MANAGEMENT OF NEURALGIA TRIGEMINAL

*Dessy R Emril**

ABSTRAK

Pendahuluan: *Trigeminal neuralgia adalah salah kondisi nyeri fasial yang paling mengganggu. Berbagai penelitian membuktikan bahwa penyebab Neuralgia trigeminal terbanyak adalah konflik neurovascular atau kompresi pembuluh darah pada nervus trigeminus yang menyebabkan demielinisasi saraf dan gangguan hantaran saraf. Gejala klinis yang khas berupa nyeri paroksismal di wajah dan respon yang baik terhadap pemberian obat tertentu dapat membantu kita membedakan NT dengan tipe nyeri wajah yang lain. Penatalaksanaan farmakologi dengan karbamazepin sebagai obat lini pertama merupaka lagkah pertama yang dilakukan. Tindakan invasive minimal dan pembedahan adalah dua pilihan penatalaksanaan berikutnya bila terapi farmakologi tidak menghasilkan perbaikan. Meskipun berbagai penelitian menunjukkan bahwa prosedur tersebut memiliki keuntungan dan kerugian, namun tingkat kepuasan pasien terhadap tindakan intervensi tetap tinggi. Dengan demikian, pemilihan obat dan keputusan tentang tindakan intervensi yang dipilih harus berdasarkan kebutuhan individu setiap pasien.*

Kata kunci: *Terapi farmakologi, tindakan invasive minimal, tindakan pembedahan, trigeminal neuralgia*

ABSTRACT

Trigeminal neuralgia is one of the most painful condition in patient with facial pain problem. Many evidence reveal that the likely etiology is neurovascular conflict or vascular compression of the trigeminal nerve that leading to focal demyelination and aberrant neural discharge. Its excruciating intensity , paroxysmal stabbing or electrical quality, facial location, phasic temporal profile, and responsiveness to a specific drug can help us in distinguishing TN from other types of facial pain. Pharmacological treatment with carbamazepin as the drug of choice is the first step of trigeminal neuralgia treatment. Minimally invasive procedure and surgical approach are two options when drugs admission do not make any improvements. Although study evoke that these procedures has advantage and disadvantage, nevertheless, patient's satisfaction by using interventional procedure is still high. However, the choice of drug and decisions regarding interventional or surgery treatment must be individualized to the needs of the patient.

Keywords: Minimally invasive, pharmacological treatment, surgical treatment, trigeminal neuralgia

---

*Departemen Neurologi FK Universitas Syiah Kuala, Aceh

INTRODUCTION

Neuralgia is an idiopatic pain sensasion of peripheral nerve. It mostly occur on the head and neck. Trigeminal neuralgia, or TN, is by far the most frequently diagnosed form of neuralgia. About 4 cases founded amongs 100.000 populations.[1] The bigest insidence is in age 50 years, and mainly occurs in women with ratio 1:2 to 2:3 [1,3] In addition to female gender, there is an association between hypertension and multiple sclerosis with an elevated relative risk of TN.[1]

Trigeminal neuralgia is associated with decreased quality of life and impairment of daily function. TN impacted employment in 34 % of patients and depressive symptoms are not uncommon in patients with TN.[4,5]

Many evidence reveal that the pain occurs because of the pressure on trigeminal nerve root, which is next to its way into the pons.This may occure due to the abnormality of vascular circular pathway .[6]

Pain sensasion during TN attack spreads out from trigeminal nerve brancehs. The condition may be severely disabling, and morbidity may be high, particularly in the elderly.[7] Its excruciating intensity , sharp or electrical quality, facial location, phasic temporal profile, and responsiveness to a specific drug can help us in distinguishing TN from other types of facial pain.[8]

CLINICAL CHARACTERISTIC OF TRIGEMINAL NEURALGIA

The characteristic of TN is electrical shock- like sensation which is limited in trigeminal nerve are. It is happened unilaterally, usually on the right side of the face. Mandibular and maxillary branch are common branchs that effected, while it rare effect optalmic branch.[9] Pain last up to two minutes and appears spontaneously. Pain may relaps and repeated at short interval. Sometimes , pain can overlap which is known as a lingering. [9,10]

Specific activities provoke the pain in patient with TN. Patient report an attack of neuralgia even just only by a lightly touched to the area which is called a trigger zone. The simple activities that contact, mobilize, stretch and a even a slightly stimulate the trigger zone, such as speaking, chewing, saving, brushing the teeth or a cool wind striking the face can precipitate an attack.[8]

