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Original research article

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.

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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

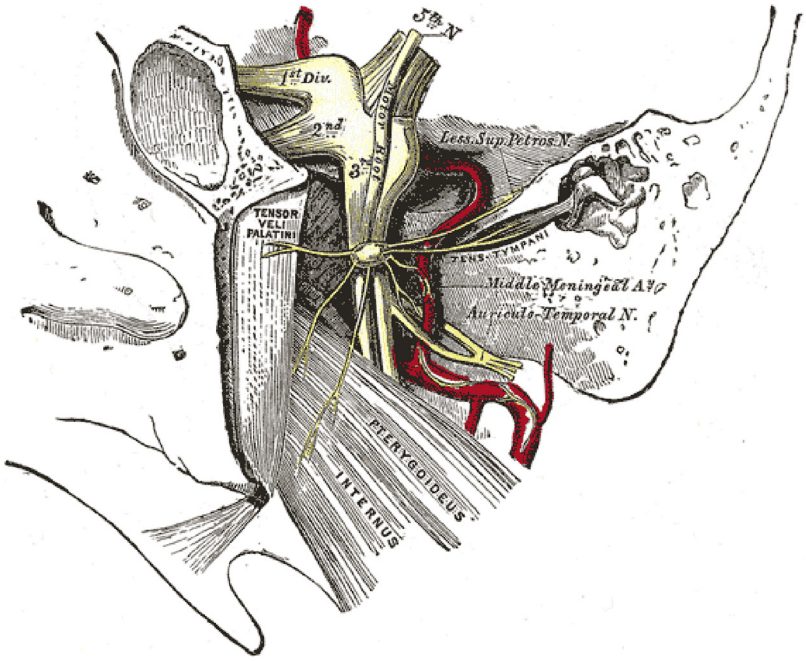

Fig. 1 – Sphenopalatine ganglion anatomy [4].

Table 1 – Patient characteristics and symptom description. M – male; F – female. NRS – pain severity expressed in Numeric Rating Scale (description in the text). V1, V2, V3 – first, second and third branch of trigeminal nerve.

No.	Sex/ age	Pain distribution	Duration of symptoms before SPG neurolysis	NRS continuous	NRS paroxysmal	Buccal numbness	Previous interventions/ cause of neuropathy
1	M/45	V1,V2,V3	7 years	5	8	Yes	Microvascular decompression × 2; V2 nerve blocks (alcohol)
2	M/56	V1,V2,V3	10 years	8	10	Yes	Microvascular decompression
3	F/75	V1,V2, V3	17 years	4	9	Yes	V2 nerve alcohol neurolysis
4	F/71	V2, V3	6 years	3	10	Yes	None/time (6 years)
5	F/53	V2, V3	3 years	3	7	Yes	Dental procedure followed by alcohol neurolysis of V2
6	F/76	V1, V2	17 years	3	8	Yes	None/time
7	M/69	V2	4 years	3	7	Yes	Alcohol V2 neurolysis
8	F/56	V1, V2, V3	20 years	5	9	No	None/time (20 years)
9	M/79	V2, V3	20 years	5	8	No	None/time (20 years)
10	M/71	V2, V3	8 years	5	9	Yes	Alcohol V2 neurolysis
11	F/52	V1, V2, V3	6 years	4	10	Yes	Alcohol V2 neurolysis
12	F/65	V1, V2	13 years	3	8	No	None/time (13 years)
13	M/52	V1, V2	10 years	4	8	Yes	None/time (10 years)
14	F/72	V1, V2	16 years	3	9	Yes	None/time (16 years)
15	M/66	V1, V2	4 years	7	9	Yes	Alcohol V2 neurolysis
16	F/59	V1, V2, V3	4 years	4	8	Yes	Trauma to temp-mandib joint, alcohol V2 neurolysis
17	F/37	V2, V3	7 years	3	10	Yes	Congenital polyneuropathy. Temp-mandib joint dysfunction
18	F/64	V2, V3	7 years	3	8	Yes	Temp-mandib joint dysfunction
19	F/69	V2, V3	10 years	3	8	Yes	None/time (10 years)
20	F/74	V2, V3	10 years	2	9	No	None/time (10 years)

phenol) or physical factors (temperature). Block techniques differ significantly considering their complexity. The least invasive approach is via nasal cavity. It is an easy technique, used for blocks performed with local anesthetics and rarely for neurolytic blocks [5].

