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SevenHills
Health Byte

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34 Years OF EXCELLENCE IN HEALTH CARE

From Editor's Desk

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It gives me immense pleasure to bring you the July 2022 issue of SevenHills Health Byte.

July 15th is celebrated as Plastic surgery day. To commemorate the day we are covering in this issue a rare case and management of Brachial Plexus injury from Plastic surgery department. The other topic in this issue is Tic Douloureux commonly known as Trigeminal Neuralgia from Neurosurgery department.

Enjoy your read on two nerve related issues from two different departments with advanced management.

SevenHills Hospital has treated all its patients' par beyond expectations with high skilled surgeons who are upgraded in recent advances in medical science.

BRACHIAL PLEXUS INJURY – FROM DESPAIR TO HOPE!!

Brachial plexus injury is the injury of nerve roots from C5 to T1 that provide motor and sensory innervation to the upper limb (Figure 1). Depending on the site of injury it may be classified as:

1. Upper plexus palsy (C5,C6) – Loss of shoulder abduction and elbow flexion.
2. Extended upper plexus (C5,C6,C7) – Above deficit with loss of elbow and wrist extension.
3. Global plexus (C5,C6,C7,C8,T1) - Complete loss of the upper limb function.
4. Lower plexus (C8,T1) – Loss of hand function.

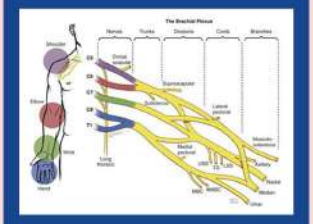

Figure 1

INCIDENCE

Almost 90% of these traumatic adult plexus injuries occur due to 2 wheeler accidents, when the shoulder hits the ground at high speed, causing deceleration and hence avulsion of the nerve roots from the spinal cord at various levels. Unfortunately these debilitating injuries commonly affect young adults in their most productive age from 16 -40 years, predominantly male. Hence it is imperative to detect and treat these injuries early on for best results.

TREATMENT

Figure 2

The treatment plan usually includes conservative management for about 2-3 months with physiotherapy which will allow the nerves affected with only neuropraxia to recover. If there is no recovery seen after this period then surgical exploration of the plexus is indicated. The best results for these injuries can be attained by Neurotisation which means transfer of functional intraplexal or extraplexal nerves to the affected nerves. After coaptation the nerve has to regenerate (at rate of 1mm /day), travel across the entire length of the recipient nerve and reach the neuromuscular junction within 18 months, after which time these junctions start to degenerate irreversibly. Hence the best results are obtained when the surgery is done within 3-6 months of the injury which is the golden period. Postoperatively the patient needs to be put on intensive physiotherapy in order to keep the neuromuscular junctions active. The first sign of success is seen usually after 3- 6 months depending on the site of nerve anastomoses which is just a flicker of movement!! Because of the prolonged nature of this condition and the need for patience during treatment, it is extremely important that the patient and his relatives are counselled about the same.

CASE

Here we present the case of a 22 year old right hand dominant male, who presented to us after 3 months of injury with Upper plexus palsy (C5,C6) leading to loss of shoulder and elbow function but with good hand function (Figure 2). A thorough clinical examination was done to assess the working muscles of the limb and plan the donor nerves. This was corroborated with an MRI of the brachial plexus and EMG/NCV study and neurotisation was planned.

The patient had good hand (intraplexal nerves), triceps (intraplexal nerve) and trapezius (extraplexal nerve) function. Hence we planned a Quadruple nerve transfer on him which included:

1) Branch of Spinal Accessory (nerve supplying the trapezius) was used to neurotise the Suprascapular nerve (supplying the Suprapinatus which initiates shoulder abduction).
2) Branch of Radial nerve (supplying the long head of triceps) was used to neurotise the Axillary nerve to restore Deltoid function (which continues the initiated shoulder abduction)
3) A fascicle each was taken from the Ulnar and Median nerves and used to coapt to branches arising from the Musculocutaneous nerve and supplying the Biceps and Brachialis muscles (which are instrumental in elbow flexion).(Figure 3)

All the nerve coaptations were done under microscope with 8-0 Ethilon. The choice of the donor fascicle was confirmed using the nerve stimulator, so that it did not lead to any critical defect in hand function.

