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Case Report

Dextrose Prolotherapy for Occipital Neuralgia Management

Taufiq Agus Siswagama[1], Buyung Hartiyo Laksono[1], Mirza Koeshardiandi[2]
[1]Department of Anesthesiology and Intensive Therapy, Faculty of Medicine,Brawijaya University, Dr. Saiful Anwar General Hospital, Malang, Indonesia
[2]Anesthesiology Department, Dr. Soedono Hospital, Madiun, Indonesia

Correspondence:
Taufiq Agus Siswagama,dr,SpAn,KMN*
Department of Anesthesiology and Intensive Therapy, Faculty of Medicine,Brawijaya University, Dr. Saiful Anwar General Hospital, Malang, Indonesia
e-mail: taufiqsiswagama@ub.ac.id

ABSTRACT

Background: Occipital neuralgia defined as a pain such as being stabbed in the skin according to the dermatomes of the greater occipital nerves (GON) and lesser occipital nerves (LON).
Case: An 80-year-old male patient diagnosed with occipital neuralgia. Previously, patients were diagnosed with lung cancer six months ago and planned for follow-up chemotherapy. Patient already receive medications including paracetamol, Non-steroidal anti-inflammatory drugs (NSAIDs), minor tranquilizers, and antidepressants, but the pain still exist. Patient then scheduled to receive blocks of GON and LON-ultrasound-guided using plain lidocaine 2% and steroids dexamethasone 10 mg. Fifteen days later, patient receive perineural deep injection along with prolo-hydrodissection in GON and LON using dextrose 15% and local anesthesia lidocaine plain 2% with a volume of 3 cc each nerve. The intervention give a positive outcomes, pain is reduced with NRS rest 0-1, NRS motion 2-3, hearing improves, and the noise in the ear disappears. The patient can sleep using a pillow.
Conclusion: Block GON and LON, perineural deep injection along with prolo-hydrodissection provides a positive outcome for occipital neuralgia pain management. This case showed an opportunity for pain specialist to develop pain intervention based on prolotherapy.

Keywords: occipital neuralgia, pain management, prolotherapy

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INTRODUCTION

Headaches contribute to nearly 20 million outpatient visits per year in the United States and become one of the most common complaints found by the primary health care provider. The prevalence of headaches is about 90% to 95%. The scalp, muscle in the head area, periosteum, dura, and blood vessels are sensitive to pain, except for the brain parenchyma, which makes there are many possible causes of headaches and facial pain.[1,2] Headaches become the most common complaint (42%) among the other neurological disorders. Data from the neuroprotective department of Dr Soetomo general hospital, Surabaya, in 2014-2015 found 1,580 (8%) of the total neurosurgery patients suffer from primary headaches.[3]

International Headache Society (IHS) defined occipital neuralgia, also known as neuralgia C2, as a pain such as being stabbed in the skin according to the dermatomes of the greater occipital nerves (GON) and lesser occipital nerves (LON). Pain originates from the suboccipital region, radiated to the vertex, especially the upper neck, behind the head, and the eyes. Pain may be accompanied by hypesthesia or dysesthesia in the affected area. A common trigger is GON or LON compression. Greater occipital nerves (GON) are more often involved (90%) than LON (10%), and 8.7% of headaches involved both of them.[4,5]

The United Kingdom's national pharmaceutical records suggest using oral, subcutaneous, intramuscular, and intravenous drugs to treat headaches. The development of intravenous drugs injection progresses rapidly, but the need for repeated administration becomes a problem since the patient will need longer hospitalization and will have to come to the ER to get therapy.[2] The use of ultrasound-guided offers an alternative treatment by following a landmark to performing injections and blocks of various nerves. Ultrasound-guide base treatment has been increasingly used in both operating rooms and pain clinics. Ultrasound can distinguish tissues and direct the needle at the targeted tissues within real-time visualization.[6]

The prevalence of chronic pain in the adult population in the US is 20.4%, and the epidemic of opioid-related deaths has created a national emergency. The authorities seek to encourage the use of new therapies to treat chronic pain,

including therapies that address the underlying causes. The main goal is to found non-opioid treatment methods and regimens for chronic pain. Prolotherapy is one of many non-opioid treatments.[7] In this paper, the author, will present a case of headache in occipital neuralgia that receives interventional pain management using ultrasound guidelines. In these patients, injection is performed on GON and third occipital nerve (TON) with two different therapy and drugs.

