Magnetic resonance imaging criteria in vascular compression syndrome

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Objectives
The aim of this work is to show the imaging criteria of the offending vessel in neurovascular compression syndrome in the cerebellopontine angle using MRI. This will increase the acceptance of the concept of vascular compression syndrome of the cranial nerves as an etiology of trigeminal neuralgia, hemifacial spasm, glossopharyngeal neuralgia, and some cases of disabling vertigo and tinnitus.

Materials and methods
Between 1994 and 2008, MRI radiographs of 782 cases of vasculoneural compression were reviewed to analyze the imaging criteria of the offending vascular loop and were correlated with intraoperative vascular decompression findings in various pathologies such as hemifacial spasm, trigeminal neuralgia, glossopharyngeal neuralgia, vertigo, and tinnitus.

Results
The specificity and high sensitivity of the T2 constructive interference in steady state or fast spin eco sequence MRI allowed us to precisely define the vasculoneural conflict in almost all cases and to highlight certain radiological criteria required for a vasculoneural conflict diagnosis in general. These criteria are mainly the presence of a vascular loop that is perpendicular to the course of the nerve and the presence of distortion of the neural structures.

Conclusion
MRI in the T2 sequence is the key assessment in neurovascular compression syndrome.

Keywords:
compression syndrome, MRI, offending vessel

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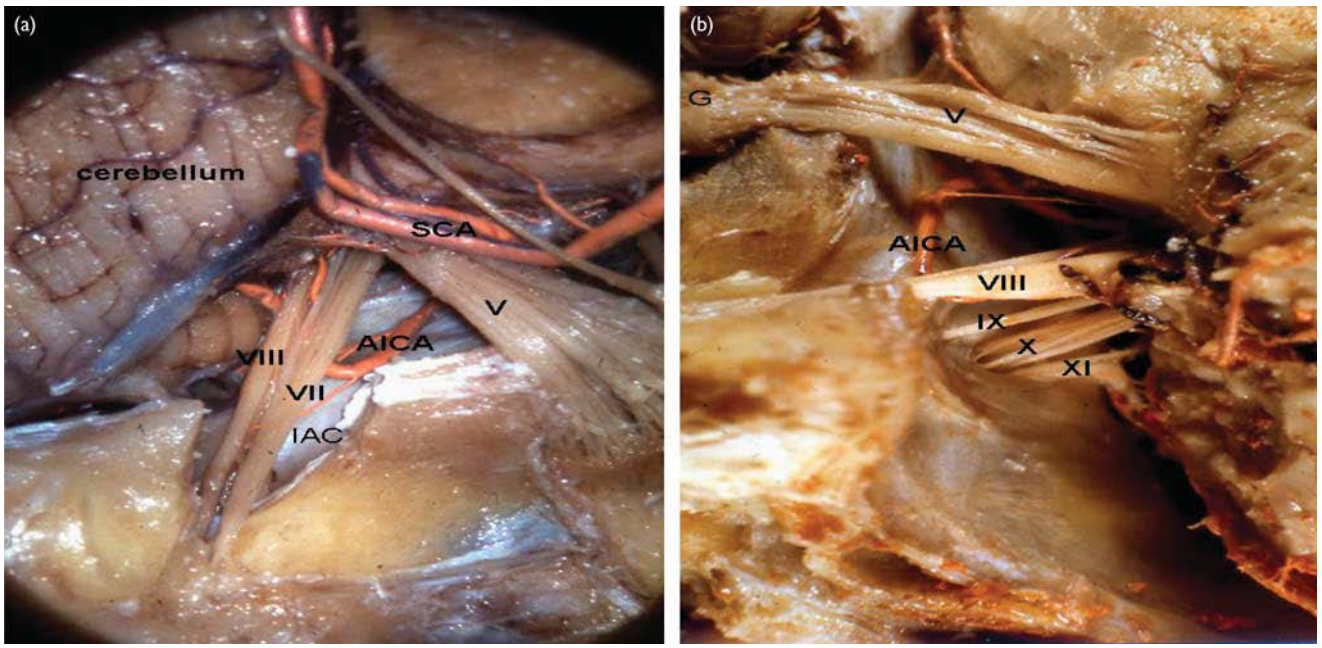
Introduction

