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Case Report

Trigeminal Neuralgia- Neuroanatomical Explanation and Case Report

K. Lakshmi Kumari[1], T.Sarada[2], P.V.S.S.Vijaya Babu[3]

[1]Department of Anatomy, Andhra Medical College, Visakhapatnam, Andhra Pradesh, India.
[2]Department of Anatomy, Rangaraya Medical College, Kakinada, Andhra Pradesh, India.
[3]Department of Biochemistry, Andhra Medical College, Visakhapatnam, Andhra Pradesh, India

Corresponding Author: K. Lakshmi Kumari

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ABSTRACT

Trigeminal neuralgia or tic douloureux is one of the most disabling neurologic pains. This is characterized by sudden, severe, sharp electric shock like pain along the branches of trigeminal nerve. The incidence of trigeminal neuralgia is 4 in 1,00,000 population per year. Knowledge of anatomical course and distribution of trigeminal nerve is important in diagnose and treatment. Trigeminal nerve is the largest of all cranial nerves which is mixed type of nerve. Sensory root of trigeminal nerve is divided in to 3 divisions, ophthalmic, maxillary and mandibular divisions. These three divisions carry pain, touch, temperature sensations from scalp, forehead, eye, lateral surface of nose, cheek, mandibular skin excepting angle of mandible and part of auricle. Commonest cause of trigeminal neuralgia is due to vascular compression by nearby artery of trigeminal nerve. Demyelination of trigeminal nerve is next cause. In this case report we discussed about 49 year old lady with intense, sudden electric shock like pain on her chin and cheek. The pain aggravated with trigger factors which is characteristic of trigeminal neuralgia. After physical and radiological examinations she had been diagnosed as trigeminal neuralgia. She had started with medical treatment. If medical treatment fails surgical treatment like micro vascular decompression, Gasserion ganglion ablative therapy will give good results.

Key Words: Trigeminal neuralgia, sensory supply, vascular compression, demyelination.

INTRODUCTION

The first known description of trigeminal neuralgia or a similar condition was written in second century AD by Aretaeus of Cappadocia a contemporary of Galen. Jujari, Arab physician in 11th century mentioned unilateral facial pain causing spasm and anxiety in his writings. He suggested that the cause of pain is the proximity of artery to the nerve. In 1773 John Fothergill described unilateral face pain evoked by eating or speaking, starting and ending abruptly. Earlier Nicolaus Andre had used the term tic douloureux.

Trigeminal neuralgia or tic douloureux is intractable pain in the area of distribution of one or all divisions of trigeminal nerve, usually with periods of remission and exacerbation. The cause of neuralgia is local and sometime unknown. This is the most disabling and relatively distressing neuropathic disorder with an estimated incidence of 4 to 5 per 1,00,000. [1] It is more common in women than men.

The medical and psychosocial impact of this disorder is such that it was once referred as 'suicidal disease'. [2] The peak incidence was shown to be in the 50-60yrs age group with recent evidence suggestive of an overlap by five years towards the younger as well as elder side of the group. [3,4]

The pain in the trigeminal neuralgia is described by international association for the study of pain as sudden, unilateral usually, severe, brief, stabbing, recurrent episodes of pain in the distribution of one or more branches of trigeminal nerve. [5] Attacks usually last only few seconds. But many recur repeatedly within short period of time. The attacks are often but not always precipitated by mild sensory stimulation of so called trigger zones, which may be located anywhere within the territory of the trigeminal nerve. Aetiology of trigeminal neuralgia is difficult to evaluate. To evaluate the cause of trigeminal neuralgia one should be aware of branches of trigeminal nerve. Trigeminal nerve is divided in to 3 divisions, ophthalmic, maxillary and mandibular divisions. The local reasons may be acute glaucoma or frontal sinusitis affecting ophthalmic division, dental caries of upper jaw, malignant growth or empyema of maxillary sinus affecting maxillary division and dental caries of lower jaw, ulcers or cancer of the tongue involving the mandibular division. Advances in imaging and correlative studies have shown 80-90% of classical trigeminal neuralgia have vascular compression at root entry zone. [6]

