

Reporte de Caso

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Vértigo incapacitante: Descompresión microvascular del nervio vestibular dentro del meato auditivo interno. Descripción técnica

Disabling vertigo: microvascular decompression of vestibular nerve inside internal auditory canal. Technical description

Resumen

Introducción. El vértigo incapacitante, originado por una compresión vascular del nervio vestibular (NV), puede beneficiarse de la descompresión microvascular (DMV) del NV. La mayoría de las compresiones del NV se observan en la zona de Entrada / Salida de la raíz nerviosa (REZ), adyacentes al tronco cerebral o en la cisterna pontocerebelosa. Sólo unos pocos casos del conflicto vascular son reportados dentro del meato auditivo interno, pero sin descripción detallada de la técnica. Reportamos un caso de este tipo con la descripción técnica.

Caso clínico. Evaluamos un paciente con vértigo incapacitante. La resonancia magnética no mostró ningún tipo de compresión vascular en la REZ o en la cisterna pontocerebelosa, pero si evidenció un bucle de la arteria cerebelosa anteroinferior (AICA) a nivel del meato auditivo interno entre el 7o y 8o nervio craneal. La descripción técnica es detallada e ilustrada. Después de la cirugía los episodios incapacitantes de vértigo desaparecieron. La evolución post operatoria fue satisfactoria.

Conclusión. La descompresión del nervio vestibular secundario a un bucle intrameatal de AICA puede ser el tratamiento del vértigo posicional debido a un conflicto neurovascular intrameatal evidenciado mediante imágenes de resonancia magnética de alta resolución.

Palabras clave

Descompresión microvascular,
vértigo, nervio vestibular, nervio
vestibulococlear

Abstract

Introduction. Disabling positional vertigo, likely to be originated by vestibular nerve (VN) vascular compression, may benefit from microvascular decompression (MVD) of VN. Most VN compressions were observed at Root Entry / Exit Zone (REZ) and adjacent brainstem or in cerebellopontine cistern. Only a few cases have been reported, conflict being within the internal auditory canal (IAC), but without detailed technical description. Authors hereby report a case with technical description.

Case report. The patient consulted for disabling vertigo. Magnetic resonance imaging did not show any vascular compression at REZ or in cerebellopontine cistern, but a loop of anterior inferior cerebellar artery (AICA) in IAC. Technical description is detailed and illustrated. After surgery vertigo attacks disappeared. The outcome was good.

Conclusion. Vestibular nerve decompression from an intrameatal loop of AICA may be the solution for disabling positional vertigo due to intrameatal neurovascular conflict evidenced by high-resolution imaging.

Keywords

Microvascular decompression surgery, vertigo, vestibular nerve, vestibulocochlear nerve

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Introducción

Disabling positional vertigo is defined as disequilibrium without fluctuating hearing loss, precipitated with head movements and not fatigable, likely to be originated by vestibular nerve vascular compression. Jannetta was the first to treat this syndrome with microvascular decompression (MVD), and he and others reported a success rate of up to 80%.^{1, 6, 11}

The compressive vessels may be vertebrobasilar artery, posterior inferior cerebellar artery, anterior inferior cerebellar artery (AICA) or a combination of them. Most vestibular nerve compressions were observed at the Root Entry / Exit Zone (REZ) and adjacent brainstem or in the cerebellopontine cistern. Only a few cases were reported, the conflict being within the internal auditory canal (IAC). Two publications, each one of a large series, mentioned neurovascular conflicts due to AICA loop in the IAC; but they did not give precision on number, anatomical relationships with the nerve and surgical procedure done.^{1, 6, 11} McCabe and Harker reported 8 cases of vestibulocochlear nerve compression hypothesized in relation with the intrameatal segment of AICA. Patients were treated by vestibular neurectomy.¹⁰ Another publication reported one case in whom compression was by a vein in coexistence with a small schwannoma. The author performed decompression by resection of the IAC posterior wall and insertion of Teflon between the nerve and the vein loop; schwannoma was not removed.¹⁵

Relationships of AICA with the vestibulocochlear nerve complex have a large number of variants. Presence of AICA within the IAC was reported at 40-50%.^{8, 9} Existence of a labyrinthine artery in close inferior or anterior relationships with the vestibulocochlear nerve inside the IAC was noticed in 21 - 30% of the cases.⁸ Relationships between the nerve and AICA and its branches, mainly the labyrinthine artery, are important to take in account when considering vascular decompression, as loop manipulation would entail a high risk of

labyrinthine artery damage. The present report is a case of vestibular nerve decompression from an intrameatal loop of the AICA.

