Microvascular Decompression for Trigeminal Neuralgia with Special Reference to Delayed Recurrence

Tomokazu GOYA, Shinichiro WAKISAKA and Kazuo KINOSHITA

Department of Neurosurgery, Miyazaki Medical College, Miyazaki

Abstract

Thirty-five patients with trigeminal neuralgia underwent microvascular decompression. Complete remission was obtained in 33 patients, while one was fair and another unchanged postoperatively. The clinical and operative findings were reviewed, analyzing the direction of vascular compression of the trigeminal nerve and the distribution of pain in the peripheral regions. There were some weak correlations between the direction of vascular compression and the distribution of pain. Neuralgia in the region of second branch of the trigeminal nerve (V2) or in the regions of V2 and third branch of the nerve (V3) was caused by compression from the ventral or ventro-rostral direction, in the region of first branch of the nerve from the ventro-caudal direction, and in the V3 region from the ventral, rostral, and dorsal directions of the nerve in general. In two patients who had had complete remission after first operation, trigeminal neuralgia recurred. They had typical intermittent painful attacks with a background of continuous dull pain or painful dysesthesia caused by Ivalon® sponges inserted between the nerve and the offending vessel. Complete remission was again obtained after removal of these sponge pieces. We would like to stress continuous dull pain or painful dysesthesia in cases of delayed recurrence as indicators for re-exploration.

Key words: trigeminal neuralgia, microvascular decompression, delayed recurrence

Introduction

Microvascular decompression (MVD) through a suboccipital retromastoid small craniectomy has recently become the usual treatment for idiopathic trigeminal neuralgia because of the elaborate operative findings established by Jannetta.4,5> Typical trigeminal neuralgia such as electric or shooting pain and atypical neuralgia such as dull pain with pressure sensation are suspected to originate from vascular compression of the trigeminal nerve at its root entry zone.9,10

In our service, 35 patients of trigeminal neuralgia underwent MVD (36 operations). We report the results of clinical analysis in these cases. We also present unusual clinical courses in two recurrent cases, both of which were successfully treated by a second operation.

Clinical Analysis

Thirty-five patients with trigeminal neuralgia underwent MVD (36 operations) between 1982 and 1988. Twelve patients were male and 23 female, and their ages ranged from 31 to 75 years (mean, 57.8 years). The affected side was the right in 15 cases and the left in 20. Trigeminal neuralgia concomitant with hemifacial spasm was observed in two cases. The period from onset of the pain to operation ranged from 3 months to 25 years and averaged 6 years.

I. Distribution of trigeminal neuralgia

Nine patients had trigeminal neuralgia in the second division (V2) only, and four in the third division (V3) only. In eight cases, the neuralgia expanded from V2 toward V3, and in five, from V3 toward V2. In one case only, the neuralgia expanded from V3 to V2 and V1, and in another, from V1 toward V2 and V3. In three cases, the neuralgia expanded from V1 to V2, and in three, from V2 to V1. In one case, the neuralgia was restricted to V1. Neuralgia in the
combination of V1 and V3 was not observed. The preoperative facial sensation was normal in all patients.

II. Offending vessels and direction of vascular compression

The most frequently observed conflicting vessel was the superior cerebellar artery (SCA) (28 cases, 80%). Figure 1 shows four types of compression caused by the SCA. In type a, the trigeminal nerve was compressed from the superior aspect, and in type b, from the ventral aspect. In type c, one branch of the SCA looped around the nerve and compressed it at the inferior ventral portion. In type d, the offending vessel was located at the dorsal aspect of the nerve. The former two types were the most frequently observed, and the latter two were rare types.

Another less frequent conflicting vessel was the anterior inferior cerebellar artery (AICA) (3 cases, 8.6%). In two cases, the rostral loop of the AICA simply compressed the nerve at the inferior aspect. In one peculiar case, the AICA penetrated the nerve between the portio major and minor from ventral to dorsal.

In three cases, the trigeminal nerve was impinged upon by both the SCA and AICA from both the rostral and caudal directions. In these cases, the vessel causing the neuralgia was difficult to identify. Therefore, all possible vessels were carefully separated from the trigeminal nerve.

Four cases had venous compression of the nerve, as illustrated in Fig. 1. The findings were similar for all these cases. The so-called pontine vein or satellite trigeminal vein directly entered the dura in the close vicinity of Meckel’s cave and, on the way to the dura, compressed the nerve forming an obvious indentation in the nerve. At surgery, these veins were also cauterized and cut.

