Long-term effectiveness of an ad hoc tailored titanium implant as a spacer for microvascular decompression in the treatment of trigeminal neuralgia caused by megadolichoectatic basilar artery anomaly: 9-year follow-up

Case report

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An enlarged, elongated, ectatic, and sclerotic aberration of the vertebrobasilar system is known as a megadolichoectatic basilar artery (BA) anomaly. The anomaly is often involved in the pathological process of trigeminal neuralgia by compressing and distorting the trigeminal nerve. First-line medical treatment includes drug therapy, but a second-line surgical procedure could be effective in medication-resistant cases. The authors report the case of a 65-year-old man with a 12-year history of progressing trigeminal neuralgia who underwent microvascular decompression after the first-line drug treatment had failed. This case is unique because an in situ tailored titanium microplate was used as a spacer to alleviate compression by the BA on the trigeminal nerve. The titanium implant provided durable and sufficient retraction for the sclerotic arterial complex when the trigeminal nerve was placed in the tunnel of the implant.

The 9-year follow-up examination proves the safety and long-term efficacy of titanium implants in the treatment of trigeminal neuralgia caused by a megadolichoectatic BA anomaly. The method applied in this case was not intended to be and certainly is not an alternative to routine microvascular decompression—this surgical solution may be reserved for some extreme cases.

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KEY WORDS • trigeminal neuralgia • megadolichoectatic basilar artery anomaly • microvascular decompression • titanium implant • long-term effectiveness • functional neurosurgery

MEGADOLICHOECTATIC basilar artery (BA) anomaly represents an enlarged, elongated, ectatic, often sclerotic aberration of the vertebrobasilar system, also known as vertebrobasilar dolichoectasia or tortuous vertebrobasilar system. By neurovascular compression of the trigeminal nerve root and/or pontine entry zone, this anomaly may contribute to the development of trigeminal neuralgia (TN). However, smaller vessels (e.g., the superior cerebellar artery [SCA] or the anterior inferior cerebellar artery [AICA]) are more often involved in mechanical distortion of the trigeminal nerve instead of isolated vertebrobasilar compression. Treatment of TN usually begins with drug therapy, but second-line procedures may be required as the disease progresses. The aim of this case report is to present the long-term efficacy of using a titanium microplate for microvascular decompression (MVD). To the best of our knowledge, this is the first successful long-term use of such a makeshift surgical implant in the treatment of TN caused by megadolichoectatic BA anomaly.

Case Report

History. A 65-year-old man with hypertension and a 7-year history of left-sided TN was referred to our hospital. Intermittent pain was characterized primarily within the supply area of the maxillary nerve and occasionally in the supply area of the mandibular nerve. As a result of drug therapy (including carbamazepine, bac-
lofen, tiapride, and clomipramine) initiated at that time, although painful episodes remained, the patient better tolerated the sequelae for a long time.

**Examination.** Five years after initiation of drug therapy, the patient was admitted again to our hospital due to worsening and drug-resistant left-sided pain with extension to the entire left side of the face. Admission CT and MR scans demonstrated compression of the trigeminal nerve caused by a considerably enlarged, elongated, tortuous, and atherosclerotic BA (megadolichoectatic BA anomaly) (Fig. 1). Considering the radiological and clinical findings, MVD was performed.

**Operation.** Surgery was performed by the senior author (I.N.) on October 5, 2004. After making a left lateral suboccipital craniotomy and opening the dura mater layer, the upper cerebellopontine angle was exposed with gentle retraction of the cerebellum. An oversized, sclerotic, curved BA was noticeable, medially displacing the pons and the lateral part of the cerebellar hemisphere, and also causing tension of the fifth, seventh, and eighth cranial
nerves. On dissection of the petrosal vein and arachnoid layer, it was clearly visible that the S-shaped megadolichoectatic BA with the branching SCA strongly compressed the root of the trigeminal nerve against the tentorium and tensed the cochlear nerve (Figs. 2 and 3A). The extraor-
dinarily thick, sclerotic AICA distended the facial nerve and could be identified by the collateral internal auditory artery. The whole vascular complex including the BA and tributaries was immobile to such an extent that the usual techniques—separating the vascular and nervous part and cushioning with a small piece of muscle—seemed inappropriate for decompression.

