Microvascular Compressicaude by Slinging a Tortuous Vertebrobasilar Artery to the Petrous Dura in Hemifacial Spasm: Technical Note

Decupressão microvascular por derrame de uma artéria vértebro-basilar tortuosa a dura petrosa no espasmo hemifacial: Nota técnica

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Abstract

Objective   In cases of hemifacial spasm caused by a tortuous vertebrobasilar artery (TVBA), the traditional treatment technique involves Teflon (polytetrafluoroethylene), which can be ineffective and fraught with recurrence and neurological complications. In such cases, there are various techniques of arteriopexy using adhesive compositions, 'suspending loops' made of synthetic materials, dural or fascial flaps, surgical sutures passed around or through the vascular adventitia, as well as fenestrated aneurysmal clips. In the present paper, we describe a new technique of slinging the vertebral artery (VA) to the petrous dura for microvascular decompression (MVD) in a patient with hemifacial spasm caused by a TVBA.

Method   A 50-year-old taxi driver presented with a left-sided severe hemifacial spasm. A magnetic resonance imaging (MRI) scan of the brain showed a large tortuous left-sided vertebral artery impinging and compressing the exit/entry zone of the 7th and 8th nerve complex. After a craniotomy, a TVBA was found impinging and compressing the entry zone of the 7th and 8th nerve complex. Arachnoid bands attaching the artery to the nerve complex and the pons were released by sharp microdissection. Through the upper part of the incision, a 2.5 × 1 cm temporal fascia free flap was harvested. After the fixation of the free flap, a 6–0 prolene suture was passed through its length several times using the traditional Bengali sewing and stitching techniques to make embroidered quilts called *Nakshi katha*. The 'prolenated' fascia was passed around the compressing portion of the VA. Both ends of the fascia were brought together and stitched to the posterior petrous dura to keep the TVBA away from the 7th and 8th nerves and the pons.

Result   The patient had no hemifacial spasm immediately after the recovery from the anesthesia. A postoperative MRI of the brain showed that the VA was away from the entry zone of the 7th and 8th nerves.

Keywords
► hemifacial spam
► microvascular decompression slinging of the vertebral artery
► arteriopexy to the petrous dura

Conclusion The 'prolenated' temporal fascia slinging technique may be a very good option of MVD in cases in which the causative vessel is a TVBA.

Resumo

Objetivo   Em casos de espasmo hemifacial causado por uma artéria vértebro-basilar tortuosa (AVBT), a técnica tradicional de tratamento envolve Teflon (politetrafluore-tileno), que pode ser ineficaz e repleta de recorrência e complicações neurológicas. Nesses casos, existem várias técnicas de arteriopexia utilizando composições adesivas, "Laços de suspensão" feitos de materiais sintéticos, retalhos durais ou fasciais, suturas cirúrgicas passados ou através da adventícia vascular, bem como aneurismas fenes-trados clipes. No presente artigo, descrevemos uma nova técnica de colagem da artéria vertebral (AV) à dura-máter para descompressão microvascular (DMV) em um paciente com espasmo hemifacial causado por um AVBT.

Método   Um taxista de 50 anos apresentava um espasmo hemifacial grave do lado esquerdo. Uma ressonância magnética (RM) de varredura do cérebro mostrou uma grande artéria vertebral tortuosa do lado esquerdo, colidindo e comprimindo a zona de saída / entrada do sétimo e oitavo nervos complexos. Após uma craniotomia, um TVBA foi encontrado colidindo e comprimindo a zona de entrada do sétimo e oitavo nervos complexos. Bandas aracnóides ligando a artéria ao complexo nervoso e a ponte foram liberadas por microdissecção acentuada. Aravés de seu comprimento várias vezes usando as técnicas tradicionais de costura e costura Bengali para fazer colchas bordadas chamado Nakshi katha, a fachada "prolonada" foi passada em torno da porção de compressão da AV. Ambas as extremidades da fáscia foram unidas e costuradas à dura porção posterior petrosa para manter o AVBT longe do sétimo e oitavo nervos e da ponte.

Palavras-chave
► spam hemifacial
► descompressão microvascular slinging da artéria vertebral
► arteriopexia a dura petrosa

Resultado   O paciente não apresentou espasmo hemifacial imediatamente após a recuperação da anestesia. Uma ressonância magnética pós-operatória do cérebro mostrou que a AV estava longe da zona de entrada do sétimo e oitavo nervos.

Conclusão   A técnica de sling de fáscia temporal "prolenada" pode ser uma opção muito boa de DMV nos casos em que o vaso causador é um AVBT.

Introduction

Hemifacial spasm results from the compression of the facial nerve root usually by cerebellar arteries, but sometimes major blood vessels, such as the vertebral and basilar arteries, may also cause hemifacial spasm. In hemifacial spasm, compression of the facial nerve fibers by a tortuous/ectatic vertebral artery (VA) is relatively common.[1-7] Dilation and elongation of the vertebral and basilar arteries with significant tortuosity is usually called megadolichovertebrobasilar anomaly, doli-choectasia of the vertebrobasilar artery, vertebrobasilar doli-choectasia, or vertebrobasilar artery tortuosity. It can manifest as intracranial hemorrhage, obstructive hydrocephalus, ische-mic strokes, spastic tetraparesis, trigeminal neuralgia, hemi-facial spasm or vagoglossopharyngeal neuralgia.[8-10] In such cases, complete decompression of the cranial nerve roots is very difficult and complicated, since the walls of a compressing vessel are rigid and sometimes densely atherosclerotic. There-fore, neurovascular decompression may be accompanied by several complications and recurrence of symptoms. Various modifications in the surgical technique of vascular decom-pression are used in these cases of tortuous vertebrobasilar artery (TVBA), from the placement of metal implants (between the nerve root and the rigid vessel) to adhesive fixation of the displaced vertebrobasilar artery.[1-7,11-26]

In the present paper, we describe a new technique to sling a TVBA to the petrous dura for microvascular decompression (MVD) in a patient with hemifacial spasm caused by a TVBA.