III. Relationships between direction of vascular compression and distribution of pain

The direction of vascular compression confirmed during operations was compared with the distribution of pain in the 35 cases, but one in which the direction was not clearly determined (Fig. 2). In cases of V1 pain, vascular compression was from the inferior-ventral direction and involved narrow region. Vascular compression in cases of V2 pain was from the superior-ventral direction, and for V3 pain from the general direction of the ventral, superior, and dorsal aspects of the nerve. In the most frequent cases with the combination of V2 and V3 pain, the direction of the vascular compression was from the superior-ventral aspect of the nerve, as indicated by the superimposed range of semicircles of V2 and V3 in Fig. 2. From these correlations, it is possible that the distribution of pain suggests the direction of vascular compression and the offending artery.

IV. Results and complications of MVD

The follow-up periods ranged from 6 months to 6 years. Complete remission was obtained in 33 (94%) of 35 cases. Pain reduced by half in one case and another was unchanged. In two cases, the pain recurred after complete remission by MVD and again completely disappeared after a second operation. These two cases will be discussed later.

As a complication of MVD, hearing loss developed in the initial three cases. In one case, cerebrospinal fluid leakage into the middle ear cavity occurred after insufficient packing of the opened mastoid air cells. No such complication has been experienced since this event. The petrosal vein was sacrificed in some cases without problems.
V. Delayed recurrence

In our two recurrent cases, interesting clinical courses and findings were observed.

**Case 1:** A 65-year-old female presented with intermittent pain in the right cheek, especially exacerbated by mouth opening, since August, 1984. This paroxysmal pain continued for 1 or 2 minutes only and there was no pain during the interphase. This pain gradually expanded to the right upper lip. In August, 1985, she had a nerve block at the infraorbital foramen and the pain was resolved until recurrence after 8 months. As severe intermittent pain occurred again, MVD was performed in March, 1987. Two branches of the SCA, together compressing the trigeminal nerve from above, were separated from the nerve by inserting a small piece of Ivalon<sup>®</sup> sponge between the brainstem and these branches (Fig. 3). Although this was sufficient for decompression, another piece of sponge was also placed between the proximal portion of the SCA and the nerve in the vicinity of the dura, as the possibility of compression of the nerve by the caudal loop of the SCA in the future was considered. The pain completely disappeared just after the operation.

Seven months after the operation, however, paroxysmal pain suddenly recurred in the right upper lip while taking a meal. Four months later, paroxysmal sharp pain had begun to superimpose on dull throbbing pain persisting all day long. As carbamazepine was ineffective for the pain, she was readmitted to our service. At the second operation, performed just 1 year after the first MVD, one of the two sponges previously inserted was tightly conglutinated to the nerve and, furthermore, compressed and distorted it downward at the petrous ridge. This Ivalon sponge was carefully separated and removed from the nerve. The neuralgia completely disappeared just after the operation and her facial sensation remained normal.

**Case 2:** A 61-year-old female noticed tingling...
sharp pain periodically in the left cheek and oral cavity since February, 1985. The paroxysms became more frequent and severe. The pain had always been typical tic douloureux, including the presence of trigger area around the left nasal labial angle. Five months after the onset of pain, the first MVD was performed in another hospital. The pain completely disappeared after this operation.

One year and 2 months later, the tingling pain recurred intermittently on the left side of the nose and cheek. Continuous dull pain gradually developed, and then, as in Case 1, paroxysmal sharp pain occurred with the background continuous dull pain. In February, 1988, 3 years after the first MVD, she was admitted to our service. The neurological examination was entirely normal. There was no evidence of sensory deficit in the distribution of the left trigeminal nerve. A skull x-ray film (Towne's projection) revealed a bone defect caused by the previous craniectomy and a faint calcified lesion on the petrous ridge (Fig. 4 left). This calcification was confirmed by a computed tomographic (CT) scan (Fig. 4 right). There were no symptoms suggesting the presence of a space-occupying lesion. At the second operation, one small artery located on the dorsal surface of the trigeminal nerve had been well separated from the nerve by an Ivalon sponge placed between this artery and the nerve. Therefore, the entry zone of the trigeminal nerve could not be directly observed because of the relatively large mass of sponge. After careful separation from the nerve and complete removal of this sponge, the trigeminal nerve was observed to be depressed toward the prepontine cistern and had become flat. Histologically, there were fine calcified flecks within this Ivalon sponge, as indicated by x-ray. At the ventral aspect of the root entry zone of the trigeminal nerve, a branch of the SCA was compressing the nerve. Presumably, this arterial compression had been overlooked at the first operation. This branch of SCA was pushed ventrally toward the prepontine cistern and a small Ivalon sponge was inserted between the brainstem and the artery. The neuralgia was completely relieved without neurological deficit.

