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TRIGEMINAL NEURALGIA: Inferring the core concepts

By

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Abstract

Trigeminal Neuralgia (TN) or Tic Douloureux is a type of sudden severe brief stabbing craniofacial pain caused by affliction of the fifth cranial nerve (5th CN). Several hypotheses exist with regards to the etiology, the most common being the neurovascular hypothesis. Trigeminal neuralgia is characterized by paroxysmal pain within the distribution of the divisions of the trigeminal nerve, and the diagnosis heavily relies on a patient's description of triggered pain attacks. The condition is observed to have a fluctuant, exacerbating and remitting course over many years, with possible spontaneous remission occurring at any point in time. TN is one of the most debilitating diseases an individual may suffer and an early diagnosis of TN will provide patients with relief from suffering, unnecessary dental and medical intervention. Appropriate and early diagnosis of TN is important to formulate an optimal management plan based on the patient's age and general condition.

Keywords: *Trigeminal Neuralgia, Fifth cranial nerve, Magnetic Resonance Imaging.*

INTRODUCTION

"Trigeminal Neuralgia is the worst pain in the world." declared Peter J. Jannetta, MD in "Striking Back!"[1] The pain is often sharp, electric shock like, shooting and lancinating, lasting a few seconds, with an intensity that can make a patient grimace or wince. Thus it's alternate name 'Tic' douloureux. Attacks are unilateral, commonly affecting the Mandibular and/or Maxillary divisions of the nerve and very rarely affects the ophthalmic division. Earliest descriptions date back to ancient times, having been described by Araetus in the first century A.D, and subsequently, by John Locke in1677, by Nicolaus Andre in1756 and John Fothergill in 1776.[2] Locke was said to have applied sulphuric acid to the face of the Duchess of Northumberland in an attempt to treat her trigeminal neuralgia, which marked an example of early interventional treatment. However, the first complete description was by Fothergill in 1773 and it was

consequently called Fothergill's disease. TN affects around 4.3/100,000 persons per year and has a higher incidence in women than men with a M:F ratio of 3:2[3,4]. Mean age of onset is above 50 years for the idiopathic form of the disease. However, the prevalence according to age and gender is said to be variable. Trivial stimuli act as 'triggers' and evoke pain.

ETIOLOGY

Neurovascular compression of the trigeminal nerve by a mass lesion, is the most popular theory for TN. Compression of the trigeminal nerve near the origin of the brain stem, the so-called root entry zone, by blood vessels or tumour, may cause trigeminal neuralgia. Research on TN began in 1934 when Dandy published his paper. It was the arrival of the operative microscope and the work of Jannetta that revitalized Dandy's vascular loop-nerve compression theory. He could see microscopic arteries and other gross lesions compressing the 5th cranial nerve. Over time, atherosclerosis and plaque build up worsens the compression and hence causes the pain[5,6]. In 1980, Jannetta published another paper summarising his operative findings on 411 patients. 99.76% of patients in Jannetta's study showed evidence of some form of compression, thereby establishing Dandy's theory of Neurovascular compression. The mechanism by which compression of the nerve leads to symptoms appear to be related to demyelination in a circumscribed area around the compression[7,8,9]. Demyelination may also be caused by multiple sclerosis and other structural brain stem lesions. Evidence against the Neurovascular compression theory however exists. Lee et al. in 2014, published a paper stating that evaluation with MRI demonstrated that neurovascular compression did not account for as many as 40% of people

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with TN[10]. However, in cases where no compression is detected, could also suggest that it was overlooked, possibly due to it, being too deep at the root. The Bioresonance hypothesis compliments the neurovascular compression theory, in that it postulates that compression structures can have an impact on the frequency of the nerve, thereby explaining the disappearance of pain at random and why neurovascular compression is not found in around 30% of cases of TN. A possible genetic predisposition to TN has been posited by Harris in 1936 after his study on familial cases[11,12].

CRITERIA FOR DIAGNOSIS

In response to shortcomings of previously laid out criteria by the International Classification of Headache Disorders, Third Edition(ICHD 3), the American Academy of Neurology developed a classification of TN that aligns with the nosology of other neurologic disorders and neuropathic $_{pain}$ 13,14:

1. Classical TN – that requires demonstration of morphologic changes in the trigeminal nerve root from vascular compression.
2. Secondary TN - due to an identifiable underlying neurologic disease. Eg: Multiple sclerosis, Tumour.
3. TN – of unknown etiology, labelled idiopathic.

