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The effectiveness of neurolytic block of sphenopalatine ganglion using zygomatic approach for the management of trigeminal neuropathy

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ABSTRACT

This study was performed to present the outcomes of trigeminal neuropathy management with the application of neurolytic block of sphenopalatine ganglion. This type of procedure is used in cases where pain is not well controlled with medical treatment. Twenty patients were treated with sphenopalatine ganglion neurolysis after their response to pharmacological management was not satisfactory. Significant pain relief was experienced by all but one patient and they were able to reduce or stop their pain medication. The time of pain relief was between a few months and 9 years during the study period. Number of procedures implemented varied as some of the patients have been under the care of our Pain Clinic for as long as 18 years, satisfied with this type of management and willing to have the procedure repeated if necessary. It appears that neurolytic block of sphenopalatine ganglion is effective enough and may be an option worth further consideration in battling the pain associated with trigeminal neuropathy.

1. Introduction

Due to its complex pathophysiology, the facial pain is a clinical challenge. One of the most common causes of unilateral facial pain is trigeminal neuralgia (TN). In some rare cases the clinical picture of TN change and progress to trigeminal neuropathy, which is characterized by constant pain accompanied by sensory disturbances, with only episodes of typical, neuralgiform pain. The cause of neuropathy may be an extreme duration of TN and the destruction of peripheral rami of the trigeminal nerve related to neurodestructive procedures or tumors and trauma.

It should not be confused with trigeminal neuralgia, where episodes of shooting pain prevail, with no sensory or motor deficits between them. Although the pain is usually predominant in the clinical picture, its constant character and sensory deficits that appear as the condition progresses – serve to differentiate between the two. It should also be noted that a damage to the rami of trigeminal nerve may be caused by

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many pathological processes, like malignant lesions [1]. Therefore, a thorough clinical evaluation is mandatory in all cases with facial numbness before symptomatic management is employed.

As pharmacological management is moderately effective in trigeminal neuropathy, substantial number of patients will not be satisfied with pharmacotherapy or develop some significant side effects. In these cases the invasive procedures are mandated. In our center we use the neurolytic block of sphenopalatine ganglion (SPG). This procedure had been performed in our center since 1990 and neurolytic agent used is 65% ethanol. There were attempts to replace it with radiofrequency thermocoagulation (RT), but it was abandoned due to the worse outcomes. In every case of trigeminal neuropathy treated with RT of SPG, the alcohol neurolysis was later performed, as improvement achieved with RT was not satisfactory.

Trigeminal neuropathy is the most common indication for neurolytic block of SPG in our center. We also use this procedure in Horton migraine and tumor-related facial pain in the area of trigeminal nerve innervation. It was used with success in typical trigeminal neuralgia (TN) in the past, but was replaced by RT of Gasserian ganglion more than 10 years ago, as the latter proved to be much more effective in TN. It is a safe procedure, which – unlike neurolytic blocks of peripheral branches of TN – does not carry a risk of sensory deficits.

Sphenopalatine ganglion (SPG), or pterygo-palatine ganglion (PPG) and termination of its function in pathogenesis and treatment of facial pain has been an issue of interest for more than a 100 years, since in 1908 Sluder described and performed its block for the first time [2]. He had been using SPG blocks in patients with unilateral facial pain, located at the bridge of the nose, radiating to periorbital area, zygomatic process, mastoid process and occipital area. This pain was to be accompanied by autonomic symptoms (running nose, lacrimation, blood-shot eyes) and was eventually named *Sluder's neuralgia*. Recent research has confirmed the importance of SPG in pathophysiology of many types of facial pain and headaches, as well as stroke and cerebral vasospasm [3]. In spite of a 100 years of history, there is very limited number of papers reporting the long-term outcomes of its neurolytic blocks.

1.1. *Sphenopalatine ganglion anatomy*

SPG consists of a large number of neurons that form the triangular structure of approximately 5 mm. It is located on the outside of the cranium, in the pterygopalatine fossa (PPF). Pterygopalatine fossa contains SPG, maxillary artery with some of its branches, venous plexus and maxillary nerve.

SPG is of mixed character: sensory, parasympathetic, and sympathetic. Its sensory root is provided by the sphenopalatine nerves from maxillary nerve. They contain dendrites of the neurons located in trigeminal ganglion (hence the beneficial effect of SPG block in TN). Sympathetic root is formed by the efferent (postganglionic) fibers provided by deep petrosal nerve (a target of neurolytic block in trigeminal neuropathy). Parasympathetic root is derived from facial nerve through the greater petrosal nerve. It is formed by dendrites of the neurons located in the upper salivary nucleus (blocking parasympathetic fibers of SPG is indicated in trigeminal autonomic cephalalgias, like cluster headache) [4]. SPG anatomy is presented in Fig. 1.

1.2. *SPG blocks*

SPG block is usually performed with the use of local anesthetics (cocaine, lidocaine, bupivacaine) and steroids. Neurolytic block is achieved with either chemical (ethanol or

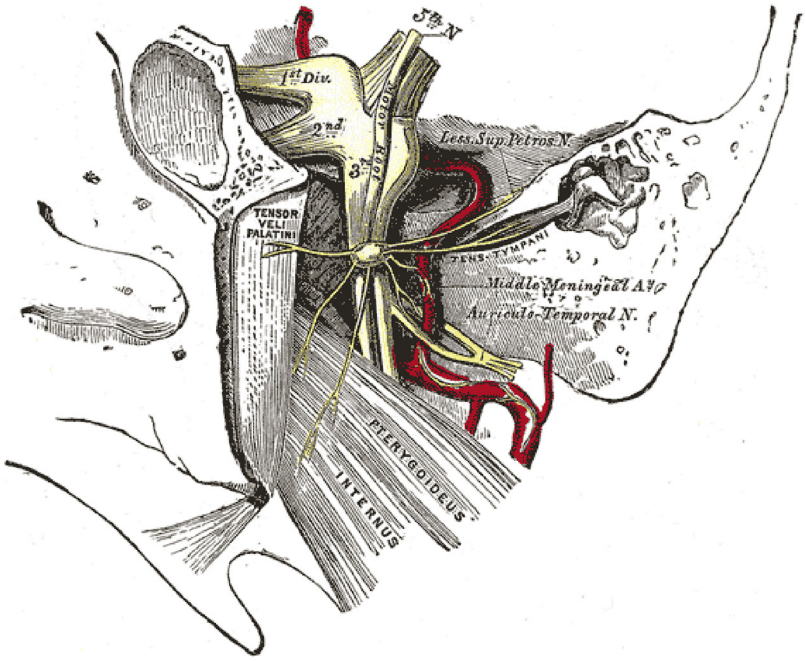

Fig. 1 – Sphenopalatine ganglion anatomy [4].