CASE

An 80-year-old male patient complained of pain in the left-back head. The pain was felt for almost four months. Pain is felt like being stabbed by a sharp object and sometimes feels very stinging. Pain radiates from the back of the head to the back of the ear and the top of the head. Pain is often felt and suddenly appears. The patient feels pain every time the upper neck and back of the head are touched or depressed. Pain is often triggered by touching or pressing the upper neck and back of the head. The pain gets worse if the patient lies down using a pillow at night. Patients also complain of hearing loss and impaired hearing. The pain disorder makes the patient suffer from insomnia and lack of energy. The patient had a consultation with psychiatry because he felt depressed with his illness. The patient has also been consulted for ear, nose, and throat (ENT) examinations. The ENT examination indicates normal ear conditions. There were no complaints related to auroras and photophobia.

Previously, patients were diagnosed with lung cancer six months ago and planned for follow-up chemotherapy. Various medications have been administered, including paracetamol, Non-steroidal anti-inflammatory drugs (NSAIDs), minor tranquilizers, and even antidepressants, but the pain is not reduced. The patient's used to work in the BUMN company.

History of disease and treatment :

a) History of lung cancer
b) History of hypertension is present and controlled
c) Smoking history exists
d) History of falls denied
e) History of allergies denied
f) History of taking antihypertensive drugs, anti-pain, antidepressants.

Physical examination

General status

General condition	: Look in pain
Awareness	: Compos Mentis
GCS	: E4V5M6
Degree of Pain	: NRS rest 3-4, NRS relapse 9-10

Vital sign

Blood pressure	: 160/90 mmHg
Pulse	: 90x/minutes,
RR	: 18x/ minutes
Skin	: Turgor skin is enough
Head	: Normocephali, Alopecia
Eye	: Anemis conjunctiva (-/-), sclera icteric (-/-)
Neck	: Symmetrical, deformity (-), enlargement of the thyroid gland (-), trachea deviation (-), nuchal rigidity (-)spasm of the back muscles of the neck (+)
Pulmo	: Normochest, symmetrical chest wall, no deformity
Abdomen	: Flat, supel (limp)
Extremity	: Edema of the limbs (-/-), cyanosis (-).

Localist Status

Head and Neck inspection	
Skin	: No abnormalities found
Deformities	: No deformities
Muscle reduction	: No reduction in neck and shoulder muscles found
Range Of Motion	: Pain of the back head when heading forward, hyperextension, and rotation
Palpation	: Palpation on the back area of the head: edema (-), pain when getting a little touch or pressure, increase in skin temperature in the nerves area GON, LON, and TON (-), spasm of the neck and back of the head muscles (+), sensory within normal limits

Table 1. Additional examination

Examination	Right	Left
Tinnel Test	(-)	(+++)
Flexion motion of neck rotation extension	Hyperextension and rotation pain Head forward pain	
Spurling test	(-)	
Touch sensation	(-)	(+++)
Cold sensation	(-)	(+++)

Additional examination:

Hematology laboratory examination, clinical chemistry (liver and kidneys), hemostasis in the normal range. Electrolytes (Na, K, Cl, Ca) in the normal range. Inflammatory (C-reactive protein quantitative) 6.44 increase by the normal value of <0.3, blood sugar 130. Computed tomography (CT) scan of the head showed destructive osteolytic lesions on the left side of the clivus os, left condylus occipitalis, os temporal (pars petrous, mastoid) with invasion to the left jugular foramen and encasement of carotid arteries Interna ec metastases.

