TRIGEMINAL NEURALGIA – DIAGNOSIS AND TREATMENT: A REVIEW

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Abstract

The trigeminal nerve (V) is the fifth and largest of all cranial nerves and it is responsible for detecting sensory stimuli that arise from the craniofacial area. The nerve is divided into three branches: ophthalmic (V1), maxillary (V2), and mandibular (V3); their cell bodies are located in the trigeminal ganglia and they make connections with second-order neurons in the trigeminal brainstem sensory nuclear complex. Trigeminal neuralgia (TN), also known as tic douloureux, is a chronic pain condition characterized by recurrent brief episodes of electric shock-like pains affecting the fifth cranial (trigeminal) nerve, which supplies the forehead, cheek, and lower jaw.Diagnosis is essentially clinically; magnetic resonance imaging is useful to rule out secondary causes, detect pathological changes in affected root and neurovascular compression (NVC). Carbamazepine is the drug of choice; oxcarbazepine, baclofen, lamotrigine, phenytoin, and topiramate are also useful. Multidrug regimens and multidisciplinary approaches are useful in selected patients. Microvascular decompression is surgical treatment of choice in TN resistant to medical management. Patients with significant medical comorbidities, without NVC and multiple sclerosis are generally recommended to undergo gamma knife radiosurgery, percutaneous balloon compression, glycerol rhizotomy, and radiofrequency thermocoagulationprocedures. Partial sensory root sectioning is indicated in negative vessel explorations during surgery and large intraneural vein. Endoscopic technique can be used alone for vascular decompression or as an adjuvant to microscope. It allows better visualization of vascular conflict and entire root from pons to ganglion including ventral aspect. The effectiveness and completeness of decompression can be assessed and new vascular conflicts that may be missed by microscope can be identified.

Keywords: Trigeminal neuralgia, craniofacial, neurovascular compression, tic douloureux, Microvascular decompression

Introduction

The trigeminal nerve is one of 12 pairs of nerves attached to your brain. It's responsible for transmitting touch and pain sensations from your face and head to the brain. There are three branches in the trigeminal nerve, all of which are responsible for transmitting nerve impulses to different parts of your face:

- **The ophthalmic branch.** The top portion of your face, including your forehead.
- **The maxillary branch.** Everything in the middle of your face, including your cheeks, nostrils and upper lip.
- **The mandibular branch.** The lower portion of your face, including your lower lip and jaw area.

Trigeminal neuralgia (TN) is defined as sudden, severe, brief, stabbing, and recurrent pain within the distribution of one or more branches of the trigeminal nerve (TR N). [1] This condition is almost always unilateral and can involve one or more divisions of the trigeminal nerve. TN is a syndrome characterized by paroxysmal facial pain. The term "tic douloureux" was given by the French physician Nicolaus Andre in 1756 because of the facial spasms that can sometimes accompany severe pain attacks.[2]

Etiology

The trigeminal nerve starts at the pons. Most cases of trigeminal neuralgia are due to the compression of the trigeminal nerve root within a few millimeters of its entry into the pons. Between 80% and 90% of the cases of TN are caused by compression by an adjacent artery or a vein.[3] The blood vessel, which has been mostly implicated in about 75% to 80% of the cases, is the superior cerebellar artery. Other blood vessels that are known to cause TN include the anterior inferior cerebellar artery, the vertebral artery, and the petrosal vein.Some of the other causes of nerve compression include meningioma, acoustic neuroma, epidermoid cyst, and rarely an arteriovenous malformation or a saccular aneurysm.Multiple sclerosis is a risk factor for TN, and it is reported in about 2% to 4% of patients with TN. This is secondary to the demyelination of the trigeminal nerve nucleus by multiple sclerosis.[4]

Central causes of the disease have also been proposed for TN; reduced basal ganglia μ-opioid receptor[5], altered gray matter (GM) in sensory, and motor cortex has been implicated.[6] The dysfunction of multiple modulatory mechanisms probably plays a key role in the pathophysiology.[7] Demyelination, dysmyelination giving increases to electrical hyper excitability, spontaneous and triggered ectopic impulse and cross excitation among neighboring afferents have been proposed in ignition hypothesis.[8] According to the bio resonance hypothesis, TR N fibers are damaged when the vibration frequency of nerve and surrounding structure becomes close to each other.[9] The brain sagging/ arterial elongation hypothesis is also believed to cause nerve

compression.[10]