Case Report

Clinical presentation

A 32-year-old man presented at our institution with history of recurrent positional vertigo lasting from several minutes to hours, secondary to head position changes during the day and not lessened with rest. The vertigo was not accompanied with visible nystagmus, tinnitus or hearing loss. Patient was treated with antivertiginous drugs without effective response. Neurological, audiometric and vestibular examinations were normal. Magnetic Resonance Imaging (MRI) showed no abnormality on right side. On left side, there was no obvious vascular compression at brainstem or cerebellopontine cistern; there was an image of AICA loop inside the IAC, compatible with vestibular nerve compression (*Figure 1*). Because of disability due to extremely frequent vertigo attacks, the patient was referred for attempt at microsurgical decompression.

Anatomic findings and operative procedure

Installation was in lateral decubitus position (*Figure 2A*). Under general anesthesia with tracheal intubation, approach was a left retromastoid craniotomy (*Figure 2B*), consisting of a 2.5 cm bone flap with additional bony resection to "skeletonize" the transverse and sigmoid sinuses (*Figure 2B, 2C, 2D, 2E*). A dural flap was turned toward the sigmoid sinus. Under the operative microscope arachnoid was opened, first in front of the XIth, Xth and IXth nerves, then posteriorly to the VIIIth and VIIth (*Figure 3A*) and up to the Vth nerve (*Figure 3B*). An AICA loop was entering deeply into the IAC as observed on MRI. Because of the AICA loop intrameatal location, the posterior wall of the IAC was removed (*Figure 3C-D*). Dura covering the IAC posterior wall was incised so that VIIth and VIIIth nerves were totally exposed. The loop passed between the

vestibulocochlear nerve complex posteriorly and the facial nerve anteriorly. The labyrinthine artery arose from the apex of the loop to penetrate into the IAC fundus (*Figure 4B-4C*). Care was taken not to stretch the artery, which was short and tense. Dense arachnoid adhesions were surrounding the vascular-nervous complex. Intense congestion of the nerves was noted. Dissection and liberation of each of the different vascular and nervous elements from arachnoid adhesions were carried out (*Figure 5A-5B*). Nerves, especially the vestibular one, had a greyish aspect at the site of the compression (*Figure 4A*). Then a small bundle of 2 mm in width and 1 cm in length made of Teflon felt fibers was interposed between the AICA loop and the VIIth nerve on one side and the AICA loop and the VIIIth nerve on the other side (*Figure 5D*). Although AICA loop was only minimally mobilized because of the risk

of stretching the labyrinthine artery, a mechanical vasospasm occurred, that was reversed within the next minute by topical application of a few droplets of 10% papaverine saline solution. A petrous bone cells opened was occluded with fat that was affixed by fibrin glue (*Figure 5C, 5D*). Dural closure was tightly done with a patch of fascia lata. Bone flap was put back; aponeurotic, subcutaneous and skin layers were closed with interrupted sutures.

Postoperative course and outcome

On awaking, a facial palsy was present of Brackman-House (B-H) grade 3. Vertigo sensation still present up to discharge, i.e., during the four first postoperative days, resolved completely during week following hospitalization. At first outpatient visit, on 30th postoperative day, wound was well sealed and vertigo attacks had disappeared.

