Management Options in Occipital Neuralgia: A Review

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Abstract

International Headache society has provided a comprehensive classification of the various headache syndromes under the International Classification of Headache disorders-3 β (ICHD-3) published in 2013. Occipital neuralgia (ON) although less common compared with the common migraine, represents a significant disease burden in terms of the severity of pain. Multiple treatment options exist for the treatment of ON with highly variable outcomes. This review emphasizes on the various treatment options and their efficacy in the treatment of ON.

Keywords

- greater occipital nerve
- lesser occipital nerve
- nerve stimulator
- radiofrequency ablation
- neurectomy
- phenol neurolysis

Introduction

Two Spanish doctors José Benito Lentijo and Mateo Martínez Ramos should be credited for the description of occipital neuralgia in the early 19th century in 1821.[1] A study in the Dutch general population reported a relatively low incidence of 3.2 per 100,000.[2] The occipital neuralgia (ON) is defined as: unilateral or bilateral paroxysmal, shooting or stabbing pain in the posterior part of the scalp, in the distribution(s) of the greater, lesser and/or third occipital nerves, sometimes accompanied by diminished sensation or dysaesthesia in the affected area and commonly associated with tenderness over the involved nerve. In majority of the cases, the cause for ON is not discernable and hence termed idiopathic. Uncommon but notable causes include; vascular loop of posterior inferior cerebellar artery, fenestrated vertebral artery causing irritation of C1 and C2 roots, bony hypertrophy, degenerative changes of the Atlantoaxial joints, ligamental sclerosis or tumors of the cervical vertebrae even Schwannomas of the occipital nerves is reported to cause ON.[3-6]

Anatomy of the Occipital Nerves

The possible pain generators in ON are:

1) Greater Occipital Nerve (GON)–Accounts for ~90% of cases
2) Lesser Occipital nerve (LON)
3) Third Occipital nerve (TON)

The GON is the largest pure sensory nerve in the body arising from the medial branch of the dorsal ramus of the C2 and sometimes with contributions from C3 root. The GON innervates the medial portion of the posterior scalp as far anterior as the vertex. It exits between the C1 and C2 archs looping around the inferior oblique muscle. The nerve first courses between the inferior oblique, Rectus Capitis Posterior Major and the semispinalis capitis (SSC) muscles for variable distance to emerge out piercing through the SSC and lie between the SSC and Trapezius muscles. Following a short course between these muscles, the GON pierces the trapezius and finally the fasciotendinous attachments of Trapezius and becomes subcutaneous at the level of superior nuchal line. At this point, the nerve lies just medial to the occipital artery. The LON arises

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from the ventral ramii of C2 and C3. It is responsible for providing sensory innervation to the superior ear, post-auricular skin, and skin of the lateral neck.[7-9] The TON is the continuation of the dorsal ramus of C3 root. The third occipital nerve provides sensory innervation to the medial posterior scalp and neck. There exists a rich interconnection of nerves between the dorsal ramii of 1st, 2nd, 3rd and rarely 4th cervical spinal nerves known as the Cruveilhier plexus.[10]

Applied Anatomy

Technically the GON is prone to be compressed at 4 potential sites;[11]

1. Piercing of the tendinous attachment of the Trapezius muscle where the nerve becomes subcutaneous.
2. Piercing point into the Trapezius muscle.
3. Piercing the Semispinalis capitis (SSC).
4. Just after its exit between C1 posterior arch and C2 lamina at the inferior border of Obliqus capitis inferior muscle.

The point number 1 is most often implicated in producing the ON and also the site for diagnostic Occipital nerve block. Few researchers also mention the compression that may occur due to occipital arterial crossing of the GON as the 5th potential compression site (►**Fig. 1**).

