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Dextrose Treats Optic Neuritis

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Abstract
The sphenopalatine ganglion—also known as pterygopalatine ganglion, Meckel's ganglion, Sluder's ganglion and nasal ganglion—is the largest of the four parasympathetic ganglia associated with the trigeminal nerve. It is considered one of the largest neuron collection in the head outside of the brain, being exposed to the environment via the nasal mucosa. Classically, refractory head and face pain were treated with a series of ineffectual medications with intolerable side effects – cycling from one to the next based on trial and error. Although the sphenopalatine ganglion is a little-known region in the face, pain management specialists believe that it is very effective in the treatment of many conditions. It is a life changing, safe and established procedure that offers the pain sufferers an immediate relief from their pain. Dextrose 5% concentration in a neutral pH sterile water solution treats the neurogenic inflammation and stops the neuropathic pain by blocking the TRPV1 ion channels. In this paper, a 32 years old lady was suffering from severe headache, with an impaired vision of the left eye. The Visual Evoked Potential (VEP) showed an attack of optic neuritis. After 5 sessions of treatment with buffered dextrose in 5% concentration, the VEP showed a resolved attack of optic neuritis.
Conclusion: *Buffered Dextrose in 5% concentration gave marvelous results in the treatment of headache and optic neuritis, and helped the patient to regain her vision.*
Keywords: *Sweet nasal treatment, Lyftogt perineural injection treatment, optic neuritis, headache, dextrose.*

Introduction

The pterygopalatine ganglion of Meckel, the largest of the parasympathetic ganglia associated with the branches of the maxillary nerve, is deeply placed in the pterygopalatine fossa, behind the middle turbinate of the nose and close to the sphenopalatine foramen. It is triangular or heart-shaped, of a reddish-gray color, and it is situated just below the maxillary nerve as it crosses the fossa[(1)].

It receives sensory, parasympathetic and sympathetic roots. The sensory root is derived from the sphenopalatine branches of the maxillary nerve. The parasympathetic root is derived from the nervus intermedius (a part of the fascial nerve) through the greater petrosal nerve. The post-ganglionic axons (vasodilator and secretory fibers) are distributed with the deep branches of the trigeminal nerve to the lacrimal gland, the glands of the mucosa of the nasal cavity, paranasal sinuses, hard and soft palate, tonsils, uvula, roof

of the mouth, lips, gums and upper part of the pharynx. The sympathetic root consists of the efferent post-ganglionic fibers of the superior cervical ganglion[1].

The sphenopalatine ganglion has been associated with a wide variety of pain problems that range from pain in the head and neck such as trigeminal neuralgia[2],[3], temporomandibular joint (TMJ) pain[3], sphenopalatine neuralgia, migraine headaches, cluster headaches, atypical facial pain [4], cancer pain of the head and neck, tongue pain, gum and mouth pain, teeth pain, Sluder's neuralgia[5], paroxysmal hemicranias[6], post-herpetic neuralgia[6], herpes zoster[7], vasomotor rhinitis, complex regional pain syndrome (CRPS) [8], [9], [10], post-traumatic headache [11] to low back pain [12].

Beginning with the early part of the 20th century, Sluder reported the first case of headache being relieved by sphenopalatine ganglion block with local anesthetic[13],[14]. Patients with chronic recurring head and face pain were treated with intranasal phenolization or cauterization of the sphenopalatine ganglion for the treatment of Sluder's neuralgia [15] with 90% relief from their pain.

The brain itself is not sensitive to pain, because it lacks pain receptors. However, several areas of the head and neck do have pain receptors and can thus sense pain. These include the extracranial arteries, middle meningeal artery, large veins, venous sinuses, cranial and spinal nerves, head and neck muscles, meninges, falx cerebri, parts of the brainstem, eyes, ears, teeth and lining of the mouth [16], [17].

Headaches often result from traction to or irritation of the meninges and blood vessels. Blood vessel spasms, dilated blood vessels, inflammation or infection of meninges and muscular tension can stimulate nociceptors and cause pain[16]. Once stimulated, a nociceptor sends a message up the length of the nerve fiber to the nerve cells in the brain, signaling that a part hurts.

The sensory innervation of the meninges is primarily by meningeal branches of both the trigeminal and vagus nerves with a smaller contribution from the upper cervical spinal nerves [18],[19]. The supra-tentorial dura mater is mainly supplied by the ophthalmic division of the trigeminal nerve[20].