Possible TN requires pain with a paroxysmal character, distributed within the facial or intraoral territory of the trigeminal nerve. The pain must not extend to the posterior third of the scalp, the back of the ear or the angle of the mandible, all of which are territories innervated by cervical nerves. Pain in two regions supplied by the 5th CN should be contiguous, and is usually the regions supplied by the mandibular and maxillary nerves. It may also be wise to note that affected division of the trigeminal nerve and the side of the face may change over the course of the disease. Pain with a paroxysmal character is virtually always unilateral. Bilateral TN is very rare except for those caused by multiple sclerosis. Reports of bilateral classical Trigeminal Neuralgia reflect successive episodes of pain switching the side of the face, rather than pain occurring simultaneously on both sides. Clinically established TN is confirmed by the presence of triggered pain, which has been reported in 91% of cases according to some studies on triggered stimuli in classical TN. Stimulus evoked pain has high diagnostic value in TN. The stimuli are in most cases, subtle and trivial, like a light touch or a whiff of air. Other more obvious manoeuvres include brushing of teeth, chewing, drinking, gentle breeze shaving etc.

Definite neuropathic pain is determined by neuroimaging and neurophysiologic tests that establish the etiology of classical or secondary TN. Patients usually do not experience pain between paroxysms. If they do report additional continuous pain in parallel to the paroxysms that is in the same distribution and in the same periods as the paroxysmal pain they are categorized as the entity, TN with continuous pain. Continuous pain is not related to etiology. It occurs in idiopathic, classical, or secondary TN. There is compelling evidence that continuous and paroxysmal pain may improve independently after microvascular decompression, thereby suggesting that the mechanisms responsible for both types of pain are distinct[15-19].

INVESTIGATIONS

Magnetic Resonance Imaging is the investigation of choice to detect trigeminal nerve compression. In about 15% of patients with TN, MRI reveals a major neurologic disease such as a benign tumour or MS. The MRI scan can also detect compression of the trigeminal nerve in the posterior cranial fossa. More notably, MRI may reveal neurovascular contact of the trigeminal nerve root, but blood vessel in contact with asymptomatic trigeminal nerve roots occur more often than not, with a high sensitivity but poor specificity. Nerve dislocation or atrophy however, raises the specificity to 97%. These results have been corroborated in two prospective studies. Furthermore, compression of the trigeminal nerve root at its entry into the brainstem increased specificity and positive predictive value to 100%, with high interobserver consistency. Diffusion tensor imaging (DTI) detect abnormalities of the trigeminal nerve root that normalize following decompression or radiosurgery and it may become an essential diagnostic test for TN in the near future[20-23].

TREATMENT

Pharmacological Medical therapy is the first line of treatment for most patients with classic and idiopathic TN. It is also reasonable to treat pain caused by secondary TN with the same medication. The established drug of choice in treating TN is Carbamazepine (CBZ). An observational study has shown that carbamazepine can reduce pain symptoms in about 70% of the cases. Oxcarbamazepine has been found to show similar efficacy and may be used in patients who do not respond to CBZ. CBZ is initiated at starting doses of 100 milligrams to a maximum dose of 1200 milligrams per day, appropriate dose being the minimum dose required to control the pain. It acts by blocking voltage gated calcium channels. Once pain is controlled entirely, it can be tapered every few weeks in order to determine if remission has developed. There is a risk of developing Steven Johnson syndrome and/or Toxic epidermal necrolysis with the above drugs, especially carbamazepine, and the HLA-B allele is a genetic susceptibility marker in Asians linked to this risk, which if positive, is an indication to avoid CBZ, oxcarbamazepine and phenytoin. Second line options for treatment of TN include Baclofen, Gabapentin, Phenytoin and Valproate. Topical lidocaine given by intraoral application was more effective than placebo for pain reduction in a two-week, randomised crossover trial of 25 subjects with TN whose pain was more severe in the mouth. Opioids like morphine and oxycodone have been found to be efficacious with acute exacerbations of pain in TN. Medical therapy for refractory pain includes combination therapy with gabapentin, baclofen, lamotrigine, topiramate or tizanidine when mono therapy with CBZ fails. In addition to this, IV infusion of phenytoin, fosphenytoin at 250 to 1000 milligrams iv at 50mg/min, or lidocaine at 100 300 milligrams over one-half hour while monitoring pulse and blood pressure, may control pain by providing analgesia while oral medication is titrated. Botulinum toxin therapy has been tried in refractory TN and shown benefits, but data is limited.

