BRAINSTEM COMPRESSION SYNDROME CAUSED BY VERTEBROBASILAR DOLICHOECTASIA

Microvascular repositioning technique

Arthur de Azambuja Pereira-Filho1, Mario de Barros Faria1, Cristina Blei2, Jorge Luiz Kraemer3

Verteobasilar dolichoectasia (VBD) is an anatomic variant that consists of enlargement and dilatation, often associated with a tortuous and elongated vessel. The anomaly is probably due to a marked thinning of the internal elastica lamina and media, most likely as a consequence of prolonged systemic arterial hypertension. It accounts for approximately 3 to 5% of all cerebellopontine mass lesions. A variety of clinical syndromes have been related due to pulsatile compression by the aberrant vessel: cerebellar dysfunction, hydrocephalus, ischemic stroke, transient or permanent motor deficits, central sleep apnea, trigeminal neuralgia, as well as brain stem compression syndrome. Microvascular decompression surgery was introduced in the 1960s and was initially used to treat trigeminal neuralgia, hemifacial spasm and glossopharyngeal neuralgia. Lately, it was used to treat brainstem dysfunction caused by an ectatic vessel. Nowadays, microvascular decompression with repositioning of the ectatic vessel is a new technique that has been used successfully.

The purpose of this study is to report and discuss a rare case of brain stem compression syndrome caused by verteobasilar dolichoectasia successfully treated with microvascular decompression repositioning technique and documented by computed tomography angiography (CTA) and magnetic resonance imaging (MRI).

CASE
A 60-years old man with a past medical history of diabetes mellitus type 2 sought neurological treatment after experiencing mild progressive disatraxia for eight months. He did not have other complaints.

The patient's neurological examination revealed, besides the speech abnormality, left side pyramidal syndrome with hiperreflexia and Babinski's sign. All the other aspects of the neurological examination were intact.

A MRI (Fig. 1 - Axial) showed an elongated and tortuous verteobasilar artery that crossed the ventral aspect of the medulla oblongata causing mechanical compression at the left side.

Imaging investigation
MRI and CTA showed an elongated and tortuous vertebrobasilar artery that crossed the ventral aspect of the medulla oblongata causing mechanical compression at the left side (Figs 1 and 2).

Surgical technique
The patient was placed in the prone oblique (park bench) position, and a left far lateral suboccipitometastamoidea approach was performed with left vertebral artery exposure. The dura was opened, and cerebrospinal fluid was released at the cisterna magna to provide a capacious working environment.

Arachnoid dissection revealed a large vascular structure, identified as the basilar dolichoectatic artery, dislocating and compressing the brain stem (medulla oblongata) in its left ventral region. As soon as the neurovascular conflicting area was

SÍNDROME COMRESSIVA BULBAR CAUSADA POR DOLICHOECTASIA VERTEBROBASILAR: TÉCNICA DE REPOSIÇAO MICROVASCULAR

1Medical Resident in Neurosurgery, Hospital São José/Compleeto Hospital Santa Casa de Porto Alegre, Porto Alegre RS, Brazil (FSC/CHSC); 2Medical Student at Fundação Faculdade Federal de Ciências Médicas de Porto Alegre, Porto Alegre RS, Brazil (FFCFM/CHSC); 3Postgraduate Professor, FFCFM/CHSC, Neurosurgeon HSJ/CHSC.

Received 5 November 2007, received in final form 17 April 2008. Accepted 28 April 2008.

Dra. Cristina Birlem Blei - Rua Henrique Sclar 284 - 91220-520 Porto Alegre RS - Brasil. E-mail: crisblei@terra.com.br
identified, microvascular decompression repositioning technique, adapted from Yoshimoto et al, was performed and the tortuous dolichoectatic vertebrobasilar artery was pulled toward and fixed with unabsorbable nylon on the nearby dura mater, achieving a very satisfactory decompression result (Fig 3).²

**DISCUSSION**

Vertebrobasilar dolichoectasia is a potentially severe condition that may cause severe disability due to ischemic or compressive dysfunction in the posterior fossa. It is an uncommon entity that affects less than 0.05% of the population, and accounts for approximately 3 to 5% of all cerebellomedullary angle mass lesions. Small cases series observed that the survival rate in VBD after 3 years follow-up was found to be 60%³. The epidemiological data about VBD are limited. Ubogu et al. reviewed 1440 magnetic resonance angiography (MRA) between 1995 and 1997, and found a VBD prevalence of 4.4% in their series. The same author also found that VBD is more common in women, 32 of 45 cases, and equally distributed between whites and african-american. Among their sample, 42% of the patients had incidental VBD findings on MRA performed on various indications other than posterior circulation dysfunction⁴. Resta et al. observed 132 patients with dolichoectatic vertebrobasilar artery among 2256 angiographies, and only 7.73% have had neurological symptoms⁵. Necropsies series showed that 30% of the patients with the abnormality had symptoms, which lead us to think that the real incidence of vertebral ectasia and the frequency by which this anatomic feature induces neurological symptoms are still uncertain⁶.