The cerebellopontine angle (CPA) is a small compact area characterized by multiple vascular and neural structures, which are normally in close relation to each other sometimes, the close contact of a vascular loop to a neural structure becomes symptomatic, leading to the so-called neurovascular compression syndrome, and the vascular structure becomes an offending vessel. Trigeminal neuralgia, hemifacial spasm, glossopharyngeal neuralgia, tinnitus, and vertigo are examples of the neurovascular compression syndrome. Many surgeons were a little bit reluctant to believe in the vascular compression syndrome. Nowadays, we believe that the term idiopathic trigeminal neuralgia or hemifacial spasm is no longer accepted and neurovascular compression is the replacement description and a vascular decompression operation is a solution to this problem. Nowadays, vascular compression syndrome is well established thanks to the assessments of preoperative images and the results of minimally invasive neurotologic surgery. MRI transformed the diagnostic of a vasculoneural conflict; to analyze cranial nerves and vascular structures crossing the CPA, good knowledge of the anatomy is required and an excellent MRI technique allows such a study. Inframillimetric axial and sagittal slices in T2 constructive interference in steady state (CISS) sequences are essential for assessment of the vasculoneural relationship. The aim of this study is to show the imaging criteria of the offending vessel in neurovascular compression syndrome in the CPA using MRI. This will increase the acceptance of the concept of vascular compression syndrome of the cranial nerves as an etiology of trigeminal neuralgia, hemifacial spasm, glossopharyngeal neuralgia, and some cases of disabling vertigo and tinnitus.

Optimizing the knowledge to the cranial nerves in the posterior cranial fossa requires an understanding of the relationship of these nerves with the cerebellar arteries, brain stem, and cerebellar surfaces [1]. When examining these, three neurovascular complexes can be defined: an upper complex related to the superior cerebellar artery (SCA); a middle complex related to the anterior inferior cerebellar artery (AICA); and a lower complex related to the posterior inferior cerebellar artery (PICA) and vertebral artery (Fig. 1).

The upper complex includes the trigeminal nerve, SCA, midbrain, cerebellomesencephalic fissure, superior peduncle, tentorial surface of the cerebellum, and oculomotor and trochlear nerves. The trigeminal root joins the

Figure 1



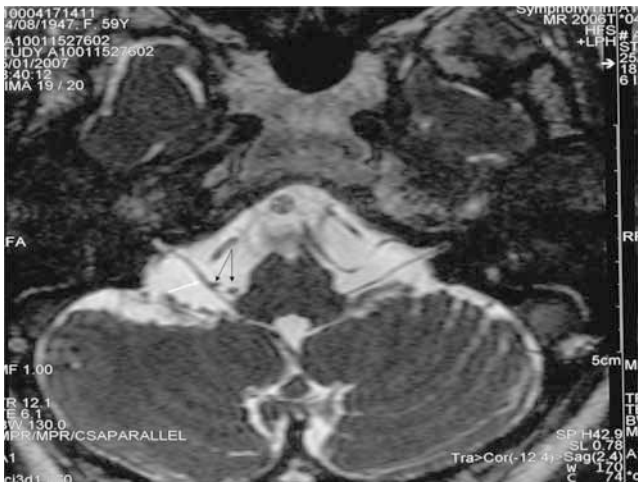
(a) Right superior view and (b) right posterior view; shows the anatomy of cranial nerves in the posterior cranial fossa. Superior cerebellar artery (SCA), anterior inferior cerebellar artery (AICA), facial nerve (VII), cochleovestibular nerve (VIII), internal auditory canal (IAC), trigeminal nerve (V), gasserian ganglion (G), glossopharyngeal nerve (IX), vagus nerve (X), and accessory nerve (XI).

brain stem about halfway between the lower and the upper borders of the pons. In its intradural course, the trigeminal nerve uniformly runs obliquely upwards from the lateral part of the pons toward the petrous apex. It exits the posterior fossa to enter the middle cranial fossa by passing forward beneath the tentorial attachment to enter Meckel's cave. The SCA arises in front of and encircles the midbrain, passes below the oculomotor and trochlear nerves, and above the trigeminal nerve to reach the cerbellomesencephalic fissure, where it runs on the superior cerebellar peduncle and terminates by supplying the tentorial surface of the cerebellum.

