

Original Article

Main causes of trigeminal neuralgia and corresponding surgical strategies

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Abstract: Background: The clinical data of patients with trigeminal neuralgia who received treatment were retrospectively analyzed, and the surgical strategy for cases without vascular compression was investigated. Methods: Clinical data from 98 patients with trigeminal neuralgia who underwent MVD surgery were retrospectively analyzed. All patients underwent preoperative examinations of 3D-TOF-MRA and 3D-CISS to identify the offending vessel and its site of compression of the trigeminal nerve. Results: In 21 cases (21.5%), no direct vascular compression of the trigeminal nerve was found intraoperatively. Of these cases, 17 showed thickened arachnoid adhere, the trigeminal nerve traveled at an angle and twisted, and five showed vascular adhesions with the trigeminal nerve at an angle by the arachnoid. An additional three cases showed unknown sinus vein compression on the dura of the outer arachnoid of the trigeminal nerve at the lower edge of Meckel's cave. Finally, one case showed suprameatal nodular hypertrophy, with direct compression of the trigeminal nerve by the bony structure together with the unknown vessel. The patients in each group were followed for 3-24 months, and one case showed recurrence during follow-up, yielding a recurrence rate of 1.0%. Conclusion: Vascular compression on the trigeminal nerve is the main cause of trigeminal neuralgia, but vascular compression may not be found during surgery. In this case, complete isolation of the cisternal segment in the trigeminal nerve and restoration of the normal traveling morphology of the trigeminal nerve can effectively treat trigeminal neuralgia.

Keywords: Trigeminal neuralgia, surgical strategies, microvascular decompression

Introduction

In the early 20th century, Cushing studied the etiology of trigeminal neuralgia and proposed the hypothesis of "mechanical compression", which Dandy further improved by proposing the "vascular compression theory" in 1932 [1] that hypothesized the presence of arterial compression on the root entry zone (REZ) at the root of the trigeminal nerve, i.e., nerve fibers were exposed, causing the onset of trigeminal neuralgia. Medical developments have confirmed that trigeminal neuralgia is not confined to arterial compression of the root of the trigeminal nerve. In clinical practice, we find various types of compression of the offending vessels, including arterial compression, venous compression, or evenovascular compression [2-5]. The causes of pain are complex and diverse, but few studies of cases without vascular compression

have been reported. This study summarized and analyzed a number of special circumstances other than vascular compression that cause trigeminal neuralgia.

Materials and methods

General information

Clinical data from 98 patients with primary trigeminal neuralgia who underwent surgical microvascular decompression (MVD) at the Xuanwu hospital from January 2014 to December 2015 were included in this study. These patients included 46 males and 52 females, age 39-88 years, with a disease history of 3-20 years. The trigeminal neuralgia was right-sided in 45 cases, left-sided in 51 cases, and bilateral in 2 cases. Temporomandibular arthralgias and glossopharyngeal

neuralgia were excluded in all cases. Among all patients, five patients had pain at the V1, 2, and 3 zones, 15 patients had pain at the V1 and 2 zones, 41 patients had pain at the V2 and 3 zones, 19 patients had pain at the V2 zone, and 18 patients had pain at the V3 zone. All patients in this study received effective treatment with oral carbamazepine, while 48 patients underwent other treatments, including blockage of the peripheral branches of the trigeminal nerve branch, radio frequency thermocoagulation, Chinese traditional medicine, acupuncture, thread embedding, and gamma knife treatment, but the side effects were not tolerated or the efficacy was not satisfactory.

Imaging study

All patients underwent CT or MRI examination to exclude secondary trigeminal neuralgia caused by space occupying lesions such as cholesteatoma of the cerebellopontine angle, meningiomas, and schwannomas. Three-dimensional time of flight magnetic resonance angiography (3D-TOF-MRA) and three-dimensional nuclear magnetic resonance of constructive interference in steady state (3D-CISS) were performed in all cases.

Surgical treatment

All surgeries were performed by the same surgeon. Electromyograms (EMG) for the orbicularis oculi muscle and orbicularis oris muscle recorded using a subcutaneous electrode were performed intraoperatively to monitor the facial nerve, and the function of the brainstem was monitored by brain stem auditory evoked potential (BAEP). After successful anesthesia intubation, the patient was put in the lateral position, with the shoulders loping downward. A curved incision was created in the posterior suboccipital sigmoid sinus of the lesion side, up to 1.5 cm above the linea nuchae superior and down to 1 cm below the level of the tip of the mastoid sinuses. After conventional disinfection and draping, the skin and subcutaneous tissues were cut in layers. With the intraoperative infusion of 250 ml of 20% mannitol, the occipital bone was drilled to remove approximately 3 cm × 2 cm of the squama occipitalis, front to the sigmoid sinus and up to the transverse sinus. After releasing the cerebrospinal fluid, sharp separation of the arachnoid around the trigeminal nerve was performed to expose