Case Report

A 50-year-old taxi driver presented with left-sided facial twitching that increased progressively over the course of two years; it was initially intermittent, but over the past eight months it was almost continuous, and persisted during sleep. He was treated several times with botulinum toxin injec-tions, and his condition was complicated by a case of keratoconjunctivitis. His hearing was reduced (between 30 to 40%) on the left side, with occasional tinnitus. He had no other systemic disease. Upon clinical examination, he had persistent hemifacial spasm on the left side marked by deviation of the angle of the mouth, with closure of the eyelids on the left side. He had significant sensorineural hearing loss on the left side. The Hoffman sign was bilaterally positive. Another neurological examination revealed no abnormalities.
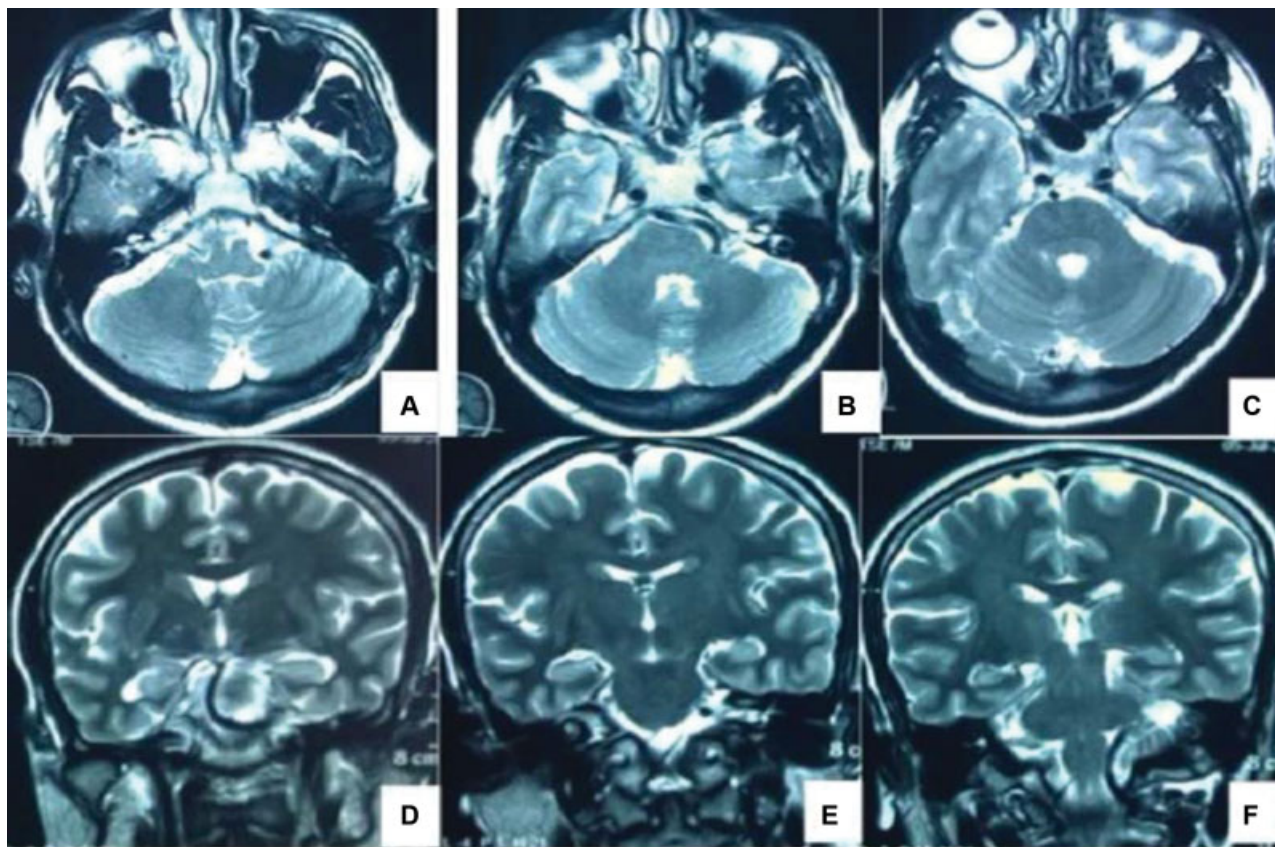
Fig. 1 Preoperative T2-weighted images of a magnetic resonance imaging scan of the brain in axial (**A, B, C**) and coronal (**D, E, F**) views showing that the exit/entry zones of the left-sided 7th and 8th nerves were deeply impinged by the tortuous vertebrobasilar artery (TVBA).

A magnetic resonance imaging (MRI) scan of the brain showed a large tortuous left-sided VA impinging and compressing the entry zone of the 7th and 8th nerve complex (►**Fig. 1**). Pure tone audiometry (PTA) showed left-sided moderate sensorineural hearing loss. After appropriate counseling, the patient underwent a surgical intervention.

Operative Techniques

Under general anesthesia with endotracheal intubation, the patient was positioned in 'three quarter prone' by keeping the left side up. Nerve monitors were put in position. A left-sided retromastoid retrosigmoid lateral suboccipital craniotomy was performed. The dura was opened under the operating microscope. Upon exploration of the left cerebellopontine angle, a large tortuous VA was found. The artery was impinging and compressing the entry/exit zone of the 7th and 8th nerve complex (►**Fig. 2 & 3**). There were thick and tight arachnoid bands attaching the artery to the nerve complex and the pons, which were released by sharp microdissection. The dissection started just above the lower cranial nerves and ascended progressively (using the bottom-up technique) to free the entry/exit zone of the 7th and 8th nerves. The arterial walls were thick and rigid but not atherosclerotic. The compressing portion of the VA was free of branches and could easily be mobilized from the nerve entry/exit zone with a microdissecting instrument,

but it returned to a point close to its original position after the removal of the dissecting instrument.

