

Endoscopic criteria of offending vessel in neurovascular compression syndrome

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Objectives

The aim of this work was to detect the anatomical relationship criteria of the offending vessel with respect to the cranial nerve in neurovascular compression syndrome using the endoscopic-assisted minimally invasive retrosigmoid approach for microvascular decompression (MVD); these criteria help the surgeon to define the actual conflicting vessel during surgery for better results.

Materials and method

Between 1994 and 2009, we have performed 782 cases of MVD surgeries using endoscopic-assisted minimally invasive retrosigmoid approach (453 MVD surgeries for hemifacial spasm, 269 for trigeminal neuralgia, 56 for tinnitus and vertigo, and four decompressions for glossopharyngeal neuralgia). During these surgeries we recorded the anatomical relationships between the offending vessel and the affected cranial nerve to detect the endoscopic criteria of the offending vessel.

Results

There are certain criteria for the anatomical relationship between the offending vessel and affected cranial nerve; these criteria include common features such as a perpendicular contact between the vascular loop and the cranial nerve along two different perpendicular planes at its root exit zone, distortion of the nerve course, and distortion or compression of adjacent neural structures mainly brain stem. Other criteria include the vascular loop causing impression of the nerve or encircling the nerve causing reduction of its diameter.

Conclusion

Certain criteria for the anatomical relationship between the offending vessel and the affected cranial nerve should be fulfilled to diagnose the actual conflicting vessel during MVD surgery in vascular compression syndrome using endoscopic-assisted minimally invasive retrosigmoid approach.

Keywords:

compression syndrome, endoscopic criteria, offending vessel

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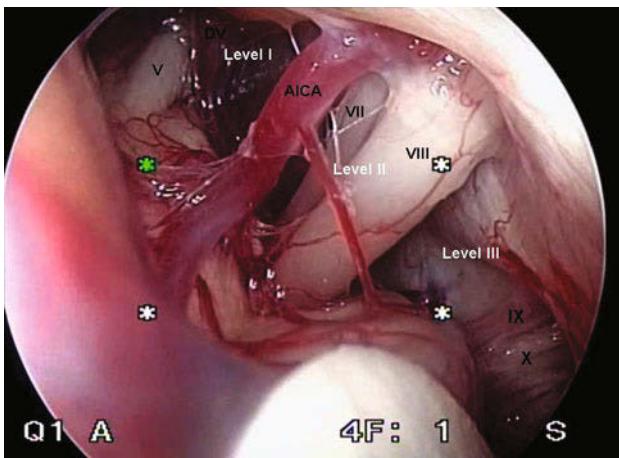
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Introduction

Neurovascular compression syndrome (NVCS) is a disease caused by presence of contact between a vascular loop in the cerebellopontine angle (CPA) and one of the cranial nerves. Normally, the CPA is characterized by presence of many vascular and neural structures, which are normally in contact with each other without causing a problem; however, sometimes this contact causes a problem to the patient and becomes symptomatic leading to the so-called NVCS, the symptom of which depends on the compressed cranial nerve and the compressing vessel is called an offending vessel [1]. Vascular compression syndrome of the cranial nerves, first suggested in 1934 by Dandy [2] and popularized by Jannetta in the 1970s [3], are gaining acceptance with the improvement in MRI assessment and the success of endoscope-assisted microvascular decompression (MVD) procedures; they are commonly described in trigeminal neuralgia and hemifacial spasm, but other disorders such as glossopharyngeal neuralgia and

disabling positional vertigo (DPV) 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 [4].

Operative endoscopic anatomy (Fig. 1) is clarified through retrosigmoid approach using endoscope; the acousticofacial nerve bundle (level II) is the reference level, crossing the middle of the CPA, giving two separate endoscope-assisted surgical areas. Superiorly, the trigeminal area (level I) is concerned with the treatment of trigeminal neuralgia. Inferiorly, the lower cranial nerve area (level III) is concerned with the treatment of hemifacial spasm and glossopharyngeal neuralgia. Auditory nerve with cross conflicts with tinnitus or DPV requires alternative endoscopic control from above and below the acousticofacial nerve bundle, and finally (level IV) an inferior extension of the CPA in which the lower medulla is readily apparent [5].

Figure 1

Endoscopic anatomical levels. Level I (superior compartment): V trigeminal nerve, dandy vein (DV). Level II (middle field compartment): VII facial nerve, VIII cochleovestibular nerve. Level III (inferior compartment): IX glossopharyngeal nerve, X vagus nerve.