Second and more complicated option is an access via greater palatine foramen. The foramen is located at the level of the third molar tooth. Dental 120° needle is then introduced through the mouth and greater palatine foramen and advanced approx. 2.5 cm superiorly and slightly posteriorly. There is no data on the use of this technique for neurodestructive procedures.

The third and most challenging technique is the zygomatic access, which is used for fluoroscopically – guided neurolytic block of SPG in our center.

2. Materials and methods

20 patients (7 males and 13 females) were treated with neurolytic block of SPG for trigeminal neuropathy from 2004 until 2015. Their age was 42–79 years at the time of first block. Table 1 shows the data regarding duration of symptoms, pain intensity and most likely causes of neuropathy. Pain intensity was assessed using 11-point Numeric Rating Scale (NRS), where 0 means no pain and 11 – worst pain imaginable. The procedure was proposed to patients when pharmacologic therapy failed to provide sufficient pain control. All technical aspects of the procedure, as well as potential risk and benefits were thoroughly explained before informed consent was obtained. Coagulopathy, psychiatric disorders, opioid addiction, local infection at the site of needle entry and lack of patient's consent were contraindications. The block was considered successful if resulted in cessation of constant pain and reduction or cessation of paroxysmal pain both immediately after the procedure and at the follow-up visit after 14 days.

In our center we implement the zygomatic approach. It is performed as an outpatient procedure, with the use of C-arm fluoroscopic guidance. On the day of procedure patients use a mouthwash with potassium permanganate, as the needle may pass through the upper recess of the oral cavity. The patient is positioned in supine position, with the C-arm around his/her head. The head is then rotated until the mandibular rami and both perygo-palatine fossae are superimposed on each other. The view of pterygo-palatine fossa then reflects the shape of an upside-down vase.

25 gauge spinal needle is introduced approx. 1 cm below the zygomatic arch and approx. 1 cm anterior of the coronoid process of mandible, advancing it slightly superior, medial and posterior, toward the PPF. Block technique and crucial anatomy points are shown in Fig. 2. In AP view the needle is superimposed on the maxillary sinus and the injected contrast is seen as a point over the view of maxillary sinus. AP and lateral views of accurate needle positioning as confirmed by contrast injection is shown in Figs. 3 and 4, respectively. After needle position is confirmed, the neurolytic agent (2 ml of 65% ethanol with lidocaine) is injected. Due to complexity of the procedure it should be performed under fluoroscopic guidance, preferably by pain specialists who are experienced in invasive pain management. Inappropriate needle placement may result in a number of complications, most serious of them being the ulceration of cornea if the needle is placed too deep (inferior orbital wall) and facial nerve paralysis if the needle is

Fig. 2 – Zygomatic approach used to block SPG.

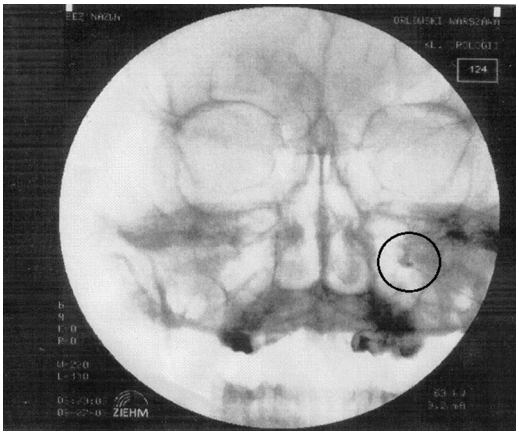

Fig. 3 – Properly positioned needle as confirmed by contrast injection: A–P view.