The middle complex includes the acousticofacial nerve bundle, the AICA, pons, the middle cerebellar peduncle, cerebellopontine fissure, petrosal surface of the cerebellum, and the abducense nerve. The acousticofacial nerve bundle arises from the brain stem near the lateral end of the pontomedullary sulcus. The facial nerve arises in the pontomedullary sulcus 1–2 mm anterior to the point at which the vestibulocochlear nerve joins the brain stem at the lateral end of the sulcus. The interval between the vestibulocochlear and the facial nerves is greatest at the level of the pontomedullary sulcus and decreases as the nerves approach the meatus. In the CPA, the facial and vestibulocochlear nerves run forward and lateral to the posterior surface of the petrous bone to enter the internal auditory canal. During their cisternal course, the facial nerve is anteromedial and the vestibular nerve is superolateral, whereas the cochlear nerve is inferolateral. As they approach the porus of the internal auditory canal, the facial is superomedial, the cochlear is inferomedial, and the vestibular nerves are lateral. The AICA arises at the pontine level and courses in relationship with the abducens, facial, and vestibulocochlear nerves to reach the surface of the middle cerebellar peduncle, where it courses along the cerebellopontine fissure and terminates by supplying the petrosal surface of the cerebellum.

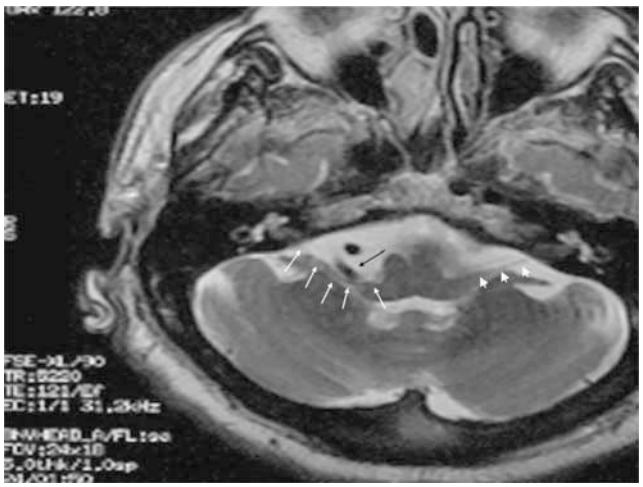
The lower complex includes the lower cranial nerves (the glossopharyngeal, vagus, spinal accessory, and hypoglossal nerves), the PICA, medulla, inferior cerebellar peduncle cerbellomedullary fissure, and the suboccipital surface of the cerebellum. The glossopharyngeal arises as one rootlet, or rarely as two rootlets, from the upper medulla, posterior to the upper one-third of the olive, just caudal to the origin of the facial nerve. It courses ventral to the choroid plexus, protruding from the foramen of Luschka on its way to the jugular foramen. The vagus nerve arises inferior to the glossopharyngeal nerve as tightly packed rootlets along a line 2–5.5 mm in length posterior to the superior one-third of the olive. The vagus is composed of multiple combinations of large and small rootlets that pass ventral to the choroid plexus protruding from the foramen of Luschka on their way to the anteromedial part of the pars venosa. The spinal accessory nerve arises as a widely separated series of rootlets that originate from the medulla at the level of the lower two-thirds of the olive and from the upper cervical cord. The cranial rootlets of the spinal accessory nerve arise as a line of rootlets just caudal to the vagal fibers. The spinal accessory fibers pass superolaterally from their origin to reach the jugular foramen. The PICA arises at the medullary level, encircles the medulla passing in relation to the glosspharyngeal, vagus, spinal accessory, and hypoglossal nerves to reach the surface of the inferior cerebellar peduncle, where it dips into the cerbellomedullary fissure and terminates by supplying the suboccipital surface of the cerebellum.

