Trigeminal Neuralgia; Chemical Ablation of Gasserian Ganglion Experience from Bangladesh

Chandra Shekhar Karmakar*, Ashraful Hoque, Lutfur Rahaman and ABM Muksudul Alam

1Department of Anesthesia, ICU & Pain Medicine, Shaheed Suhrawardy Medical College Hospital, Bangladesh

2Center for Medical Biotechnology, MIS, DGHS, Bangladesh

Abstract

Trigeminal Neuralgia (TN) is painful one of the commonest cause of neurogenic fascial pain after 50 years of age. The pain can be described as a sudden, paroxysmal severe pain which is usually triggered by a non-painful stimulus like touch, eating, drinking, washing and shaving or by any thermal changes. There is a lack of certainty regarding the etiology and pathophysiology of Trigeminal neuralgia. The treatment of this condition can be very challenging even the numerous options patients and physicians can choose from. This multitude of treatment options sometimes poses the question as to which treatment fits which patient best. For patients who are refractory to medical therapy multiple techniques such as Gasserian ganglion percutaneous techniques, gamma knife surgery and microvascular decompression are the most promising invasive treatment options. Among them some of the commonly performed interventions Pain physicians are doing currently to provide pain relief are balloon compression, Glycerol rhizolysis and RF (Radio Frequency) rhizotomy. We report a case of a forty five years old female patient who presented with left sided lancinating pain in lower jaw for 3 years which was not relieved by medication at all. Investigations and regular follow up has revealed classical trigeminal neuralgia pattern and percutaneous glycerol rhizolysis has been done in our centre with very good results.

Keywords: Trigeminal neuralgia; Interventional treatment; Minimally invasive; Pain management; Gasserian ganglion; Glycerol rhizolysis

Introduction

Trigeminal neuralgia is a quite common classic neuralgia and commonest cause of facial pain after 50. It is known to have the worst possible pain in the world; previously it was termed as suicidal pain. The incidence of Trigeminal Neuralgia (TN) is 4.3 per 100,000 persons per year, with a slightly higher incidence for women (5.9/100,000) compared with men (3.4/100,000). It is almost exclusively unilateral neuropathic pain. The pain is located in the distribution of trigeminal nerve. The burst of pain can occur spontaneously or from a stimulus in the specific area of the face known as trigger zone. Sometimes it is difficult to identify the trigger zone. Patient usually avoid touching, brushing or chewing to avoid stimulation of trigger zone. Pain may occur daily for week, months and then cease, sometimes for months to years this is called remission period. Evidence suggests that pain occurs because of pressure on the trigeminal nerve root at the entry zone into the pontine region of the brain stem [1,2]. Compression by tumor or blood vessel may cause local pressure, leading to demyelination of the trigeminal nerve roots. Experimental studies suggest that de-myelinated axons are prone to ectopic action potential generation [1,3]. Demyelination has also been shown in cases of TN associated with multiple sclerosis and tumor compression of the trigeminal nerve root. The condition may be severely disabling with high level of morbidity, particularly in the elderly patients [4].

Diagnosis

The diagnosis of Trigeminal Neuralgia (TN) is purely clinical. No medical test is available to confirm the diagnosis. The International Association for the Study of Pain (IASP) and International Headache Society (IHS) both has suggested their own diagnostic criteria for Trigeminal neuralgia [5]. These are remarkably similar and highlight the sudden, explosive nature of the pain. For further descriptions of the condition, both classifications allude to vascular compression, MS and tumors as known an etiological cause. The IASP classification makes a distinction between classic Trigeminal Neuralgia (TN) (including MS) and secondary neuralgias (caused by structural lesions...
and injuries, but not including MS), while IHS separates idiopathic Trigeminal neuralgia from the symptomatic TN depending on the presence of a structural lesion. IASP defined trigeminal neuralgia as “Sudden, usually unilateral, severe brief stabbing recurrent pains in the distribution of one or more branches of the Vth cranial nerve [5].” Painful unilateral affliction of the face, characterized by brief electric shock like pain usually limited to the distribution of one or more divisions of the trigeminal nerve. Pain is frequently evoked by minimum stimuli including washing, shaving, smoking, talking, and brushing the teeth, but interestingly it may occur spontaneously. The pain is abrupt in onset and termination and may remit for varying periods [6]. Differential diagnoses for facial pain include headache disorders, temporomandibular joint pain, dental pain, chronic sinusitis, otitis media, as well as myo-facial pain.

