Short communication

Sungho Moon, Myoungjin Ko, Sehun Kim, Hyojoong Kim and Daeseok Oh*

Superior cervical sympathetic ganglion block under ultrasound guidance promotes recovery of abducens nerve palsy caused by microvascular ischemia

https://doi.org/10.1515/sjpain-2019-0096
Received July 2, 2019; revised August 21, 2019; accepted August 28, 2019; previously published online September 21, 2019

Abstract: The abducens nerve palsy is most likely caused by microvascular issue. Spontaneous recovery of vasculopathicabducens nerve palsies was common at 3–6 months. But recovery time was longer when many risk factors were present. Several patients had residual esotropia or abduction deficit. Cervical sympathetic block has an established use in treating patients with disorders related to cranial circulatory insufficiency. It causes a significant increase in cerebral blood flow. We report a case of a 67-year-old man with acute horizontal diplopia and right periocular pain. He had been diagnosed with right abducens nerve palsy caused by microvascular ischemia. We performed ultrasound-guided superior cervical sympathetic ganglion blocks. After 4 weeks, the symptoms had been completely resolved. We introduce ultrasound-guided superior cervical sympathetic ganglion blocks for management of abducens nerve palsy caused by microvascular ischemia, which could be an effective novel method to promote recovery from diplopia.

Keywords: abducens nerve; cervical sympathetic block; superior cervical ganglion.

1 Introduction

Isolated motor nerve palsy is most likely caused by microvascular issue, defined as having at least one vascular risk factor, and among ocular motor nerves, the abducens nerve was the most frequently affected [1–3]. The diagnosis of microvascular ischemic palsy is generally presumptive and clinically based [4]. Atherosclerotic changes and transient ischemia of the microvessels supplying the motor nerves are probably important pathogenetic factors [5, 6].

Cervical sympathetic block has been established as an effective procedure in treating patients with symptoms related to sympathetic nervous system dysfunction or circulatory insufficiency of the head, neck, and upper extremities. Stellate ganglion block (SGB), a type of cervical sympathetic block, is effective for treatment of facial nerve palsy in patients with type 2 diabetes mellitus [7]. Several reports have suggested that a cervical sympathetic block causes a significant increase in cerebral blood flow [8–10].

We present a patient who experienced acute horizontal diplopia as a symptom of abducens nerve palsy caused by microvascular ischemia. We performed superior cervical sympathetic ganglion (SCSG) blocks under ultrasound guidance to promote recovery from diplopia caused by isolated microvascular ischemic sixth nerve palsy.

2 Case report

A 67-year-old man presented with acute horizontal diplopia and right periocular pain during the past 3 days. He said his symptoms appeared after several weeks of overworking, and he had no history of trauma. He had been treated for diabetes mellitus, hypertension and ischemic heart disease, and had 45 pack-years of smoking history. In addition, 20 months earlier, he was diagnosed with and treated for idiopathic right facial nerve palsy due to microvascular ischemia.

An ophthalmologic examination revealed that he had equal pupils with normal light and convergence reactions. Ocular motility showed the restriction of an abduction in the right eye in the attempted right lateral gaze, and the rest of his eye movements were full (Fig. 1). There was no facial muscle weakness. All laboratory tests were within normal limits, including the cerebrospinal fluid analysis.

***Corresponding author: Daeseok Oh,** Department of Anesthesia and Pain Medicine, Inje University Haeundae Paik Hospital, 875, Haeun-daero, Haeundae-gu, Busan, Republic of Korea, E-mail: yivangin@naver.com
Sungho Moon: Department of Anesthesia and Pain Medicine, Inje University Busan Paik Hospital, Busan, Republic of Korea
Myoungjin Ko, Sehun Kim and Hyojoong Kim: Department of Anesthesia and Pain Medicine, Inje University Haeundae Paik Hospital, Busan, Republic of Korea