Differential diagnosis

1. Migraine
2. Cluster Headache
3. Tension Headache
4. Hemicrania Continua

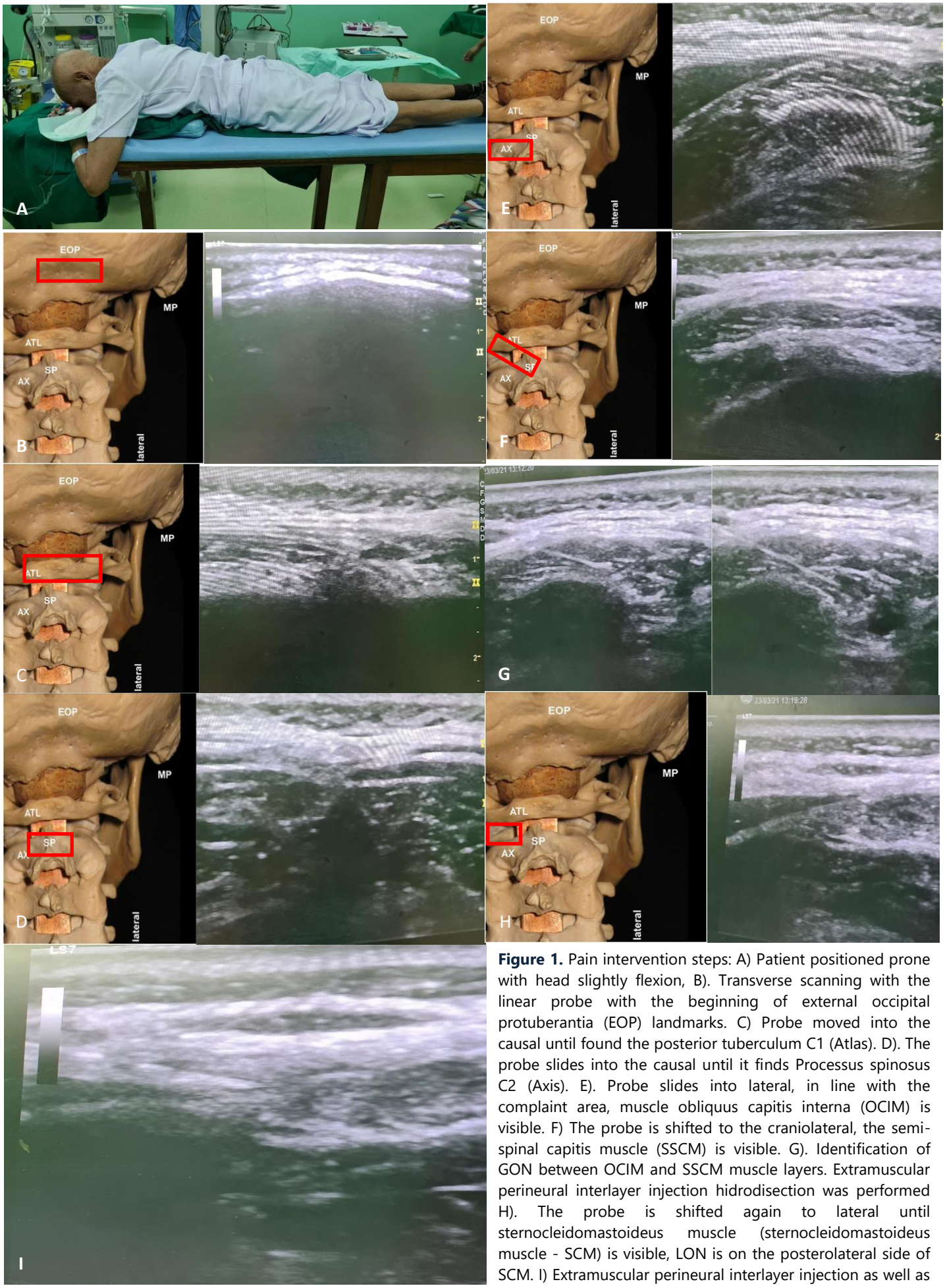

Figure 1. Pain intervention steps: A) Patient positioned prone with head slightly flexion, B). Transverse scanning with the linear probe with the beginning of external occipital protuberantia (EOP) landmarks. C) Probe moved into the causal until found the posterior tuberculum C1 (Atlas). D). The probe slides into the causal until it finds Processus spinosus C2 (Axis). E). Probe slides into lateral, in line with the complaint area, muscle obliquus capitis interna (OCIM) is visible. F) The probe is shifted to the craniolateral, the semi-spinal capitis muscle (SSCM) is visible. G). Identification of GON between OCIM and SSCM muscle layers. Extramuscular perineural interlayer injection hidrodisection was performed H). The probe is shifted again to lateral until sternocleidomastoideus muscle (sternocleidomastoideus muscle - SCM) is visible, LON is on the posterolateral side of SCM. I) Extramuscular perineural interlayer injection as well as hidrodisection was performed. ATL = atlas, AX = axis, EOP = external occipital protuberance, MP = mastoid process ,SP = spinous process of axis.

Diagnosis
Patients diagnosed with occipital neuralgia

Pain intervention management

1. On 8/3/2021: Performed blocks of GON and LON using ultrasound-guided, the drug used plain lidocaine 2% and steroids dexamethasone 10 mg.
2. On 23/3/2021: Perineural deep injection along with prolo-hidrodisection in GON and LON using dextrose 15% and local anesthesia lidocaine plain 2% with a volume of 3 cc each nerve. Interventional pain management procedures can be seen in the figure 1.