This point is localized using various methods based on the surface landmarks:

a) corresponds to a point 1.5 cm lateral and 2–2.5 cm inferior to the EOP.
b) A point 1/3rd the distance from External Occipital Protuberance to mastoid line.
c) By palpating the Occipital artery which lies just lateral to the GON on the superior nuchal line at the level of EOP

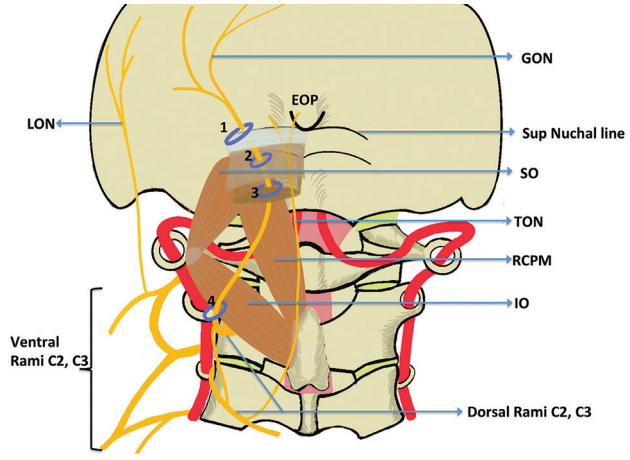

Fig. 1 Artistic representation of Posterior view of Occipitocervical junction and Anatomy of the occipital nerves: The blue rings (**1**) at the piercing of the fasciotendinous attachment of trapezius, (**2**) at the piercing of trapezius muscle, (**3**) at the piercing of Semispinalis capitis, (**4**) at the looping around the Inferior oblique muscle. GON, greater occipital nerve; LON, lesser occipital nerve; TON, third occipital nerve; EOP, external occipital protuberence; SO, superior oblique; IO, inferior oblique; RCPM, rectus capitis posterior major.

Clinical Presentation

The occipital neuralgia is defined as: Unilateral or Bilateral paroxysmal, shooting or stabbing pain in the posterior part of the scalp, in the distribution(s) of the greater, lesser and/or third occipital nerves, sometimes accompanied by diminished sensation or dysaesthesia in the affected area and commonly associated with tenderness over the involved nerves.[12]

The International Classification of Headache Disorders 3rd Edition (ICHD-3) Diagnostic Criteria for ON

A. Unilateral or bilateral pain in the distribution(s) of the greater, lesser and/or third occipital nerves and fulfilling criteria B-D
B. Pain has at least two of the following three characteristics:

1. Recurring in paroxysmal attacks lasting from a few seconds to minutes.
2. Severe in intensity.
3. Shooting, stabbing or sharp in quality.

C. Pain is associated with both of the following:

1. Dysaesthesia and/or allodynia apparent during innocuous stimulation of the scalp and/or hair.
2. Either or both of the following:

a) Tenderness over the affected nerve branches.
b) Trigger points at the emergence of the greater occipital nerve or in the distribution of C2.

D. Pain is eased temporarily by local anesthetic block of the affected nerve(s).

Tinels sign: Electric shock like sensations on tapping over the course of the nerve may be positive.

Pillow sign: Pain may be elicited with the head on the pillow with neck hyperextension or rotation.

Trigger points: Pressure on the exit point of the GON (located at 1/3rd distance from occipital protruberence to mastoid) may lead to reproduction of headache that may propogate upto the vertex.[13]

Diagnosis

The ON is most often primary and must be differentiated from the following related headache syndromes which can be broadly divided into:[3-6]

I. Organic Causes with serious implications: Posterior fossa, the Craniovertebral Junction (CVJ) and Cervical spinal abnormalities like:

1. Tumors (Schwannomas/Meningiomas), Neurinoma of the GON
2. Vascular lesions like aberrant branch of posterior inferior cerebellar artery (PICA), aberrant vertebral artery (VA) course impinging on C2 root, dural arteriovenous fistula (DAVF) of the high cervical spine, cervicomedullary

cavernomas, giant cell arteritis involving occipital artery (OA)
3. CVJ anomalies like Chiari malformation, Basilar Invagination (BI), Platybasia, etc.
4. Infections like CVJ tuberculosis and other inflammatory conditions.
5. Degenerative disease involving C1/2 joints, Rheumatoid arthritis.
6. Post traumatic C1/2 degeneration, Callous formation.
7. Iatrogenic neuroma formation following posterior fossa surgery, especially the Retrosigmoid approach for cerebellopontine (CP) angle pathologies.