Epidemiology

The lifetime prevalence of TN is estimated to be 0.16%–0.3%,[11] while the annual incidence is 4–29 per 100 000 person-years.[12-14] It is more prevalent in women than in men (F:M ratio 3:2).[13-15] The incidence increases with age, with a mean age of onset of 53–57 years and range of 24–93 years in adult series.[16] Furthermore, a recent paediatric headache clinic of 1040 identified five children in the age range 9.5–16.5 years with TN.[17]

Symptomatology

In early descriptions of TN, the disorder was called tic douloureux[18], addressing the characteristic wince that TN patients may exhibit at a pain paroxysm; TN pain is not only extremely painful, it is also characteristic that the pain is sudden and unexpected, and short-lasting, hence the term pain paroxysm. The pain quality is stabbing, electrical shock-like, or shooting. Although one single pain paroxysm may only last a fraction of a second, these paroxysms may recur, after a refractory period, many times a day, and they may come in a series of attacks with many paroxysms close together. Approximately half of the TN patients also have concomitant continuous pain: an aching or dull or burning background pain of lower intensity in the same area as the paroxysmal pain[19-21]. The continuous pain is usually present during the same periods as the paroxysmal pain. This background pain is most common in women[19,22,23.] Refractory period and trigger factors Many patients experience a refractory period after a paroxysmal attack where new attacks cannot be elicited. The pathophysiological mechanism of this phenomenon is unknown. It has been proposed that it is caused by hyperpolarisation of the sensory neuron.[24] In early studies by Kugelberg and Lindblom, the presence and duration of the refractory period in TN was a function of the intensity and duration of the preceding attack.[25] It is highly characteristic that pain is triggered by innocuous sensory stimuli to the affected side of the face. Sensory stimuli may be extraoral and intraoral. The most frequent trigger factors involve normal daily activities such as light touch, talking, chewing, brushing teeth and cold wind against the face.[26,27] It has been suggested that apparently spontaneous pain paroxysms may in fact be elicited by very subtle sensory stimuli or movements. Localisation TN most frequently affects the 2nd and/or 3rd trigeminal division and the right side is slightly, but significantly, more often affected than the left side.[26] Bilateral TN is very rare in classical TN, and should raise suspicion of secondary TN. Natural history There are very few studies examining the natural history of TN. It has been proposed that pain may worsen with time and that TN in its chronic state is characterized by longer lasting, medically refractory pain, sensory disturbances and progressive neuroanatomical changes of the trigeminal root.[23] Several studies have now challenged this notion; Di Stefano et al. found that in the majority of TN patients the pain does not increase in frequency or duration, nor did it become refractory to medication, and the dosage needed to relieve pain did not increase with time.[28] Maarbjerg et al. found that concomitant persistent pain and neuroanatomical morphological changes were not related to age or to duration of disease.[19,22] A feature that is also very characteristic to the course of TN is unpredictable periods of complete remission that may last months or even years. This unusual phenomenon in neuropathic pain is most likely attributed to a reduction in excitability and partial remyelination.[24] Autonomic symptoms in facial pain Traditionally, autonomic symptoms such as tearing and rhinorrhea have not been associated with TN. However, it is now known that a large proportion of TN patients have autonomic symptoms from time to time.[26,27,29] Keeping in mind that the trigeminovascular reflex can be elicited by intense facial pain in general,[30] it is not surprising that there can be sporadic autonomic symptoms in TN. The challenge is related to differential diagnosis; short-lasting triggered stabbing pain with pronounced and consistent autonomic symptoms is characteristic of short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT), and short-lasting unilateral neuralgiform headache attacks with autonomic symptoms (SUNA).[31]