Observation of Therapy Results:
One day after the intervention:

1. Complaints: pain is reduced a lot, hearing improves, and the noise in the ear disappears. The patient can sleep using a pillow
2. Pain Scale: NRS rest 0-1, NRS motion 2-3, and there has been no relapse during post-action observations

DISCUSSION

The International Headache Society defined occipital neuralgia as a stabbed sensation in the distribution of GON, LON, and or TON dermatomes with often severe pain intensity and causing weakness, associated with a functional quality of life. Pain originates from the suboccipital region and extends throughout the top of the head. Hypo or dysesthesia in the affected area can accompany the pain.[8,9]

Based on the examination, patient complaints refer to the symptoms of occipital neuralgia syndrome, which is pain felt like shooting or stabbing, paroxysmal, and stinging in the back of the head. Pain radiates to the back of the ear and the top of the head, according to the dermatome of GON and LON. The greater occipital nerve (GON) arises from the primary dorsal ramus of the second cervical nerve with the contribution of the third cervical nerve. GON supplies sensory innervation to the medial part from posterior scalp to anterior, i.e., to vertex, and also supplies motor innervation in semispinalis capitis.[8] Compression in GON often occurs when nerves pass through inferior obliquus capitis muscles, semispinalis, and trapezius muscles.[1]

Lesser occipital nerve (LON) arises from the primary ventral ramus of the second nerve with the contribution of the third cervical nerve. Lesser occipital nerve (LON) runs along the posterior boundary of the sternocleidomastoid muscle, supplying the cutaneous innervation towards the lateral part of the scalp and the cranial surface of the auricula. The most common compression location is when passing through the fascia of the sternocleidomastoid muscle. The third occipital nerve (TON) arises from the inside towards the medial branch of the hemp dorsal of the third cervical nerve, leading to the medial of the GON, and is associated with the occiput and GON rounds at the inferior edge of the inferior capitis oblique. TON supplies sensory innervation to the skin covering the rostral part of the neck and occipital near the external occipital protuberance. Locations that often cause TON compression, i.e., in the facet joint C2-3.[8]

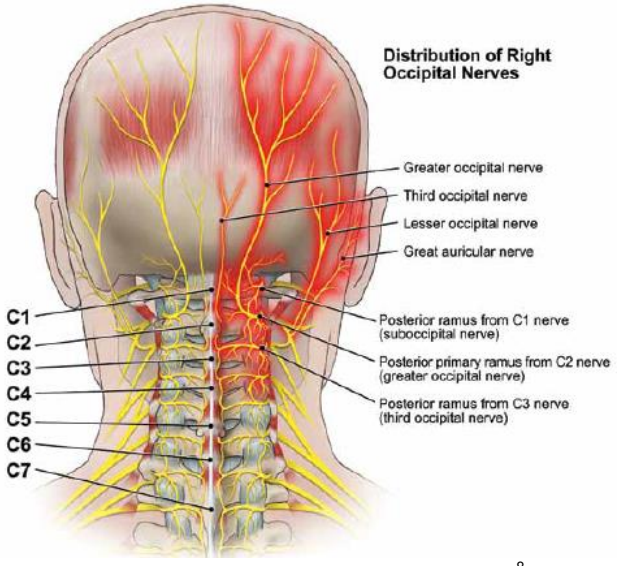

Figure 2. Right occipital nerve distribution[8]

In these patients, the distribution of pain is in accordance with the dermatomes (Figure 2), so our examination heading to occipital neuralgia. The diagnostic criteria used are in accordance with the International Headache Society (Table 2). Type of pain, radiation and distribution, onset, Tinnel test (+), and the first therapy with local anesthetic block test obtained a complaint improvement in accordance with occipital neuralgia.

Table 2. Diagnosis criteria of occipital neuralgia according to The International Headache Society in the occipital neuralgia[8]

Criteria
A shooting or stabbing sensation, paroxysmal with or without persistent pain,
between those paroxysmal pain in the occipital nerve mayor, minor, or spinalis nerve C3
Pain above the involved nerves
Temporal pain can be relieved by a local anesthetic block in the affected nerve

Other complaints that can occur in occipital neuralgia are visual impairment (67%), tinnitus (33%), dizziness (50%), nausea (50%), and nasal congestion (17%). These complaints may arise due to contact with cranial nervus VIII, IX, and X and truncus simpaticus cervical. In these cases, patients experience other complaints, including tinnitus and hearing loss.[10]

Based on etiology, occipital neuralgia can be distinguished into structural and idiopathic etiology. Structural etiology includes trauma to GON and or LON, compression of GON, LON, and or TON, compression of nerve roots C 2-3 by degenerative processes, cervical disc disease, and tumors at the nerve root C 2-3. The literature says most of its etiology is idiopathic in the absence of structural abnormalities.[1,8] Trescot et al. stated that tumor abnormalities or metastases in the bones can be a contributing factor to pain.[9] In this case, a history of malignancy in the lungs with metastases was obtained, and a CT scan showed metastases in the cranium occipitoparietal area. This factor is likely to be a structural cause of occipital neuralgia suffered by patients.