Figure 2



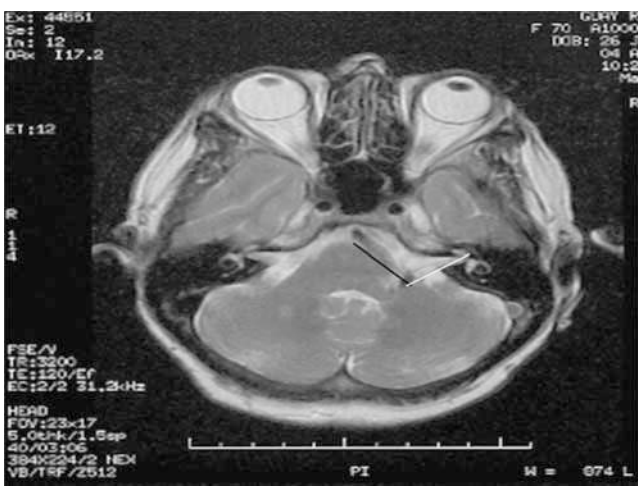
MRI T2 axial cut shown on the right side; PICA (black arrows) in contact with the glossopharyngeal nerve (white arrow). PICA, posterior inferior cerebellar artery.

Figure 4



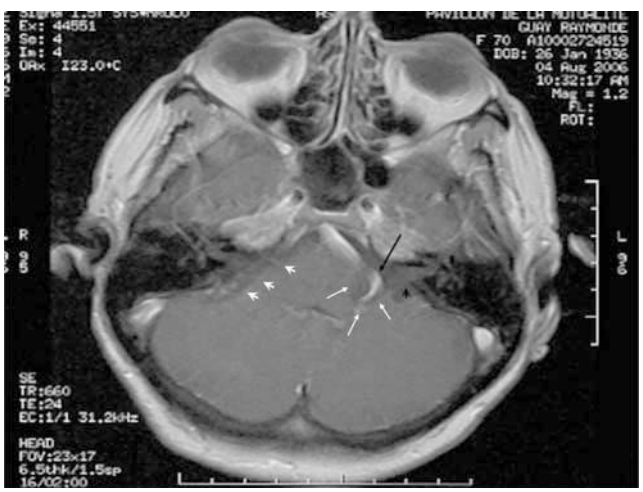
MRI T2 axial cut showing right vertebral artery (black arrow) causing distortion of the course of the right facial nerve (white arrows); white arrow heads show the straight undistorted left facial nerve.

Figure 3



MRI T2 axial cut shown on the left side: vertebral artery and pica (black line) in perpendicular contact with the facial nerve (white line); in addition, pica distorts the brain stem at the REZ of the facial nerve. REZ, root exit zone.

Figure 5



MRI postcontrast T1 axial cut showing left PICA (black arrow) at root exit zone (REZ) of left facial nerve (black arrow head) embedding of the pons (white arrows); white arrow heads show the straight edge of the pons on the right side. PICA, posterior inferior cerebellar artery.

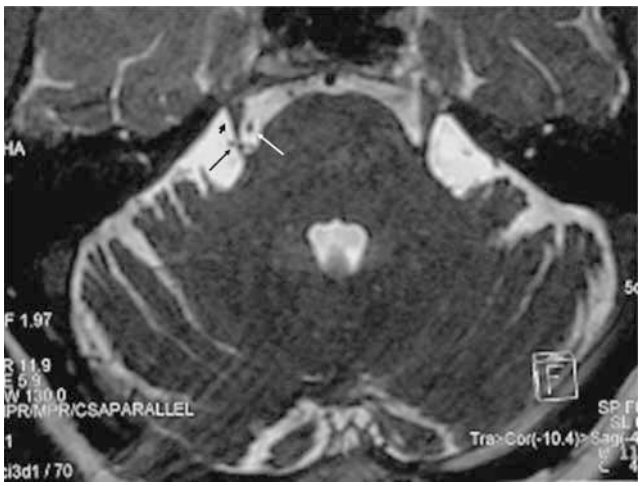
Materials and methods

Between 1994 and 2008, at our center (Hopital Nord, Marseille, France), we have performed 453 microvascular decompression (MVD) surgeries for hemifacial spasm, 269 for trigeminal neuralgia, 56 for tinnitus and vertigo, and four decompressions for glossopharyngeal neuralgia. The MRI radiographs of these 782 cases were reviewed to determine the criteria of the conflicting vessel that cause neurovascular compression syndrome.

