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Case Report

Fully endoscopic microvascular decompression for painful tic convulsif secondary to vertebrobasilar dolichoectasia

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Abstract: Vertebrobasilar dolichoectasia is a rare condition that can compress the trigeminal and facial nerves, resulting in trigeminal neuralgia and hemifacial spasms. When these symptoms occur on the same side of the face, the condition is termed painful tic convulsif. Painful tic convulsif secondary to vertebrobasilar dolichoectasia is occasional. This paper presents a case of painful tic convulsif successfully treated by fully endoscopic microvascular decompression. The patient, an older man, experienced paroxysmal pain and involuntary convulsions on the left side of the face for 2 years. Clinical presentation and magnetic resonance imaging results confirmed a painful tic convulsif diagnosis caused by vertebrobasilar dolichoectasia. The patient underwent fully endoscopic microvascular decompression, during which a dilated and tortuous vertebral artery was separated from the affected nerves using a Teflon pad. After surgery, the left-sided facial pain and convulsions were completely relieved without any complications. After 17 months of follow-up, no recurrence was reported. Hence, fully endoscopic microvascular decompression was an effective treatment for painful tic convulsif secondary to vertebrobasilar dolichoectasia.

Keywords: Endoscopy, microvascular decompression, vertebrobasilar dolichoectasia, trigeminal neuralgia, hemifacial spasm, painful tic convulsif

Introduction

Vertebrobasilar dolichoectasia (VBD) is a cerebrovascular disorder characterized by the vertebral basilar artery's substantial expansion, elongation, and tortuosity [1, 2]. It is relatively rare, usually affecting older adults, with an incidence of approximately 0.5%. Moreover, a higher prevalence is reported in men than in women [3, 4]. VBD diagnosis primarily depends on computed tomography, magnetic resonance imaging (MRI), and digital subtraction angiography. No widely recognized quantitative standard has been established for VBD. Nonetheless, basilar artery lengthening is diagnosed when its height extends above the upper clivus or dorsal sella or when its apex and the bilateral posterior cerebral arteries exceed the suprasellar cistern level. A basilar artery diameter over 4.5 mm indicates dilatation [5]. VBD is usually asymptomatic. However, it can present as ischemic stroke, cranial nerve and brainstem compression, cerebral hemorrhage, or hydrocephalus [6, 7]. Because of the proximity of the extended and dilated vertebrobasilar artery to the trigeminal and facial nerves, trigeminal neuralgia (TN) and hemifacial spasm (HFS) are the most common conditions [8-10]. Compression of other cranial nerves, such as the glossopharyngeal, abducens, and oculomotor nerves, is relatively rare [11-13]. When TN and HFS occur on the same side of the face, this condition is termed painful tic convulsif (PTC) [14]. PTC is a rare disease caused by vascular compression [15, 16]. PTC secondary to VBD is rare. Previously, diphenylhydantoin, carbamazepine, and ethanol injection into the Gasserian ganglion provided TN relief, whereas saline neurolysis of the facial nerve provided HFS relief [17]. However, these methods address TN and HFS separately. Microvascular decompression (MVD) is the most effective and lasting treatment for PTC [18]. MVD is conducted using a microscope and has gradually incor-

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porated endoscopic technology, thus yielding good results [19-21]. This paper presents a case of PTC secondary to VBD successfully treated with fully endoscopic MVD. The results elucidate the efficacy of this approach and its advantages over traditional microscopic techniques in PTC management.

Case presentation

In July 2021, a 68-year-old man without a history of hypertension, diabetes, or other comorbidities presented with paroxysmal pain and involuntary left-sided twitching, persisting for 2 years. Initially, these symptoms were partially improved with carbamazepine and oxcarbazepine; however, over time, their relief diminished, resulting in worsening symptoms and a substantial decline in his quality of life. The symptoms showed a gradual onset and progressively intensified. Neurological examination results were normal, and pathologic signs were absent. All laboratory test results, including blood count, liver and kidney function tests, immune function tests, and blood coagulation evaluation, were within normal ranges. The patient had no history of trauma or a family history of genetic diseases. Preoperative cranial MRI showed a tortuous, dilated, and extended vertebral basilar artery. The left trigeminal and facial nerves were in proximity to the tortuous basilar artery, besides being compressed and displaced (**Figure 1A**, **1B**). These results confirmed a diagnosis of PTC secondary to VBD. This diagnosis was based on the patient's clinical symptoms and MRI results. Considering the poor effect of drug treatment and the ongoing poor quality of life, the patient underwent fully endoscopic MVD through the suboccipital retrosigmoid approach.