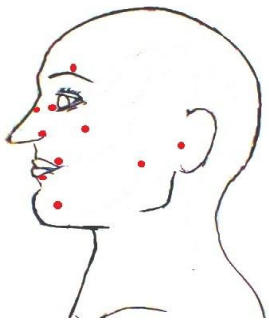

Fig-1 Trigger areas

An important hall mark of trigeminal neuralgia is the presence of 'trigger areas' which are zones of exquisite sensitivity to cutaneous stimuli. Light touch and vibration are most provocative sensory stimuli. Trigger factors may be shaving, applying makeup, exposure to wind or intra oral pressure causing conditions such as brushing of the teeth, chewing food and drinking. The episodes of pain do not occur during sleep. The distribution is usually unilateral. There may be exacerbations during morning hours. To know the distribution of pain one should know the sensory pathway of trigeminal nerve. The distribution of pain in the trigeminal neuralgia follows the anatomical boundaries of trigeminal nerve. The second and third divisions of trigeminal nerve are most commonly involved.

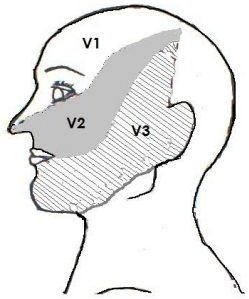

Fig-2 Trigeminal Nerve Branches distribution

Trigeminal Nerve is largest of all cranial nerves and it is mixed type of cranial nerve. It is attached to the ventral surface of the pons at its junction with the middle cerebellar peduncle by two roots-motor and sensory. Motor is small and lies medial to sensory root. The sensory root is connected with a trigeminal ganglion which contains the unipolar sensory neuron. Fibres of sensory root of trigeminal nerve on reaching the pons mostly bifurcate in to short ascending and long descending branches. The descending branches are thinly myelinated forms somatotopically arranged

tract and terminate in the spinal nucleus of trigeminal nerve. The ophthalmic fibres are most ventral and terminate in the caudal part of the nucleus. The maxillary fibres occupy intermediate position and terminate in the middle. Whereas the mandibular fibres are more dorsal and terminate in principal sensory nucleus, the ascending fibres are thickly myelinated and terminate in principal sensory nucleus. Second order neurons from spinal nucleus and principal nucleus cross the midline and end at ventero posteriomedial nucleus of thalamus. The peripheral process of ganglion cell form the ophthalmic (V_1), maxillary (V_2) and mandibular (V_3) divisions of trigeminal nerve and the central process form the sensory root. Cutaneous branches from these three divisions of trigeminal nerve are altogether eleven in number. Five from ophthalmic, three from maxillary and three from mandibular divisions. The ophthalmic zone includes tip and side of the nose, upper eye lid and fore head. This zone is supplied by lacrimal, supraorbital, infratrochlear and external nasal branches. The maxillary zone comprises upper lip, part of side of nose, lower eye lid, malar prominence and small portion of temple. This zone is supplied by three nerves- infra orbital, zygomatico facial and zygomatico temporal. The mandibular zone includes the lower lip, chin, skin overlying the mandible excluding its angle. Solitary involvement of first division is less than 5% of cases and was previously known as symptomatic trigeminal neuralgia [1,7] Sensory portion of trigeminal nerve convey pain, temperature and touch from the skin of face and scalp as far as the vertex, teeth, mucosa of gums, oral and nasal cavities with paranasal air sinuses, cornea and conjunctiva. The key feature of trigeminal neuralgia pain is very dynamic in nature which is difficult to explain in purely anatomical terms. [7] In 1829 Bell attributed the paroxysmal pain in trigeminal neuralgia to the dysfunction of trigeminal nerves after defining the specific functions of trigeminal and facial nerves. [8,9]

Dandy believed that the compression or distortion of trigeminal nerve by an artery while performing surgeries in and around posterior fossa was responsible for trigeminal neuralgia. [10]