To achieve our goal, a rosette-like microplate (Fig. 4; Bioplate, Inc.) used for cranial bone flap fixation was bent to form a tunnel and was placed with one side against the tentorium while the other side distracted the vascular complex, letting the fifth cranial nerve traverse within the tunnel (Figs. 3B and 5). Pressure of the BA conveyed through the makeshift spacer to the tentorium kept the whole structure in place. We used a specially designed U-shaped titanium microplate to accommodate the trigeminal nerve at its segment that was subjected to the arterial hammering effect. Due to the geometry of the microplate used, contours of the construction after bending remained rather rounded (Fig. 3B and C, and Figs. 4 and 5); however, the flat metal surface of the implant facing the vessel and all edges were cushioned with gelatin sponge (Spongostan; Ethicon, Inc.) and tiny muscle flaps harvested from the exposed nuchal muscle (Fig. 3C). Closure was made in the standard fashion.

Postoperative Course. Postoperatively no signs of complications or neurological dysfunction were evident, and TN disappeared completely. Postoperative fluoroscopy (Fig. 6 left) and MRI (Fig. 6 right) scans showed that the titanium plate remained in place and clinically provided sufficient decompression for the trigeminal nerve.

At the 9-year follow-up, the patient reported no recurrence of any symptoms. CT angiography (CTA) scans (Fig.
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7) demonstrated no displacement of the implant, and the trigeminal nerve remained intact within the titanium tunnel.

Discussion

Microvascular decompression is considered to be a long-ranging, effective surgical treatment option for TN caused by vertebrobasilar compression. If smaller, mobile vessels are involved, separation from the nerve trunk can be done easily with cautious dissection, and the complication rate including minor trigeminal hypalgesia, transient diplopia, and hearing loss remains low. Several materials have been reported to isolate the trigeminal nerve successfully from arteries, including fenestrated aneurysm clips, aneurysmal cuff clips, vascular tapes, or Teflon felts. However, extended neurovascular manipulation can contribute to serious postoperative complications, and some of the implants may aggravate the neural compression in cases of an enlarged, sclerotic BA. Furthermore, if the smaller collateral arteries of a tortuous vertebrobasilar system also participate in mechanical distortion, occasionally satisfactory decompression of the trigeminal nerve may not be achieved in the usual way.

In our case, the patient had all the risk factors (hypertension, male sex, and older age) for megadolichoectatic BA anomaly that are associated with a higher tendency of atherosclerosis to contribute to the development of this anomaly. Left-sided occurrence was noticed because the left vertebral artery (VA) dominates in most patients. After TN was diagnosed in our patient, first- and second-line drugs for medical treatment were successfully applied for 5 years. As the disease progressed to the entire left side of the face and the pain worsened, MVD was performed. Because intraoperative findings revealed a hard, unmovable, compressing mass formed by the BA complex, another solution was needed. Even using a rubber sling fixed to the basal dura and pulling away the vessel, which many times has been successfully applied to prevent VA–medulla compression, seemed useless in our case. The appropriately bent titanium microplate provided sufficient retraction of sclerotic arteries from the trigeminal nerve. The stable, wide tunnel of titanium plate prevented further neural compression and facilitated satisfactory neural relief.

To minimize the potential risk of vessel wall erosion stemming from the pulsating contact between the sclerotic BA and the rigid metal, we created damping layers. The metal surface of the implant facing the vessel and all edges were cushioned with muscle flaps. We also hypothesized that, with time, the interposed muscle might be transformed into connective tissue, providing additional protection. At the 9-year follow-up evaluation, CT and CTA scans demonstrated no displacement of the implant (Fig. 7), and the patient remained free of symptoms.

Cases similar to the one presented here may warrant the construction of even more appropriate spacers to achieve satisfactory decompression. The method applied in this case was not intended to be and certainly is not an alternative to routine MVD. Our surgical strategy can be considered in some extreme cases and was devised to remedy an otherwise unmanageable condition. In such rare cases of a megadolichoectatic BA causing TN, further experience is needed to make a conclusion about a widely acceptable method. To the best of our knowledge, this is the only reported case of the use of such an implant in the literature.

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Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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