In these two recurrent cases, Ivalon sponges placed as separators had become tightly bound to the nerve and compressed it, presumably causing the recurrence of trigeminal neuralgia.

Discussion

I. Fiber arrangement in the cisternal portion of the trigeminal nerve

There is some controversy concerning the fiber arrangement in the cisternal portion of the trigeminal nerve. Afferent fibers from three peripheral branches of the trigeminal nerve are reported to run in specific constant portions of the nerve bundle at the pontine cistern. Other reports suggest that, although there is some regular distribution, fibers from each peripheral branch intermingle within the nerve in this portion. In cases of partial resection of the nerve, facial hypesthesia expands from V3 to V2 according to the degree of incision. For neuralgia
As mentioned above, there is no consensus about the fiber arrangement within the bundle of the trigeminal nerve. Therefore, the AICA was dislocated distally toward Meckel's cave with a small sponge and muscle pieces. The neuralgia disappeared after this procedure.

III. Venous compression

Idiopathic trigeminal neuralgia is thought to be caused by arterial compression, but indentation of the trigeminal nerve by simultaneous venous compression associated with arterial compression can be observed. In 1981, Jannetta \(^4\) reported that there were 57 venous abnormalities and 96 complex abnormalities of both arteries and veins, out of a total 411 patients undergoing MVD for trigeminal neuralgia. Apfelbaum \(^5\) also confirmed venous compression alone in 12 of 55 patients undergone MVD. This suggests that the occurrence of venous compression may be quite high. The lateral pontine vein and the transverse pontine vein, or so-called aberrant trigeminal vein, run from the pons to the dura in the vicinity of Meckel's cave, and join the petrosal vein or enter the dura directly. Before entrance, these veins sometimes compress the nerve and form an indentation mostly at the peripheral side near Meckel's cave. In our experience, however, importance of venous compression for the neuralgia and its characteristics, compared to arterial compression, was difficult to assess: Continuous dull pain in the territory of V2 was induced by venous compression alone and this pain was not a typical trigeminal neuralgia.

IV. Recurrence of pain after MVD

If pain relief is not obtained immediately after the operation, the cause may be overlooking of offending vessels or inadequate implants. A common cause of recurrence within 6 months after the operation is said to be recollateralization of intrinsic pontine veins. Late recurrence appears to be most commonly due to the continuous elongation of arteries which enter the root entry zone of the nerve or displacement of the implant from the nerve.\(^3\)\(^,\)\(^9\) In our Case 1, the neuralgia recurred because one of two Ivalon sponges, inserted between the nerve and the offending vessels, had adhered to the nerve and compressed it. Continuous painful dysesthesia or dull pain, with a different nature from the pain caused by arterial compression, developed due to the adhesion. In addition to this continuous compression, the so-called "secondary missile phenomenon"\(^1\)\(^,\)\(^2\) occurred. Therefore, typical trigeminal neuralgia was superimposed on the background continuous dull pain. Interestingly, there was no sensory disturbance on the face, although compression of the nerve by an Ivalon sponge was apparent.
In Case 2, the offending artery located on the dorsal surface of the trigeminal nerve was well decompressed by insertion of an Ivalon sponge between this vessel and the nerve. There was no pain for more than 1 year. However, the Ivalon sponge gradually compressed the nerve and displaced it ventrally. Finally, this nerve contacted the caudal loop of the SCA located ventrally to the nerve and suffered pulsatile compression. It was supposed that typical trigeminal neuralgia recurred due to this arterial compression, in addition to the continuous compression caused by the adhering Ivalon sponge.

In these two cases, a common characteristic of the pain on recurrence was that typical intermittent trigeminal neuralgia was superimposed on the background continuous dull pain. Therefore, the following precautions are important to prevent recurrence: The size of the separator should be the minimum essential. The separator should not be introduced between the nerve and the offending vessel and should not contact the nerve. If late recurrence of trigeminal neuralgia occurs in cases who had complete remission for a while after MVD, we should consider such causes as occurred in our cases.

References

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Address reprint requests to: T. Goya, M.D., Department of Neurosurgery, Miyazaki Medical College, 5200 Kihara, Kiyotake-cho, Miyazaki-gun, Miyazaki 889-16, Japan.