Unusual recurrence of trigeminal neuralgia after microvascular decompression by muscle interpositional

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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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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 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, peristeum, 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.

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