Diagnostic considerations

The diagnosis of TN is primarily based on patient history, as there are no definitive laboratory or diagnostic tests. When obtaining patient history, special attention should be paid to the potential pitfalls leading to misdiagnosis such as a symptomatic cause of pain, odontogenic pain and associated autonomic symptoms (Table 1). When obtaining patient history, one should pay special attention to the onset of pain; if the pain was preceded by or coincided with a herpes zoster rash in the ipsilateral trigeminal distribution, painful trigeminal neuropathy attributed to acute herpes zoster should be considered.[32] In pain preceded by a relevant trauma to the ipsilateral side of the face, such as invasive dental procedures or fractures, painful posttraumatic trigeminal neuropathy (PPTN) is more likely the correct diagnosis. Studies have shown that pain in PPTN may be comparable to TN pain with short, intense triggered pain, but in PPTN there are usually clear cut sensory abnormalities, including both loss and gain of function, corresponding to the damaged peripheral nerve.[33] Also important when obtaining the patient history is the location of pain; pain originating distinctly or diffusively from the teeth should be evaluated by a dentist because, for example, a cracked tooth may present with TN-like pain evoked by chewing hard foods. In bilateral constant pain located in the temporomandibular area, tension-type headache, temporomandibular joint disorder and persistent idiopathic facial pain should be considered. If the shortlasting, intense stabbing pain is isolated to the scalp or occipital area, diagnoses such as occipital neuralgia, primary stabbing headache and paroxysmal hemicrania should be considered. Glossopharyngal neuralgia is located to the back of the tongue, the soft palate and the pharynx, and nervusintermedius neuralgia is located deep in the ear. Finally, associated symptoms are important; if each pain attack is accompanied by autonomic symptoms such as conjunctival injection, miosis or lacrimation, SUNA, SUNCT or paroxysmal hemicrania are important differential diagnoses.

The symptomatology of trigeminal neuralgia is typically very characteristic, with patients reporting intense stabbing touch-evoked unilateral facial pain in the cheek, the area of the nostrils, teeth or jaw. Primary and secondary, i.e. pain secondary to multiple sclerosis or space-occupying lesion, TN may be indistinguishable based on pain characteristics. Meanwhile, in patients with secondary TN, neurological deficits, extra-trigeminal symptoms, bilateral pain and young onset are more frequent.

Primary and secondary headache and facial pain differential diagnosis includes:

- **Glossopharyngeal neuralgia** causes evoked stabbing pain located to the back of the tongue, the pharynx or deep in the ear. Trigger factors are somewhat different from TN and include swallowing, coughing, sneezing
- **Painful posttraumatic trigeminal neuropathy** can cause stabbing and touch-evoked pain similar to TN, but pain is by definition preceded by trauma and there are usually clear-cut neurological abnormalities of both gain and loss of function corresponding to the affected peripheral nerve
- **Persistent idiopathic facial pain** causes touch-evoked or spontaneous dull or aching constant pain
- **Painful trigeminal neuropathy attributed to acute herpes zoster** causes burning and stabbing pain preceded by a herpetic rash in the trigeminal distribution. Tingling sensations and neurological abnormalities with both gain and loss of function are frequent
- **Short-lasting unilateral neuralgiform headache attacks with autonomic symptoms (SUNA), short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT)** or **paroxysmal hemicrania** cause touch-evoked and spontaneous stabbing orbital, supraorbital or temporal pain accompanied by ipsilateral autonomic symptoms. Unlike TN, pain may change side
- **Cluster headache** causes orbital, supraorbital or temporal pain accompanied by ipsilateral autonomic symptoms and restlessness. Duration is from 15–180 minutes. Unlike TN, pain may change side
- **Primary stabbing headache** causes stabbing spontaneous pain in the scalp and is not accompanied by autonomic symptoms

Odontogenic differential diagnosis:

- **Cracked tooth** can cause evoked shooting pain when chewing hard foods
- **Caries** or **pulpitis** can cause evoked pain at intake of sweet, cold or hot foods. The pain can last from ten minutes up to hours

[a]Autonomic symptoms are conjunctival injection, lacrimation, rhinorrhea, nasal congestion, sweating, miosis, ptosis and eyelid edema.