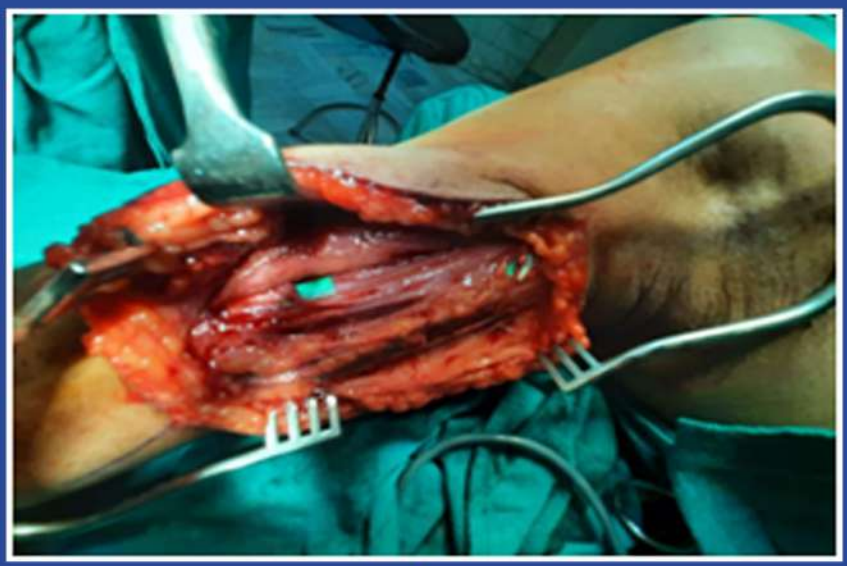

Figure 3

In the post operative period physiotherapy was continued along with targeted electrical stimulation of both donor and recipient muscles.

The patient achieved a favourable result at the end of 1 year (grade 3 / 5 restoration of function) and this will only improve over time. Timely intervention in this young patient helped give him this result so that he could go back to society productively. (Figure 4(a),4(b))

DISCUSSION

This patient had a favourable outcome because of early intervention. However very often the patient presents late, which happens due to lack of awareness about the problem among both, the patient and the treating doctors. Hence, the golden period is lost and this greatly hampers the recovery.

The best results can be achieved only with neurotisation but if the patient presents late, neurotisation cannot be done due to irreversible damage to the neuromuscular junctions. Such patients can then be offered only tendon transfers and free functioning muscle transfers, the results of which are much inferior to nerve surgeries. In our data itself, for each such patient who has benefited from early surgery with a good outcome, there are at least 3 patients who have presented later than a year, just hoping for spontaneous recovery and hence losing crucial time and the opportunity to have the best outcome. Hence, the need of the hour is to keep a very high index of suspicion for these injuries at the first or subsequent evaluation of a polytrauma patient. Once the diagnosis is made, early referral to a brachial plexus surgeon is critical so that treatment can be planned at the earliest.

Figure 4 (a,b)

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TIC DOULOUREUX – THE LANCINATING PAIN THAT COULD KILL

Trigeminal neuralgia (TN), also called tic douloureux, is a chronic pain condition that affects the trigeminal or 5th cranial nerve, one of the most widely distributed nerves in the head. TN is a form of neuropathic pain (pain associated with nerve injury or nerve lesion.) The incidence of new cases is approximately 12 per 100,000 people per year; the disorder is more common in women than in men.

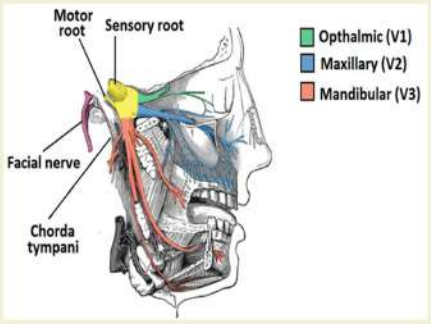
The typical or "classic" form of the disorder (called "Type 1" or TN1) causes extreme, sporadic, sudden burning or shock-like facial pain that lasts anywhere from a few seconds to as long as two minutes per episode. These attacks can occur in quick succession, in volleys lasting as long as two hours.