the REZ of the trigeminal nerve. If vascular compression of the REZ of the trigeminal nerve was clearly identified, a Teflon cottonpad was placed after the vessel was properly disassociated. If a thickened arachnoid adhesion was found between the trigeminal nerve and the tentorium and the trigeminal nerve was traveling at an angle and showed twisting, the arachnoid was cut to fully release and disassociate the trigeminal nerve. The arachnoid around Meckel's cave was cut in 360° for exploration. If compression of an unknown sinus vein at the outer arachnoid dura on the trigeminal nerve was observed, the trigeminal nerve was disassociated with the unknown sinus vein using a cotton cushion. If suprameatal nodular hypertrophy was observed, the hypertrophic nodules were milled and removed using a diamond grinder to completely release compression of the trigeminal nerve by bone. With strict hemostasis, dural closure was performed using continuous water tight sutures.

Results

Preoperative 3D-TOF and CISS revealed that the trigeminal nerve was adjacent to a blood vessel in 73 cases (74.5%) and not adjacent to a blood vessel in 25 cases (25.5%); intraoperatively, compression of an offending artery was identified in 59 cases (60.2%), and compression of an offending vein was found in 18 cases (18.4%). Among these 77 cases, seven showed mixed arterial and venous double compression. All the compression sites were separate during a Teflon cotton pad. In the remaining 21 cases (21.5%), no direct vascular compression of the trigeminal nerve was found. Among these 21 cases, 17 showed thickened arachnoid adhesions between the trigeminal nerve and the tentorium, so that the trigeminal nerve traveled at an angle and twisted; five cases showed vascular adhesion with the trigeminal nerve at an angle by the arachnoid when the arachnoid was cut intraoperatively, and after full release of the trigeminal nerve to restore its normal morphology, the vessel had no contact with the trigeminal nerve. Teflon cotton pads were not used in these 17 cases. The arachnoid around Meckel's cave surrounding the trigeminal nerve was cut in 360° for exploration. The compression of an unknown sinus vein at the outer arachnoid dura on the trigeminal nerve was observed in three cases, and the trigeminal

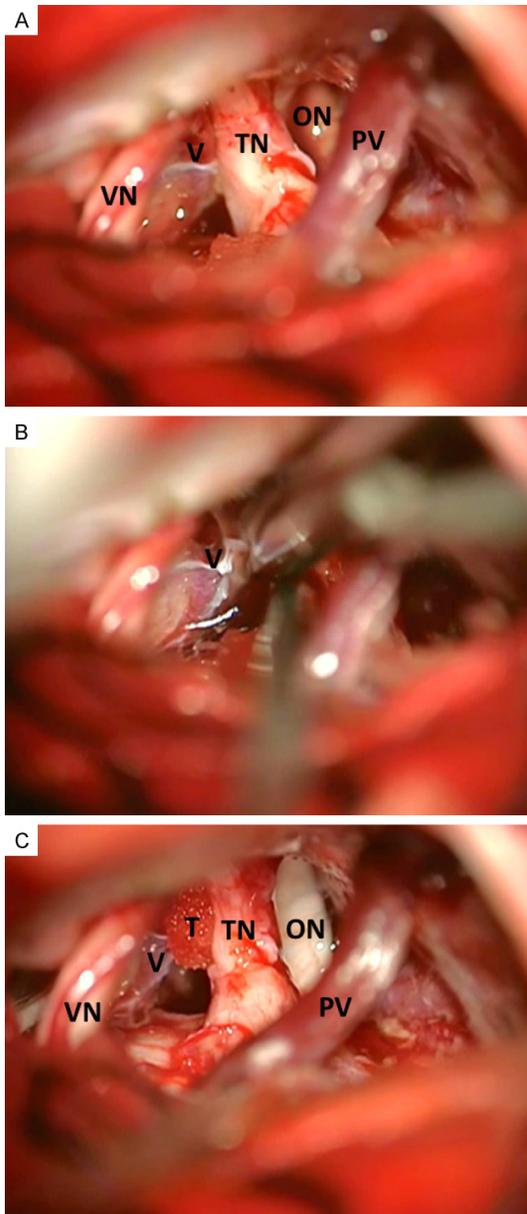


Figure 1. Microvascular decompression of the left trigeminal nerve. A: V is an unknown venoussinus on the dura of the outer arachnoid at the lower edge of Meckel's cave, showing compression on the trigeminal nerve. VN, the vestibular nerve; ON, the oculomotor nerve; TN, the trigeminal nerve; PV, the petrosal vein. B: Sharp separation was performed for the trigeminal nerve from the venoussinus. V, the venoussinus. C: After the Teflon cotton cushion was used to separate the offending vessel, the original morphology of the trigeminal nerve was restored. VN, the vestibular nerve; ON, the oculomotor nerve; TN, the trigeminal nerve; PV, the petrosal vein; V, the sinus vein; and T, the Teflon cotton cushion.

nerve was disassociated with the unknown sinus vein using a cotton cushion (**Figure 1**).