Through the upper part of the incision, a 2.5 × 1 cm temporalis fascia free flap was harvested. After fixation and stabilization of the free flap, a 6–0 prolene suture was passed through its length several times using the traditional Bengali
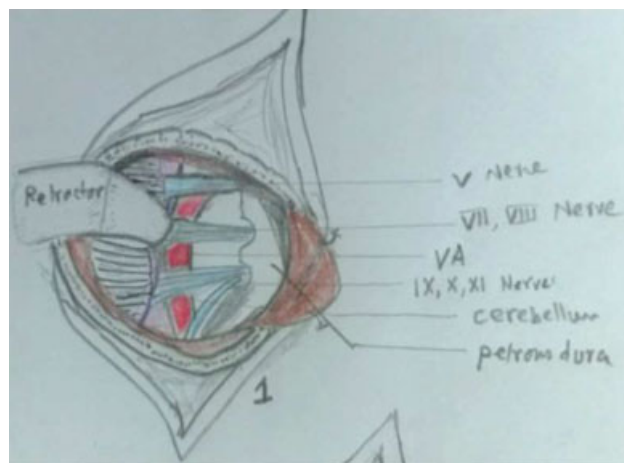

Fig. 2 Schematic (pencil drawing) perioperative picture of the operative findings, that is, compression of the root exit/entry zone of the 7th and 8th nerves at the cerebellopontine angle by the tortuous vertebrobasilar artery (TVBA).
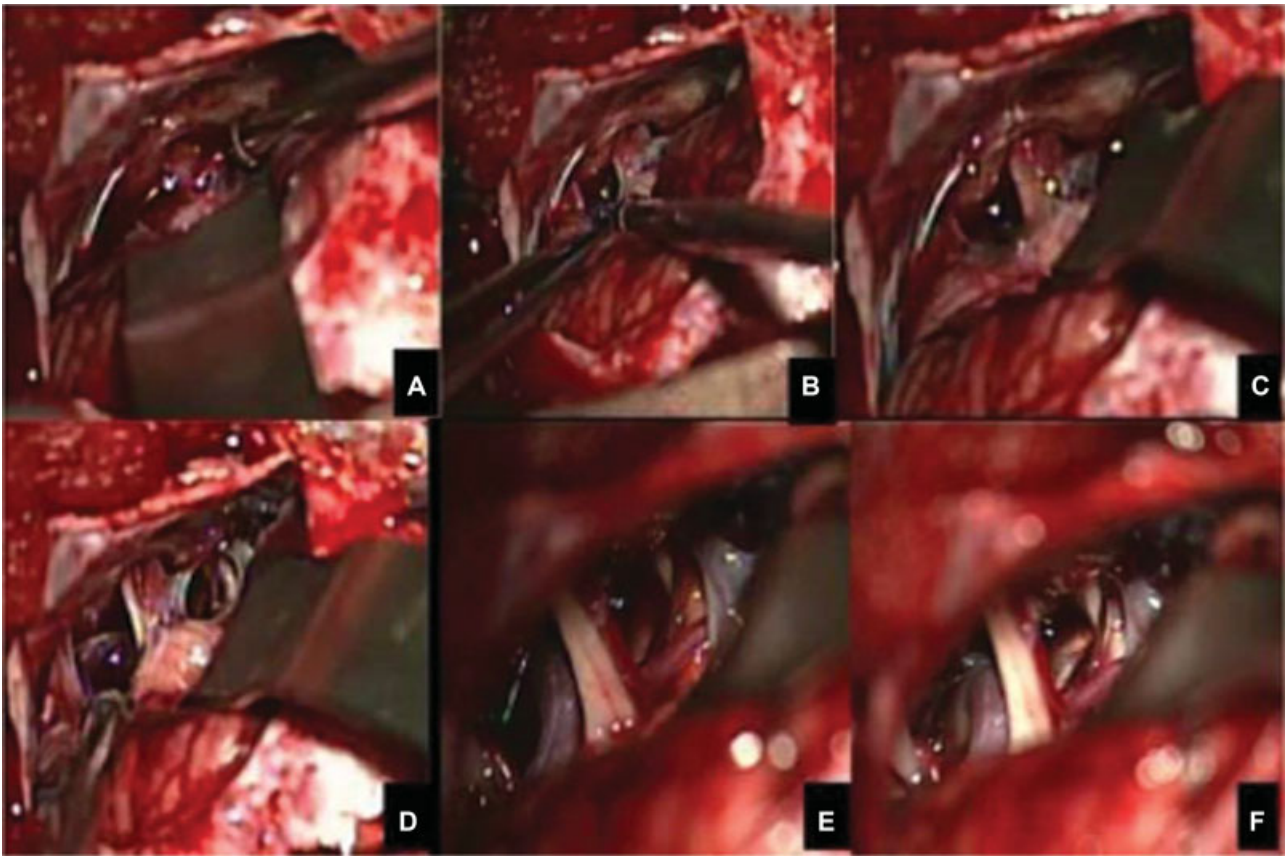
Fig. 3 (**A, B, C, D, E** and **F**) Sequential perioperative pictures showing exposure of the compression of the nerve exit/entry zone (7th and 8th nerves) by the tortuous vertebrobasilar artery (TVBA) at the left cerebellopontine angle.
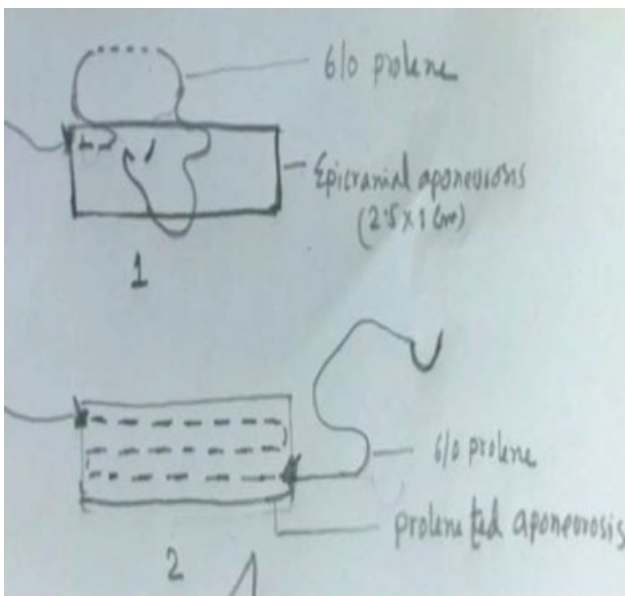