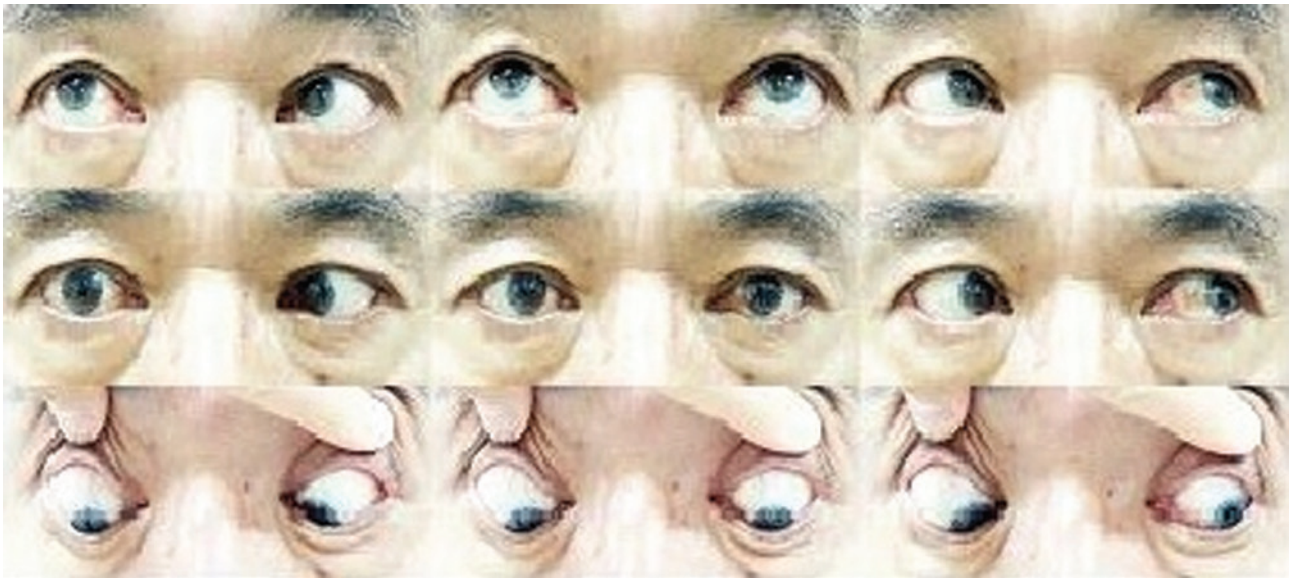

Fig. 1: Binocular movement of a 67-year-old patient. Initial presentation of right abducens nerve palsy showing the restriction of abduction in the right eye in right lateral gaze and normal ocular motility in left lateral gaze (center). Rest of the movement were full.

He was evaluated by a neuroimaging test. Brain computed tomography and magnetic resonance imaging were unremarkable, and no space-occupying lesion was reported. But magnetic resonance angiography revealed moderate stenosis in both proximal parts of the carotid artery, and there was a luminal irregularity in both of its distal parts. A neurologist suggested that we allow an observation of the progression in the right abducens nerve palsy due to microvascular ischemia. The ophthalmologist explained that about 80% of patients with idiopathic abducens nerve palsy caused by microvascular ischemia improve spontaneously in about 3–6 months. However, the patient complained of a horizontal double vision and wanted to recover the horizontal diplopia as soon as possible because he had to drive a heavy truck as part of his business.