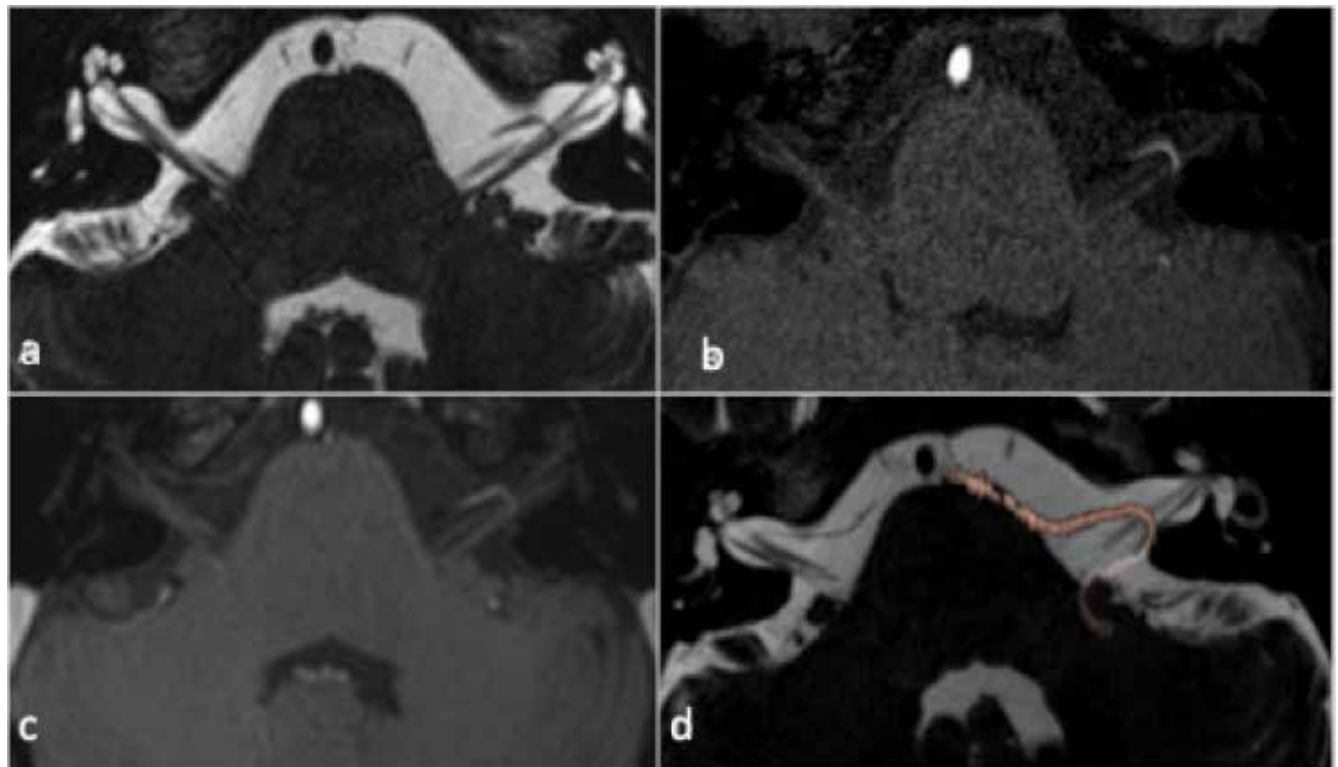


Figure 1. High resolution MRI with the three following sequences in association showing AICA (antero inferior cerebellar artery) loop cross-compressing VIIIth nerve inside the internal auditory canal (IAC). (a) T2 Drive sequence shows fine cisternal and intrameatal anatomy. (b) TOF-Angio sequence shows no vascular conflict on right side. On left side, there is no vascular conflict at brainstem / REZ but intrameatal loop cross-compressing VIIIth nerve inside IAC. (c) T1-Gado sequence shows on left side the AICA loop and no venous conflict. (d) 3D fusion - reconstruction shows on left side the intrameatal AICA loop course and its contact with the vestibulocochlear nerve.

Neurological examination revealed that the gait / walking was normal in speed, without any deviation even with eyes closed. No nystagmus was found, either spontaneous or in lateral gaze. No disturbance in upper limb coordination was observed. Facial palsy had improved from B-H grade 3 to 2. Patient could close eyes and whistle. Audiometric testing showed unimpaired hearing.