Of these 782 patients, we have selected 16 cases that best represent the different and various pathologies (10 cases of hemifacial spasm, four cases of trigeminal neuralgia, one case of glossopharyngeal neuralgia, and one case of vertigo), operated through an endoscope-assisted MVD using a minimally invasive retrosigmoid approach; their MRI radiographs were analyzed and correlated with intraoperative endoscopic findings to confirm the imaging criteria of the offending vessel.

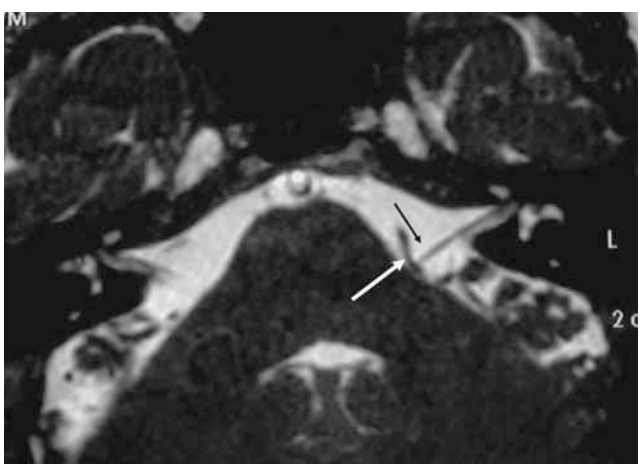
Imaging of the CPA is carried out using serial thin slices of 0.4 mm thickness. The axial plane offers comprehensive and demonstrative images. MRI axial and coronal 0.4 mm cuts using T1 and T2 three-dimensional Fourier transform is the most effective method for the assessment of neurovascular structures in the CPA and the internal auditory canal. T2 is carried out using CISS sequence. Postcontrast T1 reformatted turbo-flash in the axial plane helps delineate the conflicting vessel (angiographic MRI sequences).

Figure 6



MRI T2 axial cut showing right trigeminal nerve (black arrow head), compressed (reduced caliber) by the superior cerebellar artery (SCA) (white arrow) and an aberrant trigeminal nerve (black arrow); compared with the normal caliber of the left trigeminal nerve.

Figure 7



MRI T2 axial cut showing PICA (white arrow) in contact with the REZ of the left facial nerve (black arrow). PICA, posterior inferior cerebellar artery; REZ, root exit zone.

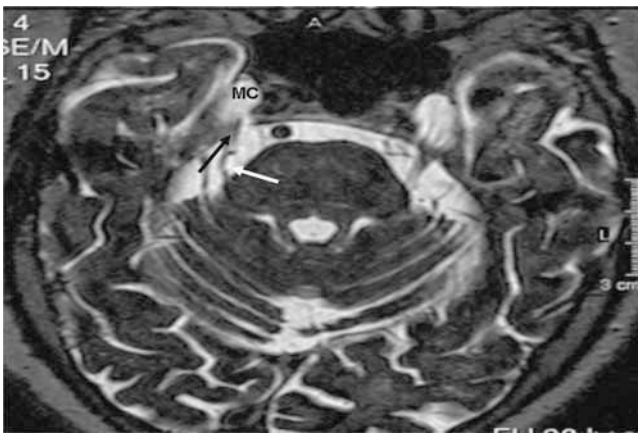
Results

In our study, using the endoscope-assisted MVD technique, the offending vessels were detected in all cases, whereas MRI documented vascular contact in 98% (766 cases) and the vessel induced distortion in 73% (570 cases).

MRI has a sensitivity of 97% (756 cases) and a specificity of 100% (782 cases); the positive predictive value is almost 100% (782 cases) and the negative predictive value is 60% (469 cases). MRI is thus highly sensitive for depicting vascular compression, and is the key method for the assessment of the indication for surgical treatment.

The following are the imaging criteria of a conflicting vessel in neurovascular compression syndrome in CPA:

Figure 8



MRI T2 axial cut showing SCA (white arrow) in contact with the right trigeminal nerve (black arrow) along its course from REZ to Meckel's cave (MC). REZ, root exit zone; SCA, superior cerebellar artery.