II. Benign causes: Migraine, Cluster headaches, Tension type headaches and Cervicogenic headaches.

Clinical History and Physical examination

A thorough clinical evaluation will point toward the diagnosis in majority of the cases. The characteristic neuropathic pain (shooting/stabbing type) in the distribution GON/LON/TON provides lead toward the diagnosis of ON. However, migraine headaches, in isolation or may be associated with ON, need to be differentiated from ON as it is among the most common type of headaches. Hence, ruling out other common types of headaches due both serious and benign, prior labeling it as ON, is of paramount importance.[3-6,12]

Diagnostic Block

The point where the GON becomes subcutaneous by piercing the tendenous attachment of Trapezius muscle, at the level of superior nuchal line and lies ~1.5 cm off midline and 2–2.5 cm below the external occipital protruberence (EOP). The headache alleviates nearly all the patients. The cervicogenic and migraine headaches also respond to the block in more than 50% of cases.[13,14]

Imaging

Plain X-rays: Dynamic CVJ X-rays in cases of Atlanto axial dislocations (AAD).
CT CVJ/ CT Angiography: To rule out bony abnormalities like AAD, BI, Platybasia, Osteomas and callous formation of the posterior C1/2 arches.
MRI brain and Cervical Spine/ MR Angiography: In suspected cases posterior fossa lesions, Chiari malformations, Vascular anomalies and lesions.

Management

The management options for ON can be broadly divided into: Non Invasive and Invasive methods:

Non–Invasive

Pharmacological Therapy[14]

1. Tricyclic antidepressants: Amitryptyline, Nortryptyline.
2. Anticonvulsants: Carbamazepine, Gabapentin, Pregabalin, Baclofen, Phenytoin.
3. Opioid medications and nonsteroidal anti-inflammatory drugs

Invasive

Invasive therapy is considered when all the conservative measures have failed to relieve the pain. The following options can be tried in cases of ON refractory to medications and other non-invasive methods.

Local Anesthetic Block

This is the first invasive modality to be offered in medically refractory cases. Occipital nerve blockade (ONB) is used both for diagnostic and therapeutic purposes. Pain relief following ONB is one of the diagnostic criteria of ON as per the ICHD-3. Local anesthetics especially; Lignocaine (1%, 2% and 5%)/ Bupivacaine (0.25–0.5%), either in isolation or with Steroid (methyl prednisolone/Betamethasone/Triamcinolone) combinations along with clonidine/epinephrine have been tried in the literature. The pain relief although reported temporary in 80–90% of cases persisting for up to 1–2 weeks. The pain relief may be extended in 15–36% patients for up to 2 months. The most common side effect with Lignocaine injection was dizziness. Cushingoid features were observed in prolonged usage of steroid combinations.[13,15]

Techniques

Position: This procedure is performed either in sitting or prone position with head flexed.

Free hand technique using surface landmarks

Multiple injection points have been suggested in the literature. Based upon the course of the GON, injection 2 cm away from the EOP along the Superior nuchal line seems more logical (Fig-). The GON is often prone to compression at this point, as the nerve pierces the tendinous attachment of Trapezius and Semispinalis capitis muscles to become subcutaneous. The opening here is often tight and results in ON from constriction. Other landmarks tried are; 1/3rd distance from EOP on the OP mastoid line, ½ distance from EOP on the OP mastoid line and 2 cm lateral and below the EOP.[13,15,16]

Ultrasound guided ONB: The accurate localization of the GON, to account for the variation in the anatomical course, is the key in achieving excellent results. Ultrasonography with high-resolution machines currently available aids in precise localization and thereby provides a real time location of the nerves. The Doppler imaging of the occipital artery, which accompanies the GON, is always found just lateral to the nerve and provides a confirmatory clue. Comparing free hand with USG guidance.[17-19]