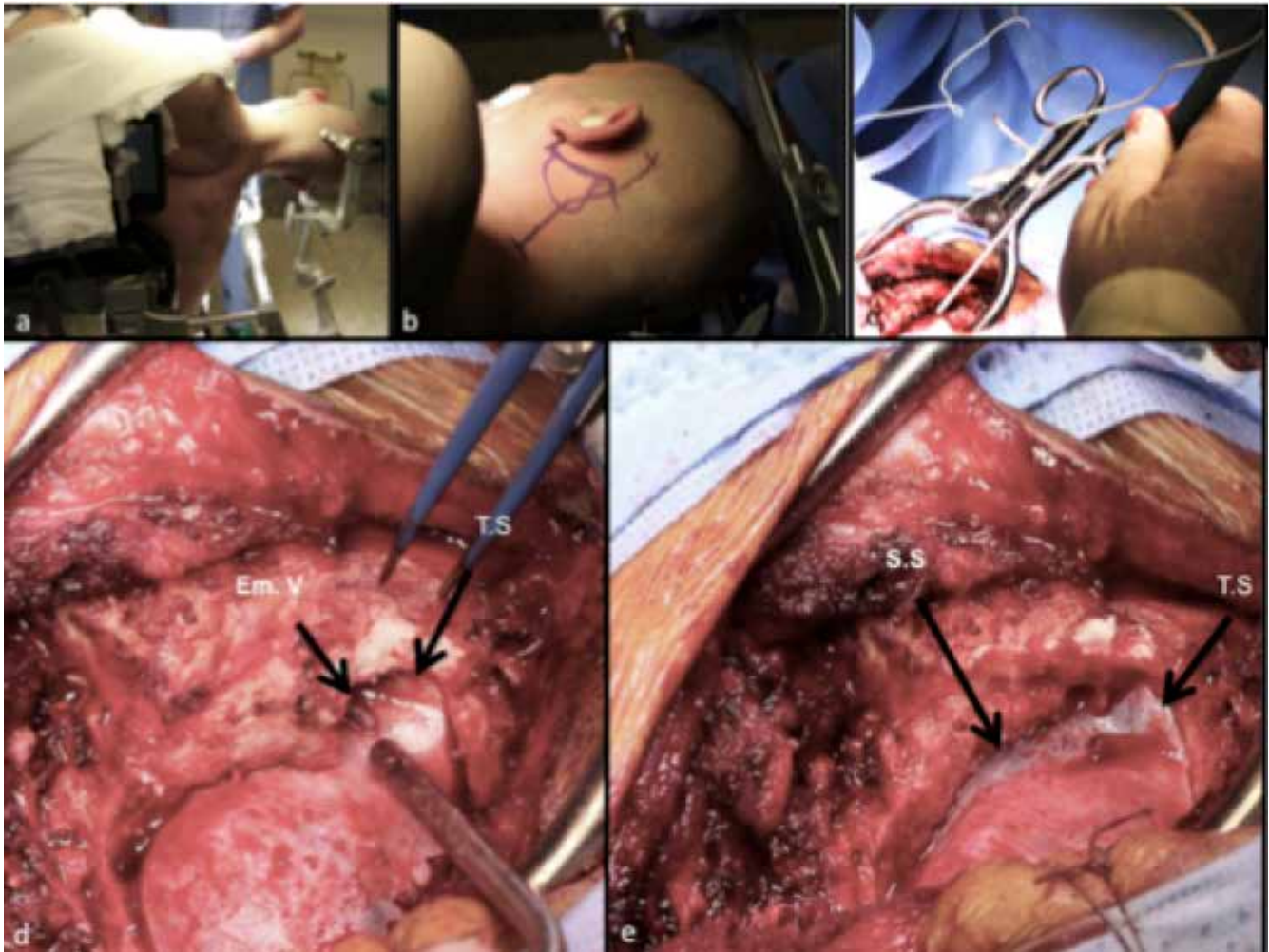


Figure 2. (a) Park-Bench decubitus lateral positioning with contralateral neck inflexion to avoid surgeon's view obliterated by shoulder. (b) Landmarks for incision and drawing of craniotomy posterior to mastoid tip for infra-floccular approach. (c) Identification of the transverse sinus and sigmoid sinus using intraoperative doppler. (d) After transverse sinus (TS) was skeletonized, emissary vein (EV) is identified and coagulated. (e) Completed exposure of the TS, which has a solid wall, and of the sigmoid sinus (SS), which has a fragile wall.