After general anesthesia, the patient was placed in the right lateral decubitus position, with the head fixed in a Mayfield stent with slight bending. A 5-cm-long vertical incision was made behind the ear at the asterion level. The skin and muscle were incised to entirely expose the mastoid root. The bone removal window measured approximately 3 × 3 cm, exposing the inferior and medial aspects of the transverse and sigmoid sinuses. When mastoid air cells were encountered, they were meticulously sealed with bone wax, and the dura mater was incised. An endoscope was introduced, and cerebrospinal fluid (CSF) was slowly drained from the cisterna magna to allow the cerebellar hemisphere to collapse naturally and create sufficient surgical space. Endoscopy involved a combination of fixation and handheld techniques. For complex operations requiring both hands, the endoscope was stabilized. However, for deeper structures, the endoscope position was dynamically adjusted through handheld techniques. Furthermore, the cerebellopontine angle around the lateral cerebellum was observed. The arachnoid surrounding the trigeminal and facial nerves was dissected to ensure complete exposure (**Figure 1C**). The trigeminal nerve was severely compressed, which was characterized by nerve thinning. Considering the dilatation and elongation of the left vertebral artery, simple separation was ineffective. Hence, a gradual decompression approach was used. First, a Teflon pad was inserted into the proximal end of the left vertebral artery, utilizing the principle of displacement to lower the impact of vascular pulsation on the affected nerves (**Figure 1D**). Second, spherical Teflon pads were placed between the left vertebral artery, facial nerve root, and trigeminal nerve using to achieve sufficient decompression (**Figure 1E**). Endoscopy indicated small arterioles adjacent to the trigeminal nerve. These arterioles were separated from the nerve, with spherical Teflon pads placed to prevent compression. The absence of offending vessels and the size and placement of Teflon pads were confirmed. Hemostatic gauze was applied to the surface of the flocculonodular-lobe and cerebellum. Finally, the dura mater was sutured, and artificial meninges were used for double-layer repair to prevent CSF leakage. Four titanium plates and screws were used to repair the cranial defect, and the incision was closed in layers. Despite no complications during surgery, standard measures were employed to prevent risks, such as nerve injury, vascular damage, and CSF leakage. Surgeons should be familiar with the distribution of cerebral vessels and cranial nerves, use artificial meninges for double-layer repair, and use titanium plates and screws to reinforce cranial defects and minimize complications.

The evaluation of MVD results was based on the standardized MVD grading approach proposed by Kondo et al. [22]. For TN, postoperative pain evaluation yielded an E-0 score (completely pain-free), whereas the evaluation of