The objective of this study was to describe the anatomical criteria of the offending vessel (endoscopic anatomical relationship), which causes NVCS, through analysis of 782 cases during endoscopic MVD through keyhole minimally invasive retrosigmoid approach (MIRA): 453 MVD for hemifacial spasm, 269 for trigeminal neuralgia, 56 for tinnitus and vertigo, and four decompressions for glossopharyngeal neuralgia.

Materials and methods

Our study was built on a retrospective manner of analysis of 782 cases of neurovascular decompression surgeries to detect the endoscopic intraoperative criteria of offending vessel, which produces symptoms when in contact with a cranial nerve (produce the so-called NVCS). The measured criteria are the anatomical relationship between the offending vessel and the affected cranial nerve, as normally the CPA is a closed space characterized by presence of many vascular and neural structures that are normally in contact with each other; when the vascular structure comes in contact with the cranial nerve in a special anatomical manner, it may become hazardous and produce symptoms. These anatomical relationships have been recorded and mentioned as offending criteria of the offending vessel.

The MIRA was first described by senior author, J. Magnan, and his teachers Bremond and Garcin in 1974 [6,7]. We have been performing operations since 1990 using the combined endoscopic and microsurgical technique.

Between 1994 and 2009, at our hospital (Hospital Nord, Marseille, France), we have performed 453 MVD surgeries for hemifacial spasm, 269 for trigeminal neuralgia, 56 for tinnitus and vertigo, and four decompressions for glossopharyngeal neuralgia.

In all cases, we used the MIRA, also described as 'keyhole approach'; MIRA has been performed by combining the use of an endoscope along with microscope to achieve a safer, more reliable approach to CPA surgery and detecting the conflicting vessel and its manner of contact with the affected cranial nerve.

Retrosigmoid craniotomy

The MIRA in our department is performed as follows:

For the area to be shaved, only 2–3 cm behind the ear is sufficient. Body position should be face up and to protect the face, the forehead of the collateral side should be fastened with a gel sheet, without a two-point bracing of the skull.

Design the so-called Frankfurter line, which is an extension toward the back of the line joining the outer canthus to the superior border of the external auditory canal, and a line along the posterior margin on the mastoid. A keyhole 2 cm in diameter is drawn below and backward from the point where two lines cross. This is the position of the craniotomy.

For skin incision, an arc 6–8 cm in length is drawn with the convex side toward the back at a position one finger's width in back of the oblique line by passing the backside of the keyhole.

The keyhole designed in item 2 is burred. Ensure that the emissary vein comes into the center and then bur the hole with the vein as a marker.

When the operation reaches the posterior fossa dura and posterior margin of the sigmoid sinus, use the microscope to make the incision of the dura matter. With use of a surgical knife, incise in a U-shape, and, while sucking the cerebrospinal fluid, wait for spontaneous cerebellar retraction. Place neurosurgical cotton, 1.5 cm wide by 5 cm long, on the cerebellum and gently press the middle part with the suction pipe toward the back and open the posterior cistern.

For monitoring facial nerve function, the NIM response (Medtronic USA Inc., Minneapolis, Minnesota, USA) is used.

Endoscopic surgery in cerebellopontine angle

The endoscopes used are mainly the following four: 6 cm in length, 4 mm in diameter, 0°; 6 cm in length, 4 mm in diameter, 30°; 11 cm length, 4 mm (and 2.7 mm) diameter, and 30° angle. We use these endoscopes according to the situation, but mainly the one of 11 cm length and 30° angle is used. When the operator is not familiar with endoscopic procedure, we recommend using only 6 cm length, which is safer and prevents injury. The endoscope and the optical fiber cable used are completely sterilized by autoclaving. By covering the camera cable and the camera adaptor with sterilized polyethylene cover, endoscopes can be easily exchanged under clean handling conditions.

Results

We did analyses of the anatomical relationship of the offending vessel to the affected cranial nerves, during MVD using endoscope-assisted MIRA in vascular compression syndrome in 782 cases (453 hemifacial spasm, 269 trigeminal neuralgia, 56 for tinnitus and vertigo, and four glossopharyngeal neuralgia) from 1994 to 2008.