Fig. 4 Schematic (pencil) drawing of techniques of 'prolenation' of fascia or aponeurosis for slinging of TVBA.

sewing and stitching techniques to make embroidered quilts called *Nakshi katha* (►**Fig. 4** & **5**). The 'prolenated' (sutured with prolene) fascia was passed around the compressing portion of the VA. Then, both ends of the fascia were brought together and stitched to the posterior petrous dura (taking great care not to injure the surrounding neurostructures) to keep the TVBA away from the 7th and 8th nerves and the pons (►**Fig. 6** & **7**). After an arteriopexy, Surgicel (Ethicon, Bridgewater, NJ, US) was placed between the VA and the nerves. The wound was closed accordingly, without a drain.

Postoperative Course

The patient had no hemifacial spasm immediately after the recovery from the anesthesia, but he developed facial paresis (Brackmann-House (B&H) grade 2), a more intense tinnitus (in comparison with the preoperative status), and further hearing deterioration on the left side.

Three months after the operation, the patient's facial paresis improved to B&H grade1, and the hearing also improved, but was still worse in comparison with the preoperative state. Though improved, he had occasional annoying tinnitus.

During the follow-up after 6 months, the patient reported further improvement of the tinnitus, with less intensity and frequency. The other neurological statuses were stable, with absence of the Hoffman sign. An MRI scan of the brain showed that the VA was away from the entry zone of the 7th and 8th nerves (►**Fig. 8**).

During the follow-up after 12 months, the patient had no hemifacial spasm, but a persistent occasional tinnitus in adoptive form.
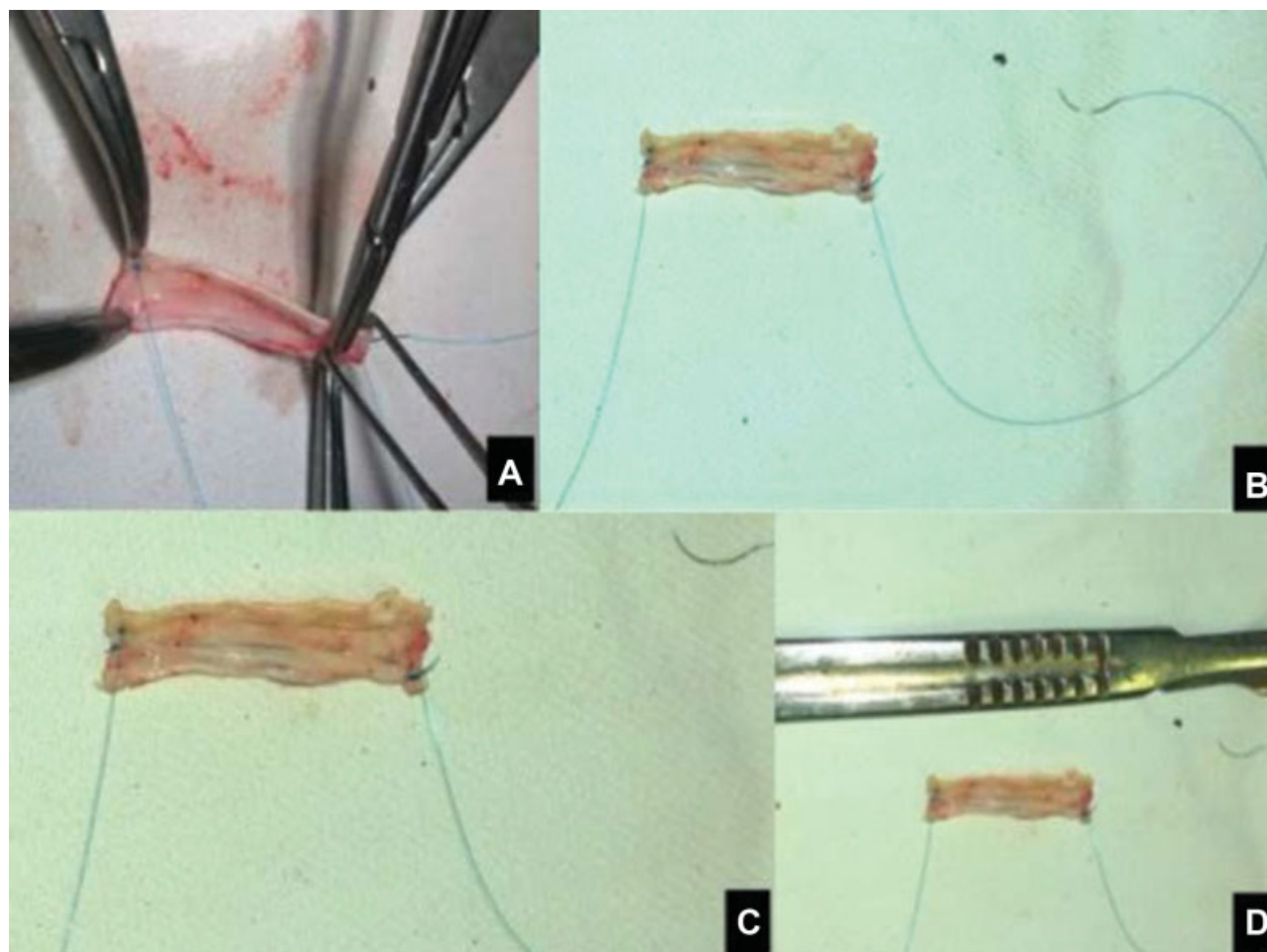
Fig. 5 (A, B, C and D) perioperative pictures of 'prolenation' of temporalis fascia with 6-0 prolene in the techniques of 'Nokshi katha' stitching.
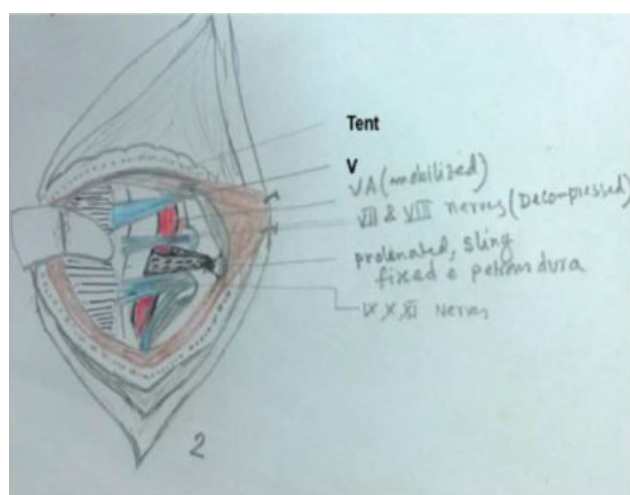