(1) Presence of vascular contact with the cranial nerve (Fig. 2), but this criterion alone is not sufficient.
(2) Orthogonal (perpendicular) contact of the vessel with the cranial nerve (Fig. 3).
(3) Distortion of the course of the cranial nerve (Fig. 4).
(4) Distortion of the adjacent neural structure (brain stem) (Fig. 5).
(5) Reduction of the nerve caliber (Fig. 6).
(6) Site of contact between the vessel and the cranial nerve; for the facial nerve, the weakest point is the root exit zone (REZ) as it is the transition zone between central and peripheral myelin, and for the trigeminal nerve, usually any site along its course from the root entry zone (REZ) to the pes caves (Meckel's cave) (Figs 7 and 8).

Discussion

Vascular compression syndromes of the cranial nerves, first suggested in 1934 by Dandy and then popularized by Jannetta in the 1970s [2], are gaining acceptance with the improvement in MRI assessment and with the success of endoscopy-assisted MVD procedures. They are commonly described in trigeminal neuralgia and hemifacial spasm, but other disorders such as glossopharyngeal neuralgia and disabling positional vertigo can also be treated successfully by MVD of the respective cranial nerves. It has also been shown, although in small series, that some cases of disabling tinnitus can be alleviated by MVD.

The two most commonly encountered vascular compression syndromes are trigeminal neuralgia and hemifacial spasm. Trigeminal neuralgia is characterized by short periods of excruciating pain in specific areas of the face [3]. It is more common than hemifacial spasm, with an estimated incidence of about 4.7 per 100 000 for both sexes in the white population [4]. The pain is usually triggered by a light touch to a specific area of the face or

inside the mouth, and it does not respond to conventional analgesics [5].

As for hemifacial spasm, it is a rare disease (incidence: 0.74 per 100 000 in white men and 0.81 per 100 000 in white women) [6] with an onset relatively late in life. It is characterized by periods of facial spasm, between which there is essentially normal function facial mimetic musculature. Hemifacial spasm usually develops according to a specific pattern: brief periods of spasm around the eye occur first, and over several years, the spasm gradually increases in intensity and spreads downward over the face so that 6–10 years following its onset, the entire half of the face is involved, including the platysma, but often excluding the forehead [7]. In about 8% of patients, however, hemifacial spasm is atypical in onset, beginning in the buccal muscles and progressing upward over the face [8]. Spontaneous remissions are rare [9].

The two prevailing hypotheses that claim to explain the symptoms and signs of hemifacial spasm focus on the cross-talk (ephaptic transmission) between individual nerve fibers of the facial nerve where it is compressed by a blood vessel [10] and, second, on abnormal hyperactivity on the facial motor nucleus [11]. Nielsen [12] reported that preoperatively there was a statistically significant prolonged latency of R1 components of the blink reflex on the affected side of the patients with hemifacial spasm, which would indicate a rather severe demyelination of the facial nerve. He also found that the amplitudes of the R1 components were about twice as high on the affected side as on the unaffected side, and that more motor neurons than normal fire in response to stimulation of the supraorpital nerve. These findings would support the hypothesis of the presence of ephaptic transmission between injured nerve fibers. For a long time, this hypothesis that ephaptic transmission at the site of vascular compression causes the symptoms of hemifacial spasm was favored [13], but more recently, the results of several studies [14] have provided evidence that hyperactivity of the facial motor nucleus is the cause of the disorder. Although there seem to be histologically detectable changes in the facial nerve of individuals with hemifacial spasm that can be related to the vascular compression, the degree of demyelination noted does not seem to be sufficient enough to explain the spasm and synkinesis in the hemifacial spasm on the basis of the ephaptic transmission hypothesis.

