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Surgical Techniques for Treating Trigeminal Neuralgia without Vascular Compression Using Magnetic Resonance Imaging

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Abstract

Introduction: To depict the careful technique, remedial impact and postoperative intricacies of trigeminal neuralgia (TN) without vascular pressure utilizing head attractive reverberation imaging (X-ray).

Methods: From January 2018 to March 2021, we collected complete clinical data from 184 TN patients who had been admitted to Zhoukou Central Hospital's Department of Neurosurgery. A preoperative MRI revealed that there was no vascular compression in 35 patients (19.0 percent). 16 (45.7%) of them underwent surgery with simple venous compression, 9 (25.7%) underwent surgery with venous compression and arachnoid adhesion, 5 (14.3%) underwent surgery with arachnoid adhesion, 3 (8.6%) underwent surgery with neither venous compression nor arachnoid adhesion, and 2 (5.7%) underwent surgery with simple artery compression. The appropriate surgical approaches for the various kinds of venous compression were developed during the procedure. The Barrow Neurological Institute pain intensity and facial numbness scores were used to evaluate postoperative efficacy.

Results: The appropriate decompression techniques were found to effectively relieve or eliminate pain (97.1%), and the postoperative effect was relatively satisfactory, according to the four types of compression that were discovered during the procedure but did not reveal obvious vascular compression on the preoperative head MRI. There was no repeat following 1-2 years of follow-up. Two patients (5.7%) reported occasional pain following the procedure, one patient (2.9%) reported minor pain, and two patients (5.7%) reported facial numbness. In addition, one patient (2.9%) experienced aseptic meningitis, and another patient (2.9%) experienced transient tinnitus or hearing loss on the side that was affected.

Conclusion: Based on microvascular decompression and adequate trigeminal nerve decompression findings, various surgical strategies can be implemented for patients with (TN) without vascular compression on head MRI prior to surgery. Our findings demonstrated that these strategies were capable of producing contented postoperative outcomes without increasing the number of postoperative complications.

Keywords: Magnetic resonance imaging • Trigeminal neuralgia • Microvascular decompression • Surgical strategy

Introduction

Trigeminal neuralgia (TN) is a severe form of recurring paroxysmal pain resembling an electric shock or a knife cut that affects one or more sensory distribution areas of the trigeminal nerve on one face side. This condition is also known as "the first pain in the world," and it is referred to as "the most severe pain ever experienced by a human." The majority of patients experience unilateral onset and the attack may have a "trigger point" and end abruptly. On the affected side, there may be simultaneous tearing and salivation. Carbamazepine has a powerful analgesic effect. TN is currently caused by a variety of factors, including: vascular compression, nerve compression, facilitation caused by changes in central plasticity, myelin degeneration, viral infection, and other space-occupying lesions like arteriovenous malformation, aneurysm, vestibular schwannoma, meningioma, and other types of cysts and tumors. The majority of researchers currently accept the theory that neurovascular compression is the primary cause of TN. Vascular compression around the intracranial segment of the trigeminal nerve is thought to play a significant role in the pathogenesis of TN, according to this theory. A few studies have found no obvious arterial compression on magnetic resonance imaging (MRI), indicating that the majority of the responsible blood vessels are arteries. This study evaluated the surgical strategies, curative effects, and postoperative complications of 35 patients with TN whose clinical MRI data did not show any obvious arterial compression [1].

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Materials and Methods

Population of patients

Between January 2018 and March 2021, 184 patients with concurrent operations were admitted to the Zhoukou Central Hospital, China, Department of Neurosurgery. The nerve root was subjected to microvascular decompression (MVD) during all of the operations, which were carried out by the same surgeon. Before the surgery, a cranial MRI was done to rule out secondary TN caused by bone compression and other factors. The results showed that none of the 35 patients had any obvious vascular compression. During the procedure, simple venous compression was observed in 16 patients; during the procedure, nine patients experienced venous compression and arachnoid adhesion; During the procedure, 5 patients experienced arachnoid adhesion; During the procedure, three patients did not experience venous compression or arachnoid adhesion; during the procedure, two patients' arteries were compressed. The "International Classification of Headaches" (Third Edition) diagnostic criteria for TN [2].