CT Fluoroscopy Guided Selective C2 C3 Root Blockade

This is another accurate method for obtaining ONB practiced in all the cases of refractory ONB prior to performing Cervical dorsal rhizotomy (CDR)[20]

Botox–An Injection

Botulinum Toxin A (BOTA) has been therapeutically used for treatment of spasticity, dystonias, chronic migraine, blepherospasm, hemifacial spasms. It has been extensively used to treat Trigeminal neuralgia, postherpetic/post-traumatic neuralgias and diabetic neuropathy. The anti-nociceptive action of BOTA is attributed to the inhibition of release of substance P and Calcitonin Gene-Related Peptide (CGRP) at the nerve endings. Both these proteins are responsible for producing pain

There have been 2 series enrolling 6 patients each suffering from Occipital neuralgia. In both the studies 5 units of BOTA was used for pain relief. One study reported sustained pain relief in 5 out of 6 patients for a period of four months. The other study noted improvement in the pain for few weeks but none reported complete cessation of pain.[20-22]

Chemical Neurectomy

The role of causing neurolysis using ethyl alcohol (95%), phenol and glecyrol has been published in volumes in the literature. Studies on chemical neurectomy involving the nerves of the scalp are lacking. Kulkarni performed bilateral greater occipital neurolysis using 1ml of 8% phenol for a case of refractory ON. The patient remained pain free at 1year of follow up, weaned off medications.[23] Similarly, Kawale et al, reported excellent result in a case of adiposis dolorosa, with bilateral GON neuralgia at 9 months of follow up. The cause of the neuralgia could have been the compression of the GON by the adipose tissue extending in bilateral parieto-occipital region. They used 1ml of 95% ethyl alcohol for achieving neurolysis of the bilateral GON. The authors reported sensory loss in the distribution of the nerve and patches of alopecia at the injection site.[24]

Larger studies in the future to look into the safety and efficacy of the procedure are awaited, as it might offer a minimally invasive method. It can also be performed as an office procedure, more acceptable to patients, in resource limited settings offering a cheaper alternative to the patients.

Cryoneurolysis

The use of cold temperature for inducing tissue damage, also known as cryotherapy, is a well-known treatment modality. Its application for the treatment of headaches is limited by sparse number of studies in the literature. The mechanism of nerve injury in cryotherapy is through the formation of microcrystals in the vasa nervosum of the nerves leading to endoneural edema without damaging the endoneurium. This progresses to wallerian degeneration and nerve damage.[25]

A double blinded randomized controlled trial conducted by Kwarstein et al, to evaluate the efficacy of cryoablation in 52 patients of cervicogenic headache. They randomized the patients into 2 groups: One group (n = 31) received Cryoablation and the other group (n = 21) received a combination of steroid and local anesthetic. Although the authors found that Cryoneurolysis provided temporary pain relief but was not superior to the steroid/ local anesthetic group. The following limitations might be accounted for the equivocal results obtained; the lesioning was preformed using surface landmarks and stimulation results, the sample size was small and also the patient population was heterogenous like; majority of the patients suffered accompanying migraine headaches.[26]

Kim and collegues, collected a data of 38 patients, retrospectively treated for refractory ON with cryoablation. They reported > 50% pain relief in 68% of patients sustained for a period of 6 months. There were no major adverse events reported.[27]

Kastler et al reported CT guided cryoablation in 6 patients diagnosed with ON. They noted 5 of the 6 patients to have encouraging results in terms of pain freedom.[25]

Surgical Neurolysis of GON

Few studies have documented excellent outcomes following the neurolysis of the GON, as this is frequently responsible for the ON in majority of the subjects. The GON has 5 potential sited of compression as depicted in the (►**Fig. 1**). This procedure retains the advantages of preserving the GON and releasing the nerve free from any external compression. More than 50% pain relief was noted in 80–90% patient population with ON undergoing neurolysis. Ducic et al, in a retrospective study involving 206 patients with ON, documented pain relief of more than 50% in 80% patients for a period of 12 months. Gille and collegues performed the sectioning of the inferior oblique muscle along with neurolysis of the GON routinely and achieved excellent results in 7 out of ten patients.[28-30]

Dorsal Rhizotomy

Sindou et al advocated the concept of partial rhizotomy. The logical basis for performing partial rhizotomy was based upon the landmark study by Sindou and collegues. In this study the demonstrated that the nociceptive small fibers are laminated ventro laterally while the large fibers carrying the cutaneous sensations and proprioception were located dorso-medially. Hence, this forms the rationale for performing partial rhizotomy.