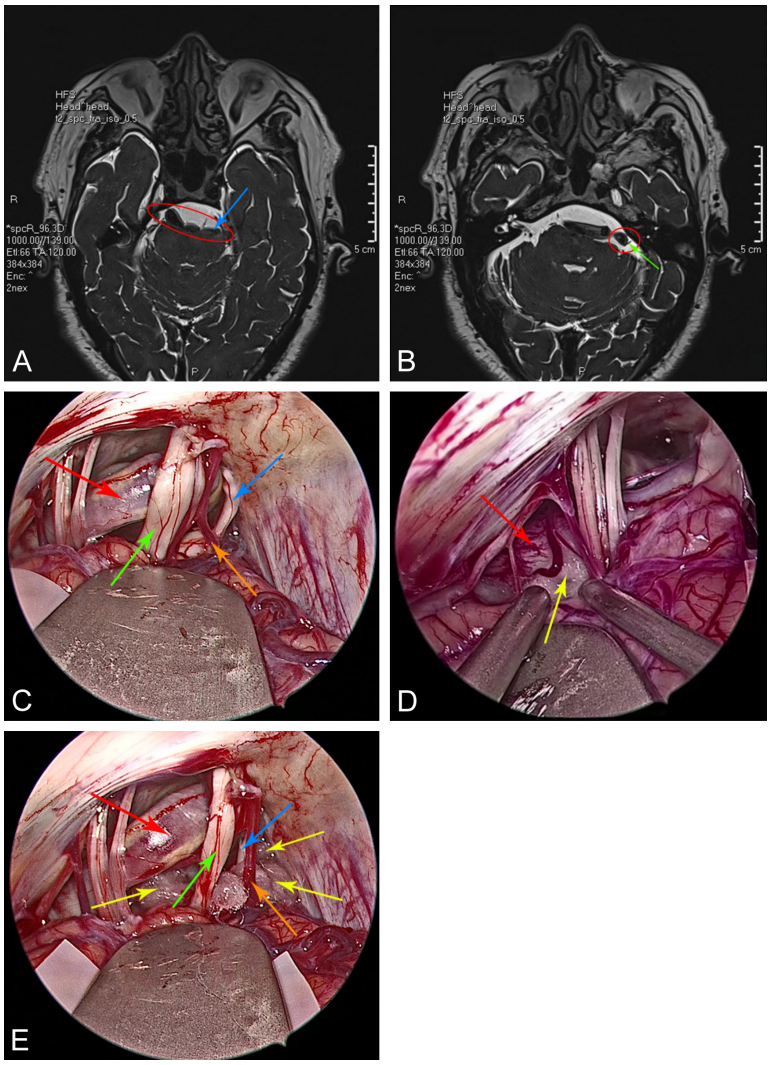

Figure 1. A, B. Preoperative head magnetic resonance imaging of the patient. The left vertebral artery compressed the trigeminal and facial nerve root. C. The offending vessels, left trigeminal and facial nerve, were clearly visible under the endoscopic view. D. A Teflon pad was inserted into the proximal end of the left vertebral artery. E. The Teflon pad was placed between the offending vessels and left trigeminal and facial nerve under the endoscopic view. Red circle indicates the lengthening and dilatation of the left vertebral artery, blue arrow indicates the left trigeminal nerve, green arrow indicates the left facial nerve, the red arrow indicates the left vertebra artery, the orange arrow indicates the small artery, and the yellow arrow indicates the Teflon pad.

complications yielded a C-0 score (no deficits, or only minor subjective complaints). The total evaluation result was classified as T-0 (excellent). For HFS, postoperative evaluation of involuntary movement yielded an E-0 score (complete disappearance of spasm), whereas the evaluation of postoperative complications yielded a C-0 score (no, or only minimal cranial nerve dysfunction). The total evaluation result was classified as T-0 (excellent). Postoperative neurological examinations at 1, 6, and 17 months confirmed normal nerve function without any recurrence of symptoms. After 17 months, the patient reported a marked improvement in quality of life. This result indicates the effectiveness of fully endoscopic MVD in treating PTC secondary to VBD.

Discussion and conclusions

VBD is a rare cerebrovascular disease, with an unclear etiology. Several possible mechanisms have been proposed to describe its onset. Because VBD primarily affects older men, hypertension-induced atherosclerosis may be its primary cause [23]. However, VBD may also occur in younger people, who are less likely to develop atherosclerosis. Therefore, some researchers have proposed that VBD is caused by congenital factors. VBD has been associated with autosomal recessive polycystic kidney disease, Fabry disease, sickle cell anemia, Marfan syndrome, Ehlers-Danlos syndrome, and PHACES syndrome [24, 25]. Additionally, syphilis, acquired immunodeficiency syndrome, and Hodgkin's lymphoma have been implicated in VBD pathogenesis [24, 25]. VBD results from an interaction between congenital factors and acquired factors. No effective treatment for VBD exists, and most patients have a poor prognosis. Several patients die of ischemic stroke, making anticoagulation treatment a possible treatment option. Nonetheless, excessive anticoagulation may increase the risk of fatal bleeding [4]. Cranial nerve compression - presenting as TN and HFS - is one of the most common VBD hallmarks. VBD accounts for approximately 2% to 7.7% of TN cases [26] and approximately 0.7% of HFS cases [27]. The simultaneous onset of