Some authors suggest that VBD may be a congenital vasculopathy of the elastic layer of the arterial wall, and may be cause of posterior circulation dysfunction independent of atherosclerosis disease affecting the intimal layer⁷. Others suggest a hypothesis that try to explain

---

**Fig 2. MRI (T1 Gadolinium - Sagittal) showed an elongated and tortuous vertebrobasilar artery that crossed the ventral aspect of the medulla oblongata causing mechanical compression at the left side.**

---

**Fig 3. Left for lateral suboccipitotonsillar approach transoperative photography showing the microvascular repositioning technique. M.O., medulla oblongata; P, posterior inferior cerebellar artery; V.A., Vertebral artery; XII, hypoglossal nerve; X, vagus nerve.**
the posterior circulation ischaemia by the induced athro
erosclerosis in dolichoectatic arteries at the regions of
maximum angulation. Some still believe in the corre-
lution between prolonged systemic arterial hypertension
and the marked thinning of the internal elastic lamina and
media of the abnormal vessel.

Padget et al., in 1954, proposed a developmental origin
as a consequence of the zig zag pattern that the verte-
bral artery assumes during its development, to explain
the artery tortuosity, although degenerative changes in
the vessel might be considered. Obougu et al. suggest that
VBD is part of a more widespread vasculopathy. These au-
tor also studied the radiological features of the VBD and
demonstrated that the major VBD location is the basilar
artery alone (40%), followed by bilateral vertebral arter-
es, 22% basilar artery and both vertebral arteries, 16%.
Basilar artery and single vertebral artery presentation, as
demonstrated in the present case, account for only 4%
of VBD location.

A cohort study define VBD as a basilar artery or verte-
bral artery diameter >4.5 mm or deviation of any portion
of them higher than 10 mm from the shortest expected
course, or basilar length >29.5 mm or intracranial verte-
bral artery length >23.5 mm. Smoker et al. judged a basilar
artery to be elongated if, at any point along its course, it
lay lateral to the margin of the clivus or dorsum sellae or
bifurcated above the plane of the suprasellar cistern.

Since there are no well established imaging criteria
for VBD, some authors suggest that the clinical diagno-
sis of symptomatic VBD should be made based on poste-
rior circulation dysfunction in the absence of significant
stenotic or occlusive disease of the posterior circulation
with an ectatic and tortuous vessel present on angiogra-
phy, and no other potential cause for the symptoms. The
usefulness of neuradiological methods such as computed
tomography and MRI are still controversial. MRI sensitiv-
ity and specificity to diagnose VBD are unknown. Some
authors emphasized that good results depend on the cor-
correct MRI projection (oblique sagittal) and the appropriate
gradient echo sequences. However, this is an acceptable
method for diagnose and reduces morbidity of an invasive
procedure.

The VBD anomaly can cause two kinds of symptoms: those resulting from the compression of structures ad-
 antic to the aberrant vessel, such as the brainstem and
cranial nerves roots compression and those resulting of
 ischemic events. Cerebellar dysfunction, hydrocephalus,
trigeminal neuralgia, ischemic stroke and medulla oblon-
gata compression are some of these syndromes that have
been associated with the pulsatile compression produced
by the ectatic vessel. Levine et al. previously reviewed lit-

interposing Teflon felt or padding or other synthetic implants between the offending vessel and the nerves.  

Brainstem compression caused by VBD can not be treated using an ordinary technique of microvascular decompression. In the surgical strategy of this condition, it is not sufficient to achieve decompression only by insertion of prosthesis between the vessel and the brainstem, because the size and stiffness of the dilated artery makes standard microsurgery difficult. Moreover, the overall size of the ectatic vessel and its increased pulsatility do not allow an effective separating action by the prosthesis. Besides, an increased neural compression may be expected with the placement of prosthesis in the conflict area in such condition. Therefore, alternative surgical techniques should be considered.

Different techniques have been described by some authors in the literature. Ogawa et al. reported the use of a synthetic vascular graft sutured to the clival dura to move the vessel away from cranial nerves. A silicone sling sutured to the petrous dura to reposition the basilar artery away from the trigeminal nerve was used by Stone et al. A vascular clip graft or a Silastic ring around the trigeminal nerve to isolate it from the offending vessel was reported by Laws et al. and Yoshimoto et al., respectively. A Silastic rubber sling stitched to the dura was used by Rawlins and Coakham in patients with hemifacial spasm. Fujikawa and Kondo reported the use of Teflon slings and pieces of Surgicel to mobilize the offending vessel and promote fibrous adhesions of the Teflon to the dura. Recently, alternative techniques for neurovascular decompression of the brain stem have been reported, however its long-term success rate is still unknown and other studies are needed in order to prove its efficacy.

In the present case, we used a very simple microsurgical repositioning technique: the tortuous dolichoectatic vertebrobasilar artery was pulled toward and fixed with an absorbable nylon on the nearby dura-mater, achieving a very satisfactory brain stem decompression result. The follow-up period in this case is not so long, but the excellent postoperative outcome obtained until today make us believe that the cure in this patient is very likely to be achieved.

In conclusion, vertebrobasilar dolichoectasia is a very rare cause of brain stem compression and a satisfactory outcome can be obtained with microvascular repositioning technique.

REFERENCES
22. Hikitaonuma T, Matsubahima T, Inoue T. Microvascular decompression for treatment of trigeminal neuralgia, hemifacial spasm, and glossop-