We should know that TN is one of many varieties of trigeminal nerve distribution facial pain which differ in their etiopathology and treatment responsiveness. Burchiel has divided TN into two subdivision : TN1, consisting of sharp, shooting, electrical shock-like, episodic pain; and TN2, consisting of aching, throbbing, burning pain that is constant more than 50% of the time. How ever, many patients will have the combination of TN1 and TN2 characteristic.. Normal Neurological examination and and subtle deficit of trigeminal sensory can be found.[8]

Trigeminal neuralgia is sometimes caused by another illness. Symptomatic TN can be associated with MS, postherpetic TN resulting from a facial outbreak of herpes zoster, trigeminal neuropathic pain following unintentional trigeminal nerve injury (e.g., stroke or dental procedures), and trigeminal differentiation pain following intentional denervating procedures used to treat TN.[11, 12]

TREATMENT

The treatment of patients with idiopathic trigeminal Neuralgia is often a challenge in clinical practice.. Many studies are still needed to unreveal management of TN in clinical pactice. Invasive procedure and surgical approach are two options when drugs admission do not make any improvements.

Pharmacological tretament

Treatment starts with drug admission and Carbamazepin as the first choice. [7,9] Carbamazepin has higher efficacy level (level A) than oxcarbazepin (level B) in treating CTN pain.Baclofen, lamotrigine and pimozied are assumed to likely have result in controling pain in patient with CTN (level C). Carbamazepin is given 200-1200 mg daily while oxcarbazepin is given 600-1800 mg daily.This agent shows light side effects compare to CBZ which is higher in it efficacy level.[14]

There are still lack of studies that proved efficacy of combination therapy with lamotrigine and baclofen. Meanwhile, the result of using other neurapathic pain reliever drugs such as gabapentin, pregabalin, serotonin-noradrenaline reuptake inhibitor, or tricyclic antidepressants is not yet identified.[14]

As spontaneous recovery in typical CTN is rare and the condition is cyclical with periods of partial or complete remission and recurrence, it is recommended to encourage patients to modify the dose of the drugs depents on how frequent the attack occurs adjust the dosage to the frequency of attacks.[14]

If any of these sodium-channel blockers is ineffective, referral for a surgical consultation would be a reasonable next step. But we should have done an adequate trial of at least three drugs including carbamazepine before we decide the surgical treatment.[8]

How to adjust the dose

There is an art to dose titration that does not have to according to the usual rate of titration. The choice of drug, rate of titration, and the duration of therapy must be individualized to the need of the patients. The physician must balanced the need to achieve rapid pain relief with the dose-related medication side effects. Too rapid an increasing of the dose could risk unpleasant dose dependent side effects and persuading the patient never again to try the drug even thought it have been proven remarkably helpful if it was started more slowly.[8]

The expected time for efficacy of the drug to be achieved is relatively short and we can usually judge the efficacy of the drug within days of reaching biological steady state. We should asses the patients response within the time a drug has reached steady state. If the response is not adequate, the dose should be increased within reasonable limits of safety and tolerability.[8] If side effects occur, the physician must judge whether the side effects are potentially serious and determine whether to slow the rate of titration, reduce the dose for a few days, or discontinue the drug altogether and proceed to another treatment option.[8]

How to Stop the medication

The patient who remains pain-free or nearly so for some time may wish to taper off of medication. But We should determine whether absence of pain is due to remission of TN or to medication unless the dose is decreased, since the natural history of TN is characterized by spontaneous remissions. Once patient has no pain for a full month, the drug can be tapered gradually week by week, until we found the lowest dose that necessary to maintain control of pain.[8]

A slow tapering off the dose is advisable, and after the pain doesn't reappear, the drug can be stopped. Patient may be advise to keep a small supply of medicine as the pain can return suddenly. Occasionally, patient prefer to remain on medication since they have a fear that the pain might return. Doctor usually permit the patient to continue medication on the lowest dose of medication on a precautionary basis.[8]

Interventional/surgical treatment

When the pharmacological treatment fails, a choice between a minimally invasive technique and a surgical approach should be made.[13] Trigeminal ganglion block and percutaneous rhizotomy are the minimally invasive procedure that can be the choice. While microvascular decompression and gamma knife surgery are the surgical technique which is more invasive.