At a week after the onset of diplopia, we considered performing an SCSG block to increase cerebral perfusion. The patient was placed in a supine position, and his head was turned approximately 45° to the left side. A model LOGIQ S7 5-MHz, multi-frequency linear ultrasound transducer (General electric healthcare, Milwaukee, WI, USA) was applied after skin preparation with betadine and draping were conducted. The transducer was transversely oriented over the right side of his neck, and the transverse process of C6 was located to the cricoid cartilage level. The transducer was moved in the cephalad direction along the neck to locate the common carotid bifurcation. After identifying the oval-shaped and hypoechoic structure between the longus capitis muscle and the starting point to branch off the common carotid bifurcation, a 23-gauge needle was introduced slowly from a lateral approach with the in-plane technique using ultrasound guidance. The needle was inserted until it approached the target site, and it was positioned immediately adjacent to the structure appearing to be the SCSG. We performed the SCSG block with 5 cc of 0.2% ropivacaine (Fig. 2), and then we identified Horner's sign (ipsilateral miosis, ptosis, and conjunctival hyperemia) on the same side after the injection. He received blocks in the same manner once every 3 or 4 days due to personal circumstances. After 1 week, the right periocular pain had subsided. Within 3 weeks,

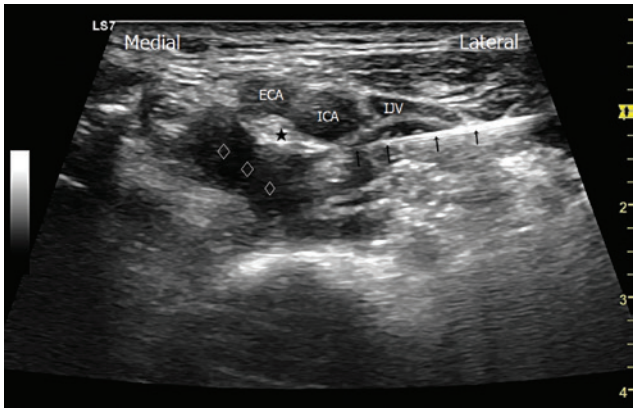

Fig. 2: Ultrasound image of spreading local anesthetic (diamond) below superior cervical sympathetic ganglion (asterisk). External carotid artery and internal carotid artery are just starting to branch off the common carotid artery. The black arrows indicate the needle placement. ECA, external carotid artery; ICA, internal carotid artery; IJV, internal jugular vein.

the diplopia had been reduced considerably, but it persisted in the extreme right gaze, and the abduction motility in the right eye was not completely recovered. At the 4-week follow-up, it was found that the residual diplopia had been completely resolved. He had been given nine sessions of the SCSG block for 4 weeks total. There were no complications of nerve injury, hematoma, infection related to the procedures, or toxicity of local anesthetics. One time of transient change in voice was observed.

3 Discussion

There has yet to be a complete elucidation of the pathophysiology of microvascular ischemic ocular motor nerve palsy. The suggested mechanism involves thickening and hyalinization with luminal narrowing of the nutrients vessels that provide the blood supply to peripheral nerves, which are referred to as vasa nervorum [5]. These regularly arise from adjacent arteries, which results in ischemic demyelination of a portion of the nerve [5, 6]. That is, transient decreased perfusion in the vascular border zones of the nerve may play a role in microvascular ischemic palsy [5]. Advanced age, hypertension, diabetes mellitus, hyperlipidemia, tobacco use, or a combination of these may be vascular risk factors for abducens nerve palsy-revealed ischemic lesions in the course of six nerves with normal neuroimaging [4]. Our patient also had multiple vascular risk factors including advanced age, hypertension, diabetes mellitus and a history of heavy smoking. Angiography identified artherosclerotic changes in cerebral vessels.

The abducens nerve arises from abducens nucleus in pons, near the midline and beneath the upper part of the floor of the fourth ventricle [11]. It emerges in a groove between the pons and the pyramidal and runs upward and laterally before bending forward over the apex of the petrous temporal bone [11]. It then traverses the cavernous sinus and reaches the orbit through the superior orbital fissure and it supplies the lateral rectus muscle, which rotates eye outward [11]. It can be affected anywhere along its course. The abducens nerve is mainly supplied by the internal carotid artery, basilar artery, and their branches [12]. Especially, the perfusion in the internal carotid artery is related to both the intracranial and extracranial supplies of the abducens nerve and opthalmic artery [12]. The ophthalmic artery, which is a branch of internal carotid artery, supplies the intraorbital segment of the abducens nerve [12].