To confirm the diagnosis of an offending vessel, the presence of single neurovascular contact is not sufficient. Several criteria are required, with some variability depending on the involved cranial nerves.

The common criteria include:

- (a) A perpendicular contact between the vascular loop and the cranial nerve along two different perpendicular planes at its root exit zone (REZ),
- (b) Distortion of the nerve course, and
- (c) And distortion or compression of adjacent neural structures mainly brain stem.

Besides the common criteria of the offending vessel, we found that a vulnerable site of contact varies from one cranial nerve to another, meaning that each nerve has special site in its course, which becomes more vulnerable.

For the trigeminal nerve, we found that it is vulnerable in all parts of its course (Table 1): at root entry zone in 129 cases (48%) (Fig. 2), at the cisternal course of the nerve in 107 cases (40%) (Fig. 3), and at the exit of the nerve at the entrance of Meckel's cave in 67 cases (25%) (Fig. 4).

For the facial nerve, we found that it is vulnerable mostly at the REZ in 430 cases (95%) and at the porus in 23 cases (5%) associated with one or more of the general criteria mentioned before (Table 2). The REZ

of the facial nerve is the weakest point of the facial nerve as it is the site of junction between central and peripheral myelin (Figs. 5 and 6).

For the vestibule-cochlear nerve, the single presence of a neurovascular contact is not sufficient; the presence of one or more of the common criteria is mandatory to diagnose an offending vessel. Another important finding is the presence of an imprint in the vestibulocochlear nerve and reduction in its diameter by vascular loop encircling the nerve (Figs. 7 and 8), and finally the presence of an arterial loop in the internal auditory canal may cause tinnitus or DPV (Fig. 9).

In our series of study, we found the cause for neurovascular compression in trigeminal neuralgia (Table 3): the superior cerebellar artery alone either the main trunk or its two branches in 46.5% of cases, the anterior inferior cerebellar artery (AICA) alone in 2.5% of cases, the vertebral/basilar artery in 2% of cases, vein alone in 11% of cases, and multiple combination in 38% of cases. The cause for neurovascular compression in hemifacial spasm (Table 4) was: the posterior inferior cerebellar artery (PICA) alone in 37.2% of cases, vertebral artery (VA) alone in 11.6% of cases, PICA+VA in 32.4%, AICA alone in 8.1% of cases, vein alone in 1.9% of cases, and multiple combinations in 8.8% of cases.

Table 1 Vulnerable parts of trigeminal nerve

| | Site | | |
|-----------------|-----------------|----------------|---------------|
| | Root entry zone | Cisternal part | Meckel's cave |
| Number of cases | 129 | 107 | 67 |
| Percentage | 48 | 40 | 25 |

Table 2 Vulnerable parts of facial nerve

| | Site | |
|-----------------|----------------|-------|
| | Root exit zone | Porus |
| Number of cases | 430 | 23 |
| Percentage | 95 | 5 |

Table 3 Causes of neurovascular compression in trigeminal neuralgia

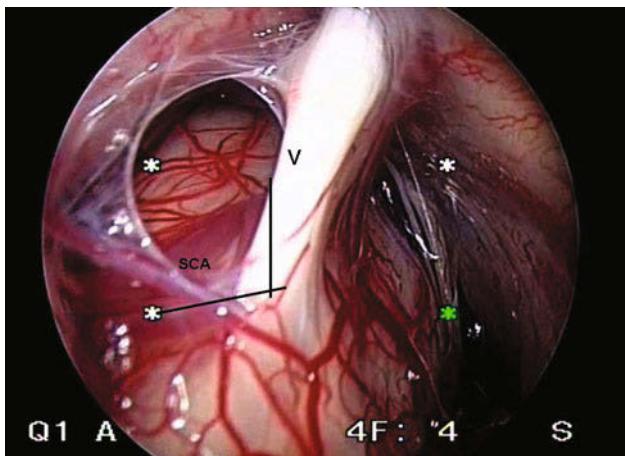
| Percentage | Vessel | | | | |
|------------|--------|------|-----|------|----------|
| | SCA | AICA | VBA | Vein | Multiple |
| 46.5 | 2.5 | 2 | 11 | 38 | |

AICA, anterior inferior cerebellar artery; SCA, superior cerebellar artery; VBA, vertebrobasilar artery.