Pathological specimens or samples, form the leading hypothesis of pathogenesis in classical trigeminal neuralgia. Zones of demyelination in and around the area of vascular compression bordering root entry have been shown. The aspect of vulnerability of the thinly myelinated A-Delta fibres has been high lightened. [11,12] Most common trigeminal neuralgia is focal compression of the trigeminal nerve root .So the probable cause of trigeminal neuralgia is either displacement of trigeminal nerve by the compression of artery or demyelination leading to ectopic generation of spontaneous nerve impulses. Demyelination of nerve is due to multiple sclerosis or deficiency of myelination forming proteins result of autoimmune disease. This explains the paroxysmal nature of the symptomatology of trigeminal neuralgia, while the extent of involvement as well as progression of symptoms may be attributed to the non-synaptic transmission of nerve impulse. [13]

CASE REPORT

This is a case of 49 year old female. She started experiencing right sided sudden intense electric shock like facial pain. The pain was primarily located in the right cheek and jaw region and is aggravated by chewing, laughing and other facial movements. In a matter of few weeks symptoms were severe enough to effect normal daily activities. There were exacerbations of pain in morning hours. On clinical examination she experienced allodynia in V2-V3 region i.e. maxillary and mandibular divisions of trigeminal nerve.

She has not experienced pain in night times. She had been evaluated by dentist and even after repeated visits also pain was not relieved. Then she proceeded for neurological examination and was diagnosed with trigeminal neuralgia. She was sent for MRI scan by neurosurgeon.MRI scan revealed no evidence of tumour or other pathological changes that causes compression of nerve. Finally this case was diagnosed as classical trigeminal neuralgia. She had been started with carbamazepine 200 mgs in divided doses.

DISCUSSION

Trigeminal neuralgia is the term implies to pain in the area of distribution of trigeminal nerve or nerve root. The characteristic pain of the Trigeminal neuralgia includes paroxysmal, explosive, severe and electric shock like lancinating quality. The feature should be highly distinctive in contrast to atypical facial pain, a study aching or throbbing pain frequently described as pressure and sensation of swelling of the face. To identify the nerve of neuralgia, it is best to identify anatomical site of neuralgia. Additional history, thorough physical examination and imaging techniques like MRI should help the physicians to diagnose the underlying Cause.

The characteristic features of pain episodes that include brief, intense nature, stereotyped, trigger areas and a specific facial pain suggest the diagnosis of trigeminal neuralgia. Patients with dental ache or pulpitis usually have persistent throbbing pain with occasional sharp character localized to that area and may have heightened tooth sensitivity are not demonstrable in this case. Temperomandibular dislocation may be detected by dull aching pain at Temperomandibular joint and the presence of click sound elicited on wide opening of jaw. Neuropathic trigeminal affliction is associated with persistent aching or throbbing with focal deficits in the distribution of the nerve. The syndrome of atypical facial pain better referred to as persistent idiopathic facial pain comprises all patients who neither fulfill the diagnostic criteria of classical trigeminal neuralgia nor have a secondary cause for the ailment. The occurrence of pain in this syndrome is bilateral and beyond the distribution of trigeminal nerve without cutaneous trigger area. It is usually associated with filbromyalgia and history of depression. Trigeminal neuralgia can be differentiated from the Trigeminal autonomic cephalgia (TAC), especially short unilateral neuralgiform pain with autonomic symptoms and short unilateral neuralgiform pain with conjunctival tearing by the absence of autonomic symptoms. It is important to note that short lasting pain syndrome commonly involve peri orbital areas and not shown the phenomenon of refractory period. MRI can detect if a tumor or multiple sclerosis is irritating the trigeminal nerve. However, unless a tumour or multiple sclerosis is the cause for trigeminal neuralgia, imaging of brain is usually normal. The vessel touching the nerve root is difficult to see even on high resolution MRI. Trigeminal neuralgia is usually diagnosed by symptoms. This case is characterized by severe, brief shock like pain with distribution of pain along the V2, V3 divisions and aggravated by chewing and laughing which act as trigger factors, mostly in favour of trigeminal neuralgia. Her clinical examination and imaging studies are also in favour of trigeminal neuralgia

CONCLUSION

Trigeminal neuralgia is difficult to diagnose and treat. This condition is distinguished by short bursts of lancinating unilateral pain in one or more divisions of

the trigeminal nerve. Most common cause of trigeminal neuralgia is focal compression of nerve root by the artery. Comprehensive clinical history and physical examination are imperative in assisting in the differential diagnosis.

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