The determination of pathological compression location of the GON is very important since the contribution of GON in occipital neuralgia is 90%. There are several areas of occipital nerve compression: (1) where GON arises from the dorsal root ganglion C2 (DRG), between the atlas and axis (2) between inferior obliquus muscle and semispinalis muscle

capitis (3) where nerves penetrate semispinalis capitis, and (4) where nerves come out of the aponeurosis of the trapezius muscle.

Base on physical examination, the patient's head posture tends to be upright towards the back. If the head is forward (wearing a pillow), hyperextension or rotation will cause pain. From the literature, this postural is called a "pillow sign," where GON compression occurs in the inferior obliquus muscle area. Tinnel test shows positive results in compression area. The usual location of GON compression is seen in the image below (Figure 3A):[9]

The planned intervention are diagnostic and hydrodissection blocks on GON and LON. After diagnostic block using local anesthesia and dexamethasone steroids, we performed a definitive pain intervention by administering a 15 % dextrose injection on the extra muscular and perineural interlayer layer simultaneously prolohydrodissection GON and LON. The definitive therapy is performed two weeks after diagnostic block. Consideration of time lag can be adjusts based on the patient admission as well as provide a duration for dexamethasone half-life bioavailability, so it will not affect the next modality.

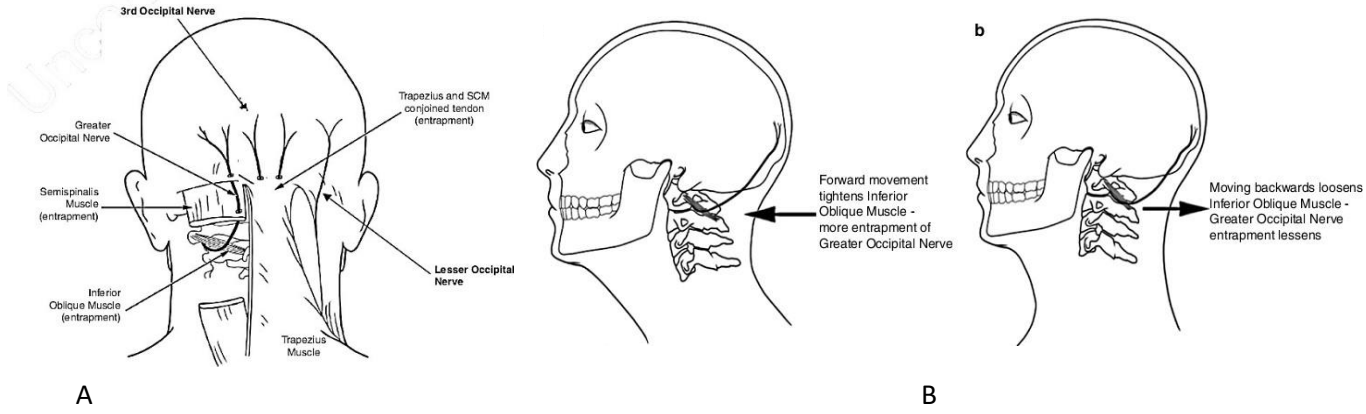

Figure 3. A. The occipital nerves compression area, B). The mechanism of GON compression by inferior obliquus muscle[9]

Several clinical trials were perform to underlie dextrose therapeutic injection. The new drug ingredients are used in several injection-based modalities for the pain management. Basic science and recent clinical research suggest dextrose can reduce pain, improve overall function, and restore connective tissue function. While the mechanism of action of dextrose is not yet well understood on a cellular level. Mobile-level research is still being conducted, but the publication is limited. Clinical trials have assessed three therapeutic-related modalities using dextrose and reported positive clinical effects compared to controls. The techniques used are as follows:[11]

1. Prolotherapy: Hypertonic dextrose injection to treat chronic musculoskeletal pain. Hypertonic dextrose is the most commonly used injection; the purported mechanism focuses on the proliferative repair.
2. Perineural Injection Therapeutic (PIT) with dextrose: Dextrose injection of peripheral nerve areas to reduce neuropathic pain. Dextrose solution is almost isotonic (dextrose 5% in water; D5W) most commonly used. The purported mechanism is related to sensorineural effects. It is also mentioned in some literature under the name Neuronal Prolotherapy.
3. Hydrodiseccion with dextrose: Dextrose is injected near the peripheral nerve with ultrasound guidance to release the peripheral nerve from the fascia that wraps it to give a decompression effect. From other literature also called Perineural deep injection.[12]

Those three mechanisms are hypothesized to have the following effects: [11]

1. Slow down, stop or even reverse degenerative changes in ligaments, tendons, and joints;
2. Simultaneously localize and treat primary nociceptive sources with proper diagnostic injection;
3. Reduce peripheral sensitization in neuropathic pain; and
4. It directly releases nerve entanglement and reduces neurogenic inflammation without the risk of anesthesia toxicity.

