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Case Study

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Unusual recurrence of trigeminal neuralgia after microvascular decompression by muscle interposal

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

Giovanni Grasso[1AE], Francesco Meli[1B], Rosario Maugeri[1EF], Francesco Certo[1F], Gabriele Costantino[1F], Filippo Giambartino[2BC], Domenico G. Iacopino[1D]

[1] Neurosurgical Clinic, Department of Clinic Neurosciences, University of Palermo, Palermo, Italy
[2] Department of Anaesthesiology, University of Palermo, Palermo, Italy

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Summary

Background: Patients with trigeminal neuralgia (TN) and persistent or recurrent facial pain after microvascular decompression (MVD) typically undergo less invasive procedures in the hope of providing pain relief. However, re-operation should be considered in selected patients.

Case Report: A 48-year-old woman presented with recurrent trigeminal neuralgia (TN) 3 years following microvascular decompression (MVD). The patient underwent brain magnetic resonance angiography (MRA), which did not reveal neurovascular compression; therefore surgical re-exploration was carried out. During the operation, the fifth cranial nerve was seen without impingement from any blood vessels; however, a very firm tissue was observed and identified as the muscle fragment from the previous MVD procedure. The fifth cranial nerve was carefully separated from the muscle. Thereafter, the right SCA was dissected out from the muscle and suspended by a periosteum tape sutured to the nearby dura.

Conclusions: Our findings, along with similar cases reported in the literature, support the development of new inert materials and alternative surgical strategies that can limit TN recurrence.

key words: **trigeminal neuralgia • microvascular decompression • recurrence**

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Author's address: Giovanni Grasso, Neurosurgical Clinic, Department of Clinic Neurosciences, Policlinico Universitario,Via del Vespro129, 90100 Palermo, Italy, e-mail: ggrasso@unipa.it

Background

The concept of vascular compression of cranial nerves in the posterior fossa has developed from several lines of evidence. Over the past few years, microvascular decompression (MVD) has been shown to be effective [1–3].

To date, MVD is the first surgical treatment option for trigeminal neuralgia (TN) and other cranial nerve hyperactive dysfunctions. Although this technique has a high rate of success with respect to pain relief and long-term benefit [4], pain can recur and re-exploration may be indicated in a subgroup of patients [5]. There have been several reports of recurrent TN, some of which are related to the prosthesis used for separating the offending vessel and the nerve [6–8].

In this paper we report on a patient with pain recurrence after MVD for TN caused by a muscle fragment piercing the trigeminal nerve and encasing the offending artery.

Case Report

A 48-year-old woman presented with a long history of right TN within the ophthalmic and mandibular divisions, which failed to respond to medical management. The patient underwent brain magnetic resonance angiography (MRA), revealing neurovascular compression at the dorsal root entry zone of the right fifth cranial nerve and also underwent MVD with right SCA displacement and muscle interposition. The neuralgia resolved immediately after surgery, but recurred 3 years later with the same distribution. The pain was refractory to medical management. MRA was performed again but did not show any trigeminal vascular compression. The patient underwent percutaneous rhizotomies, performed at another centre, without effect.

Considering that most recurrences occur within 2 years following surgery, and that new arterial loop compression, regrowth of veins, or incomplete decompression at the first surgical treatment are the main causes, a decision was made to conduct surgical re-exploration.

During the operation, the fifth cranial nerve was seen without impingement from any blood vessels. However, inferiorly to the nerve and directly against it, a solid tissue was observed that was identified as the muscle fragment from the previous MVD procedure. This tissue was distorting and stretching the nerve and encasing the right SCA (Figure 1). The fifth cranial nerve was carefully separated from the muscle. Thereafter, the right SCA was dissected out from the muscle and suspended by a periosteum tape sutured to the nearby dura.

The patient had no intraoperative or postoperative complications. At 2-year follow-up the patient was pain-free without medication.