Table 1: Differential diagnosis in trigeminal neuralgia

Medical Treatment

Carbamazepine (CBZ) is drug of choice in TN; baclofen, lamotrigine, clonazepam, oxcarbazepine, topiramate, phenytoin, gabapentin, pregabalin, and sodium valproate can be used.[34-36]Multi drugs are useful when patients are unable to tolerate higher doses of CBZ.[37]With an availability of increasing number of anticonvulsant drugs, it is likely that surgical option may not be offered for many years.[38]

Intravenous infusion of a combination of magnesium and lidocaine can be very effective in some patients.[39] Five percent lidocaine plaster[40]and 8% capsaicin patch[41]can be useful in some TN. The 5-HT R3 antagonists, neurokinin-1 antagonists, or mast cell stabilizers may have role in the treatment of TN. A multidisciplinary approach using antidepressants and anti-anxiety drugs such as amitriptyline42and duloxetine is needed for the management of emotional status.[43]

Botulinum toxin Type A injections may be offered before surgery or unwilling to undergo surgery, and in failed drug treatment.[44]Tetracaine nerve block as an additional treatment after CBZ, acupuncture and peripheral nerve stimulation can be used.[45-47]Deep brain stimulation of the posterior hypothalamus may be considered as an adjunctive procedure for refractory TN of first division,[48] especially in MS.[49]Motor cortex stimulation can be used in certain neuropathic or deafferentation pain.[50] Treatment of associated tumor, AVM, epidermoid, aneurism, and hydrocephalus in Chiari malformation can resolve TN.[51]

Gamma knife radiosurgery

Radiation may block the conduction of excessive sensory information responsible for triggering pain attacks.[52] Radiosurgery results in about 50% drop in FA values at the target with no significant change in outside the target nerve. Radiosurgery primarily affects myelin sheath.[53]

TN after a failed MVD, significant medical comorbidities, and MS are generally recommended to undergo GKRS. It is indicated in typical or atypical TN,[54] with or without vascular compression,[55] and in recurrence after GKRS, glycerol rhizotomy (GR), radiofrequency thermocoagulation (RFTC), and percutaneous balloon compression (PBC).[56-58] Repeat GKRS provides a similar rate of pain relief as the first procedure. The best responses are observed when there is good pain control after first procedure, with new sensory dysfunction and in single division nerve distribution typical TN.[56-59]

GKRS can be given using one or two isocenters[60]and targeting radiosurgery posteriorly at dorsal REZ,[61] or anteriorly in retrogasserian zone.[62] 80 Gy,61 85 Gy,[63] and 90 Gy can be used. Lower dose to the root are associated with less side effect, whereas higher doses provide better pain control with less risk of recurrences but more side effect such as facial numbness. The benefits and risks of higher dose must be carefully discussed with patients, since bothersome facial numbness, may be an acceptable for patients with severe pain.[64]

Radiosurgery can be given using single fraction; multiple fractions can deliver comparatively higher doses. Although hypofractionated stereotactic radiotherapy is not associated with any facial numbness, single fraction radiosurgery provides better pain relief and a lower recurrence rate as compared to hypofractionated technique.[65] Radiosurgery can be given with or without frame-based method,[66] with MRI or computerized tomography (CT) planning when there is contraindication to MRI.[67]

Initial pain relief is 77%–96%, which takes about 1–3 weeks (sometimes 10 weeks or longer). Results are better in typical neuralgia with single nerve distribution pain.[59]About 37%[68] and 95%[69] of patients become pain free within 48 h, and 10 days of procedure, respectively. Although the long-term results of GKRS are not as satisfactory as MVD,[70,71] it is an effective alternative with more than 50% long-lasting pain relief.[72,73] Pain control rate is inferior in vertebrobasilarectasia highlighting need for multimodality management.[74] Although GR provides urgent pain relief than GKRS, Gamma knife provides better long-term pain relief with less morbidity.[75]

Recurrence can be seen in about 15% and 50% at 32 months[69]and long-term follow up, respectively.[71,72] Trigeminal sensory deficit is observed in 30%–35%[69]which is more in diabetes mellitus, after RFTC, pain relief coming after 30 days of GKRS,[68] failed MVD or GRand repeat GKRS.[58] The cyber knife provides the high precision of dose with the sparing of healthy tissues. Effectiveness and safety of frameless stereotactic radiosurgery (SRS) using cyber knife system are comparable to frame bases SRS. X-knife radiosurgery also provides effective pain relief with a low complication rate.