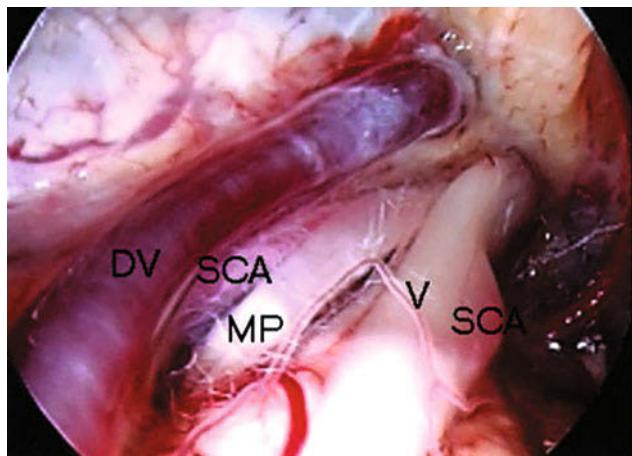
Table 4 Causes of neurovascular compression in hemifacial spasm

| Percentage | Vessel | | | | | |
|------------|--------|------|---------|------|----------|------|
| | PICA | VA | PICA+VA | AICA | Multiple | Vein |
| 37.2 | 11.6 | 32.4 | 8.1 | 8.8 | 1.9 | |

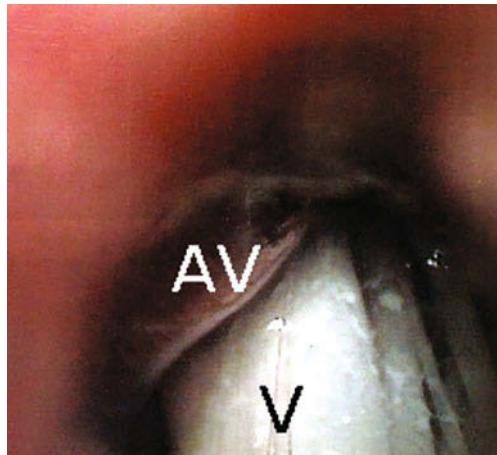
AICA, anterior inferior cerebellar artery; PICA, posterior inferior cerebellar artery; VA, vertebral artery.

Figure 2

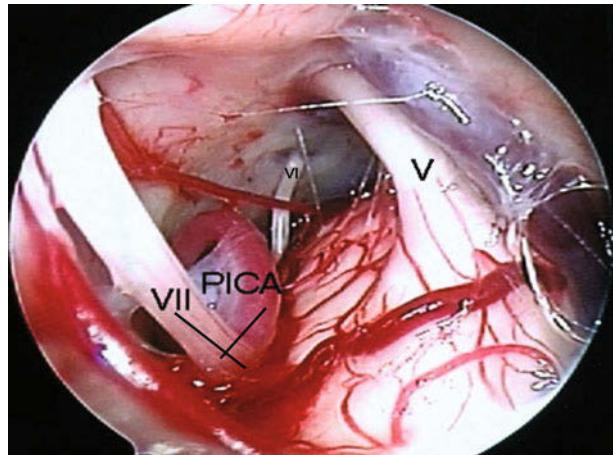
Superior cerebellar artery (SCA) in contact with trigeminal nerve (V) at root exit zone in a perpendicular plane.

Figure 3

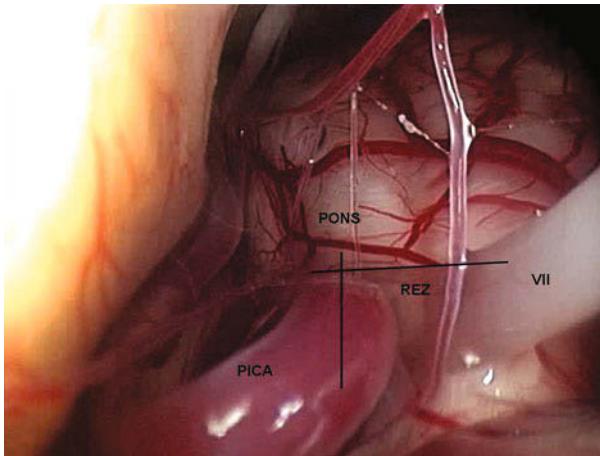
Superior cerebellar artery (SCA) in contact with the cisternal part of the trigeminal nerve (V). MP: motor part, DV: dandy vein.