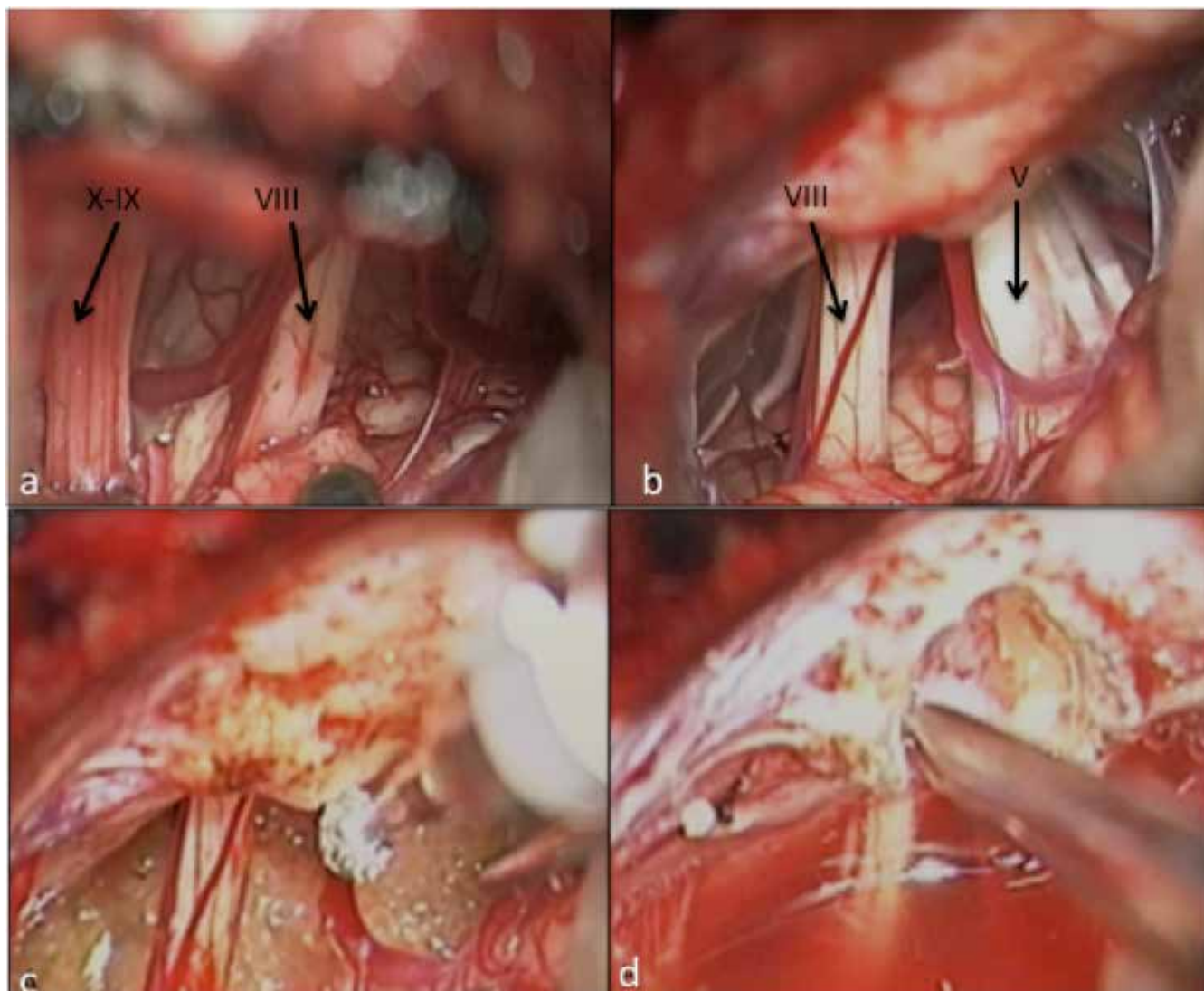


Figure 3. Microsurgical exposure in left CPA cistern of Xth - IXth rootlets and VIIIth nerve (a). VIIIth and Vth nerves, and the AICA loop entering IAC (b). Note that the posterior wall of IAC masks the intrameatal vascular conflict. The IAC posterior wall is drilled out (c) and rongeur (d) until the VIIth - VIIIth nerves and the AICA are exposed.