TN and HFS symptoms on the same side of the face is rare. In 1920, Harvey Cushing coined the term "painful tic convulsif" to describe this condition [14]. PTC is a rare disease. A 2021 statistical report by Yin et al. encompassing 192 patients with PTC suggested that vascular compression accounted for 85% of cases, with the superior cerebellar artery being the most commonly offending vessel. Interestingly, VBD accounted for only 1.7% of PTC cases [15]. Drug treatment is the mainstay for VBD-induced TN. Similar to TN treatment, carbamazepine is the first choice for alleviating paroxysmal pain [28]. For VBD-induced HFS, botulinum toxin injection helps to control symptoms [29]. Nevertheless, when drug treatment does not effectively provide symptom relief, surgical treatment can be considered. MVD is the most effective and safe method to treat cranial nerve compression symptoms caused by VBD [30]. Sun et al. reported on 15 MVD cases for TN secondary to VBD [26]. All patients experienced immediate facial pain relief after surgery, with no recurrence during the average follow-up period of 29.8 months. Only two patients reported facial numbness, without other complications. Likewise, Zhao et al. studied 36 patients with TN secondary to VBD [31]. After MVD, 97.8% of patients experienced complete facial pain relief. After 1, 3, 5, and 7 years of follow-up, 95.7%, 85.1%, 74.2%, and 74.2% of patients, respectively, reported good outcomes. Ehab et al. studied 11 patients with HFS secondary to VBD [32]. After receiving MVD, all patients experienced complete symptom relief. Additionally, Gabriel et al. reported a case of left TN and HFS caused by a dolichoectatic left vertebral artery compression [18]. After MVD, all symptoms were relieved immediately, without any recurrence at the 1-month follow-up. In the present case study, PTC was successfully treated with MVD under full endoscopy. Hence, MVD is an effective treatment for PTC secondary to VBD. However, conducting MVD in patients with VBD is challenging. The enlarged vascular diameter and fragile arterial walls, along with the need for anticoagulation treatment, increase the risk of bleeding and vascular injury. MVD is conducted using a microscope. Nevertheless, advances in endoscopic technology have facilitated its use in MVD. Compared with a microscope, an endoscope offers better illumination, improved visual angles, expanded visual fields, and high-quality resolution, helping surgeons accurately identify offending vessels and reduce the possibility of missing compressions. Chen et al. studied 167 patients with TN and reported that approximately 14.74% of the affected vessels were undetected under the microscope but successfully identified under the endoscope [33]. In this study, imaging examinations identified only the left vertebral artery as the offending vessel. Nonetheless, endoscopy identified an additional small artery, elucidating the advantage of endoscopy in identifying offending vessels. Additionally, good illumination and an expanded visual field offer a more comprehensive view of relevant anatomic structures and avoid damage to surrounding brain tissue, blood vessels, and nerves, reducing postoperative complications. Compared with a microscope, an endoscope requires less cerebellar retraction. This feature is advantageous to patients with VBD, where the large diameter of the offending vessels limits the surgical space. Finally, endoscopy offers a great advantage in evaluating the adequacy of decompression. Surgeons can observe the placement of the Teflon pad from multiple angles, ensuring sufficient decompression while mitigating damage to the peripheral tissues, nerves, and blood vessels. Therefore, fully endoscopic MVD is an effective method to treat PTC secondary to VBD. However, endoscopy has limitations. The limited intracranial space and the space required by the endoscope can damage the surrounding structures. The 2D images lack depth perception. Moreover, intraoperative bleeding can contaminate the lens, affecting image quality and complicating the procedure. Additionally, the light source emitting heat and sharp edges may injure nearby tissues. Finally, mastering this technique requires a steep learning curve and extensive training.

This paper presents a case of TN and HFS secondary to VBD, which was successfully treated using a fully endoscopic MVD. Therefore, fully endoscopic MVD is an effective method to treat TN and HFS secondary to VBD. Despite its challenges, surgeons can master this technique through adequate training, hands-on experience, and continuously updated equipment. This case had certain limitations. First, the results were based on a single case, limiting their generalizability. Therefore, researchers should expand the sample size and conduct

multi-center, large-scale clinical trials to validate the effectiveness and safety of fully endoscopic MVD. Second, although PTC secondary to VBD was successfully treated, the long-term effects of fully endoscopic MVD warrant further investigation. Finally, while this case confirmed the effectiveness of fully endoscopic MVD for PTC secondary to VBD, its applicability to other vascular abnormalities or more complex cases remains elusive. Therefore, future studies should explore the application of endoscopic techniques across different pathologic findings and compare their outcomes with those of traditional surgical methods.

Disclosure of conflict of interest

None.

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