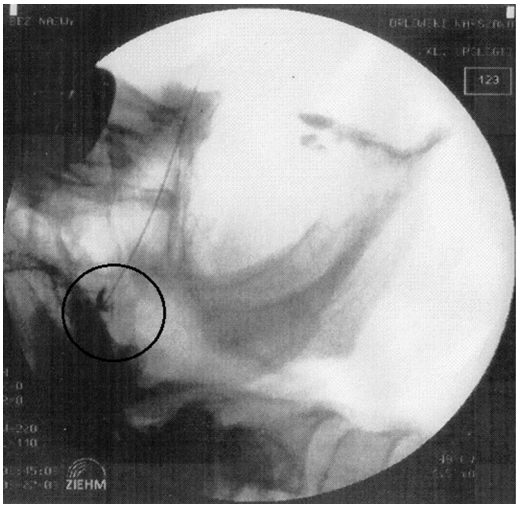

Fig. 4 – Lateral view of the needle and contrast injected in PPF.

placed too shallow and neurolytic agent spreads toward the alar foramen. Inadvertent puncture of the maxillary artery in PPF may result in formation of hematoma, which may take up to 3 weeks to resolve. Infraorbital spread of the neurolytic agent may also effect in diplopia, which resolves spontaneously within a few days.

It does happen occasionally to postpone the procedure after a few unsuccessful attempts to place the needle in correct position. It is usually due to the abundance of vessels present in PPF. SPG block with local anesthetic is then performed; neurolytic block is planned in a few weeks time, which is then usually uncomplicated and successful.

3. Results

Sixty-eight neurolytic blocks were performed in cases described. A success rate was very high, as only three procedures were postponed due to inability to place the needle in correct position under fluoroscopic imaging. In all three cases the procedure was successful with later attempt. Similarly, the effects of the procedure were spectacular. Summary of the management introduced is presented in Table 2. With the neurolytic agent injected, pain relief was noted in 19 out of 20 patients. Similarly, all 19 patients confirmed the improvement brought by the block at the follow-up visit after 14 days. Unsuccessful case was referred for surgical intervention. Neurosurgical decompression was done, which brought some reasonable pain relief for a period of 3 years. After that the radiofrequency ablation of Gasserian ganglion was performed, but no positive outcome achieved. As presented in Table 1, the most common cause of neuropathy was peripheral nerve damage, either due to invasive intervention (9), trauma (2) or congenital nerve abnormality (1). In the remaining eight cases it was impossible to identify the specific cause of neuropathy. All these patients have suffered from TN for many years before symptoms of neuropathic pain became evident, like constant pain and/or sensory disturbances, some of them for as long as 20 years. Therefore in these cases the long lasting neuralgia was considered the cause of neuropathy. All procedures were unilateral and patients remained under the care of our Pain Clinic for a period of time that varied from a few months to many years. Consequently, the number of procedures performed varied from 1 to as many as 10. Recorded duration of pain relief was between 2 months and more than 9 years at the time this report was being prepared. No serious complications were noted, apart from one case of diplopia which resolved within a few days and one episode of bruising at the site of needle entry. All but one of our patient was satisfied with the results of the procedure and – when indicated – would be happy to have the procedure repeated if the pain returns.

4. Discussion

Trigeminal neuropathy may present as a major therapeutic problem in cases where typically indicated pharmacologic therapy is ineffective. Here we present the management implemented in 20 cases of unilateral trigeminal neuropathy

Table 2 – Summary of the management. M – male; F – female. Age: at the time of first block. Pain relief given in months, if sustained – means sustained during the study period. NRS – pain severity expressed in numeric rating scale (description in the text).