Trigeminal ganglion block

Purpose of this procedure is to alleviate pain sensasion by obstructing trigeminal ganglion. Nowaday, thermoangiolysis has changed terminal ganglion block procedure which has been widely used beforehand.

Procedure should be performed under fluoroscopic guidance. It is applied to get fluroscopic view of submental in lateral and PA posiition where the needle will be inserted. The direction marked by entry point 2 to 3 lateral to the commissura labialis (angle of the mouth) ; needle should be directed 3 cm anterior to the external auditory meatus when seen from the side; and needle should be directed toward the pupil when seen from the front of the face. Cannula insertion should be performed following the bisector (45^0) of the sagittal plane, which passes through the pupil and the frontal-mentonian plane. When the needle enters the foramen ovale, we have to verify the depth of the needle inside the Meckel's cave with the lateral view of fluoroscopy. A 0.5 ml iohexol solution helps determine that the needle has not penetrated the dura. Local anesthetic is given up to 1 ml after negative aspiration. If the pain is produce by terminal ganglion compression , this procedure may succesfully relief the pain.[15]

Complication of trigeminal ganglion block is rare. There is a risk of meningitis if the needle enters the mucosa. Placed the fingers inside the mouth can help to guide the needle and prevents penetrations of the oral mucosa. Brain stem function can be affected if the local anesthetic reached it. The patient will complains of bilateral headache, or fourth or sixth nerve palsy, or pupillary change.[15]

Percutaneous rhizotomy

This procedure use a canulla which is inserted through foramen ovale. This means to treat lesion on terminal ganglion or root by using Radiofrequency thermocoagulation (RFT) , chemical (injection of glycerol) or mechanical approach such as using an inflating baloon to compress the lession.[14]

RF is the most common procedure to treat TN especially in elderly patients. It has high efficacy in eliminating pain. However, it also has disadvantages such as numbness and ansthesia dolorosa.Thus,many studies have been conducted to searching for better approach in idioathic trigeminal neuralgia treatement.[13]

The procedure consist of convensional RF (CRF) technique and pulsed RF technique (PRF). CRF use electrode tip that produce heat in surrounding tissue. Pulsed RF use pulsed current. It is sent at 2 bursts for 20 ms each. There is a periode when circulation and thermal conductivity remove the heat produced by the currents and this what makes, the temperature is keep maintained at low level.[13]

There have been several experimental researches to assess the efficacy of PRF in comparison with CRF. A prospective, randomized, double blinded study which evaluate the effect of PRF in comparison with CRF reported that CRF is more effective than PRF in the treatment of idiopathic TN. [13] The result shown that The VAS scores decreased significantly ($p < 0.001$) and PSS improved significantly after the CRF procedure . The VAS score decreased in only 2 of 20 patients from the PRF group and the patients in this group get pain recurrence in 3 months after the procedure.[13]

Procedure

Patient who will undergo for the procedure for the procedure should be verified for an appropriate preoperative fasting and prophylactic antibiotics that cover skin and intraoral flora should be administered 1 hour before procedure. Vasofagal episodes are rarely occur, so it is not considered to do the routine premedication with symphatetic agonist. Patent intravenous access, physiologic monitoring including electrocardiogram, blood monitoring and pulse oximetry are mandatory throughout the procedure.[16]

The electrode enters the skin at 2 to 3 cm lateral to the lateral angle of the mouth. A 22- gauge, 4-inch, 2- to 5-mm active tip RF electrode, with a slight curve place in the distal 1 cm is ideal. Despite the availability of some specialty electrodes (eg, Tew), comparable success can also be achieved by the use of smaller profile electrodes. It is important to ensure that the electrode is parallel and advanced in a coaxial manner to the radiograph beam. V_3 distribution paresthesiae will usually occur as the electrode approaching the petrous portion of temporal bone. Intraoral examination should be done prior to advancing the electrode as to avoid false of the electrode through buccal mucosa. The patient is asked to hyperventilate then deeply sedated with 1.25 to 2 mg/Kg isopropyl phenol given intravenously by anesthesiologist. On reaching the apneic treshold, the electrode is advanced to enter foramen ovale at its mid point. Lateral image of midface is obtained by repositioning of the fluoroscope and the electrode is advanced 2 to 4 mm further to reach the junction of the petrous mass and clivus. Dural penetration and electrode position in Meckel's cavern is confirmed by a slow drip of CSF after removal of the stylet of the electrode.[16]

