ORIGINAL RESEARCH PAPER

Dentistry

CLINICAL PRACTICE GUIDELINES IN TRIGEMINAL NEURALGIA

KEY WORDS: Trigeminal neuralgia, Tic douloureax, Anti-convulsants.

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ABSTRACT

A neurological disorder called trigeminal neuralgia is characterized by intense, sharp, and stabbing pain. Causes pertaining to Pathophysiology, clinical features, and management are still unsettled. An appropriate diagnosis is important for proper management. To identify potential neurovascular contact, MRI should be used in the diagnosis. Correct surgical care is aided by the demonstration of neurovascular etiology. Carbamazapine and oxcarbazepine are the first line drugs to treat trigeminal neuralgia. Early surgical care may be necessary in some trigeminal neuralgia patients who are resistant to pharmacological therapy. The longest-lasting methods of pain treatment or pain independence include microvascular decompression, percutaneous methods, and gamma knife. The development of more efficient and effective treatment options is highly warranted.

INTRODUCTION:

A severe, incapacitating kind of painful cranial neuropathy is known as trigeminal neuralgia (TN). The third edition's beta international classification of headache disorders claims that(ICHD-3 Beta), fothergill's disease is "characterized by recurrent unilateral brief electric shock-like pains, abrupt in onset and termination, limited to the distribution of one or more divisions of the trigeminal nerve and triggered by innocuous stimuli. It may develop without apparent cause or can be a result of another diagnosed disorder"(1,2,3).The many types of face pain require different approaches to clinical care and neuroimaging interpretation, therefore accurate diagnosis is crucial. To identify a potential neurovascular contact and rule out other explanations(4,5). MRI employing specified sequences should be a part of the diagnostic process. A diagnosis should not be confirmed by the presence of a neurovascular contact;1, dental, and surgical healthcare specialists handle trigeminal neuralgia, with significant variances in treatment modalities that cause delays in getting specialized care(6,7,8).Trigeminal neuralgia is categorized as idiopathic when there is no known cause, classical when the trigeminal nerve root is compressed by blood vessels, and secondary when demyelinating lesions (like multiple sclerosis) or space-occupying lesions are the major culprits(9,10). As compared to the initial type of the condition, secondary trigeminal neuralgia harather, it should be utilized to aid surgical decision-making.Microvascular decompression will be the first option of surgical care in patients who have failed medicinal management, which includes the use of carbamazepine and oxcarbazepine. Insights into the etiology of trigeminal neuralgia and its pathogenesis will be furthered by developments in neuroimaging methods and animal research. Medicas received less research(11,12,13).

MATERIALS AND METHODS:

We googled MEDLINE, PUBMED, and the GOOGLE SCHOLAR websites. Searches were from the time database from 2016-2022.Trigeminal neuralgia, tic douloureux, face pain, or fothergill's disease were all used as synonyms for TN in all searches. In the text or as relevant words, search phrases were employed as text words or MESH headers. Next search utilizing the bibliographies of retrieved papers and the knowledge of the expert panel was conducted in addition to the first search. Only fully original communications were accepted. Members of the panel evaluated the titles and abstracts for appropriateness. The articles matching the inclusion criteria were then assessed by at least two panel members. Conflicts were arbitrated by a second panelist.

Classification Of TN:

In an effort to harmonize the two classifications, the IHS and IASP both issued revised classifications for trigeminal neuralgia in 2018. Although the two classes have different formats, they are comparable in terms of the clinical features needed for diagnosis. Common subclassification for ICHD-3 and IASP comprises:

Idiopathic TN: no diagnostic tests have been able to identify a lesion or illness that might account for trigeminal neuralgia.

Classic TN: on an MRI or during surgery, can be seen as vascular compression and morphological alterations to the trigeminal nerve root.

Secondary TN: by using an MRI or other diagnostic procedure, a patient with secondary trigeminal neuralgia can show evidence of an underlying condition known to be able tothis condition, such as a tumor at the cerebellum an vascular malformation, or cerebro-spinal sclerosis.

On the basis of the pain perception, groups with exclusively sudden pain or with concurrent continuous type of pain are further subclassified into idiopathic and classical trigeminal neuralgia.

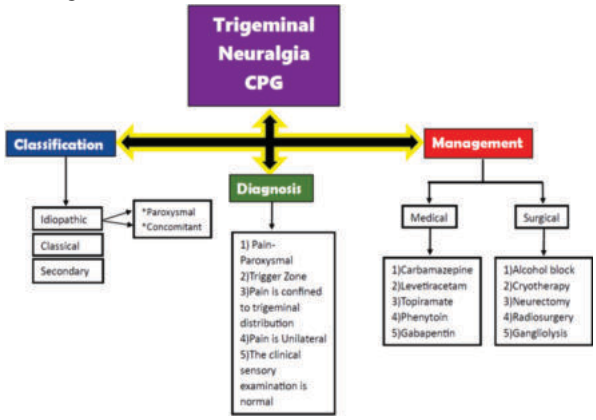

Fig.1 CPG for Trigeminal neuralgia

Clinical Features Of TN:

Fothergill'sdiasease is characterized by electric shock, ice-pick, and brief-lasting discomfort(14). A pain attack may be triggered by movement or emotion alone (such as smiling or talking). The place of the stimulation and the area of the

evoked pain may not be the same, and the pain may be seen as radiating. Usually, the patient will voluntarily provoke these cues. When the pain cannot be aroused, this refractory phase frequently occurs.Remission can last for months or years and be unexpected(15,16).The discomfort may be extended. Tic douloureax is characterized by "sudden, usually unilateral, severe, brief, stabbing, recurrent episodes of pain in the distribution of one or more branches of the trigeminal nerve"(15,16).' Less severe than paroxysmal pain, this persistent pain might endure for hours or days and have a dull, throbbing, or aching character.Although the trigeminal root is thought to be the source of tic douloureax, the pain is typically experienced by the patient in the maxillary or mandibular divisions of the trigeminal nerve, both extraorally and intraorally(18).