The optic nerve is the second of twelve paired cranial nerves and is technically part of the central nervous system. The optic nerve is en-sheathed in all three meningeal layers (dura, arachnoid, and pia mater) rather than the epineurium, perineurium, and endoneurium found in peripheral nerves [21].

The sensocrine nervous system in the body represents 50% of the somato-sensory system. It is formed of the small nerve fibers "nervi nervorum" (un-myelinated C and some myelinated Aδ nerve fibers) being widely distributed everywhere in the body, it is responsible for the interoceptive sensation which reflects the metabolic disturbance in the micro-environmental atmosphere in order to support the tissue homeostatic control function, it is very rich in transient receptor potential vanilloid type 1 (TRPV1), and it carries the sensory nerve impulses in both orthodromic and antidromic directions[22].

Sensocrine nervous system, from its name, has a dual action (sensory and endocrine). TRPV1 are trans-membrane, non-specific, polymodal ion channels that become stimulated and up-regulated on exposure to nociception (mechanical, thermal or chemical), acidosis (H+), hypoxia and GLYCOPEANIA [oxygen and glucose are the principle homeostatic molecules]. When stimulated, these ion channels allow: (1) Influx of sodium (Na+) leading to propagation of action potential (neuropathic pain). (2) Influx of calcium (Ca2+) leading to release of neuropeptides CGRP and SP (neurogenic inflammation). (3) Efflux of potassium (K+)[22].

Patients and Methods

A 32 years old female, mother of two kids, suddenly could not see anything with her left eye except white color and black floating spots which lasted for seconds, then her vision went back to

normal, this was on the 5th of May, 2018. One week later, on the 12th of May 2018, she developed a severe headache with the feeling of a severe pressure on her left eye. Since that day, she was feeling that her left eyesight was getting worse on getting up every morning, till one day she woke up and could not see anything with her left eye except identifying colors with cloudy vision, this was on the 19th of May 2018. On the 3rd of June 2018, she was sent for a visual evoked potential (VEP) [an evoked potential is an electrical potential recorded from the nervous system following presentation of a stimulus, detected by electroencephalography (EEG)]. The VEP pattern according to ISCEVs standards revealed that both eyes showed well formed response with average implicit time on two different size stimuli with reduced amplitude in the left eye compared to the right eye (OD: 90,97ms and OS: 95,98ms) denoting affection of conduction in visual pathway. The impression was that the right eye was within normal optic nerve function and the left eye showed a resolving attack of optic neuritis. She started the sweet nasal treatment on the 4th of June 2018: The patient lays down on her back (supine position) with the neck in the hyper-extended position out of the bed, a 5 ml of buffered dextrose 5% concentration was applied in each nostril, then the patient stays in this position for 10-15 minutes. (Buffered dextrose solution is obtained by adding 2.5 ml of 8.4% of sodium bicarbonate to a 500 ml bottle of dextrose 5% concentration). On getting up after the treatment, the patient has to bring her head on the side to not aspirate the solution from the nose into the lungs. The patient repeated again the treatment on the 5th and 6th of June, then again on the 8th, 9th and 10th of June, then again on the 12th, 13th, 14th and 15th of June, 2018. The patient felt that her left eye vision started going back to normal since the first session, and she kept on improving after each session till she could read the billboards on closing the right eye. On the 8th of June 2018, the VEP was redone at the same place, with the same person and same machine and the VEP pattern according to ISCEVs standards revealed that the left eye showed well formed response with average implicit time on two different size stimuli (OS: 96,98ms) and improved amplitude compared to the first visit denoting good conduction in visual pathway. The impression was that the VEP response showed improved amplitude compared to the first visit returning to normal limits (Resolved attack of optic neuritis).

Conclusion

1) Perineural treatment (Sweet Nasal technique) is very effective and gives marvelous results in the treatment of optic neuritis.
2) Perineural treatment (Sweet Nasal technique) is a very strong and effective treatment for headaches, migraines, facial pain, chronic sinusitis, snoring, ear tinnitus, eye lacrimation and dizziness.
3) Perineural treatment (Sweet Nasal technique) is very promising in the treatment of the neck, shoulder and arm problems.
4) Perineural treatment (Sweet Nasal technique) is the challenging ease that solved the unsolved back and lower limbs problems secondary to neck problems.
5) Perineural treatment (Sweet Nasal technique) is a very powerful technique in the treatment of the hard and serious problems that were not treated completely in the past. It is very promising in the Regenerative Medicine and therefore more research work is needed in the treatment field of the high cognitive functions of the brain, strokes, memory problems, dementia and Alzhimer's disease.

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