Fig. 6 Schematic (pencil drawing) perioperative picture after slinging of TVBA by 'prolenated' fascial sling with petrous dura.

Discussion

Hemifacial spasm was first described by Gowers in 1884. It is a segmental myoclonus of facial muscles innervated by the 7th cranial nerve. This disorder usually presents in the fifth or sixth decades of life, and occurs almost always unilaterally.

Bilateral involvement can occur rarely. Hemifacial spasm usually starts with short clonic contractions of the orbicularis oculi, and spreads to other facial muscles over the years (corrugator, frontalis, orbicularis oris, platysma, and zygomaticus muscles).[27,28]

It is believed that irritating stimuli of the 7th nerve nucleus in the pons causes hyperexcitability of the nucleus, while such irritation to the proximal segment of the nerve may cause ephaptic transmission within the 7th nerve. Both mechanisms explain the involuntary rhythmic myoclonus observed in hemifacial spasm.[29]

Tumor, arteriovenous malformation, Paget disease, or other compressive lesions, as well as stroke, multiple sclerosis and basal meningitis, or other non-compressive lesions, may present clinically as hemifacial spasm. Most cases are idiopathic, and are due to compression by aberrant vessels such as the distal branch of the anterior inferior cerebellar artery or a VA on the root exit zone of 7th nerve in the cerebellopontine angle.[27]

Injections of botulinum toxin are the initial treatment in most cases of hemifacial spasm. Transient relief of the spasm lasts for 3 to 6 months, and begins within 3 to 5 days after the injection. Sometimes, it may present some serious complications, such as ptosis, persistent spasm, diplopia, exposure keratitis/ulcer, facial asymmetry etc.[27,28]
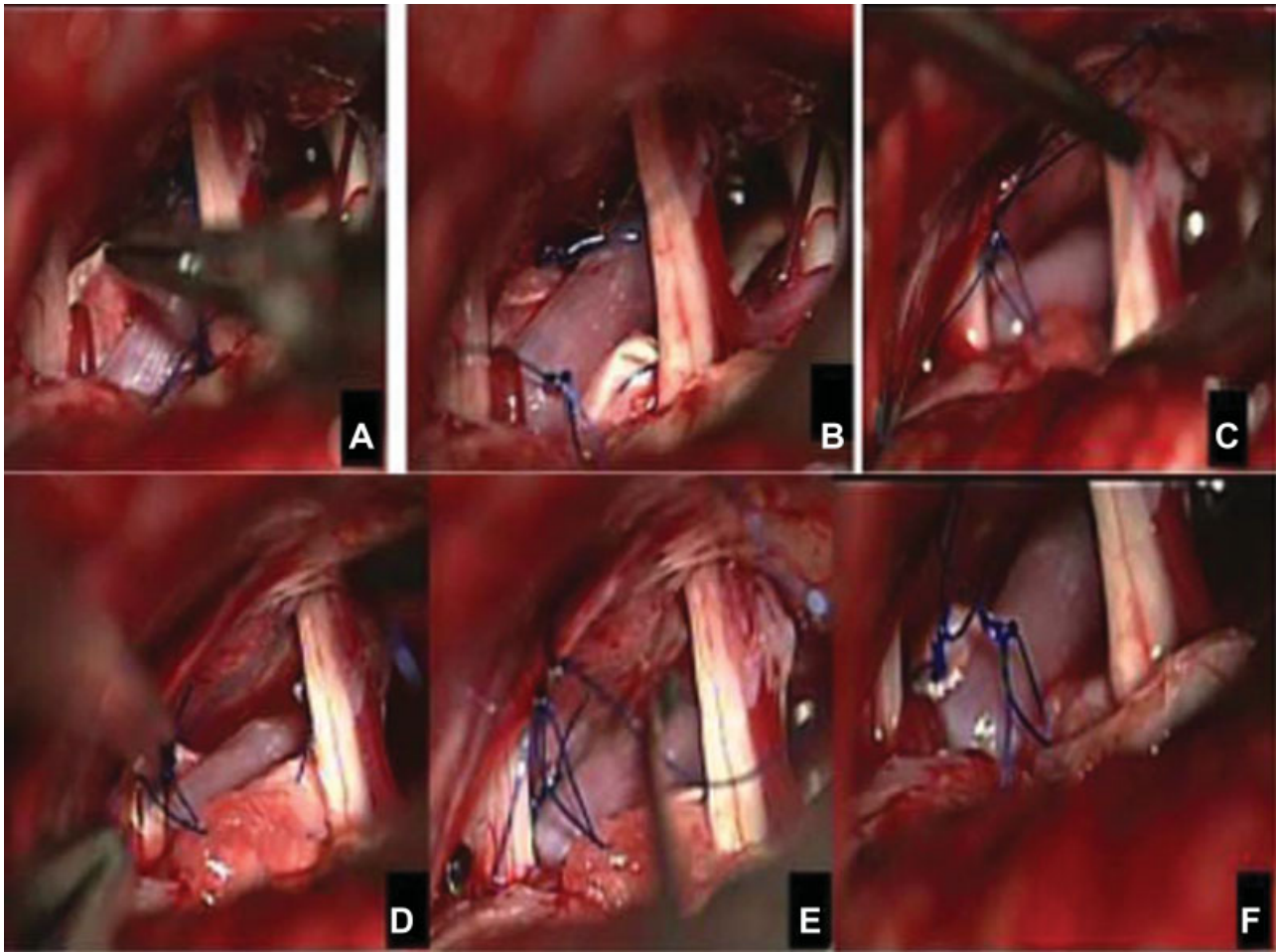
Fig. 7 (A, B, C, D, E and F) Sequential perioperative pictures showing the slinging of the tortuous vertebrobasilar artery (TVBA) by a 'prolenated' fascial sling to the petrous dura at the left cerebellopontine angle decompressing the nerve exit/entry zone (of the 7th and 8th nerves).