Percutaneous Balloon Compression

PBC selectively avoids injury to the small unmyelinated fibers that mediate the corneal reflex. Balloon compression is indicated in patients difficult to communicate, MS, failed MVD, with significant medical comorbidity, multiple divisions including first division, without vascular compression, and in repeat PBC. PBC is reserved for patients in whom the effect of GR has been of short duration or difficult to repeat due to cisternal fibrosis

3D CT reconstructions can identify an ideal pear shape configuration to improve outcome. Procedure can be performed under local or general anesthesia.There is controversy regarding duration of compression, in one study there were no differences in outcomes between 60 s and longer times, whereas in other study, longer compression time of 70–90 s resulted in better outcome. Pear shape balloon is an indication of proper compression and higher pain-free survival, whereas persistent elliptical shape is a bad sign and an indication for aborting the procedure. There is 2% risk of technical failures.

PBC is a safe, simple, and effective method of about 90% temporary pain relief. Repeat PBC, though associated with some increase in complications, is reasonably safe. Single trigeminal division, primary procedure in the absence of previous operations, and the pear shape balloon are associated to higher pain-free survival.Results in MS patients are comparable to classic TN. About 14%, 18.9%, 29.5% recurrence is observed within 2, 3, and 5 years follow up, respectively after PBC. Symptomatic dysesthesias, masseter muscle weakness, cardiovascular stress, cheek hemorrhages, corneal ulceration, infections, and transient diplopia are also observed.

Glycerol Rhizotomy

GR is indicated in patients unresponsive to pharmacotherapy, significant medical comorbidities,MS, unilateral and bilateral pain, and after failed MVD. It is cost-effective than MVD, RFTC and GKRS. GR is a safe and efficacious method as a repeat procedure.

The immediate success rate is about 95%with 50%–60% recurrence at 24 months follow up. GR is a simple procedure, and most of the complications are reversible. There is significant positive correlation between the presence of cerebrospinal fluid (CSF) outflow and good success rate. Significant number may experience either mild numbness or dysesthesias. New facial numbness after GR is associated with excellent pain control. An-

esthesia dolorosa although rare may be observed.

Radiofrequency Thermocoagulation

RFTC can be used in bilateral pathology, elderly, recurrence after failed MVD, vertebrobasilardolichoectasia, and MS. Peripheral nerve block, and general anesthesia could relieve perioperative pain without an increase in complications. Although pulsed radiofrequency (PRF) treatment is associated with less complication than conventional RFTC, it is not as effective as the conventional procedure. Higher intraoperative PRF voltage and electrical field intensity may provide better pain relief. Combined PRF and continuous radiofrequency (CRF) can achieve comparable pain relief with lesser side effects as compared to CRF. Initial pain control rate is about 95% with about 25% recurrent pain, occasional jaw weakness, corneal anesthesia, and troublesome dysesthesia.

Peripheral Nerve Section

Peripheral neurectomy is a safe and effective procedure for elderly patients, in rural and remote centers where neurosurgical facilities are not available. Pterygopalatine fossa segment neurectomy of maxillary nerve can be used in elderly who may not tolerate craniotomy, or when RFTC and GR treatment is not possible. Pain relief can be lasting from 15 to 24 months. Loss of sensation and recurrences are associated with peripheral neurectomy.

Partial Sensory Root Section

Partial sensory root sectioning (PSRS) is indicated in MS associated with negative vessel explorations during MVDand in large intra neural vein that is difficult to mobilize.PSRS is also recommended in re exploration after failed MVD when there is no NVC. Excellent to good outcome is observed in 70% caseswith minimal sensory loss.