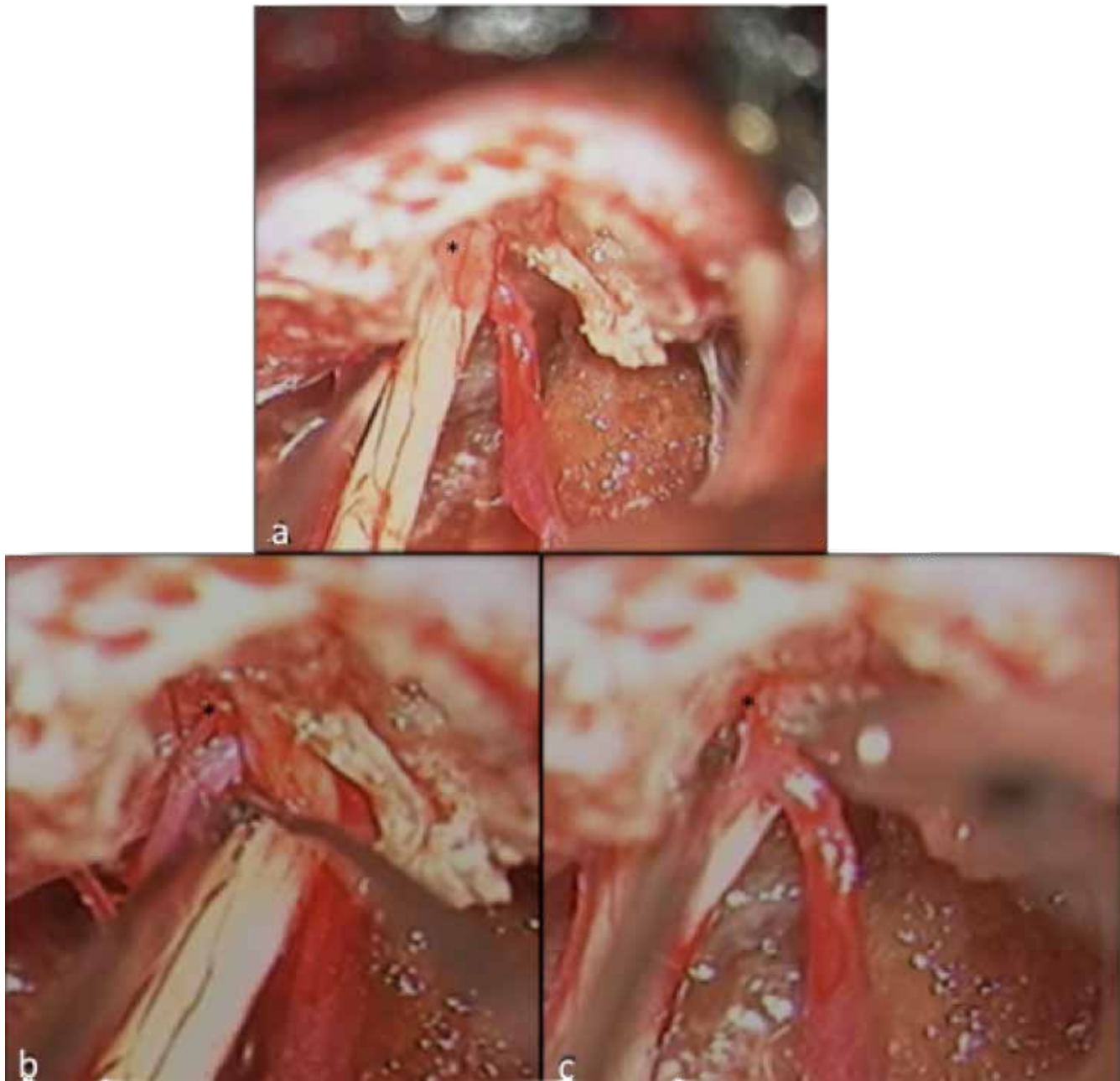


Figure 4. After resection of the IAC posterior wall and dura incised, the AICA loop is visible; it passes in between the VIIIth nerve posteriorly and the VIIth anteriorly. Note that the vestibular nerve has a greyish aspect (*) at the site of the vascular compression by the AICA loop. Note also vasospasm of AICA loop due to mechanical stress (a). The labyrinthine artery seen from below (b) and from above (c) arises from the apex of the AICA loop to penetrate into the IAC fundus. Due to its shortness and tension, its manipulation and stretching would be risky for hearing function.