Most of the time, the patients become frustrated with the injection, and want a permanent solution.

Carbamazepine and benzodiazepines are used in the treatment of non-compressive hemifacial spasm. In patients who refuse botulinum toxin injections and surgical decompression, carbamazepine, benzodiazepines, and baclofen may also be used, but this form of treatment is usually not effective. Surgical treatment is required in cases of compressive lesions. Microvascular decompression surgery can be an effective treatment for those patients who do not want/ respond to botulinum toxin or have complications after the injection.[27,28,30]

When the patient is fit for surgery and the cause is compression of the nerve exit zone by a vascular loop, the definitive choice of treatment should be MVD.

Ectatic vessels can cause hemifacial spasm by compressing the exit zone of 7th nerve. The MVD of these vessels can have an excellent outcome.[31,32]

Cases of apparent idiopathic hemifacial spasm may benefit from surgical exploration and MVD.

W. Dandy[33] was the first to detect the compression of a thickened vertebrobasilar artery as a cause of trigeminal neuralgia, though he used the term 'cirsoid aneurysm' for the vessel. E. Campbell and C. Keedy,[34] and W. Gardner and G.

Sava[35] described the compression of the nerve root entry zone of the facial nerve caused by adjacent loops of the tortuous and dilated VA.[1]The TVBA is more commonly involved in the neurovascular conflict in patients with hemifacial spasm (14%) than in those with trigeminal neuralgia (2.8–7.7%).[1,2,4,5,18]

The patients with vertebrobasilar artery compression were older and predominantly male, with predominant involvement of the left side of the face and high correlation with ipsilateral hemifacial spasm and hypertension.[18] The higher frequency of left-sided involvement can be understood by the asymmetric diameter of the VAs with higher caliber on the ipsilateral side. This branching of the left VA from the subclavian artery originating directly from the aortic arch is another important factor that results in more pronounced pulse wave transmission on the left side as opposed to the right VA originating from the brachiocephalic trunk. All of these hemodynamic factors result in tortuosity and dolichoectatic changes in the distal (intracranial) segments of the left VA and the whole vertebrobasilar artery.[1,8,9]

T. Fukushima operated 1,663 cases of hemifacial spasm, and in 232 (14%) cases, compression (of the exit zone of the facial nerve) was caused by a tortuous VA.[2] In a study by M. Samii et al,[5] who reported 143 cases of hemifacial spasm, facial nerve
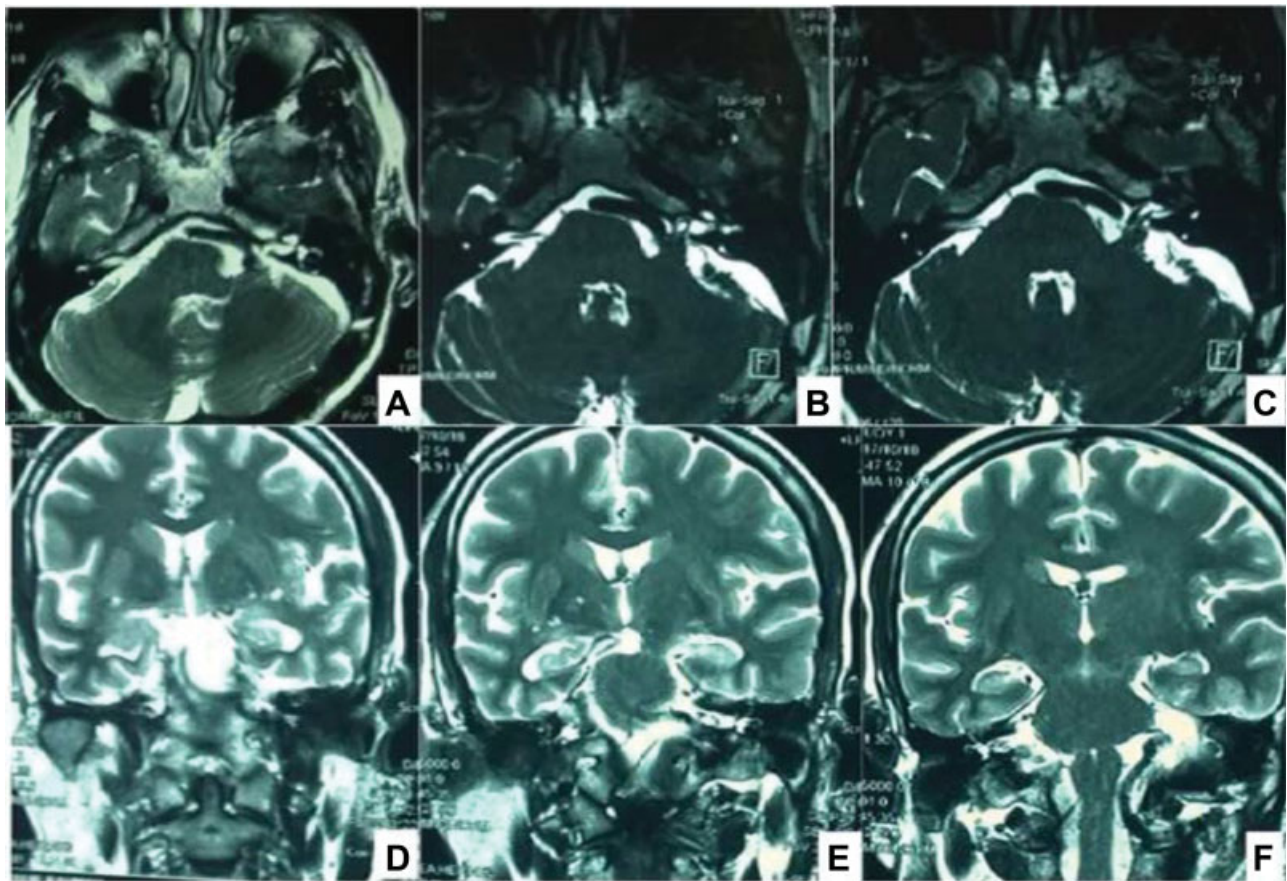
Fig. 8 Postoperative T2-weighted images of amagnetic resonance imaging scan of the brain in axial (A, B and C) and coronal (D, E and F) views showing that the left sided tortuous vertebrobasilar artery (TVBA) was shifted anterio-medially with the free exit/entry zones of the 7th and 8th nerves.