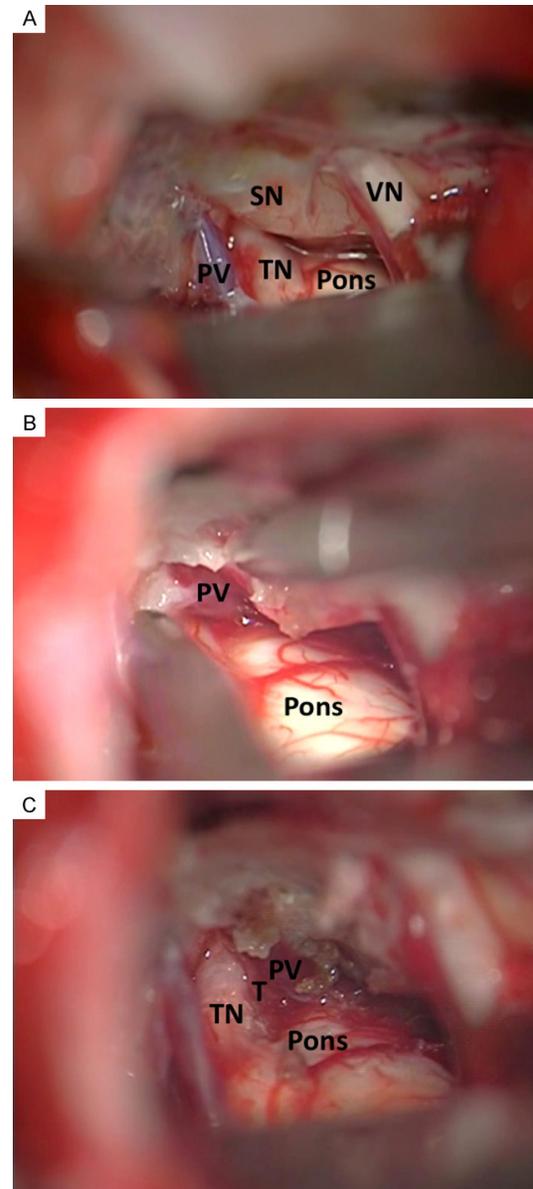


Figure 2. Trigeminal neuralgia on the right side. Microvascular decompression of the right trigeminal nerve. A: Suprameatal nodule hypertrophy caused stenosis of the prepontine cistern. SN, the supra-meatal nodule; VN, the vestibular nerve; TN, the trigeminal nerve; PV, the petrosal vein. B: PV is the petrosal vein at the anterior of the suprameatal nodule hypertrophy. After part of the hypertrophic nodule was milled and removed using a grinder, the petrosal vein with compression of the trigeminal nerve was exposed at the anterior of the nodule. C: The Teflon cotton cushion was used to separate the offending vessel from the trigeminal nerve. PV, the petrosal vein at the anterior of the nodule; T, the Teflon cushion cotton; TN, the trigeminal nerve.

Suprameatal nodular hypertrophy was observed in one case, causing stenosis of the

prepontine cistern with direct compression of the trigeminal nerve by the bony structure together with the unknown vessels. The hypertrophic nodules were ground and removed during surgery to completely release the compression of the trigeminal nerve by bone (Figure 2).

After surgical treatment, pain disappeared in all 98 patients in this study. This result occurred immediately after surgery in 89 cases (90.8%), with nine cases (9.2%) experiencing a significant reduction after surgery and gradual disappearance. The total rate of efficacy was 100%. Postoperative complications included 12 cases of facial numbness and superficial hypoesthesia, seven cases of intracranial infection, and one case of cerebrospinal fluid leakage. There were no cases of hearing loss, facial paralysis, or severe disability or death.