Case Presentation

The 45 years old, well-oriented female patient from a low socio economical status reported to Pain clinic of the department of Anesthesiology, Shaheed Suhrawardy Medical College Hospital, Dhaka, with a history of facial pain on the left lower quadrant of face for 3 years. Her pain was sharp, lancinating electric shock like. Started spontaneously and intensity increased gradually over the last 3 years. Pain was unbearable and lasts for one to two minutes. According her statement frequency of her attack increased in extreme of temperature, even brushing and chewing on affected side trigger her pain. She is normotensive and non diabetic. She had no history of trauma or surgery on her affected side of face. On examination her face is symmetrical with no bony deformity or joint abnormality detected. There was no motor or sensory deficit on her affected side. Her Oral hygiene was poor. The patient had previously sought treatment from dentist and medical doctors; initially Carbamazepine was tried by which she had no pain relief. Years back she insisted her dentist to pull out her 2nd and 3rd divisions of the trigeminal nerve. She has complete pain relief after 30 min of administration of glycerol. Patient was conscious oriented but slight dizziness was there for about 2 h. Patient was kept in post operative ward overnight for close monitoring. On next morning follow up she complain numbness in the distribution of maxillary and mandibular nerve. Otherwise she was completely fine, totally pain free. For needle pain she was prescribed Tab Paracetamol 500 mg 6 hourly for 5 days. Carbamazepine and Baclofen was withdrawn slowly over 7 days. At 15 days and on 1 month follow up she was totally pain free (VAS was 0) no motor deficit in her left side of face, corneal reflex was intact, slight sensory deficit in her left side over V3 and V2 distribution.

Discussion

Trigeminal neuralgia is not a static disease and is usually characterized by remissions and exacerbations [7]. It is very important to take a proper history and do extensive physical examination which is the most effective tool in diagnosing cases of Trigeminal Neuralgia. However, confusion can arise in determining exact cause of pain whether it is due to migraine, trigeminal neuralgia, atypical facial pain and cluster headaches with clinical certainty [8]. Jannetta et al. [9] had attributed the etiology of the neuralgic pain to vascular compression of the trigeminal nerve by arterial loops or veins. Several other studies including that of Ramesh and Premkumar have shown that the vascular contact and compression at the trigeminal root entry zone may be seen in a significant number of normal populations who never suffer from electric shock like pain that creates fallacy on vascular compression theory [10]. Loeser et al. [11], on the basis of their proposed theory of presynaptic inhibition, have shown that the etiology of Trigeminal Neuralgia (TN) could be due to focal changes in axonal diameter or in myelination of the fifth cranial nerve. There is another theory that postulates peripheral nerve injury or pathology of the trigeminal nerve, increase afferent auto firing probably by emphatic transmission between afferent un-myelinated axons and partially damaged myelinated axons. Trauma or prior surgeries in the
face have been associated with Trigeminal Neuralgia type symptoms referred to as Atypical Trigeminal Neuralgia, Trigeminal Neuropathy or Post-Traumatic TN. For management of TN Medical therapy is the initial treatment of choice which includes anti-convulsant drugs. Typical TN is usually responsive to initial medical management with Carbamazepine. The gold standard drug for treating TN is carbamazepine which is started at a low dose and be gradually increased to a level where the pain is controlled with minimum side effects. Alternative medication includes Oxcarbazepine, Baclofen, Gabapentin, Lamotrigine and Pimozide in combination. Around 75% of patients show partial improvement after taking this drug but on prolonged use it's efficiency decreases. A variety of procedures are available to treat refractory TN and for patients with intolerable side effects. The choice of the procedure depends on the symptoms of the patient, medical co-morbidities, and the age of the patient, prior treatment modalities and availability of the procedure. The goal of the treatment should be complete pain relief with an acceptable level of side effects. Different procedures like balloon compression, microvascular surgery, radiofrequency rhizotomy, glycerol rhizotomy, and stereotactic radio surgery work in different ways to induce axonal degeneration of the nerve. The patient should be pain free and the fear of recurrence. Pollock has reported in his series of 98 patients that 73% patients were free of pain at some point following any surgical procedure, with the chance of remaining totally pain-free without using medications at 61% and 50% after 1 and 3 years, respectively. In conclusion his study showed in almost all forms of treatment, the reported outcome is almost always comparable. Percutaneous glycerol rhizotomy is considered as the first line of treatment for refractory TN by many physicians because of the relative advantages it has over the other percutaneous procedures. Such as post-procedure sensory deficits rate is quite low. Anhydrous glycerol chemically ablates the so-called pathological site on the axons. Though it is not clear how anhydrous glycerol acts to relieve the attacks without producing the dense anesthesia which is seen after most neuro-lytic injections. But with glycerol, altered sensations in the form of dysesthesia or hypoesthesia, a sluggish corneal reflex, and temporary paresis of the V3 motor component in some of the patients have been observed. The most common complications of percutaneous glycerol rhizotomy are minor like development of a local hematoma, infection, sensory deficits, reactivation of labial herpes, and anesthesia dolorosa. Severe life threatening complications like chemical meningitis, infectious meningitis are rare [13]. Percutaneous glycerol rhizotomy can be done under local anesthesia with/without mild sedation, as an outpatient procedure with C-arm. The patient can go back to his daily activity in a few days and is saved from long-term costly medications and side effects.

Conclusion

Medical therapy should tried first for the treatment of TN, if medical treatment is in adequate interventional procedures can be considered in patients who have persistent pain despite drug therapy or who are unable to tolerate adverse effects of drugs. Percutaneous glycerol rhizotomy for patients with intractable TN is a good option for those patients who cannot tolerate the medications, as it is simple, safe, and cost-effective with minimum complications. It has a high efficacy, and with recurrence rates comparable to that of other procedures.

References