Prolotherapy is a modality that uses dextrose as a medicine to treat chronic pain. The term is derived from the words "proliferative" and "therapy." Basic science has not been able to explain the clear mechanisms and the exact concentration of dextrose. Some studies use a type that varies from a 10% non-inflammatory solution to a 12.5-25% inflammatory solution. There have been no studies comparing the relative proliferative effects of different concentrations.[12] The dextrose concentration thatoften used in clinical practice is between 12.5% - 25%. Dextrose is considered an ideal proliferation agent because it is water-soluble, naturally part of chemical structure of the blood, and can be injected safely in some areas and in large quantities. Hypertonic dextrose solution acts by dehydrating the cells on the injectable side, causing local tissue trauma that will attract granulocytes and macrophages and trigger healing. Dextrose proliferation has been approved by the FDA.

The mechanism of action behind prolotherapy is not yet fully known. However, a recent theory says proliferation injection will trigger the body's natural healing process by initiating a cascade of local inflammations that will trigger the release of growth factors and depositions from collagen. As shown in the image below (Figure 4).[13]

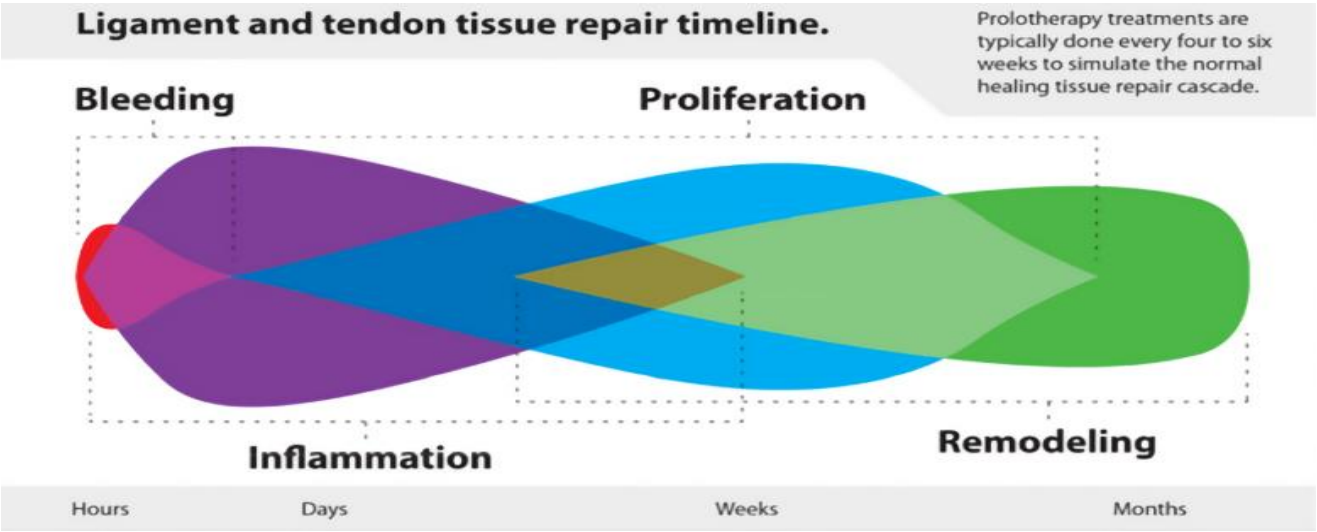

Figure 4. Timeline of the prolotherapy mechanism of action[13]

The proliferative effect of dextrose in fibroblasts has been studied in vivo using hypertonic dextrose concentrations. Research by Oh et al. reported that 10% dextrose injection, in contrast to saline control injection, led to the proliferation of subsynovial tissue. Dextrose injection also showed subsynovial tissue proliferation thicker than saline-injected control tissue.[11] A 12.5% dextrose intra-articular injection showed the growth of new cells in stage IV knee osteoarthritis as seen in the colorization of biopsy tissue. A 12.5% dextrose is known to be slightly inflammatory, but when the injection is placed in a suprapatellar pouch, it will be diluted quickly up to a concentration of 10% or less. [11]