compression by the VA was found in 32 (26.6%) cases; isolated involvement of the VA was found only in 6 (4.2%) cases; and both the anterior and posterior inferior cerebellar arteries were the culprit vessels in the rest of the cases. S. Nagahiro et al[4] have observed compression of the facial nerve exit zone by the vertebral artery in 14 patients out of 68 operated cases with hemifacial spasm, but in 11 of them the nerve root was also compressed by the cerebellar artery.

The increased frequency of postoperative complications of the cranial nerves is due to many surgical manipulations for the mobilization and transposition of the major vessels. Major-vessel displacement is associated with the action of both surgical instruments and dense arterial structures, changing relationships with adjacent neurostructures on the surrounding cranial nerves (4th, 6th, 8th, 9th and 10th cranial nerves).[1] Postoperatively, hearing loss, facial weakness, lower cranial nerve dysfunctions, and ischemic strokes of the medulla oblongata are more common in patients with hemifacial spasm caused by TVBA compared with patients with compression of the facial nerve by the cerebellar artery.[1,18]

Hemifacial spasm disappears immediately after neurovascular decompression in most of the cases. In the long-term follow-up, the symptoms recur only in a few cases. In hemifacial spasm caused by a TVBA or cerebellar artery, the success rate is similar (in the long-term follow-up), and is close to 90%.[4,18]

The primary target of the MVD is to eliminate the pulsating effects of the vessels at the entry/exit zones of the cranial nerves. The most frequently used technique is microsurgical interposition, in which a few small synthetic implants, typically pieces of Teflon (polytetrafluoroethylene) wool, are sequentially placed between the cranial nerve root and the compressing culprit vessel. This technique has been successfully used not only in patients with compression caused by cerebellar arteries with relatively small diameters, but also in cases of compression caused by bigger vessels, such as a TVBA. However, the use of the microsurgical interposition technique to eliminate the compression effect caused by big arteries has some limitations and disadvantages. Apart from the common complications, such as the displacement of the installed implant and the formation of foreign body granulomas in the postoperative period, the important characteristic of this method is the necessity to insert a significantly larger-than-usual amount of implanted material between the cranial nerve and the ectatic vessel. This MVD technique results in the displacement and deformation of the cranial nerve root itself, instead of in the retraction/displacement of the major vessel away from the primary position, due to the high density/thickness of its walls and the high intraluminal pressure. The interposition technique has a higher success rate regarding the elimination of trigeminal neuralgia, though there is a higher chance of sensory disturbances on the face (the effect of partial rhizotomy), but its use in

hemifacial spasm may almost inevitably result in postoperative paresis of the 7th nerve and hearing disturbance.[1]

In order to eliminate the compression of the nerve roots caused by a TVBA, isolation techniques in combination with the interposition technique are used in many ways. The isolation technique may be performed by wrapping the nerve roots and/or compressing vessels with many types of implants (in the form of strips and bands), as well as by implanting cylindrical and fenestrated aneurysmal clips separating the vascular and neurostructures from each other. However, this surgical technique is not fundamentally different from the interposition technique, because the implants are in contact with the cranial nerve roots, and need a much larger number of surgical manipulations. Extreme variants of this kind of surgical 'redundancy' were shown in a few cases of MVD in which the titanium implants designed for the fixation of the craniotomy bone flaps were used to isolate the TVBA from the 5th cranial nerve root.[12,24]

The transposition of the arteries involved in the neurovascular decompression with minimal surgical impact on the neurostructures is the most effective and acceptable method of MVD. During the operation, after the identification of the culprit vessels in the entry/exit zones of the corresponding cranial nerve roots, the objective of the surgery is to mobilize and displace the artery from its original site, and to place and keep it in the new position. Mobilization and placement of major vessels away from nerve roots is a complicated operation that requires skill because of the severe tortuosity and high density of the atherosclerotic walls. Sharp microdissection of the arachnoid membranes fixing the TVBA to the brainstem should begin below the level of the affected cranial nerve root. In cases of hemifacial spasm, microdissection should be started with the separation of the caudal group of cranial nerves. The method of phased 'bottom-up' arachnoid microdissection along the brainstem enables the gradual mobilization of the TVBA, the assessment of its mobility, and the timely identification of the entrances of the cerebellar and brainstem arteries hidden by nerve roots.[1]