Microvascular Decompression

MVD is indicated in Type 1or Type 2 TN, with NVC.The cure rate is higher in arterial compression compared to venous or no NVC.MVD is also indicated in MS,isolated V2 TN, ectatic vessel with neuralgia, and after SRS. MVD is recommended in younger patients with longer life expectancy and healthy elderly [Figure 1]. Although less invasive procedure may be preferable in elderly patients, as complications do tend to increase gradually with an advanced age, MVD in physiologically healthy elderly population remains a reasonable surgical option.

Dissection in MVD is not significantly difficult after GKRS.Presurgical virtual endoscopyand 3D computer graphics models can provide excellent visualization of NVC and allows simulation. The dextroscope system can also create a stereoscopic neurovascular model to shorten the learning curve.

Indocyanine green angiography could be a helpful adjunct in decompressing the TR N and can guide the surgeon to the nerve-vessel conflict. All vessels, including the transverse pontine vein near meckel's cave, in relation to the nerve should be decompressed. There may be multiple vessels related to the root.] Sacrifice of a small intraneural vein can be performed while PSRS is preferred over extensive mobilization of large vein. Wrapping techniques can better decompress intraneural artery.

Prominent suprameatal tubercle should be drilled out for better exposure of entire TR N and vascular conflicts. Laterally placed craniotomy helps to visualize the whole nerve root along with REZ. Dissection of the cerebellar horizontal fissure and rostral retraction of the superior semilunar lobule allows easy identification of the REZ with minimal traction. Supracerebellar route permit identification and dissection of the offending supracerebellar artery. Whole surface of the TR N can be observed easily by combining these two approaches.Preservation of the vestibular nerve arachnoid minimizes complications and optimizes surgical outcome.

Autologous muscle graft, oxidized regenerated cellulose,and fibrin glue alonecan be used to transpose vessel away from the nerve. Transposition of the offending vessel with Teflon wool or slings, especially in tortuous NVC, is a useful. Aneurysm clip with or without unabsorbabledural sling can be used.

Adhesion between the trigeminal root and surrounding structures, secondary to fibrin glue or prosthesis, can stretch nerve, which can cause recurrence. Prosthesis if used should be lying in subarachnoid space or cistern avoiding contact to dura matter or tentorium. Arachnoid membrane of CPA can be used as a sling to transpose the superior cerebellar artery.

Combingor PSRScan be combined with MVD when no vascular conflict is detected.Muscle pieces interposition between the duramater, artificial dura mater, cranioplasty, sealing of mastoid sinus by bone wax and muscle can be effective technique for the prevention of CSF leak.Re surgery is an effective and safe after failed MVD. The preservation of the petrosal vein and its tributaries, lateral inversion vein of ventricle IV is important in preventing the postoperative vestibular and cerebellar disorders.

Early outcome after MVD in typical TN with associated NVC is 90%–95% which drops to 75% at 1and 5 years follow up. MVD is significantly superior to GKRS. TR N combing has a much higher pain relief in patients without vascular compression than those with vascular compression. 3D models by fusing CTA and FIESTA can be used to evaluate the translational and rotational shift of the compressive artery, and decompressed distance from the root after failed MVD.

Immediate postoperative pain relief is a good predictor of better long-term outcome. Type 2 TN, presence of autonomic symptoms, MSare associated with poor prognosis. Shorter preoperative duration, older age, and typical features are good predictors of favorable outcome.Subset of patients progressed from Type 1 to Type 2 TN over time also have good outcome resembling Type 1. Low FA values can be reversed after successful MVD.Significant reduction of FA value may predict an optimistic outcome of MVD.

The trigemino-cardiac reflex due to stimulation of the TR N during MVD may result in about 50% fall in heart rate and mean arterial blood pressure, cessation of manipulation lead to normalization of parameters. Facial nerve dysfunction, hearing abnormality,and TR N dysfunction may be observed, especially after more dissection and mobilization of respective nerve. Brainstem auditory evoked potential monitoring and neuro-endoscopy during MVD can preserve hearing function.