	Sex/ age	Time under pain clinic care	Pain clinic medications prior to the block	No of neurolytic blocks	Average pain relief (min–max) – months	NRS after the single block
1	M/45	7	Carbamazepine, Baclofen	6	18.5 (12–30) and sustained	0
2	M/56	16	Amitriptyline Carbamazepine, Baclofen	8	27 (16–48) and sustained	0
3	F/75	13	Carbamazepine, Amitriptyline	3	(36–72) and sustained	0
4	F/71	6	Carbamazepine, Lamotrigine, Baclofen, Opipramol	1	Sustained (>7 years)	0
5	F/53	1.5	Carbamazepine, Amitriptyline	1	Sustained (>18 months)	0
6	F/76	18	Carbamazepine	5	31 (12–48) and sustained (>8 years)	0
7	M/69	10	Carbamazepine, Amitriptyline, Baclofen	2	24 and sustained (>9 years)	0
8	F/56	3	Carbamazepine, Gabapentin, Mianserin	1	No improvement	5
9	M/79	5	Carbamazepine, Baclofen, Mianserin	1	Sustained (>5 years)	0
10	M/71	9	Carbamazepine, Gabapentin, Baclofen, Amitriptyline	1	Sustained (>8 years)	0
11	F/52	4	Carbamazepine, Lamotrigine, Baclofen, Amitriptyline	2	12, 30 and sustained	0
12	F/65	8	Carbamazepine, Baclofen, Amitriptyline	2	30 and sustained (>6 years)	0
13	M/52	11	Carbamazepine, Amitriptyline	10	25.5 (9–60), and sustained (>1 year)	0
14	F/72	8	Carbamazepine, Lamotrigine, Baclofen, Amitriptyline	8	10 (2–24) lost to follow-up 2 years after the last block	0
15	M/66	2	Carbamazepine, Gabapentin, Baclofen, Mianserin	2	8 and sustained (>8 months)	0
16	F/59	6	Carbamazepine, Lamotrigine, Amitriptyline	1	Sustained (>6 years)	0
17	F/37	8	Baclofen, then Venlafaxine	1	Sustained (>7 years)	0
18	F/64	4	Carbamazepine, Lamotrigine, Baclofen, Amitriptyline	2	14 (19–9) Referred for surgical interv.	0
19	F/69	10	Carbamazepine, Gabapentin, Baclofen, Amitriptyline	10	13 (3–36), sustained (>2 years)	0
20	F/74	1	CArbamazepine. Lamotrigine, Baclofen, Trazodone	1	Sustained (>6 months)	0

caused by either previous interventions on peripheral nerves, chronicity of trigeminal neuralgia or congenial factors. Although analgesic block of SPG is known to provide significant advantages in many conditions, its neurolytic block appears to be uncommon and more likely to be used in malignancy-related neuropathies [5–10]. It may be due to seriousness of the procedure or the fact that trigeminal neuropathy appears to be underdiagnosed in modern societies. Nevertheless, current insights into facial sensory deficits are likely to change that [1]. Destruction of the trigeminal nerve neurons – as done in Gasserian ganglion neurolysis – is unlikely to provide long lasting improvement in patients with neuropathy, as pathogenesis of neuropathic pain is different from that of neuralgia. Most of our patients developed neuropathic pain due to neurodestruction of peripheral parts of trigeminal nerves, while long lasting neuralgia was considered the cause of neuropathy in eight cases. Both continuous and paroxysmal pain relief was achieved in all but one patient and relief time varied from a few months to a few years. It is not possible to discuss our results with regards to other studies directly, as research reports on SPG destruction used to treat trigeminal neuropathy are scarce. Varghese and Koshy used nasal approach for phenol-based neurolytic block of SPG in patients with pain related to malignant face and neck tumors and reported some plausible results [5]. Different technique and cause of neuropathy does not allow to compare their findings with ours. Gasserian ganglion radiofrequency ablation is much more popular, as well as studies evaluating the effectiveness of its alcohol destruction, but nearly all of them describe neurolytic procedures used to treat trigeminal neuralgia [11,12]. Bayer et al. reported on the use of radiofrequency ablation of SPG in patients with chronic face and head pain and found it to be sufficiently effective to warrant future studies, but no cases of trigeminal neuropathy were treated [13]. Similarly, other reports are available where SPG radiofrequency destruction is employed for various types of headache [14,15]. Peripheral trigeminal neuropathy, being relatively uncommon, is much more complex and once diagnosed – much more challenging to manage. Patients with trigeminal neuropathy are usually managed pharmacologically, using protocols that are recommended for other neuropathic pain syndromes. Therefore, the use of tricyclic antidepressants, serotonin–norepinephrine reuptake inhibitors and gabapentinoids is common. Unfortunately their effectiveness in trigeminal neuropathy is not spectacular, hence the doses used are usually significant and side effects are likely [16]. Alternative treatments, resulting in reduction of pain medication could be of great value. Recent guidelines on interventional pain management indicate that only inconclusive recommendation can be given for application of techniques of brain stimulation for facial neuropathic pain, while indication for neurodestructive procedures is given with regards to medically refractory cases of trigeminal neuralgia only [17].