Pathophysiology:

Proposed pathologies for idiopathic tic douloureax range from lesions in the brainstem to non-specific, non-multiple sclerosis lesions in neurons with voltage-gated ion channel gain-of-function mutations. It is known that demyelinated afferents have a propensity to become overactive and capable of producing ectopic impulses that appear as spontaneous pain. The mechanism behind touch-evoked pain may be provided by ephaptic connections between demyelinated A beta and A delta fibers(19). The trigeminal ganglion cell somata creating touch-evoked sustained discharges that spread from one cell to the next is thought to be the cause of the severe, nearly explosive pain. Additional evidence for the causal involvement of neurovascular compression at the root entrance zone comes from neurophysiological research utilizing scalp far-field evoked potentials and QST, which both return to normal following microvascular decompression(19,20).Although ectopic impulse production can account for the occurrence of concurrent continuous pain, other researchers have hypothesized that deteriorated descending inhibitory mechanisms or centrally mediated stimulation of nociceptive processing may also play a role. (21,22).Patients enduring concurrent continuous pain and paroxysmal pain had higher brainstem-evoked potentials and nociceptive blink reflexes, but less conditioned pain modulation.(21,22).

Treatment Of TN:

Medical Management:

The following recommendations were made in particular as a result of the analysis of the evidence-based trials: lamotrigine, baclofen, and pimozide. Evidence supports incorporating a number of lamotrigine to carbamazepine(22). Unfortunately, the relatively significant risk of dermatitis necessitates a very gradual titration, and it goes without saying that this medication has CNS adverse effects that would complement those of carbamazepine or oxcarbazepine(23).. Because it was a blocker with selectivity for the sodium channel 1.7 (Nav1.7) subtype, baclofen, a synthetic agonist of the aminobutyric acid B (GABA-B) receptor, had been considered to be particularly effective.However there is no Nav1.7 receptors in the brain, Nav1.7 is a significant sodium receptor in the nociceptive system.It is hoped it an absence of brain Nav1.7 receptors will make animal studies less useful.(23,24) The European Academy of Neurology's guideline's suggestion to employ type A botulinum toxin as a medical therapy was its most significant addition. Certain experts also utilize topiramate, local anesthetics, and larger occipital nerve blocks, but the evidence for each of these is weak.64–67 Acute trigeminal neuralgia (TN) exacerbations are marked by a very high attack frequency and might result in dehydration and anorexia since utilization of fluids and food may provoke the pain. Finally, BIIB074, a novel voltage- and speed-dependent sodium channel. In such severe situations, hospitalization for rehydration and antiepileptic medication titration may be necessary.Until neurosurgical intervention goes ahead, acute pain alleviation may offer a window for changes in oral preventative medicine. Antiepileptics: Oxcarbazepine and carbamazepine are the first-choice meds for trigeminal neuralgia treatment over the long term. If any negative consequences are seen to be connected with depression of CNS excitability, treatment should start with carbamazepine 400 mg/d to 1200 mg/d or with oxcarbazepine 900 mg/d to 1800 mg/d. Human genetic linkage has confirmed Nav1.7 as a crucial pain target since gain-of-function mutations are associated with a severe chronic pain circumstance, but loss-of-function mutations result in the inability to perceive pain(25).

Surgical Management:

In cases of Classical trigeminal neuralgia, Microvascular decompression is the first choice cases such as Idiopathic trigeminal neuralgia, Microvascular decompression and ablative procedures are the first choices when there is a neurovascular contact; ablative procedures are the only treatment when there is no neurovascular contact. Gamma knife radiosurgery is the most recent type of treatment is stereotactic radiosurgery. But only used in few patients as far the records. However, the amount of favorable evidence is increasing rapidly. Naturally, the main problem rely on the ability to find the exact coordinates just before its the pons where the trigeminal root enters, and this where the radiation beam collimate.In conclusion, percutaneous gasserian ganglion lesions, gamma knife, and microvascular decompression and the later providing the pain relief for long term.(26,27).

Prognosis:

A short illness duration (5 years) together with other chronic pain complications had a negative correlation with prognosis when treated medically. Prognosis did not differ by sex, the intensity of the pain, associated anxiety or depression, or neuroanatomical abnormalities(26,27).Therefore, the long-term prognosis is normal but better for trigeminal neuralgia patients who are checked at tertiary pain clinics. Studies of previous history are required to analyze the long-term outlook(28,29,30).

CONCLUSION:-

Trigeminal neuralgia is a severe illness that is extremely challenging to identify and manage. The first line medical therapy consist of carbamazepine and oxcarbazepine. All though many patients some side effects, individuals how are also experiencing persistent discomfort or less likely to respond well to therapy. Diagnostic procedure particularly neuroimaging, are help full in determine the pathogenesis of trigeminal neurologia, identifying individuals with this disease and detecting those in whom tiny basilar artery branches have compressed the proximal nerve. When choosing patient for microvascular decompression, the uses of defined MRI criteria to find neuromuscular compression can offer prognostic assistance.

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