Recurrences, ranging from 18% to 34%, may be seen at long-term follow up. It is more common within 2 years of surgery and thereafter at a rate of 2%–3.5% per year. Significant predictors of recurrence are younger age, and symptoms lasting longer than 10 years.Recompression due to regrowth of new vein or artery-can cause TN. Hardened Teflon can pierce nerve and produce TN, therefore the contact of prosthesis, if used, with the nerve should be avoided. Outcome can be improved by establishing center dealing TN. Late communicating hydrocephalus may be a potential complication of MVD surgery.

Endoscopic vascular decompression

Endoscopic techniques are increasingly being used in spine, skull baseand intracranial pathologies. Endoscopic technique can be used alone in TNor as an adjuvant to microscopeIt is a mini-

mally invasive technique,allows better visualization of entire root from pons to ganglionincluding ventral aspect. The endoscope is a valuable tool during MVD, especially when a bony ridge hiding the direct microscopic view of vascular conflict.Effectiveness and completeness of decompression can be better assessed. New nerve-vessel conflicts can be identified which may be missed by microscope in 7.5%–33% of patients. It is safe, requires less brain retractionand associated with improved pain relief with lower complications as compared to MVD. The vascular conflict is mostly distributed in the medial side on second division while it is in lateral area for third division in TN.[76]

Prognosis

TN is characterised by recurrences and remissions. Many people have periods of remission with no pain lasting months or years but in many, TN becomes more severe and less responsive to treatment over time, despite increasing pharmacological intervention. Most patients with TN are initially managed medically, and at our tertiary referral centre approximately 50% eventually have a surgical procedure.[77]

Conclusion

Recent advances in TN have led to an improvement in its classification on the basis of the neuroimaging findings. Better understanding and description of other neuralgiform disorders such as SUNCT and SUNA have made the differential diagnosis clearer. Improved care pathways involving multidisciplinary teams and potentially new medications is resulting in improved outcomes for patients with TN.

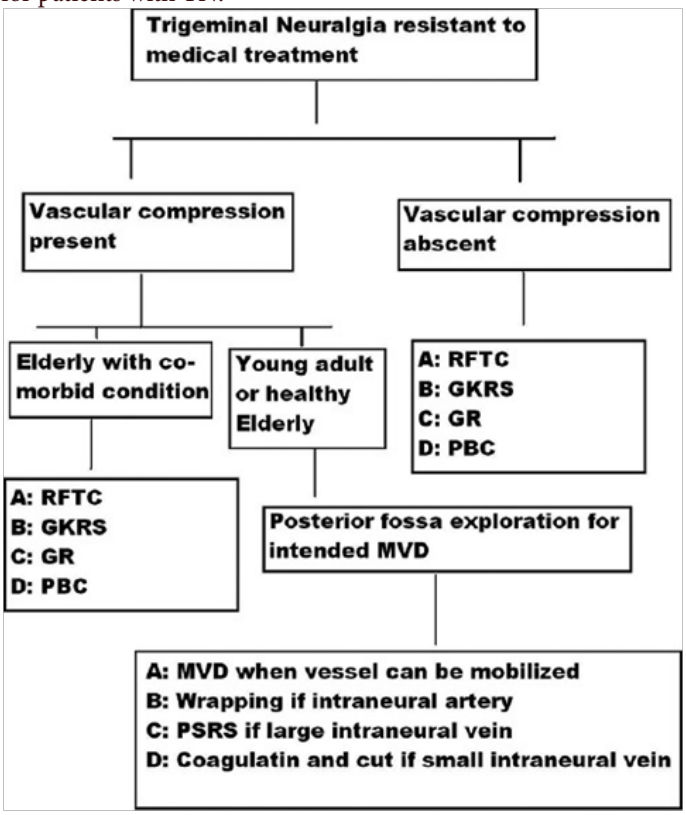

Fig. No.1: Flow chart showing treatment plan of trigeminal neuralgia resistant to medical management. GKRS = Gamma knife radiosurgery, GR = Glycerol rhizotomy, MVD = Microvascular decompression, PBC = Percutaneous balloon compression, PSRS = Partial sensory root sectioning, RFTC = Radiofrequency thermocoagulation

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