Both Partial and Complete cervical dorsal rhizotomies (CDR) have been tried in refractory cases of ON. The advantages of performing partial over complete rhizotomy are:

1. Preservation of cutaneous and proprioceptive sensations.
2. Avoidance of vertigo and imbalance as complete severance of the dorsal roots leads to loss of joint and muscle proprioceptive sensations.

However, the pain relief obtained is similar in both the procedures 65–70% complete pain relief.[31-33]

Neurectomies and C2 Ganglionectomy

Peripheral neurectomy of the GON, LON, and even the TON is a simple procedure that can be performed under local anesthesia. Depending upon the symptoms, the distribution of the pain, presence of the tender points/dysasthesias and atleast greater than or equal to 50% response, the neurectomies are performed. Although, the localization of the nerves can made on the anatomical landmarks (Fig-), precise

localization under ultrasound guidance helps easy and early identification of the nerves with minimum tissue dissection. Sharma and collegues, demonstrated excellent pain relief in 90% patients at 6 weeks and 70% for prolonged duration.[34] Ducic et al, retrospectively followed up 71 patients who had undergone greater occipital neurectomies following failed neurolysis for refractory ON. They noted 70% patients experiencing pain relief, with 41% attaining excellent pain free outcome at 33 months follow up.[35]

The disadvantage of peripheral neurectomy is recurrence of the pain over a period of time. The recurrent pain in these patients is worse than the primary pain. This is attributed to either axonal regeneration or neuroma formation. Hence, C2 dorsl root ganglion (DRG) excision is considered to be a more definitive solution to neurectomy, as the cell bodies residing in the C2 ganglion are excised completely, thereby annulling the scope of regeneration. The DRG excision is theoretically superior to dorsal rhizotomies, as it is said that few afferent fibers travel through the ventral roots and escape damage in dorsal rhizotomies.[36] Acar et al, retrospectively analyzed 20 patients wi th ON undergoing C2/C3 ganglionectomy (in isolation or both). They reported 95% pain relief within 3 months follow up, which dropped to 35% at 1 year i.e, 65% patients developed recurrence of pain, requiring adjacent level ganglionectomy. Five patients failed to respond favorably to second ganglionectomy. Overall at long term follow up 60% reported more than 50% pain relief.[37]

Radiofrequency Therapy

The application radiofrequency (RF) for the treatment of neuralgic pain has been well studied and documented in the literature, especially for the treatment of Trigeminal neuralgia. The research available in the literature, on the treatment of ON using RF is scant. The RF therapy has been tried in two ways:

i. Pulsed Radiofrequency (PRF) and
ii. Radiofrequency thermal ablation (RFA).

The major difference between both these procedures is the reversibility. The intent in PRF is to avoid a permanent damage, while in RFA permanent lesioning of the nerve is intended. The RF lesion parameters used between both the techniques is different. In RFA higher temperature (80°C) is the commonly used for a prolonged and sustained duration. The lesion diameter is usually 1.5 times the diameter of the tip of the RF probe. A temperature of 60–65°C is necessary to cause thermal necrosis in the surrounding tissue. The tip of the RF probe should attain higher temperature (at least 80°C) to achieve the necessary surrounding tissue ablating temperature. In contrast, PRF neurotomy uses a lesser temperature (up to 45°C) in multiple cycles, so as to cause microscopic disruption, without causing permanent damage or loss of function of the nerves.

