

Causal Relation between Nerve Compression and Migraine Symptoms and the Therapeutic Role of Surgical Decompression

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Background: Nerve decompression has been recently described as a therapy for migraine headaches. Multiple studies have demonstrated significant symptomatic relief or complete resolution of migraine symptoms in patients with surgical decompression. However, there is no study describing a causal relation between migraine headaches and nerve compression and resolution of symptoms with tumor removal and nerve decompression.

Methods: We were presented with a biological example of compression neuropathy causing migraine headaches due to greater occipital nerve compression by a lipoma from a remote head trauma. Included is a literature review of nerve decompression therapy for migraine.

Results: Migraine symptoms were completely resolved on removal of the mass and nerve decompression. The patient has not required any migraine medications since the surgery.

Conclusions: This case serves as a biological example to validate the true causal relationship between greater occipital nerve compression and migraine headaches. (*Plast Reconstr Surg Glob Open* 2015;3:e395; doi: 10.1097/GOX.0000000000000345; Published online 11 May 2015.)

Migraine headache affects over 35 million Americans and causes \$16 billion loss in productivity per year in the United States.¹⁻³ It generally manifests under the age of 35, and it is more prevalent in women, with a cumulative risk of 43% in women and 18% in men.^{1,2} Migraine has traditionally been perceived to be caused by neuronal dysfunction, and pharmacological therapy has been the mainstay of the migraine treatment for a

long period of time.⁴ However, extracranial trigger sites have been recently identified and multiple studies have demonstrated significant symptomatic improvement with surgical decompression of such triggers. The major peripheral trigger sites include frontal trigger, temporal trigger, occipital trigger, and nasoseptal trigger in the setting of septal deviation or turbinate hypertrophy. The trigger sites possibly exist as a result of compression of the supra-orbital and supratrochlear nerves (frontal trigger), zygomaticotemporal nerve (temporal trigger), and greater occipital nerve (occipital trigger).⁵ Chemical denervation with Botox or diagnostic peripheral nerve block is used to identify the trigger sites that would respond to surgical decompression. Despite its success, it is a relatively new approach for migraine treatment with emerging data in the literature.⁶⁻¹³ Even though these studies have established the role of surgical decompression in migraine treat-

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ment, there is no description of migraine symptoms due to physical compression of these trigger sites and subsequent effect of tumor removal and surgical decompression on migraine symptoms. Such an example could elucidate a “cause and effect” relationship between migraine symptoms, and compression of the trigger sites would further validate the role of peripheral trigger sites in migraine. We present a patient with history of chronic migraine symptoms after developing a lipoma from trauma to the back of his head resulting in compression of greater occipital nerve. His symptoms were completely resolved after removal of the mass and greater occipital nerve decompression.

INDEX PATIENT

The patient is a 51-year-old man who was seen in our clinic with an occipital lipoma that was being followed with serial imaging. Interestingly, the patient had a history of chronic migraine, which manifested soon after he was struck in the back of his head in 1985. The patient reported a hematoma from the injury, which may have developed into a lipoma over a period of time. Shortly after his injury, he started having severe headaches in the occipital area, radiating to his frontal area, with aura of “prisms” before the onset of his headaches. He also reported photophobia with improvement of his headaches on moving to a dark room. His symptoms were responsive to sumatriptan, and over past few years, he had been requiring abortive pharmacological therapy several times a week. Because of worsening severity and frequency of headaches and interval increase in the occipital lipoma on follow-up magnetic resonance imaging, he was referred to plastic surgery for consultation. After his clinic visit, he decided to undergo surgical resection of the occipital lipoma in quest of both removing the mass and curing his headache. Intraoperatively, he was found to have a 6cm lipoma, which was compressing the right greater occipital nerve (Fig. 1). After complete removal of the lipoma (Fig. 2), the right greater occipital nerve was released along its course of the trapezius fascia and along the occipital region (Fig. 3). The patient did not require any narcotics perioperatively and was subsequently discharged from the hospital without any complications. On his 4-week postoperative clinic visit, his headache symptoms had completely resolved. He had not required sumatriptan or any other medication for headache since surgery, which was significantly different from his preoperative condition. On a follow-up call 6 weeks postoperatively, he continued to be completely asymptomatic with regard to his migraine headaches.