There has been no treatment trials evaluating interventions directed toward improving the recovery of microvascular ischemic nerve palsy. Sanders et al. [13] reviewed the charts of all patients diagnosed with vasculopathic sixth nerve palsy, and they reported that 86% of patients experienced spontaneous complete resolution of their diplopia, and 14% had incomplete resolution. Generally, the area of ischemic demyelination subsequently undergoes remyelination over time, accounting for clinical recovery [5]. Therefore, the treatment of vasculopathic six nerve palsy is supportive therapy, including eye patching and a prism to relieve the patient's symptom of diplopia until spontaneous recovery [6, 11]. No other medical therapies have been proven to be of benefit. However, Jung and Kim [4] suggested that, when many risk factors were present, recovery time in patients with ischemic ocular motor nerve palsy was longer. If the palsy became chronic, extraocular muscle surgery was considered as a possible treatment option [14].

The cerebral vasculature is densely supplied with sympathetic nerve fibers, mainly originating from the SCSG [15]. The SCSG is the uppermost part of the cervical sympathetic chain, and it provides sympathetic innervation to the face and head by its vascular filaments supplied to the internal and external carotid arteries, and these filaments form a plexus around the arteries [16]. Cervical sympathetic block has been applied to treat diseases affecting the intracranial and extracranial blood supply, although its effect on perfusion of these vascular systems is not completely clear. A study that used blood flow velocity suggested that after a SGB, there was significantly increased cerebral blood flow [10]. Also, Kang et al. [9] demonstrated by magnetic resonance angiography that after an SGB in healthy volunteers there were significant changes in the extracranial vessels and the opthalmic artery, which originates from the internal carotid artery. Iwama [17] suggested the manner by which SGB increases cerebral blood flow is that local anesthetics reached the SCSG, and SGB without SCSG block does not influence cerebral circulation. That is, the SCSG block may produce a more successful sympathetic blockade to the head with less success of a sympathetic blockade to the upper extremities, and this can be more advantageous than the SGB for improving cerebral perfusion using the same dosage of local anesthetics. Treggiari et al. [18] applied SCSG blocks to improve cerebral perfusion in patients with aneurysmal subarachnoid hemorrhage, and they suggested that it is effective in improving distal cerebral perfusion and increasing cerebral circulation time as a consequence of the decrease in peripheral resistance although a change in caliber of major vessels was not established.

The SCSG is 3–5 cm in length and is located anterior to the longus capitis muscle. It lies on the transverse process

of the C2–C4 vertebrae [16]. Wisco et al. [19] suggested that the common carotid artery bifurcation is a good landmark for localizing the SCSG block using a pseudodensity heat map. The ultrasound-guided technique for the SCSG block proved to be highly accurate in human cadavers, and it can avoid radiation exposure and enables identification of the potentially hazardous structures in the needle path [20]. We considered using a cervical sympathetic block at the level of the SCSG in order to improve the effectiveness of blood perfusion to the abducens nerve, although no comparative study has been done examining the differences for cerebral circulation between an SCSG block and an SGB with same dosage of local anesthetics. We could identify a landmark for SCSG easily using ultrasound imaging. And we identified the development of Horner's sign to evaluate whether the cervical sympathetic block was well established.

According to our case, we suggest that cervical sympathetic blocks at the level of the SCSG could represent a novel method by which to promote the recovery of diplopia due to abducens nerve palsy. We think that the recovery of ischemic demyelination of a portion of the nerve is greatly assisted by an improvement in the cerebral blood flow. Further controlled studies should be conducted comparing the effects of this novel approach.

Authors' statements
Research funding: None declared.
Conflict of interest: None.
Informed consent: Written consent for submission of this case report to the journal was obtained from the patient.
Ethical approval: Not applicable.