Pulsed Radiofrequency Therapy

The PRF is a form of neuromodulation, where the nerve function is modulated without disruption. This is achieved by delivering short bursts of radiofrequency pulses, with the help of a radiofrequency probe, are delivered to the culprit nerve. These pulses are intervelled with long pauses, thereby allowing the heat to dissipate in the neural tissues. Hence the nerve suffers microscopic damage at the intracellular level (disrupting at the level of mitochondria, microtubule and microfilaments), thereby altering the function in terms of pain relief. Erdine and collegues first demonstrated this kind of disruption histologically, which was more pronounced in the small Type C nociceptive fibers.[38]

Huang et al, conducted a multicenter study, involving 102 patients of ON treated with PRF.

They used the following parameters: voltage output: 40–60 V; 2 Hz frequency; 20 milliseconds pulses in a 1-second cycle, 120-second duration per cycle; impedance range between 150 and 500 W; and 42°C plateau temperature. The authors noted more than or equal to 50% pain relief in 51% of patients at 3 months follow up.[39]

Similarly, Vanelderen obtained equivalent results in 19 patients of ON, prospectively treated with PRF. They reported slightly more than 50% patients reporting more than 50% pain relief. Here again the PRF parameters used were; 20 millisecond bursts with a frequency of 2 Hz and 45 V and was applied for 240 second per nerve. Care was taken not to exceed 42-C.[40]

Cohen et al, in a randomized trial compared PRF with steroid injection in ON and found PRF to be superior in producing pain relief. The efficacy of PRF was to the tune of 60% at 6 weeks compared with 36% for steroid injection alone. This effect although weaned off to nearly half at 3 and 6 months for both the treatment modalities. However, the PRF remained superior to the steroid injection even at extended follow up.[41] The following PRF parameters were used: voltage output 40 to 60 V; 2 Hz frequency; 20 milliseconds pulses in a 1-second cycle, 120 second duration per cycle; impedance range between 150 and 400 Ω; and 42°C plateau temperature. Most of the studies have utilized more or less the same parameters for performing PRF.[42]

Radiofrequency Ablation

The RFA induces permanent lesioning in the nerves and is therefore irreversible compared with PRF, owing to the application of RF thermal energy for prolonged time duration. Hoffmann et al, reported RFA of the GON and LON in 46 patients suffering from ON. The authors followed the RF parameters as: 80°C for 180 seconds. They recorded 70–80% pain relief in 64% of patients and at least 50% pain relief in upto 84% of the patients studied for a period of 6 months to 1 year.[43] Abd-Elsayed et al, demonstrated superior results for longer duration in patients undergoing RFA for pericranial neuralgias. Majority of these patients were treated for headaches due to GON/LON involvement.[44] Govind et al, have also utilized RFA for treating TON neuralgic headache. The authors performed RFA of TON under fluoroscopic guidance in 49 patients. Fourty three (88%) patients became pain free for a mean duration of 10 months follow up, with 14 patients requiring repeat RFA, owing to the reappearance of pain. Twelve of them attaining freedom from pain in the follow up after 9 months. The adverse effects were limited to

temporary dysasthesias and ataxia, numbness. The parameters for RFA used were 80°centigrade for 90 seconds.[45]

RFA has also been used to ablate the C2 DRG with many studies showing superior results. In a retrospective study, Hamer et al, reported RFA in 40 patients suffering from cervicogenic headache/ON. They reported nearly 90% patients experiencing more than 80% pain relief for an average duration of 6 months. The parameters for RFA used were 80°C for 90 seconds.[46]

Neuromodulation

Pulsed Radiofrequency (PRF) Application

This therapy already detailed wide supra is a form of neuromodulation therapy, where short bursts of radiofrequency pulses are delivered to the culprit nerve. The nerves are not destroyed rather their microstructure undergoes functional modification, thereby achieving control in the neuralgic pain.[39-42]

Occipital Nerve Stimulation (ONS)

Peripheral nerve stimulation for the treatment of chronic headache syndromes (CHS) is extensively studied with numerous publications available in the literature. PNS for the treatment of various headache syndromes is not approved by FDA and hence it is an off label indication. Weiner and Reed popularized the concept of PNS for treating migraine CHS, especially Migraine.[47] They demonstrated successful pain free outcomes leading to extensive use of the technique subsequently. Migraine and cluster headache syndromes are among the common diseases, where ONS has been used. The device used for ONS is the same as the one used in spinal cord stimulation (SCS). Only few studies document the utility of ONS in treating Occipital neuralgia.