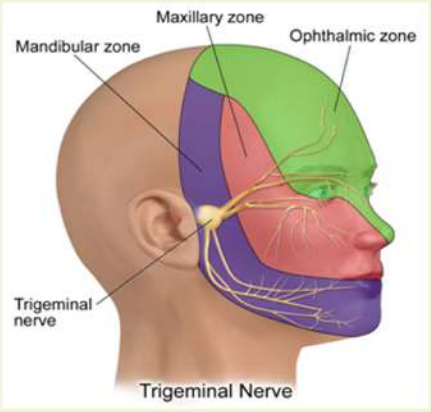
The "atypical" form of the disorder (called "Type 2" or TN2), is characterized by constant aching, burning, stabbing pain of somewhat lower intensity than Type 1. The intensity of pain can be physically and mentally incapacitating in some cases even forcing the patient to commit suicide.

Thanks to the better understanding of this condition, in the present day it can be diagnosed at an early stage and complete cure can be achieved

BRIEF ANATOMY OF TRIGEMINAL NERVE

The 5th cranial nerve nerve has three branches that conduct sensations from the upper, middle, and lower portions of the face, as well as the oral cavity, to the brain. The ophthalmic, or upper, branch supplies sensation to most of the scalp, forehead, and front of the head. The maxillary, or middle, branch stimulates the cheek, upper jaw, top lip, teeth and gums, and to the side of the nose. The mandibular, or lower, branch supplies nerves to the lower jaw, teeth and gums, and bottom lip. More than one nerve branch can be affected by the disorder.





PRESENTATION OF TRIGEMINAL NEURALGIA

Pain varies, depending on the type of TN, and may range from sudden, severe, and stabbing to a more constant, aching, burning sensation. The intense flashes of pain can be triggered by vibration or contact with the cheek (such as when shaving, washing the face, or applying makeup), brushing teeth, eating, drinking, talking, or being exposed to the wind. The pain may affect a small area of the face or may spread.

The attacks often worsen over time, with fewer and shorter pain-free periods before they recur. Eventually, the pain-free intervals disappear and medication to control the pain becomes less effective. Due to the intensity of the pain, some individuals may avoid daily activities or social contacts because they fear an impending attack.

Many times it is misdiagnosed as a dental pain and many patients end up with tooth extraction before the correct diagnosis is made.

WHAT CAUSES TGN

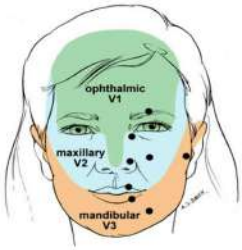
The commonest cause of idiopathic TGN is found to be due to an artery or vein compressing on the trigeminal nerve causing focal demyelination over time that results in short circuiting of the nerve and manifesting as the lancinating pain. The commonest artery found to be compressing the nerve is the superior cerebellar artery.

The other causes of TGN could be a tumour like epidermoid/schwanomma or demyelinating disease in the level of the 5th nerve nucleus in the brainstem as in multiple sclerosis.

HOW IS THE DIAGNOSIS MADE?

TN diagnosis is based primarily on the person's history and description of symptoms, along with results from physical and neurological examinations.

Usually there is no significant sensory or motor deficits with respect to the trigeminal nerve. However pain may be triggered by certain trigger points in the face.



TRIGGER POINTS

Other disorders that cause facial pain should be ruled out before TN is diagnosed. Some disorders that cause facial pain include post-herpetic neuralgia (nerve pain following an outbreak of shingles), cluster headaches, and temporomandibular joint disorder (TMJ, which causes pain and dysfunction in the jaw joint and muscles that control jaw movement).