References

[1] Park UC, Kim SJ, Hwang JM, Yu YS. Clinical features and natural history of acquired third, fourth, and sixth cranial nerve palsy. Eye (Lond) 2008;22:691–6.
[2] Tiffin PA, MacEwen CJ, Craig EA, Clayton G. Acquired palsy of the oculomotor, trochlear and abducens nerves. Eye (Lond) 1996;10(Pt 3):377–84.
[3] Richards BW, Jones Jr. FR, Younge BR. Causes and prognosis in 4,278 cases of paralysis of the oculomotor, trochlear, and abducens cranial nerves. Am J Ophthalmol 1992;113:489–96.
[4] Jung JS, Kim DH. Risk factors and prognosis of isolated ischemic third, fourth, or sixth cranial nerve palsies in the Korean population. J Neuroophthalmol 2015;35:37–40.
[5] Asbury AK, Aldredge H, Hershberg R, Fisher CM. Oculomotor palsy in diabetes mellitus: a clinico-pathological study. Brain 1970;93:555–66.
[6] Kung NH, Van Stavern GP. Isolated ocular motor nerve palsies. Semin Neurol 2015;35:539–48.
[7] Luo G, He J, Wu T, Huang Y, Miao Z, Zhao Z, Wang X, Wang Y. The therapeutic effect of stellate ganglion block on facial nerve palsy in patients with type 2 diabetes mellitus. Eur Neurol 2015;74:112–7.
[8] Kang CK, Oh ST, Chung RK, Lee H, Park CA, Kim YB, Yoo JH, Kim DY, Cho DY. Effect of stellate ganglion block on the cerebrovascular system: magnetic resonance angiography study. Anesthesiology 2010;113:936–44.
[9] Gupta MM, Bithal PK, Dash HH, Chaturvedi A, Mahajan RP. Effects of stellate ganglion block on cerebral haemodynamics as assessed by transcranial Doppler ultrasonography. Br J Anaesth 2005;95:669–73.
[10] Nitahara K, Dan K. Blood flow velocity changes in carotid and vertebral arteries with stellate ganglion block: measurement by magnetic resonance imaging using a direct bolus tracking method. Reg Anesth Pain Med 1998;23:600–4.
[11] O'Donnell TJ, Buckley EG. Sixth nerve palsy. Compr Ophthalmol Update 2006;7:215–21; discussion 223–4.
[12] Hendrix P, Griessenauer CJ, Foreman P, Shoja MM, Loukas M, Tubbs RS. Arterial supply of the upper cranial nerves: a comprehensive review. Clin Anat 2014;27:1159–66.
[13] Sanders SK, Kawasaki A, Purvin VA. Long-term prognosis in patients with vasculopathic sixth nerve palsy. Am J Ophthalmol 2002;134:81–4.
[14] Hsu CS, Closmann JJ, Baus MR. Idiopathic unilateral cranial nerve VI palsy: a case report and review of the literature. J Oral Maxillofac Surg 2008;66:1282–6.
[15] Hamel E. Perivascular nerves and the regulation of cerebrovascular tone. J Appl Physiol (1985) 2006;100:1059–64.
[16] Elias M. Cervical sympathetic and stellate ganglion blocks. Pain Physician 2000;3:294–304.
[17] Iwama H. A study of cerebral circulation following cervical sympathetic ganglion block. Masui 1992;41:1250–9.
[18] Treggiari MM, Romand JA, Martin JB, Reverdin A, Rufenacht DA, de Tribolet N. Cervical sympathetic block to reverse delayed ischemic neurological deficits after aneurysmal subarachnoid hemorrhage. Stroke 2003;34:961–7.
[19] Wisco JJ, Stark ME, Safir I, Rahman S. A heat map of superior cervical ganglion location relative to the common carotid artery bifurcation. Anesth Analg 2012;114:462–5.
[20] Siegenthaler A, Haug M, Eichenberger U, Suter MR, Moriggl B. Block of the superior cervical ganglion, description of a novel ultrasound-guided technique in human cadavers. Pain Med 2013;14:646–9.