Discussion

The concept of vascular compression of cranial nerves in the posterior fossa has developed from several lines of evidence. Dandy first proposed the fifth cranial nerve compression, at its point of entry into the pons, by the superior cerebellar artery, as a possible cause of trigeminal neuralgia [9]. Subsequent reports confirmed that patients with

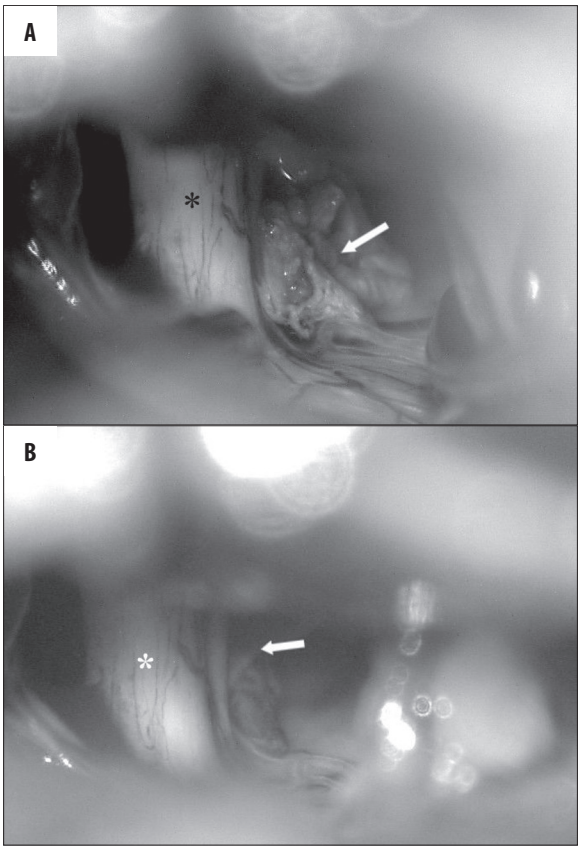

Figure 1. Intraoperative photograph at re-operation. (**A**) The image showing the right trigeminal nerve (*asterisk*) compressed by a firm tissue (*arrow*) that was identified as the muscle fragment from the previous MVD procedure. Such a tissue, was distorting and stretching the nerve and, at the same time, encasing the right SCA; (**B**) After careful partial dissection the right SCA was visualized (*arrow*).

trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia had blood vessels in close contact with the respective cranial nerve, and that separating the blood vessel from the nerve by interposing a soft implant between them (microvascular decompression) could be curative [1–3].

Pathophysiological mechanisms underlying cranial nerve hyperactive dysfunction after vascular compression have been investigated extensively and partially clarified. Briefly, it has been suggested that these clinical syndromes result from pulsatile compression by arteries at the root entry/exit zone of the cranial nerve, a junctional area between central and peripheral myelin [3]. Over the past several years, this concept has been widely accepted and has stimulated several studies addressed primarily at establishing precise patient selection criteria [10]. With the advent of magnetic resonance imaging, which, using specific three-dimensional sequences [11–13], has offered a good visualization of both cranial nerves and cerebral vessels, neurovascular compression disorders have been diagnosed with increasing frequency, thus providing additional evidence supporting MVD treatment.

To date, MVD is associated with a high incidence of pain relief and long-term success since about 70% of patients remain

pain-free and off medication for at least 10 years following the procedure [4]. Recurrence may occur in 18–30% of patients, mainly within 2 years of surgery and thereafter at a rate of 2–5% per year [4,14]. This occurrence has been attributed to several causes, including new arterial loop compression, regrowth of new veins, incomplete decompression and problems related to the interposed material [6–8,15–17]. In addition, arachnoid thickening or granulomatous severe adhesion between the nerve and the surrounding structures following the first MVD surgery has been reported [18,19]. In up to 44% of patients no factor explaining the recurrence can be identified [20,21].

In this paper we described the recurrence of TN in which autologous muscle was used as interposing material during the first operation. At the re-operation the muscle formed a very firm tissue that distorted and stretched the trigeminal nerve. It also encased the right SCA, thus transmitting the vascular pulsations into the nerve.