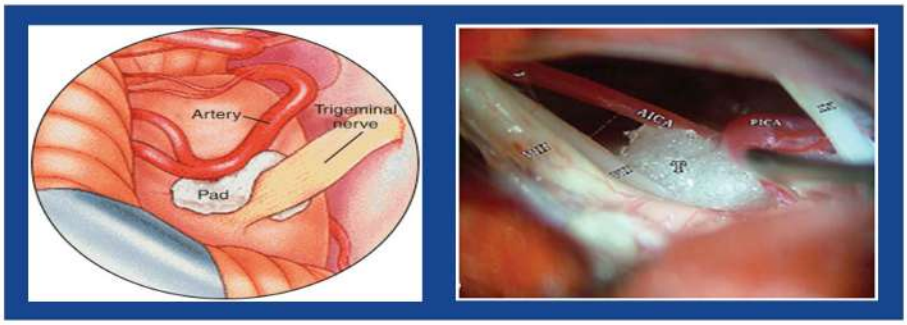
MRI brain with special sequence like 3D CISS or 3D FIESTA is mandatory to rule out tumour or other pathology and identifying the vascular loop compressing the nerve

Figure (a)

Figure (b)

Fig (a) - MRI Showing a vascular loop compressing the 5th nerve.

Fig (b) - Intraoperativepicture showing the arterial loop compressing the nerve.



TREATMENT FOR TGN

Medications : Anticonvulsants can be used to block nerve firing and are effective in treating TN1 but often less effective in TN2. These drugs include carbamazepine (commonest drug used upto 1200mg/day), oxcarbazepine, topiramate, gabapentin, pregabalin, clonazepam, phenytoin, lamotrigine, and valproic acid. This is the first line of treatment Tricyclic antidepressants such as amitriptyline or nortriptyline can be used to treat pain. Common analgesics and opioids are not usually helpful in treating the sharp, recurring pain caused by TN1, although some individuals with TN2 do respond to opioids.

Eventually, if medication fails to relieve pain or produces intolerable side effects such as cognitive disturbances, memory loss, excess fatigue, bone marrow suppression, or allergy, then surgical treatment may be indicated. Since TN is a progressive disorder that often becomes resistant to medication over time, individuals often seek surgical treatment.

Surgery : Several neurosurgical procedures are available to treat TN, depending on the nature of the pain; the individual's preference, physical health, blood pressure, and previous surgeries; presence of multiple sclerosis, and the distribution of trigeminal nerve involvement (particularly when the upper/ophthalmic branch is involved). Some procedures are done on an outpatient basis, while others may involve a more complex operation that is performed under general anesthesia. Some degree of facial numbness is expected after many of these procedures – depending on whether it is a destructive or non destructive procedure, and TN will often return even if the procedure is initially successful.

MICROVASCULAR DECOMPRESSION (MVD)

This is the most invasive of all surgeries for TN, but also offers the lowest recurrence rate. About 25 percent of individuals undergoing MVD for TN ma experience recurrent pain within 12 to 15 years. This inpatient procedure, which is performed under general anesthesia, requires that a small opening be made through the mastoid bone behind the ear. While viewing the trigeminal nerve through a microscope or endoscope, the vessel (artery or vein) that is compressing the nerve is separated off and a soft cushion like Teflon sponge is placed between the nerve and the vessel. Unlike rhizotomies, there is no numbness in the face after this surgery. Pain relief occurs almost immediately after surgery in most patients.

A selective posterior rhizotomy (also called partial nerve section), which involves cutting part of the nerve, may be performed near the entrance point of the nerve at the brain stem during an attempted microvascular decompression if no vessel is found to be pressing on the trigeminal nerve. A rhizotomy will cause more long-lasting numbness in the area of the face that is supplied by the nerve or nerve branch that is cut. However, when the operation is performed in the face, the nerve may grow back and in time sensation may return. With neurectomy, there is risk of creating anesthesia dolorosa.

Surgical treatment for TN2 is usually more problematic than for TN1, particularly where vascular compression is not detected in brain imaging prior to a proposed procedure. Many neurosurgeons advise against the use of MVD or rhizotomy in individuals for whom TN2 symptoms predominate over TN1, unless vascular compression has been confirmed. MVD for TN2 is also less successful than for TN1.

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