Among the 98 patients in this study, 92 were followed, and six were lost to follow-up; the follow-up time was 3-24 months. The 12 cases of postoperative facial numbness and superficial hypoesthesia resolved by one month after hospital discharge. During follow-up, one case showed recurrence, yielding a recurrence rate of 1.1%. During the secondary surgery, a thickened arachnoid adhesion was found at the Teflon cotton cushion, so the trigeminal nerve was pulled at an angle and twisted. The arachnoid adhesion was cut, and the excessive stuffing of the cotton pad was removed. After normal morphology of the trigeminal nerve was restored, the offending artery had no contact with the compression site, and the Teflon cotton cushion was no longer needed. Postoperatively, the painfully disappeared. None of the 98 patients in this study received trigeminal nerve lateral amputation.

Discussion

Pathology of trigeminal neuralgia

Dandy proposed the vascular compression theory in 1932 and described the demyelinating change in the lesions [1]. Hilton [6] further studied the pathological changes of the nerve root with compression and believed that compression leads to the demyelinating change. Close contact with the demyelinated axons forms a new "ectopic REZ", resulting in "false-synaptic" transduction between the tactile and pain fibers that forms a "short circuit" phenom-

enon in the excitatory transmission process. According to this theory, in MVD surgery, an implant can be placed between the offending vessel and the compressed trigeminal nerve to separate the stimulus source and the nerve segment from the damaged myelin and thereby achieve the treatment goal [7, 8].

Causes of trigeminal neuralgia

Typically, primary trigeminal neuralgia is caused by the compression of an artery, with a small number of cases caused by the compression of a vein, while secondary trigeminal neuralgia is caused by compression of tumors (common in meningioma, cholesteatoma, and nerve sheath tumor) [3, 5, 7, 9, 10]. Some special cases have been found in clinical practice in which the REZ of the trigeminal nerve and even the entire Meckel's cave have obvious vascular compression. Generally, they can be classified into the following three categories: 1. Thickened arachnoid adhesions between the trigeminal nerve and the tentorium such that the trigeminal nerve travels at an angle and twists; 2. Unknown sinus vein compression of the trigeminal nerve on the dura of the outer arachnoid at the lower edge of Meckel's cave; 3. Suprameatal nodular hypertrophy that causes stenosis of the prepontine cistern with direct compression of the trigeminal nerve by the bony structure together with the vessel.

In the patients of this study, thickened arachnoid adhesions between the trigeminal nerve and the tentorium were found intraoperatively in 17 cases, with the trigeminal nerve traveling at an angle and twisting. After the arachnoid was cut to fully release and disassociate the trigeminal nerve, the morphology of the trigeminal nerve returned to normal. When the arachnoid around Meckel's cave surrounding the trigeminal nerve was cut in 360° for exploration, compression of an unknown sinus vein at the outer arachnoid dura on the trigeminal nerve was observed in three cases, and the trigeminal nerve was disassociated with the unknown sinus vein using a cotton cushion. Suprameatal nodular hypertrophy was observed in one case. The hypertrophic nodules were ground and removed to completely release bone compression on the trigeminal nerve. In the 24 months of follow-up, one case showed recurrence, and secondary surgery was provided. All cases achieved satisfactory results.

Notes for MVD surgery

(1) A 3 cm × 3 cm bone window needs to be created to expose the rearedge of the sigmoid sinus at the front and the transverse sinus at the top. (2) Intraoperatively, the microscope optical axis should be maintained in line with the operating channel to ensure good exposure of the trigeminal nerve for the entire procedure and to minimize the pulling of the cerebellar hemisphere, thereby avoiding the rupture of the petrosal vein and other complications. (3) More than one site may be causing trigeminal neuralgia, and the offending vessel causing compression may be an artery, vein, or both, so the entire course of the trigeminal cranial nerve throughout the posterior cranial fossa needs to be carefully checked, and the original morphology of the trigeminal nerve needs to be restored after the offending vessel is separated by the cushion to relieve both adhesion and compression. (4) After the offending vessel is isolated by the cushion, the artery should not be twisted at an angle. (5) If the offending vessel is a thick, tortuous, and sclerosing vertebral artery, it is often difficult because of the narrow surgical space, and thus, decompression should be performed not only at the compression site of the trigeminal nerve but also at the vertebral artery and the brain stem surface, so that part of the compression of the vertebral artery on the trigeminal nerve can be distributed; (6) Teflon cotton pads should be used sparingly, with the least material that provides relief, and the key is to find all the pain sites and fully restore the original morphology of the trigeminal nerve.

In summary, the compression of the offending vessel in trigeminal neuralgia is diverse, and sometimes the offending vessel does not exist. The causes of pain are complex and diverse [7]. Although preoperative imaging studies may not suggest obvious compression of an offending vessel, there may be other factors that impact the trigeminal nerve. With careful intraoperative exploration and judgment based on experience, satisfactory results can still be achieved.

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Disclosure of conflict of interest

None.

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