In our center the neurolytic block of SPG is used in management of trigeminal neuropathy (including neuropathies resulting from malignant lesions of head and neck) and (much less frequently) cluster headache. It is also employed in atypical facial pain, although this indication is scarce. It has been performed in our center for more than 25 years and approximately 100 patients were treated with at least one block. Of so many attempted procedures, complete failure to perform the block occurred in three cases only, due to major vascular malformation noted in PPF. All of them were treatment-resistant trigeminal neuralgia cases. The number of complications was also negligible and none required specific medical intervention. It appears that percutaneous alcohol neurolysis of sphenopalatine ganglion using zygomatic approach appears to be safe and effective in the management of trigeminal neuropathy.

Conflict of interest

None declared.

Acknowledgment and financial support

None declared.

Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

REFERENCES

[1] Smith JH, Cutrer FM. Numbness matters: a clinical review of trigeminal neuropathy. Cephalalgia 2011;31:1131–44.
[2] Sluder G. Etiology, diagnosis, prognosis and treatment of sphenopalatine neuralgia. JAMA 1913;6:1201–16.
[3] Oluigbo CO, Makonnen G, Narouze S, Rezai AR. Sphenopalatine ganglion interventions: technical aspects and application. Prog Neurol Surg 2011;24:171–9.
[4] Gray H. Anatomy of the human body. Philadelphia: Lea & Febiger; 1918, New York: Bartleby.com; 2000.
[5] Varghese BT, Koshy RC. Endoscopic transnasal neurolytic sphenopalatine ganglion block for head and neck cancer pain. J Laryngol Otol 2001;115:385–7.
[6] Devoghel JC. Cluster headache and sphenopalatine block. Acta Anaesthesiol Belg 1981;32:101–7.
[7] Manahan A, Maleska M, Malone P. Sphenopalatine ganglion block relieves symptoms of trigeminal neuralgia: case report. Nebr Med J 1996;81:306–9.
[8] Walkman S. Sphenopalatine ganglion block – 80 years later. Reg Anesth 1993;18:274–6.
[9] Prasanna A, Murthy P. Combined stellate ganglion and sphenopalatine ganglion block in acute herpes infection. Clin J Pain 1993;9:135–7.
[10] Prasanna A, Murthy P. Sphenopalatine ganglion block and pain of cancer. Clin J Pain 1993;8:125–6.
[11] Kosugi S, Shiotani M, Otsuka Y, Suzuki T, Katori N, Hashiguchi S, et al. Long-term outcomes of percutaneous radiofrequency thermocoagulation of gasserian ganglion for 2nd- and multiple-division trigeminal neuralgia. Pain Pract 2015;15(3):223–8.
[12] HuangF B., Yao M, Feng Z, Guo J, Zereshki A, Leong M, et al. CT-guided percutaneous infrazygomatic radiofrequency neurolysis through foramen rotundum to treat V2 trigeminal neuralgia. Pain Med 2014;15(8): 1418–28.
[13] Bayer E, Racz GB, Miles D, Heavner J. Sphenopalatine ganglion pulsed radiofrequency treatment in 30 patients suffering from chronic face and head pain. Pain Pract 2005;5 (3):223–7.
[14] Sanders M, Zuurmond W. Efficacy of sphenopalatine ganglion blockade in 66 patients suffering from cluster headache: 12–70-month follow-up evaluation. J Neurosurg 1997;87:876–80.
[15] Narouze S, Kapural L, Casanova J, Mekhail N. Sphenopalatine ganglion radiofrequency ablation for the management of chronic cluster headache. Headache 2009;49(4):571–7.
[16] Haviv Y, Zadik Y, Sharav Y, Benoliel R. Painful traumatic trigeminal neuropathy: an open study on the pharmacotherapeutic response to stepped treatment. J Oral Facial Pain Headache 2014;28(1):52–60.
[17] Dworkin RH, O'Connor AB, Kent J, Mackey SC, Raja SN, Stacey BR, et al. Interventional management of neuropathic pain: NeuPSIG recommendations. Pain 2013; 154(11):2249–61.