Method of surgery

All patients underwent the previously described retrosigmoid craniotomy. They were undeniably positioned in inclined position and gone to the sound side, with the impacted side on top, after intravenous intubation and

general sedation. Behind the mastoid at the hairline, a straight incision of 5–6 centimeters in length was made. Bone window was opened below the transverse sinus, and sufficient bone wax was used around the bone edge (all possible openings for the mastoid air chamber were closed) to expose the open bone window to the edge of the sigmoid sinus down to the foramen magnum. In an inverted "T" shape, hang and fix the dura mater toward the transverse sinus, sigmoid sinus, and transverse-sigmoid sinus junction, respectively, from the opening's front and upper edges. Under the microscope, release a portion of the cerebrospinal fluid appropriately and slowly. The trigeminal nerve root was examined and completely loosened from the pontine end to the adjacent Mekel capsule after the cerebral pressure decreased and the cerebellum collapsed. The arachnoid membrane that separated the facial and acoustic nerves from the tentorium cerebelli was cut along the horizontal fissure of the cerebellum into the cerebellopontine angle area. The specific plan is as follows: The dura mater was water-tightly sutured, the muscle, subcutaneous tissue, and skin were sutured layer by layer in a standard manner, and hemostasis was strictly maintained throughout the procedure [3].

Arachnoid adhesion thickening and venous compression

Venous compression is frequently linked to arachnoid adhesion thickening. Eight patients had small veins compressed (less than 3 mm in diameter) and arachnoid adhesions of varying degrees observed during the procedure. In order to protect the surrounding veins from lumen occlusion caused by thermal damage, low-power electric coagulation was used to cut off the peripheral veins during the procedure. The trigeminal nerve root was able to be completely loosened in the axial position as a result of the complete release of the arachnoid membrane and adhesion zone. Finally, a combing knife was used to make a longitudinal comb in the direction of the nerve fiber. The range, frequency, and severity of preoperative pain episodes, as well as the degree of arachnoid adhesion observed during the procedure, were used to determine the number of combings, and only superficial nerve combing was carried out. Intraoperative combings were carried out in this study. In one patient, a bigger vein was seen during the activity, joined with thickening of arachnoid grips. The responsible vein and the trigeminal nerve were completely cut off during the procedure, and Teflon cotton was placed in between them. Finally, adequate nerve combing and arachnoid release were carried out [4].

Thickening of arachnoid adhesions

In five cases, the arachnoid adhesion was obvious, the local arachnoid membrane was thicker, and even the arachnoid frenulum was formed, which twisted the trigeminal nerve. There were no obvious responsible blood vessels. The most obvious is particularly close to Meckel's capsule. The adhesion zone and arachnoid membrane ought to be completely and abruptly released during the procedure. It needs to be completely loosened all the way from the REZ to the Meckel capsule. To return the trigeminal nerve to its original floating state, all of the arachnoid bands surrounding it should be severed. Finally, nerve grooming was performed once more, three to four times to guarantee the postoperative curative effect. No venous pressure or it was seen to thicken of arachnoid bond [4].

Compression of the arteries

In one instance, small arteries were observed to compress the trigeminal nerve, and in another instance, small arteries and arachnoid adhesions were observed to compress a patient. Teflon cotton was inserted between the artery and the trigeminal nerve during the procedure, and the adhesion zone and arachnoid membrane were sharply released to completely loosen the trigeminal nerve root [5].