Implant placement between the brainstem and the artery is technically simple. Numerous Teflon implants in the form of pellets and lumps to tampon the space formed after the mobilization of the compressing artery are used by most of the authors, which gives reliable fixation of the newly formed neurovascular relationships. Muscles, the fascia, fat, cotton, surgical materials etc. are the other materials that can be used.[1]

Grigoryan et al,[1] in 2016, published a series of MVD associated with vertebrobasilar artery tortuosity in which the TVBA was mobilized by microdissection of the arachnoid membranes between the artery and the brainstem and retracted laterally. Then, the TVBA was displaced away from the brainstem in the caudo-rostral direction. These microsurgical manipulations resulted in 'spontaneous' decompression of the cranial nerve roots without implantation of prostheses between the vessel and the nerve root entry/exit zone. In most cases (28 out of 30), they used pieces of muscle and adipose tissue, which were inserted in a phased manner for the fixation of the displaced artery away from the brainstem. The place-

ment of tissue autoimplants is easy to perform due to the possibility of arbitrary modeling of the size of the tissue pieces, and it requires no additional surgical procedures after the mobilization and displacement of the artery. During the placement of the implant in the 'bottom-up' direction and gradual TVBA retraction from the brainstem, the mobilized and displaced artery 'spontaneously' moves away from the entry/exit zone of the compressed nerve. Because of the 'spontaneous' decompression, microsurgical manipulation on the cranial nerve roots is not performed, and the implants are not inserted between the TVBA and the nerves, which avoids the development of postoperative cicatricial deformity of the cranial nerve fibers. Newly formed neurovascular relationships are further strengthened with fibrin glue, which preserves the spatial arrangement of the mobilized and displaced artery until the final fixation of the TVBA by cicatricial adhesions with the autoimplant and the dura mater. No recurrence of clinical symptoms was observed in the series.[1]

There are various techniques of microsurgical arteriopexy. Arteriopexy can be performed using adhesive compositions, 'suspending loops' made of synthetic materials, dural and fascial flaps, microsurgical sutures passed around or through the vascular adventitia, as well as fenestrated aneurysmal clips.[6,11,13–16,19–26] The double-stick tape technique for the transposition of an offending vessel in MVD in hemifacial spams was described by Ichikawa et al in 2011.[16]

The use of sling/loop fixation of the artery is the most attractive and reasonable way of performing neurovascular decompression, since: the success rate is very high; it eliminates the need to install a large implant; it reduces the chance of developing aseptic granuloma; and it reduces the chance of postoperative neurological complications that occur due to the placement of large implants.

However, the implementation of this technique requires the expansion of the surgical field by increasing cerebellum retraction, and may be accompanied by additional injury to both nervous and vascular structures, with increased number of postoperative complications. Additionally, it is a more complicated procedure.[6,11,13–16,19–26]

Ferreira et al, in 2011,[14] described vertebral arteriopexy for MVD of the facial nerve in the treatment of hemifacial spasm in 6 patients. During the operation, after the identification of the site of VA compression, the artery was adequately mobilized and displaced for the decompression. Great actions that require skill were taken to avoid kinking/damage of the perforating arteries arising from the VA. Two 8–0 nylon microsutures were passed through to the adventitia of the VA and then through the clival or petrous dura, and then tied carefully to alleviate compression on the facial nerve. Postoperatively, all patients had complete resolution of the hemifacial spasm, but one patient suffered from hearing loss, another developed a postoperative transient unilateral vocal cord paralysis, and a third patient developed a pseudomeningocele that resolved with the placement of a lumbar drain.[14]

Lin et al, in 2012,[19] performed vertebral or basilar artery mobilization and transposition using the vascular sling with a strip of non-absorbable dural tape. The vertebrobasilar artery-sling complex was then fixed to the petrous dura by

an aneurysm clip through the dural bridge. The direction and angle of traction on the vertebrobasilar artery was adjusted using different lengths of clip or the horizontal level of the dural bridge. They used this technique in seven cases with very good results. They concluded it is an easy and adjustable way to perform MVD safely and effectively.

Here we describe the new modified technique to perform the slinging of a tortuous and large ectatic VA using the 'prolenated' temporal fascial sling on the petrous dura.

Advantages of this Technique

- The temporal fascia/epicranial aponeurosis can be easily harvested from the same wound.
- It is autologous, so there is no fear of an immune response.
- It is biologically modifiable.
- Due to the use of the prolene suture, it becomes non-absorbable (to some extent) and sufficiently strong.
- As prolene is inert to the body tissue (fascia/aponeurosis), there is very little chance of developing foreign body granuloma.
- The 'prolenation' of the fascia/aponeurosis avoids the folding and twisting of the fascia/aponeurosis during the slinging to the petrous dura.
- There is an equal pressure on all surfaces of the artery that are in contact with the sling.
- The acute angulation of the artery is much less probable.
- There would be a lower chance of perforator insult (by injury, acute angulation or occlusion).
- The chance of arterial penetration is absent. Therefore, thrombosis or infarct should be less probable.
- The force, direction and angulation of the traction on the ectatic vessel are adjustable.

Disadvantages of this Technique

- It may not be applicable when sufficient perforator-free vertebrobasilar vessel is not available.
- There is a chance of iatrogenic injury.
- A high microsurgical skill is required.

Conclusion

The present paper is a technical single case report. Therefore, any decision-making comments cannot be made. But The 'prolenated' temporal fascia slinging technique may be a very good option of MVD in cases in which the causative vessel is a TVBA.

Conflict of Interests
The authors have none to disclose.

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