Figure 4

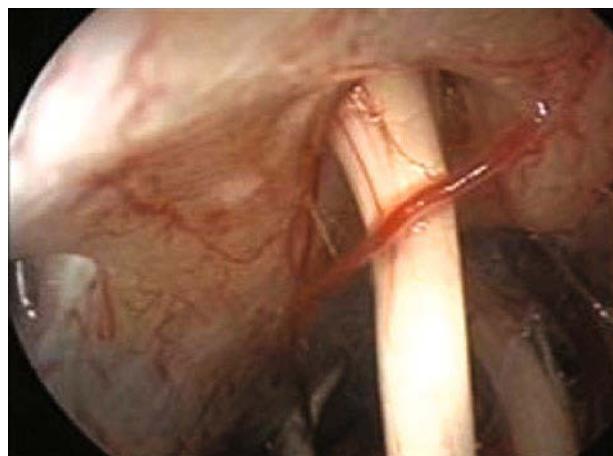
Aberrant vein (AV) in contact at the exit of the trigeminal nerve at the entrance of Meckel's cave. V is Latin number (5) 5th cranial nerve.

Figure 5

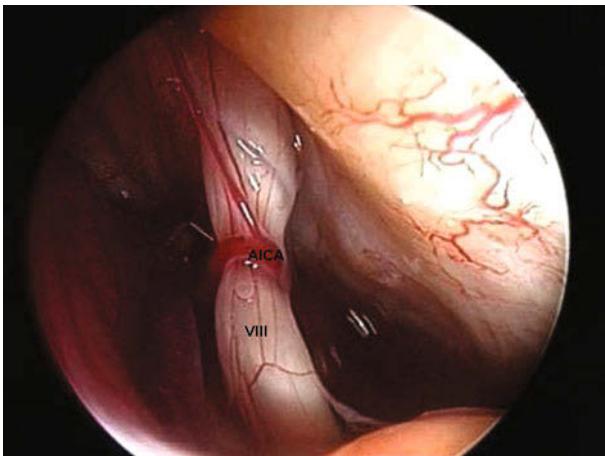
Posterior inferior cerebellar artery (PICA) in contact with the facial nerve (VII) at the root exit zone in perpendicular plane causing distortion of the course of the nerve. V VII are Latin numbers 5 7.

Figure 6

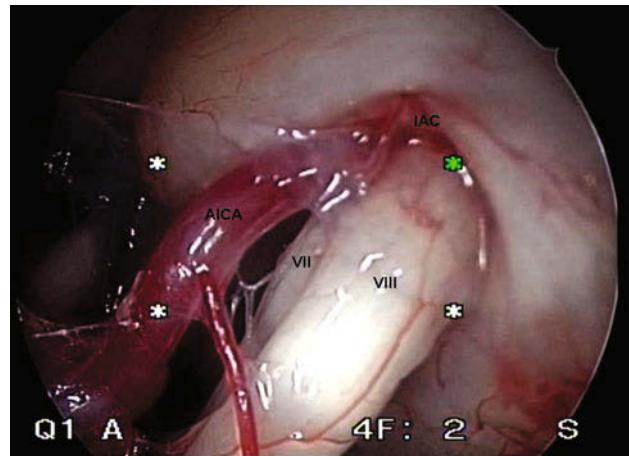
Posterior inferior cerebellar artery (PICA) in contact with the facial nerve (VII) at the root exit zone (REZ) in perpendicular plane compressing the brain stem (PONS).

Figure 7

Subarcuate artery crossing the vestibulocochlear nerve causing an impression on the nerve.

Figure 8

Loop of anterior inferior cerebellar artery (AICA) encircles the vestibulocochlear nerve (VIII) causing an impression of the nerve and reduction of its diameter.

Figure 9

Loop of anterior inferior cerebellar artery (AICA) at the internal auditory canal (IAC) between the facial (VII) and the vestibulocochlear nerves (VIII).