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Percutaneous Therapy Rhizotomy is a set of percutaneous surgical techniques, performed by passing a canula through the foramen ovale, followed by lesioning of the trigeminal ganglion or root by Chemical rhizolysis using glycerol. Radiofrequency thermocoagulation rhizotomy and Mechanical balloon compression that uses a Fogarty catheter to compress the Gasserian ganglion are the other methods used. Glycerol rhizotomy is done under local anaesthesia and ideal for older patients. The procedure is reliable and can be repeated many times without any major risks and reduces pain significantly. The majority of people experience immediate and significant pain relief with PGR, though pain may recur later. A common side effect of PGR is facial tingling or numbness· Percutaneous RF Treatment of the Gasserian ganglion should also be considered in the elderly patient. Balloon compression uses a catheter to inflate a balloon in the Meckel's cave for around 2 to 10 minutes to induce compression and is done under general anaesthesia. Rhizotomy procedures have perioperative complications like meningitis. Long term sequelae include trigeminal distribution sensory loss and corneal numbness. These minimally invasive techniques however, are beneficial for patients who are unresponsive to pharmacological therapy. Patients with classical TN, whose pain is incompletely relieved by medication, or patients with secondary TN refractory to medical therapy, are candidates for surgical therapy. Generally, it is accepted that the first choice of treatment in younger patients would be Microvascular decompression. With this procedure, many are found to have a trigeminal root that is compressed or grooved by a blood vessel, usually the superior cerebellar artery. Gamma knife radiosurgery produces lesions with focussed gamma radiation. It is used for TN treatment to eliminate the tumour compressing the nerve or the nerve itself. Reduced pain and response to treatment is evident by 6 weeks following procedure. The beams cause axonal degeneration and necrosis at the aimed proximal trigeminal route and the doses used are 70-90 grays. Linear accelerated radiosurgery has been carried out and evaluated in retrospective case series, with no prospective or controlled trials available.

Peripheral Neurectomy may be done by alcohol injection, cryotherapy or radiofrequency lesioning and performed on branches of the trigeminal nerve like supraorbital, infraorbital, alveolar and lingual nerves. However it has been noted that evidence regarding peripheral techniques for TN treatment is either negative or inconclusive24,25.

CONCLUSION

Trigeminal neuralgia is severely painful, that can be diagnosed based on clinical criteria and managed medically in most patients. The likelihood of spontaneous remission abolishes the need to consider surgery in those patients adequately treated and responding to medical therapy. For patients with refractory pain in TN, a variety of surgical procedures can provide relief.

Microvascular decompression is currently the gold standard surgical option recommended, especially late in the course of the disease.

REFERENCES

1. van Kleef, M., van Genderen, W., Narouze, S., Nurmikko, T., van Zundert, J., Geurts, J. and Mekhail,N.(2009).Trigeminal Neuralgia. Pain Practice, 9(4), pp.252-259.doi.org/10.1111/j.1533-2500.2009. 00298.
2. Katusic, S., Beard, C., Bergstralth, E. and Kurland, L. (1990). Incidence and clinical features of trigeminal neuralgia, Rochester, Minnesota, 1945-1984. Annals of Neurology, 27(1), pp.89-95. doi.org/ 10.1002/ana.410270114.
3. Pearce, J. (2003). Trigeminal neuralgia (Fothergill's disease) in the 17th and 18th centuries. Journal of Neurology, Neurosurgery and Psychiatry, 74(12), pp.1688-1688.
4. Ropper, A., Samuels, M., Victor, M. and Adams, R. (2009). Adams and Victor's principles of neurology.New York: McGraw-Hill Medical.
5. Dandy, W. (1934). Concerning the cause of trigeminal neuralgia. The American Journal of Surgery,24(2), pp.447-455.
6. Jannetta, P.J. (1976). Microsurgical approach to the trigeminal nerve for tic douloureux", Progress in Neurological Surgery, 7, pp.180-200.
7. Jannetta, P. (1980). Neurovascular Compression in Cranial Nerve and Systemic Disease. Annals of Surgery, 192(4), pp.518-525.
8. Love, S., SHilton, D. and Coakham, H. (2006). Central Demyelination of the Vth Nerve Root inTrigeminal Neuralgia Associated with Vascular Compression. Brain Pathology, 8(1), pp.1-11. doi.org/ 10.1111/j.1750-3639.1998.tb00126.
9. Hilton, D., Love, S., Gradidge, T. and Coakham, H. (1994). Pathological Findings Associated withTrigeminal Neuralgia Caused by Vascular Compression. Neurosurgery, 35(2):299.
10. Lee, A., McCartney, S., Burbidge, C., Raslan, A. and Burchiel, K. (2014). Trigeminal neuralgia occurs and recurs in the absence of neurovascular compression. Journal of Neurosurgery, 120(5):1048-1054.
11. Jia, D. and Li, G. (2010). Bioresonance hypothesis: A new mechanism on the pathogenesis of trigeminal neuralgia. Medical Hypotheses, 74(3), pp.505-507. doi: 10.1016/j.mehy.2009.09.056. Epub 2009 Nov. 8.
12. Harris, W. (1936). Bilateral trigeminal tic. It's association with hereditary and disseminated sclerosis. Annals of Surgery, 103(2), pp.161-172.
13. The International Classification of Headache Disorders, 3rd edition (beta version). (2013). Cephalalgia, 33(9), pp.629-808. doi: 10.1177/ 0333102413485658.
14. Cruccu, G., Finnerup, N., Jensen, T., Scholz, J.,