Discussion

According to the trigeminal root vascular compression theory, the trigeminal nerve root can be compressed by abnormal blood vessels, particularly in the REZ, where the sensory roots of the trigeminal nerve enter the brainstem segment, which is the weakest part of the myelin sheath and is especially susceptible to external factors like compression. The demyelination of the trigeminal nerve changes in response to a variety of inducing factors, resulting in a spontaneous abnormal action potential between nerve fibers. Based on this theory, MVD is one of the most common treatments for primary TN, with a postoperative effective rate of more than 95%, and its efficacy has been widely acknowledged. The spontaneous action potential enters the trigeminal central nucleus and accumulates repeatedly through the pseudo synaptic circuit before reaching a threshold and initiating TN. Early treatment of MVD focuses primarily on TN cases in which the artery is the responsible blood vessel. The superior cerebellar artery and the anterior inferior cerebellar artery are the most typical responsible arteries. Gradually, the significance of non-arterial factors like venous compression, arachnoid adhesion, or both (none) was realized. Ridder and co. believed that any part of the cranial nerve's blood vessels, including arteries and veins, could cause clinical symptoms, including the REZ.7 Barker et al. also reported 1185 cases of MVD patients with TN, 151 of which involved straightforward venous compression8 reported in their study that venous oppressors were found in 234 of 1476 TN cases. More and more researchers now believe that venous compression is also a cause of TN [6,7].

Many researchers believe that petrous veins and their branches are the responsible blood vessels in TN patients with venous compression. The posterior cranial fossa's petrosal vein is a crucial drainage vein. It mostly sends the venous blood from the brainstem and cerebellar hemisphere to the superior and inferior petrosal sinuses. The superior and inferior petrosal veins of the venous sinus are distinguished by their differences. The petrosal vein is a relatively thick branch tube that may block the surgeon's sight during the operation. The number, route, and location of the branches into the venous sinus vary greatly from person to person. The petrosal vein, a responsible vessel, is a relatively thick branch tube that may obscure the surgeon's vision during the procedure. The petrosal vein branch, which is the responsible blood vessel, may tightly adhere to the trigeminal nerve and compress it through multiple branches. A few researchers accept that harming or obstructing other cerebellar veins, for example, veins won't cause clear complications. A few researchers likewise suggest electrocoagulation of the little veins answerable for TN to decrease the repeat of postoperative pain. Anyway different researchers accept that injury or coagulation of the dependable vein might cause dead tissue of the cerebellar side of the equator and cerebrum stem. After the surgery, of the patients in this study experienced less or no pain at all. After the operation, one patient experienced intermittent pain without the use of medication. One patient experienced mild numbness or paresthesia on the affected side following the operation. During the follow-up period, he tolerated it well and his quality of life was unaffected [8,9].

In this study, intraoperative venous compression with arachnoid adhesion was not uncommon. It is believed that arachnoid thickening, adhesion, and arachnoid traction nerve angulation can cause nerve ischemia and nutritional disorders, which in turn affect the REZ and eventually result in myelin degeneration, medullary bulb formation, interstitial edema, interstitial collagen fibrosis, mitochondrial vacuolar degeneration, and The author clarified the TN-causing main and subordinate veins before completely separating the arachnoid membrane or adhesions during the procedure. The veins underwent the same treatment as previously mentioned. The quantity of not set in stone after full arachnoid lysis in view of the reach, recurrence, and seriousness of the patient's preoperative aggravation episodes, as well as the level of arachnoid attachment seen during the activity (the quantity of brushing was generally, and the reach was restricted to 1/3 of the back base of the trigeminal nerve). For longitudinal surface combing in the direction of the nerve fiber, the carding knife was used. In addition, postoperative compression caused by incomplete arachnoid adhesion relaxation can be avoided if only the arachnoid adhesion was thickened during the procedure. Finally, the trigemimal nerve's sensory root combing was carried out. The outcome of the surgery afterward was good [10].

Conclusion

Patients with TN who have no vascular compression on their head MRI prior to surgery may experience simple venous compression, arachnoid adhesion, arachnoid adhesion, or neither during MVD. To ensure the

postoperative curative effect and operation quality, distinct surgical strategies based on specific surgical conditions are essential.

Acknowledgement

None.

Conflict of Interest

None.

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