Perineural injection therapeutic (PIT) by giving 5% dextrose around the neural tissue indicates a clinical improvement. Clinical improvement in the absence of proliferation may be caused by sensorineural effects of dextrose on neuropathic pain generators. Clinically, pain reduced immediately or within 1-2 days after treatment. This phenomenon is a period of time that is not in line with the effect of tissue proliferation. To understand what might happen, an understanding of the relationship between nerve inflammation and chronic pain is important. Increased regulation of inflammatory mediators produced by acute changes after injury, including prostaglandins, nerve growth factors, bradykinin, interleukin, or TNFα, will modulate the potential for temporary receptors of sodium ion channels in the central and peripheral nerves (especially C peptidergic fibers) and may lead to a transition from acute to chronic pain.[11]

This transition to chronic pain is characterized by the production and release of pain-producing neuropeptides and continuous degenerative processes. These neuropeptides typically include substance P and the peptide-related calcitonin gene Calcitonin gene-related peptide (CGRP). The production and release of this neuropeptide by activation of C fibers is called neurogenic inflammation and is characterized by the absence of leukocytes.[11] The potential action of dextrose in sensorineural effects has been widely stated in various writings. In 2005, Dr. John Lyftogt observed that injection of subcutaneous dextrose without local anesthesia in the sensory pain nerve (PIT with dextrose) resulted in rapid loss of hyperalgesia and allodynia (within seconds) of the injection area. Results from several case studies showed pain reduction with subcutaneous dextrose injection of the associated sensory nerve pathways in Achilles tendinopathy, knee, shoulder, and elbow pain, and lower back pain.[11]

More clearly, the mechanism of action of dextrose in neuropathic pain can be seen in the figure 5, where it is suspected that glucose works on the Transient receptor potential cation channel subfamily V member 1 (TRPV1) capsaicin receptors in neuron cell membranes. If TPRV1 is active,

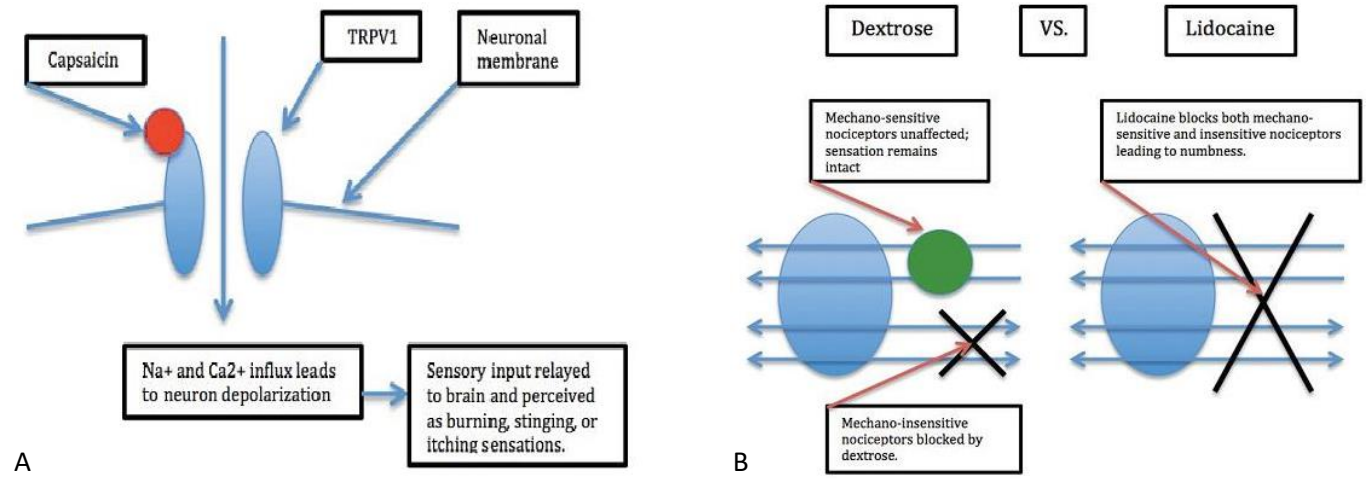

Figure 5. The glucose action in the TRPV1 of neuronal membrane. A). Capsaicin as TRPV1 activator, B). Effect of dextrose on TRPV1 receptor[14]

then there will be the production of inflammatory neurogenic chemical mediators from neurons in the form of CGRP and substance P, where this mediator will be secreted by afferent neuron cells. Incoming and transmitted signals are burning, itching, and stinging or punctured sensations typical of neuropathic pain. Glucose will inhibit these receptors and restore neuron function under normal conditions. Then against nociceptive pain or acute pain, described glucose inhibits mechano-insensitive receptors so as not to cause numbness. This is in contrast to local anesthesia, which inhibits mechano-sensitive and insensitive (Figure 5).[14]