A similar explanation has been suggested for trigeminal neuralgia. The first (peripheral) hypothesis assumes that the symptoms are caused by ephaptic transmission in the segment of the trigeminal nerve that is in close contact with a blood vessel [15]; the second (central) hypothesis claims that the symptoms have a central cause [16]. Recently, it has been claimed that the symptoms of trigeminal neuralgia can be explained by a peripheral etiology and a central pathogenesis [17]. The hypothesis assigning a peripheral cause for the pain of the trigeminal neuralgia assumes that defects in myelin result in multiple reflections of nerve impulses. Such reflections amplify peripheral nerve impulses, which, together with the dorsal root reflex, may cause presynaptic depolarization that, in turn, may elicit the pain of trigeminal neuralgia [18]. Fromm [17] proposed that the nuclear hyperactivity causing the pain of trigeminal neuralgia results from impaired segmental inhibition, which is caused by irritation of the trigeminal nerve root in combination with increased activity in afferent fibers of the nerve because of spikes and possibly cross-talk (ephaptic transmission) between injured fibers. The reduction in segmental inhibition could be cased by chronic irritation of the trigeminal nerve by a blood vessel.

The common basic etiology in these vascular compression syndromes is the contact between the offending artery and the nerve. Therefore, it is logical that elimination of this contact would resolve the patient's symptoms. MRI is an essential element in the work-up of these pathologies [19].

MRI is able to reproduce the anatomy of the CPA and to show the presence of neurovascular conflict. It also eliminates other potential causes such as a tumoral process or multiple sclerosis. MRI in both T1 and T2 three-dimensional Fourier transform sequences is the most effective method of delineating both the cranial nerves and the surrounding vascular structures in the CPA. The T2 is carried out using CISS sequences [20]. Imaging of the CPA is carried out with serial then slices of 0.4 mm thickness. The axial plane offers the most comprehensive images. To confirm the diagnosis, the single presence of a neurovascular contact is not sufficient. Several radiological criteria are required, with some variability in the cranial nerves involved.

Common criteria include a perpendicular contact between the vascular loop and the cranial nerve, visualized along two different perpendicular planes; this perpendicular contact allows maximum power of stress and pressure by the vascular loop on the cranial nerve at one point, which makes it a weak point.

Distortion of the nerve course by the vascular loop causes stretching of the nerve, which weakens the nerve; also, compression of the adjacent delicate neural structure (brain stem) at the REZ has been found to be a cause.

For the facial nerve, the location of the conflict at the REZ of the nerve and the distortion of the brain stem at this level are additional criteria; REZ is the weakest point of the nerve as it is the area of the junction between central and peripheral myelin. Thus, demylineation of the nerve is more rapid. The most conflicting vessels are PICA in 70% of patients, the dolico vertebral artery in 41%, AICA in 28%, and aberrant vein in 2% of patients, and the frequency of multiple contacts is as high as 38%.

For the trigeminal nerve, the reduction in the diameter is an additional criterion, as compression of the nerve by a vascular loop causes some ischemia and weakens the sheath at the site of compression. The most common offending vessel is the SCA in 67.5% of patients, but in 25% of cases, a venous structure (aberrant trigeminal vein) might be the cause of the conflict, and it is visible

only on T1 with gadolinium and AICA in 20% of cases. For the auditory nerve, we expect to see displacement of the nerve with a certain distance between the facial and the cochlear nerves, with an imprint on the nerve and a reduction in its diameter, and with brain stem distortion caused by vascular structure at the level of the REZ of the cochlear nerve [21,22].

It should be kept in mind that the pathological process caused by compression of the cranial nerve by the offending vessel needs a long duration of up to 10–15 years and may be more symptomatic; thus, when we perform MRI in healthy individuals with no symptoms, sometimes, we find one or more of the radiological criteria of the offending vessel with no clinical complaint as the pathological process is not well established. We have to keep in mind again that the CPA is a compact narrow intracranial space with a large number of neural and vascular structures, which normally have a close relationship and are in contact with each other; thus, only symptomatic cases with MRI criteria, after excluding other causes of the disease, are candidates of neurovascular compression syndrome to be treated by an MVD operation.

Conclusion

MRI can reproduce the anatomy of the CPA and indicate the presence of a neurovascular conflict. It also eliminates other potential causes such as a tumoral process or multiple sclerosis; MRI findings correlate very reliably with the surgical findings, and provide a rather precise idea of the vasculoneural conflict that may be encountered during surgery.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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