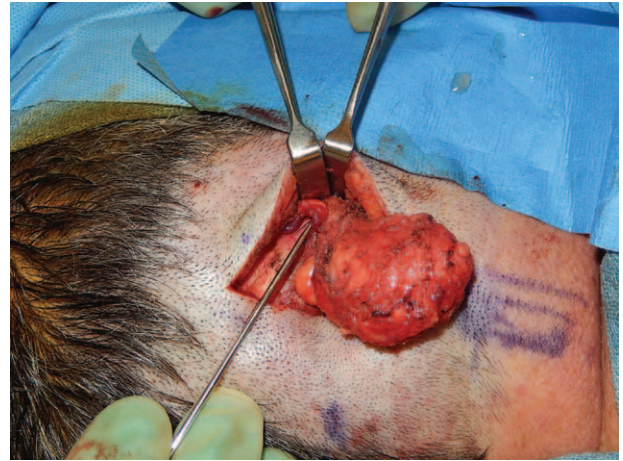


Fig. 1. Compression of greater occipital nerve from the lipoma.



Fig. 2. Dissected occipital nerve with lipoma almost removed.

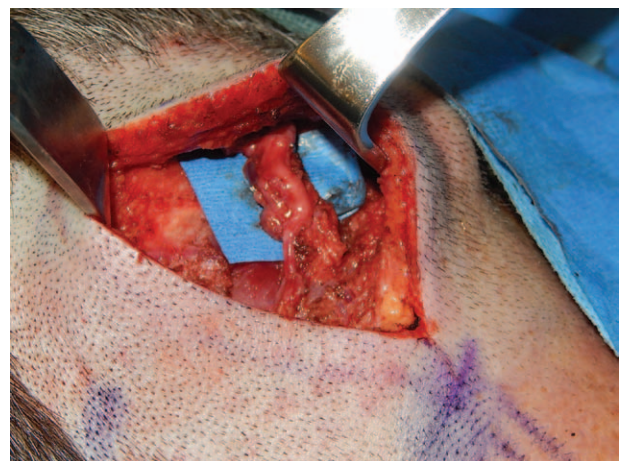


Fig. 3. Decompressed greater occipital nerve.

DISCUSSION

Surgical decompression for migraine treatment was a serendipitous finding with reports of symptomatic improvement in patients undergoing corruga-

Table 1. Preinjury, Preoperative, and Postoperative Comparison of Migraine Symptoms of the Index Patient

Migraine Headache	Preinjury	Greater Occipital Nerve Compression from Lipoma	Post Nerve Decompression and Removal of Lipoma
Location	Absent	Occipital	Absent
Nature	Absent	Pulsatile	Absent
Severity	Absent	Moderate to extremely severe	Absent
Frequency	Absent	2–3 times per week	Absent
Progression	Absent	Worsening	Absent
Aura	Absent	Present (visual)	Absent
Photophobia	Absent	Present	Present
Sumatriptan/abortive therapy	Absent	2–3 times per week	Absent
Symptomatic relief with sumatriptan	NA	Yes	NA

NA, not applicable.

tors supercilli muscle resection as a part of cosmetic browlift procedure.⁶ Guyuron et al⁷ reported complete elimination or significant improvement in more than 95% (21 out of 22) of migraine patients who underwent nerve decompression in response to the trigger points identified by preoperative Botox injection. In 2009, Guyuron et al⁸ published a disciplined randomized controlled trial with sham surgery or placebo-controlled trial. Although 84% of patients (41 out of 49) reported migraine elimination or significant improvement with nerve decompression, only 58% of patients (15 out of 26) in the sham surgery group experienced improved symptoms ($P < 0.05$). The findings of Guyuron et al⁸ were corroborated by other investigators who reported similar symptomatic improvement with decompression.^{9,10} Dirnberger and Becker⁹ reported 68% (41 out of 60) improvement in migraine headaches with surgical decompression, whereas Janis et al¹⁰ reported significant improvement or complete elimination of symptoms in 79% of patients (19 out of 24) with decompression of peripheral trigger points guided by preoperative Botox injection.