The rapid neurogenic effects of dextrose on pain-producing C fibers after the subcutaneous injection may also explain the rapid reduction of pain after intraarticular injection in prolotherapy since the same pain-producing C fibers are also found in the bone cortex.[11] The dextrose injection analgesic effect observed by Dr. Lyftogt is then reported in RCT comparing D5W with saline injection in the caudal epidural chamber in patients with back pain, buttocks, or legs. Dextrose injection produce significant analgesia for 15 minutes to 48 hours.[11]

The hydrodissection technique is a long-developed nerve decompression technique. However, the use of dextrose itself in such techniques is still very rare. Pain caused by nerve entanglement in classic and non-classical locations is increasingly suspected as the cause of chronic pain along with the development of ultrasound imaging. Bennett observed that non-press contact of connective tissue with the mouse's scimatic nerve surface resulted in functional nerve disorders and the appearance of an "hourglass" formation with nerve swelling on both sides of the connective tissue. The entanglement formation by connective tissue on the nerve supports the concept that minimal compression of nerves in the fascia layer can result in clinically important neurogenic inflammation and neuropathic pain. The use of real-time visualization with ultrasound to inject fluid adjacent to the peripheral nerve to separate the visible nerves from all fascia layers is called hydroids. Wu et al. compared the hydrodissection action of the medianus nerve in carpal tunnel syndrome with subcutaneous injection using normal saline, reporting better benefits of hydrodissection than subcutaneous injection. In other randomized controlled trials, hydrodisxia with D5W was better than hydrodissection with saline or hydrodissection with triamcinolone in copy. Thus, dextrose hydrodissection seems to offer mechanical hydrodissection and sensorineural effects in cases of carpal tunnel syndrome.[11,12]

To emphasize the potential benefits of hydrodissection in neurogenic pain. Lam et al. study various nerves or ganglia in the upper body (stellate ganglions, brachial plexus, cervical nerve root, and paravertebral space) in patients with severe neuropathic pain and obtained pain reduction results exceeding 50% in 26 consecutive participants. This high volume hydrodissection is used only dextrose, so it does not have the risk of lidocaine toxicity.[11]

Basic science and clinical studies show therapeutic effects of dextrose in conditions related to degeneration or insufficiency of connective tissue, neuropathic pain, and the presence of narrowing of the fascia or entanglement in the nerves. Most idiopathic neuropathic sufferers may experience increased symptoms due to the "double crush" effect of nerve compression, and treatment of such susceptible nerves to reduce neuropathic symptoms may be an opportunity for further clinical research. In addition, because dextrose is studied to have analgesic effects and can be used for hydrodissection without anesthesia, its use in therapeutic nerve blocks can facilitate diagnostic and therapeutic injection while preventing lidocaine toxicity.[11]

The ideal dextrose injection concentration for therapeutic applications has not been clear evidence-based. From the research data so far, we know very little about the proliferation capability of dextrose less than 10% since all in vivo studies have used concentrations of 10% or more. Dextrose may be more effective when its concentration reaches the level that triggers inflammation (12.5%), but this also requires more research. Dextrose 5% is recommended to reduce neuropathic pain, minimize the potential proliferative effects of dextrose in enclosed spaces since clinical effects do not seem to vary from 5-25%. The dextrose mechanism of action in each procedure and clinical indications tend to be multifactorial due to the complexity of chronic pain and the degree of suppression due to compression and volume at the tissue level.[11]

In patients carried out extra muscular perineural interlayer injection using dextrose concentration of 15%. Dextrose solution is diluted under local anesthesia to achieve blockade on TPRV1 neuropathic pain receptors and nociceptive acute pain receptors. The selection of dextrose concentration of 15% is a consideration of achieving proliferation and inflammatory stimulus effects. Due to the concentrated solution concentration of 15%, there will be a pain at the injection site, so local anesthesia is required. The volume given is 3 cc on each nerve to give the hydrodissection decompression. This therapy reported a significant reduction in the degree of pain and improvement of neurological complaints of hearing loss in patients. The combination of perineural deep injection therapy with prolohydrodissection proved beneficial in patients with occipital neuralgia with nerve compression. Prolotherapy treatment still requires more research of either animal research trials or further clinical trials. It is an opportunity for pain practitioners to learn more and make a promising therapy in the future.

CONCLUSION

Occipital neuralgia is caused by compression of the occipital nerve, and 90% is caused by GON compression. The use of prolotherapy is still limited and has not been widely preferred by pain practitioners. In the above case, we use a combination of three techniques to use dextrose preparations. The results are quite satisfactory, with significant improvements in patients. This case report showed an opportunity to develop prolotherapy in pain management.

ACKNOWLEDGMENT

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CONFLICT OF INTEREST

None

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