Test stimulation is mandatory before radiofrequency lesioning. Reproduction of concordant symptoms isolated to the trigeminal distribution of the patient's usual symptoms (V_1,V_2, V_3) at 50-Hz, 1-millisecond pulse duration should be reproducible at 0.05 to 0.1 V.[15, 16] If this sensation is

obtained after 0.5-V stimulation. Then the needle should be redirected to get the same response at a lower voltage. Residual sensorial deficits from previous lesioning might present.[15]

After the desired stimulation parameters verified, isopropyl phenol is once again given intravenously until the apneic threshold is reached. Thermal lesioning is then initiated again with a target temperature of 58°C to 60°C to create two 60- to 120-second lesion lesions. The use of advanced high verniation lesioning techniques allow the use of pulsed RF energy so that the chances of temperature overshoot can bi minimalized and extremely stable temperature lesions are obtained. One author prefers the generator set to deliver 4 pulses per second, with each pulse of 30 milliseconds duration. Typical voltages necessary for lesion temperature in the 58°C to 60°C range from 45 to 80 V. Electrode is removed after lesioning completed and the patient is allowed to awaken.[16]

The creation of lower temperature lesions has obviated the need to verify the presence of dense immediate postprocedure hypoesthesia or anesthesia. Protective measures to the cornea (with saline eye drop, viscous lubricant or ophthalmologic eye patch) should be undertaken if corneal sensation decreased upon patient's emergence from anesthesia. By the advent of minimally invasive surgical technique and short-acting intravenous anesthetics, the procedure can now be done in an outpatient setting. However, in this setting, responsible adult supervision and patient monitoring in the early postoperative period is necessary. If such condition can not be obtained, 24 to 48 hours admission should be done.[16]

Complete recovery is achieved soon after surgery, though some of the patients still experience postoperative pain in 2 to 3 weeks. This could be managed by giving neuromodulator agents (eg carbamazepine, gabapentine and so on) continuously untill postoperative follow up day begin. Initial treatment with additional analgetic agents be may needed in the beginning of postoperative period.[16]

Basically this procedure has good result.After percutaneous RF gasserian rhizotomy, about 80 % patients completely recover from the attack, and about 15% to 20% experience reccurency in 12 months. This procedure could be reperformed if patients experience reccurence attack. In an unmanageable trigeminal neuralgia, patient who have failed repeat RF neurotomy or when RF can not be performed, microvascular decompression should be an alternative choice.[16]

Complication can be diminished by carefully selected of the patient with attention to both somatic and psychosocial factors, and the procedure must be performed by a trained clinician in the optimal environment.[17]

The risks include anesthesia dolorosa in 0.6-6% of patients. Transient or permanent cranial nerve palsies may occur. More serious complication such as meningitis, abscess, blindness, carotid-cavernous fistula and even brainstem injury may occur but very rare. Corneal anesthesia that lead to keratitis may occure as the complication of ophthalmic division lesion. Lesion of mandibular division may reduce lingual sensation and slur speech.[17,8]

Microvascular decompression

Since Dandy theorized that vascular compression of the trigeminal neuralgia is responsible for TN, microvascular decompression (MVD) has been widely used with favorable outcomes.[18]

Craniotomy is performed in the procedure, where trigeminal nerve in posterior fossa which is compressed by the vesel is taken off and still maintaining the nerve function.[14]

Several report on this procedure result has reported that almost all of the patient is recovered. Meanwhile 80% of patients feel free from pain at one year, , 75% at three years and 73% at five years. Mortality rate from this procedure is low ; 02% to 0,5%. Post operative adverse events such as CFS leaks, infarct or hematomas occur in 4% patients. Other complication such as diplopia and bell's palsy is uncommon. But 7% of patients have reported the sensory loss. Ipsilateral hearing loss may be the prolonged complication that occurs in 10% patients.[14]

A study from Jian Hai shows that patient with atypical neuralgia attains recovery by decompression on the whole trigeminal root.26 patients with vascular decompressien were detected using magnetic resonance tomographic angiography, they then were diagnosed with atypical trigeminal neuralgia. Up to 46,2 % patients have contradictory arterial or venous formation. Predilection areas respectively are supero-medial , supero lateral and inferior arround circumference of trigeminal root. Almost half of patients achieve recovery after complete decompression, while the rest who do no have complete decompression have partial relief (30.8%) and recurency (19,2%).[18]

Although study evoke that this procedure has advantage and disadvantage. Nevertheless, patient's satisfaction by using this procedure is still high.