The uniqueness of our study lies in the natural course of the disease progression of the patient. Since his migraine symptoms appeared only after development of hematoma secondary to the trauma, it indicates the causal relationship between the nerve compression and migraine headaches. Over a period of time, his hematoma could have subsequently turned into lipoma, which was slowly increasing in size as indicated by serial imaging. This is consistent with the increasing severity and frequency of his headaches and the need for sumatriptan and abortive therapy. Finally, with the removal of the tumor and decompressing the greater occipital nerve, his migraine symptoms resolved completely. A comparison of his preinjury versus preoperative versus postoperative disease characteristic is detailed in Table 1. Even though he is only 6 weeks out of surgery currently, the acute relief in his symptoms is incredible. Based on

our literature review, there has been no other study demonstrating such causal relation of migraine headaches with nerve compression and subsequent resolution with tumor removal and nerve decompression.

CONCLUSION

Surgical decompression of migraine headaches is an effective treatment modality. This case serves as a biological example to further validate the true causal relationship between greater occipital nerve compression and migraine headaches.

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REFERENCES

1. Stewart WF, Simon D, Shechter A, et al. Population variation in migraine prevalence: a meta-analysis. *J Clin Epidemiol*. 1995;48:269–280.
2. Stewart WF, Wood C, Reed ML, et al; AMPP Advisory Group. Cumulative lifetime migraine incidence in women and men. *Cephalalgia* 2008;28:1170–1178.
3. Goldberg LD. The cost of migraine and its treatment. *Am J Manag Care* 2005;11(2 Suppl):S62–S67.
4. Cutrer FM. Pathophysiology of migraine. *Semin Neurol*. 2006;26:171–180.
5. Janis JE, Barker JC, Javadi C, et al. A review of current evidence in the surgical treatment of migraine headaches. *Plast Reconstr Surg*. 2014;134(4, Suppl 2):131S–141S.
6. Guyuron B, Varghai A, Michelow BJ, et al. Corrugator supercilli muscle resection and migraine headaches. *Plast Reconstr Surg*. 2000;106:429–434; discussion 435–437.
7. Guyuron B, Tucker T, Davis J. Surgical treatment of migraine headaches. *Plast Reconstr Surg*. 2002;109:2183–2189.
8. Guyuron B, Reed D, Kriegler JS, et al. A placebo-controlled surgical trial of the treatment of migraine headaches. *Plast Reconstr Surg*. 2009;124:461–468.
9. Dirnberger F, Becker K. Surgical treatment of migraine headaches by corrugator muscle resection. *Plast Reconstr Surg*. 2004;114:652–657; discussion 658–659.

10. Janis JE, Dhanik A, Howard JH. Validation of the peripheral trigger point theory of migraine headaches: single-surgeon experience using botulinum toxin and surgical decompression. *Plast Reconstr Surg*. 2011;128:123–131.
11. Guyuron B, Kriegler JS, Davis J, et al. Comprehensive surgical treatment of migraine headaches. *Plast Reconstr Surg*. 2005;115:1–9.
12. Guyuron B, Kriegler JS, Davis J, et al. Five-year outcome of surgical treatment of migraine headaches. *Plast Reconstr Surg*. 2011;127:603–608.
13. Poggi JT, Grizzell BE, Helmer SD. Confirmation of surgical decompression to relieve migraine headaches. *Plast Reconstr Surg*. 2008;122:115–122; discussion 123–124.