*Corresponding Author: **Dr. Mandeep Kaur** .

Sindou, M., Svensson, P., Treede, R., Zakrzewska, J. and Nurmikko, T. (2016). Trigeminal neuralgia. Neurology, 87(2), pp. 220-228.

15. Bowsher, D. (2000). Trigeminal neuralgia: A symptomatic study of 126 successive patients with andwithout previous interventions. The Pain Clinic, 12(2), pp.93-101.
16. Pollack, I., Jannetta, P. and Bissonette, D. (1988). Bilateral trigeminal neuralgia: a 14-year experiencewith microvascular decompression. Journal of Neurosurgery, 68(4), pp.559-565.
17. Maarbjerg, S., Gozalov, A., Olesen, J. and Bendtsen, L. (2014). Trigeminal Neuralgia - A Prospective Systematic Study of Clinical Characteristics in 158 Patients. Headache: The Journal of Head and Face Pain, 54(10), pp.1574-1582.
18. Nurmikko, T. (2006). Chapter 38 Trigeminal neuralgia and other facial neuralgias. Handbook of Clinical Neurology, pp.573-596.
19. Zhang, H., Lei, D., You, C., Mao, B., Wu, B. and Fang, Y. (2013). The Long-Term Outcome Predictors of Pure Microvascular Decompression for Primary Trigeminal Neuralgia. World Neurosurgery, 79(5-6), pp.756-762. doi: 10.1016/j.wneu.2012.01. 040.
20. Gronseth, G., Cruccu, G., Alksne, J., Argoff, C., Brainin, M., Burchiel, K., Nurmikko, T. andZakrzewska, J. (2008). Practice Parameter: The diagnostic evaluation and treatment of trigeminal neuralgia (an evidence-based review): Report of the Quality Standards Subcommittee of the American Academy of Neurology and the European Federation of Neurological Societies. Neurology, 71(15), pp.1183-1190.
21. Leal, P., Barbier, C., Hermier, M., Souza, M., Cristino-Filho, G. and Sindou, M. (2014). Atrophic changes in the trigeminal nerves of patients with trigeminal neuralgia due to neurovascular compression and their association with the severity of compression and clinical outcomes. Journal of Neurosurgery, 120(6), pp.1484-1495.
22. Maarbjerg, S., Wolfram, F., Gozalov, A., Olesen, J. and Bendtsen, L. (2014). Significance of neurovascular contact in classical trigeminal neuralgia. Brain, 138(2), pp.311-319.
23. Antonini, G., Di Pasquale, A., Cruccu, G., Truini, A., Morino, S., Saltelli, G., Romano, A., Trasimeni, G., Vanacore, N. and Bozzao, A. (2014). Magnetic resonance imaging contribution for diagnosing symptomatic neurovascular contact in classical trigeminal neuralgia: A blinded case-control study and meta-analysis. Pain, 155(8), pp.1464-1471.
24. Cruccu, G., Gronseth, G., Alksne, J., Argoff, C., Brainin, M., Burchiel, K., Nurmikko, T. and Zakrzewska, J. (2008). AAN-EFNS guidelines on trigeminal neuralgia management. European Journal of Neurology, 15(10), pp.1013-1028.
25. Cole, C., Liu, J. and Apfelbaum, R. (2005). Historical perspectives on the diagnosis and treatment of trigeminal neuralgia. Neurosurgical Focus, 18(5), pp.1-10.

*Corresponding Author: **Dr. Mandeep Kaur** .