In our experience, the use of autologous muscle as interposing material between nerve and vessel has been shown to be safe, with a recurrence rate similar to those cases in which Teflon and other synthetic materials have been employed. Conceptually, the use of muscle arises from the idea that an autologous material should be safer and better tolerated than a synthetic prosthesis [22,23]. However, dissolution of the implant and recurrent vascular compression of the trigeminal root entry zone related to the use of resorbable materials, such as muscle, periosteum, collagen foam, or lyophilized dura, has been reported [24]. Synthetic materials, such as Teflon or Ivalon sponges have also been associated with a direct subsequent neurovascular compression by the same vessel because of a slipped prosthesis [7]. Compression of the trigeminal root entry zone, caused by the prosthesis itself or by severe adhesions, has been reported by several authors [25,26], and even indirect vascular compression caused by fairly hard implants like Ivalon has been reported [27,28]. Many other reports have also focused on adverse reactions to synthetic materials. Teflon-induced granuloma has been documented in various reports [25,29,30].

Based on these experiences, some authors have recently advocated alternative techniques such as the "hanging technique", where the offending vessel is transposed from the nerve by using strips of autologous tissue or fenestrated clips for aneurysm surgery [31,32]. This technique, used in our case and already suggested by our group for the treatment of medulla oblongata compression by vertebral artery [33,34], seems to be a useful method, especially in cases such as we report, in which recurrence of the TN can be related to compression or adhesion caused by the material used in the first MVD.

Conclusions

In this paper we report on a patient with pain recurrence after MVD for TN caused by a muscle fragment that had pierced the trigeminal nerve and encased the offending artery.

Successful long-term outcome following MVD in cranial nerve dysfunction disease depends primarily on maintaining the isolation between the nerve and the offending vessel. For this reason development of new inert materials and use of alternative surgical strategies can limit TN recurrence.

References:

1. Gardner W, Miklos M: Response of trigeminal neuralgia to decompression of sensory root; discussion of cause of trigeminal neuralgia. J Am Med Assoc, 1959; 170
2. Hongo K, Kobayashi S, Takemae T et al: [Posterior fossa microvascular decompression for hemifacial spasm and trigeminal neuralgia-some improvements on operative devices and technique]. No Shinkei Geka, 1985; 13: 1291–96
3. Jannetta P: Neurovascular compression in cranial nerve and systemic disease. Ann Surg, 1980; 192: 518–25
4. Barker FG II, Jannetta PJ, Bissonette DJ et al: The long-term outcome of microvascular decompression for trigeminal neuralgia. N Engl J Med, 1996; 334: 1077–83
5. Amador N, Pollock BE: Repeat posterior fossa exploration for patients with persistent or recurrent idiopathic trigeminal neuralgia. J Neurosurg, 2008; 108: 916–20
6. Fujimaki T, Hoya K, Sasaki T et al: Recurrent trigeminal neuralgia caused by an inserted prosthesis: report of two cases. Acta Neurochir (Wien), 1996; 138: 1307–10
7. Liao JJ, Cheng WC, Chang CN et al: Reoperation for recurrent trigeminal neuralgia after microvascular decompression. Surg Neurol, 1997; 47: 562–68; discussion 568–70
8. Vitali AM, Sayer FT, Honey CR: Recurrent trigeminal neuralgia secondary to Teflon felt. Acta Neurochir (Wien), 2007; 149: 719–22; discussion 722
9. Dandy W: Concerning the cause of trigeminal neuralgia. Am J Surg, 1934; 24: 445–55
10. Jannetta P: Selection criteria for the treatment of cranial rhizopathies by microvascular decompression (honored guest lecture). Clin Neurosug, 1997; 44: 69–77
11. Furuya Y, Ryu H, Uemura K: MRI of intracranial neurovascular compression. J Comput Assist Tomogr 1992; 16
12. Miller J, Acar F, Hamilton B et al: Preoperative visualization of neurovascular anatomy in trigeminal neuralgia. J Neurosurg, 2008; 108: 477–82
13. Satoh T, Omi M, Nabeshima M et al: Severity analysis of neurovascular contact in patients with trigeminal neuralgia: assessment with the inner view of the 3D MR cisternogram and angiogram fusion imaging. AJNR Am J Neuroradiol, 2009; 30: 603–7
14. Olson S, Atkinson L, Weidmann M: Microvascular decompression for trigeminal neuralgia: recurrences and complications. J Clin Neurosci, 2005; 12: 787–89
15. Lee SH, Levy EI, Scarrow AM et al: Recurrent trigeminal neuralgia attributable to veins after microvascular decompression. Neurosurgery, 2000; 46: 356–61; discussion 361–62
16. Broggi G, Ferroli P, Franzini A et al: [Microvascular decompression intervention in the treatment of trigeminal neuralgia]. Recenti Prog Med, 2001; 92: 164–68
17. Kouyialis AT, Stranjalis G, Boviatsis EJ et al: Recurrence of trigeminal neuralgia due to an acquired arachnoid cyst. J Clin Neurosci, 2008
18. Kondo A: Follow-up results of microvascular decompression in trigeminal neuralgia and hemifacial spasm. Neurosurgery, 1997; 40: 46–51; discussion 51–52
19. Ishikawa M, Nishi S, Aoki T et al: Operative findings in cases of trigeminal neuralgia without vascular compression: proposal of a different mechanism. J Clin Neurosci, 2002; 9: 200–4
20. Rath SA, Klein HJ, Richter HP: Findings and long-term results of subsequent operations after failed microvascular decompression for trigeminal neuralgia. Neurosurgery, 1996; 39: 933–38; discussion 938–40
21. Meaney JF, Eldridge PR, Dunn LT et al: Demonstration of neurovascular compression in trigeminal neuralgia with magnetic resonance imaging. Comparison with surgical findings in 52 consecutive operative cases. J Neurosurg, 1995; 83: 799–805
22. Tomasello F, Alafaci C, Angileri FF et al: Clinical presentation of trigeminal neuralgia and the rationale of microvascular decompression. Neurol Sci, 2008; 29(Suppl.1): S191–95
23. Alafaci C, Salpietro FM, Montemagno G et al: Spasmodic torticollis due to neurovascular compression of the spinal accessory nerve by the anteroinferior cerebellar artery: case report. Neurosurgery, 2000; 47: 768–71; discussion 771–72