Discussion

Trigeminal neuralgia, hemifacial spasm, intractable vertigo, tinnitus, and glossopharyngeal neuralgia are disabling functional cranial nerve disorders that can impair severely the quality of the life of patients. The efficacy of conservative management usually is limited over time, and patients ultimately seek a definitive surgical solution to their problem. Although MVD is gratifying for the patient and the surgeon, it is the surgeon's duty to prevent any devastating complications in these functional disorders. In our neuro-otological experience using MIRA surgeries for indications such as vascular compression syndromes, we have very little morbidity; this should be the rule in all functional surgical indications. The most common surgical causes of incomplete cure are misjudgment of the real offending vessel and incomplete or improper replacement of the conflict vessel. The use of endoscope intraoperatively enhances the chances of visualization of the offending vessels without increasing the amount of retraction of the cerebellum, and thus reducing the chances of complications such as hearing loss and facial weakness. When there are multiple vessels running closely to the nerve, the real offending vessel causing compression of the nerve should be accurately diagnosed. If that is not possible, all the conflicting vessels (even vein) should be decompressed.

Trigeminal neuralgia was first described by Dandy [2] in 1934, as compression syndrome of the trigeminal nerve, from his operative findings made on 215 patients who underwent surgery in the CPA; he described the presence of the superior cerebellar artery in contact with the trigeminal nerve in 45% of his cases and hypothesized that the vascular compression may be

responsible for the painful phenomenon. The MVD surgery for trigeminal neuralgia was performed for the first time in 1959 by Gardner *et al.* [8,9], but it was popularized by Jannetta *et al.* [10,11] after late 1960s, who is now considered the father of 'MVD surgery'. The clinical syndrome of hemifacial spasm was first described by Gowers [12] in 1888, distinguishing it from other motor abnormalities of the face. As early as 1909, Hunt [13] explained the hemifacial spasm by a 'reflex' theory where the trigeminal nerve acted as activating factor. In 1912, Sicard and Bollack [14] proposed a supranuclear origin. In 1952, Wartenberg [15] proposed hyperactivity at the nuclear level. Alajouanine and Thurel [16] proposed the presence of a lesion along the nerve itself whether in the CPA or in the interpetrous segment. Gardner and Sava [17] in 1960 published the presence of a vascular loop as a cause of surgically reversible hemifacial spasm. Jannetta *et al.* [18,19] confirmed and developed in the 1970s the concept of the neurovascular conflict as a cause for hemifacial spasm.

Sirikei *et al.* [20] classified the anatomical relationship between the AICA and the cochleovestibular (CVN) nerve into four types: type 1 point compression (20%) where the AICA compresses only a limited portion of the CVN, type 2 longitudinal compression (40%) where the AICA approaches the CVN as both traverse parallel to each other, type 3 loop compression (20%) where vascular loop of AICA encircles the CVN, and type 4 indentation (20%) where the AICA compresses the CVN so as to make an indentation in the nerve. Using the MRI to analyze the anatomical relationship between vascular structures and the cranial nerves in VCS, common criteria include perpendicular contact between the vascular loop and the cranial nerve,

visualized along two different perpendicular planes, and distortion of the nerve or other neural structures; for the facial nerve the location of the conflict at the REZ of the nerve and distortion of the brain stem at this level are additional criteria. For the trigeminal nerve, reduction of the diameter is an additional criterion [1].

The operating microscope, despite its qualities of brightness, magnification, and depth of field, provides only axial view to the operator. Yet, the site of the conflict is to be found most often against the brain stem at the emergence of cranial nerves, which then requires a mechanical retraction of the cerebellum and other neural structures. According to the landmark paper of Jannetta *et al.* [21], the pioneer of MVD ‘turning the corner is the most dangerous stage of the operation and must be executed with patience and the utmost care’, and most complications can be attributed to cerebellar retraction. The intraoperative endoscopy, thanks to its panoramic vision and angulated view, allows a complete exploration of the CPA without retraction of the cerebellum or brain stem and leaves no hidden areas to be missed [22,23]. It is therefore the procedure of choice to locate the site of conflict without significant cerebellar retraction, positively identify the vessels responsible as well as their path, and define the best maneuver to decompress the artery.

Conclusion

Vascular compression syndrome is characterized by presence of contact between a vascular structure and a cranial nerve leading to manifestations according to the compressed nerve. The endoscopic-assisted MIRA is used successfully to correct this condition; intraoperative endoscopy has helped very much in detecting the offending vessel even in hidden areas in CPA. There are certain criteria for the anatomical relationship between the offending vessel and affected cranial nerve; these criteria include common features such as a perpendicular contact between the vascular loop and the cranial nerve along two different perpendicular planes at its REZ, distortion of the nerve course, and distortion or compression of adjacent neural structures mainly brain stem. Other criteria include the vascular loop causing impression of the nerve or encircling the nerve causing reduction of its diameter.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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