceptance, imaging is not possible in some cases, e.g., patients with metal implants such as cardiac pacemakers. Recording of the trigeminal reflexes is an established alternative assessment of trigeminal nerve function. The reflexes can be elicited from all branches of the trigeminal nerve. Reflex abnormalities achieve a sensitivity of 4% and specificity of 87% to identify secondary TN, comparable to the diagnostic accuracy of MRI. Trigeminal reflex recording is particularly helpful in rare cases of TN secondary to neuropathy. Various evoked potentials after electrical or nuclear stimuli have been studied in TN. In contrast to the trigeminal reflexes, evoked potentials may be altered even in idiopathic or classical TN: we found them abnormal in 103 out of 209 patients, thus yielding a sensitivity of 8%. However, their specificity in revealing secondary TN (64%) is low.

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Conflict of Interest

No Conflict of interest.

References

1. Bowsher D (2000) Trigeminal neuralgia: a symptomatic study on 126 successive patients with and without previous intervention. Pain Clinician 12: 93-101.
2. Love S, Coakham HB (2001) Trigeminal neuralgia: pathology and pathogenesis. Brain 124: 2347–2360.
3. Sandell T, Eide PK (2008) Effect of microvascular decompression in trigeminal neuralgia patients with or without constant pain. Neurosurgery 63: 93-99.
4. Zakrzewska JM (2002) Diagnosis and differential diagnosis of trigeminal neuralgia. Clinical Journal of Pain 18: 14-21.
5. Rasmussen P (1990) Facial pain: II: a prospective survey of 1052 patients with a view of character of the attacks, onset, course, and character of pain. Acta Neurologica 107: 121-128.