24. Klun B: Microvascular decompression and partial sensory rhizotomy in the treatment of trigeminal neuralgia: personal experience with 220 patients. Neurosurgery, 1992; 30: 49–52
25. Cho DY, Chang CG, Wang YC et al: Repeat operations in failed microvascular decompression for trigeminal neuralgia. Neurosurgery, 1994; 35: 665–69; discussion 669–70
26. Yamaki T, Hashi K, Niwa J et al: Results of reoperation for failed microvascular decompression. Acta Neurochir (Wien), 1992; 115: 1–7
27. Goya T, Wakisaka S, Kinoshita K: Microvascular decompression for trigeminal neuralgia with special reference to delayed recurrence. Neurol Med Chir (Tokyo), 1990; 30: 462–67
28. Jannetta PJ, Bissonette DJ: Management of the failed patient with trigeminal neuralgia. Clin Neurosurg, 1985; 32: 334–47
29. Megerian CA, Busaba NY, McKenna MJ et al: Teflon granuloma presenting as an enlarging, gadolinium enhancing, posterior fossa mass with progressive hearing loss following microvascular decompression. Am J Otol, 1995; 16: 783–86
30. Premsagar IC, Moss T, Coakham HB: Teflon-induced granuloma following treatment of trigeminal neuralgia by microvascular decompression. Report of two cases. J Neurosurg, 1997; 87: 454–57
31. Mitsos AP, Georgakoulias N, Lafazanos SA et al: The "hanging technique" of vascular transposition in microvascular decompression for trigeminal neuralgia: technical report of four cases. Neurosurg Rev, 2008; 31: 327–30
32. Attabib N, Kaufmann AM: Use of fenestrated aneurysm clips in microvascular decompression surgery. Technical note and case series. J Neurosurg, 2007; 106: 929–31
33. Tomasello F, Alafaci C, Salpietro FM et al: Bulbar compression by an ectatic vertebral artery: a novel neurovascular construct relieved by microsurgical decompression. Neurosurgery, 2005; 56: 117–24; discussion 117–24
34. Grasso G, Alafaci C, Passalacqua M et al: Landmarks for vertebral artery repositioning in bulbar compression syndrome: anatomic and microsurgical nuances. Neurosurgery, 2005; 56: 160–64; discussion 160–64