Gamma knife surgery

For medically refractory idiopathic TN, stereotactic radiosurgery with gamma knife surgery (GKS) has accepted to be the good treatment. It is non invasive and using beam of radiation at the root of trigeminal nerve.

Different results of this procedure have been reported from several studies.However, the interpretation of the studies is still unclear because the reports do not have the same standar outcome.[19]

In Kostas et al study, stereotactic surgery is found to be valuable in idiopatic TN treatment beside its safety. Result was collected from two groups.Group one is patients with no previous surgery.This group has respones rate 92,9%. Group 2 is patients who had undergone surgery with response rate 85,7%. Group one has better outcome either in first and second year post treatment (82,5% and 78%) while group 2 has only 69,4% in first year and 63,5% in second year.[19]

Some complications such as post treatment facial numbness, sensory loss or parasthesias have been reported. Facial numbness is commonly found after the procedure (9-37%) while sensory loss/or parasthesia only reported by 6-13% of patients. No anesthesia dolorosa has been reported.[14]

REFERENCES

1. Katusic S, Beard CM, Bergstralh E, Kurland LT. I ncidence and clinical features of trigeminal neuralgia, Rochester, Minnesota, 1945- 1984. Ann Neurol 1990;27:89-95.
2. Darlow LA, Brooks ML, Quinn PD. Magnetic resonance imaging in the diagnosis of trigeminal neuralgia. J Oral Maxillofac Surg. 1992;50:621-6.
3. Turp JC, Gobetti JP. Trigeminal neuralgia versus atypical facial pain: a review of the literature and case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1996;81:424-32
4. Tölle T, Dukes E, Sadoysky A. Patien burden of trigeminal neuralgia: results from a cross-sectional survey of health state impairment and treatment patterns in six European countries, Pain Pract. 2006;6:153-60
5. Marbach JJ, Lund P. Depression, anhedonia and anxiety in temporomandibular joint and other facial pain syndromes, Pain.1981; 11(1): 73-84
6. Love S, Coakham HB, Trigeminal neuralgia: pathology and pathogenesis. Brain. 2002;124:247
7. Stannard C, Booth S. Pain. 2nd ed. 2004: 318
8. Cheshire WP. Trigeminal neuralgia: For one nerve a multitude of treatments. Expert Rev Neurother 2007;7(11):1565-75
9. Bagher SC, Farhidvash F, Perciaccante VJ. Diagnosis and treatment of patients with neuralgia trigeminal neuralgia. JADA 2004;135: 1713-15
10. Benneto L, Patel NK, Fuller G. Trigeminal neuralgia and its management. British medical journal. 2007; 334: 201-4
11. Burchiel KJ. A new classification for facial pain. Neurosurgery.2005; 53(5): 1164-66
12. Eller JL, Raslan AM, Burchiel KJ. Trigeminal neuralgia: definition and classification. Neurosurg. Focus 18(5), E3 (2005)
13. Erdine S, Ozyalcin NS, Cimen. Comparison of pulsed radiofrequency with conventional radiofrequency in the treatment of idiopathic trigeminal neuralgia. European Journal of Pain. 2007;11:309

14. Cruccu G, Gronseth G, Alksne J. AAN-EFNS guidelines on trigeminal neuralgia management. European Journal of Neurology. 2008;15:1013-4
15. Erdine S, Racz GB, Noe C. Somatic blocks of the head and neck. In: Raj PP, editor. Interventional pain management. 2nd ed. Philadelphia: Saunders- Elsivier; 2008: 82-7.
16. Yin W. Radiofrequency gasserian rhizotomy: The role of RF lesioning in the management of facial pain.Techniques in Regional Anastesia and Pain Management 2004;8:31
17. Zundert JV, Sluijter M, Kleef MV. Thermal and pulsed radiofrequency._In: Raj PP, editor. Interventional pain management. 2nd ed. Philadelphia: Saunders- Elsivier; 2008: 61
18. Jion Hai, Shi-Ting, Qing-Gang Pan. Treatment of atypical trigeminal neuralgia with microvascular decompression. Neurology India. 2006;54:53-57
19. Fauntas KN, Smith JR, Lee GR. Gamma Knife stereotactic radiosurgical treatment of idiopathic trigeminal neuralgia: long-term outcome and complications. Neurosurg Focus. 2007;23:1