Device

The nerve stimulation device has two components:

1) Lead and
2) Internal pulse gene rator (IPG)/battery.

The leads consist of thin wires connected to multiple metallic contact points called the electrodes, come in two varieties; i) Percuteneous/Needle type and ii) the paddle type. The needle type leads are thin and cylindrical, which can be inserted percuteneously through a needle. Hence, it is minimally invasive both in terms on the incision and tissue damage, given the ease of its insertion. The Paddle type leads are broad and flat and needs large incision and creation of subcutaneous scalp pocket for its insertion. The major disadvantage of the percutaneous lead is its higher propensity to migrate compared with the paddle type lead. Recently, advanced bionics corporation, CA USA, has developed miniature leads known as the Bion Microstimulator. This is self-contained, rechargeable and implantable device without any additional internal hardware. The disadvantages are frequent recharging, development fibrosis around the device, owing to its small size, making it ineffective in delivering the desired stimulus to the target nerves.[48]

All the cases should undergo a mandatory ONB to look for the responsiveness of the pain and thereby predicting the final outcome. Once the decision to insert the ONS is made, a trial lead may be inserted prior to the implantation of permanent lead for a period of 5–7 days. This will ensure the success in terms of pain relief after the permanent lead placement on the part of the treating doctor. This will also aid the patients in experiencing the benefits and the adjustments that may be needed during the routine life following the permanent implantation. The patients can also be decisive on whether they are ready to undergo the ONS surgery. The decision to implant permanently is based on 50% or more pain relief following the trial implantation.[48-50]

Surgical Technique

The surgery is performed in two stages: The first stage i,e, placement of the lead, is performed under sedation and local anesthesia, while the second stage, i,e, battery placement, is performed under general anesthesia. The leads can be inserted either through a midline incision with the lead directed laterally or a retromastoid incision, with the lead directed medially and may be unilateral or bilateral depending upon the laterality of the pain. The most important aspect of placement is the leads are placed in the subcutaneous plane, by creating a subcutaneous pouch, at the level of C1 posterior arch. Once in place the leads are secured to the subcutaneous tissue with the anchors. The IPR/battery is then connected to the lead with the wire passed through the subcutaneous tunnel. The battery has be placed in infra-clavicular, mid axillar or abdomen, infrascapular and buttocks depending upon the body habitus of the patient.[48-51]

Complications

Implant Related

Lead migration accounts for majority of the adverse events, accounting for ~8–10% cases undergoing ONS for ON.

The other complications include hardware fracture, malposition and battery repositioning.

Patient related: Infection, wound dehiscence, skin erosion, seroma formation, pain over the battery site or discomforting pain at the lead site and very rarely severe burning pain leading to removal of the device.

Various studies have reported over 70% good response to ONS for patients suffering from ONS with pain relief up to ~80–90% for at least 6 months duration. Overall ONS is viable option, which is minimally invasive, in patients suffering from refractory ON.[48-50]

Conclusion

The management of Occipital neuralgias has been subjected to a series of refinements beginning from local blocks to neuromodulation currently. The ICHD-3 β criteria provides a definitive clue to the diagnosis. Occipital nerve block is the first step prior to undertaking any invasive surgical endeavor in all the cases of refractory ON. Reversible procedures like; pulsed radiofrequency and occipital nerve stimulation offer

a minimally invasive solution with excellent results without destroying the nerve. Subject to availability the use of image guidance during the surgery should always be considered, to improve the accuracy and subsequently the outcome. C2 ganglionectomy can be considered as a last resort when all the other modalities fail to provide relief.

Conflict of Interest
None declared.

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