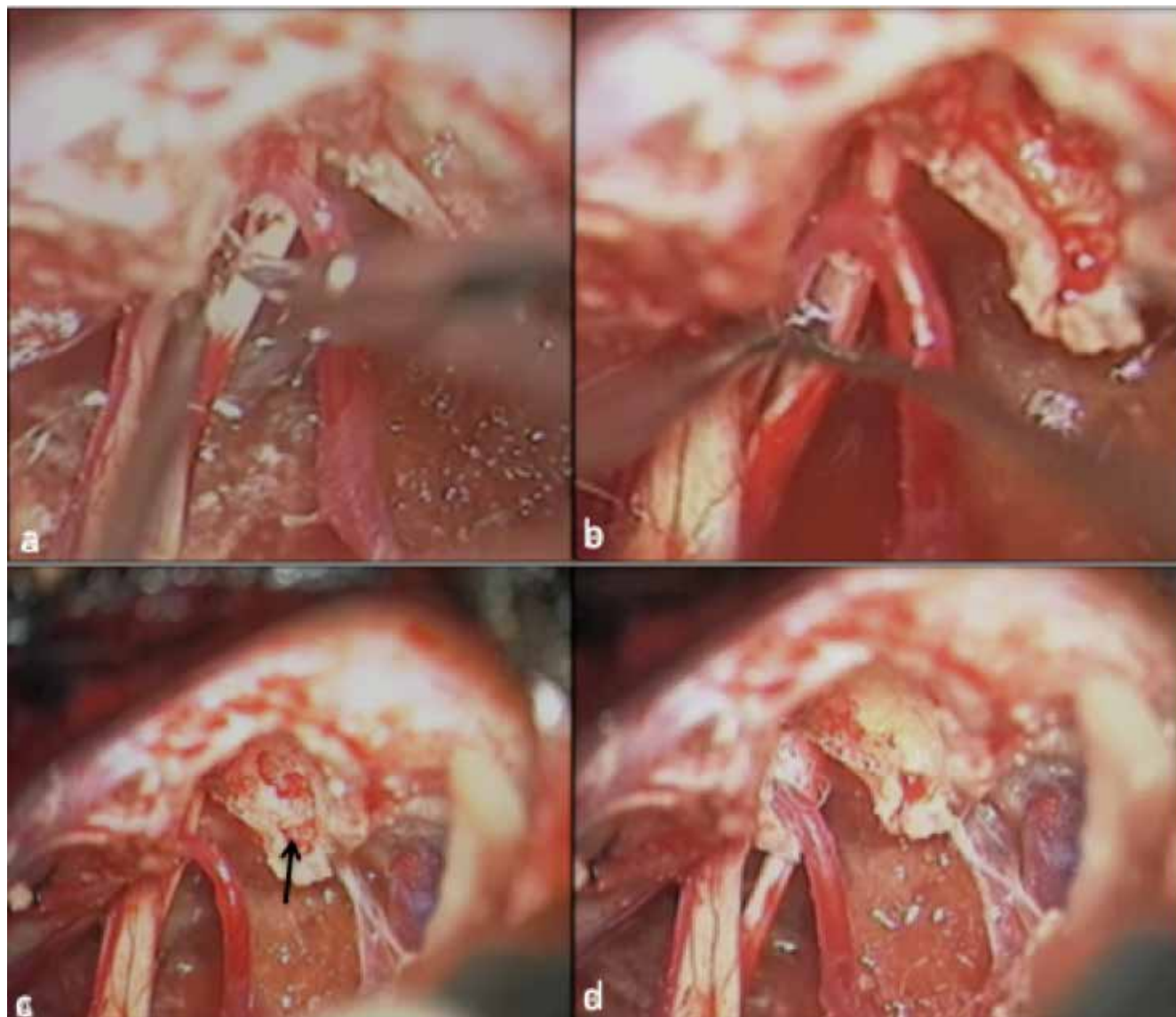


Figure 5. (a) Dense arachnoid filaments are adherent to the vascular-nervous complex. (b) Nerves and artery are freed from the adhesions. (c) A cellula of the petrous bone was opened (arrow) at posterior and superior aspect of the IAC. (d) After liberation of the nerves, a bundle made of Teflon felt fibers is interposed between AICA loop and VIIth nerve on one side and AICA loop and VIIIth nerve on the other side. Note that the petrous bone cellula was occluded with fat and fibrin glue.

Discussion

Classically, vascular compression responsible for cranial nerve hyperactive functional syndromes are mostly located at brainstem / REZ, that is along the central myelin (CM) portion of the nerve.^{4, 5} According to literature, vestibular compressions responsible for disabling positional vertigo were mostly reported not only at brainstem / REZ but also in the cerebellopontine cistern,^{1, 2, 11, 13} and very few cases at IAC. For VIIIth nerve the CM portion has a length of $11.5 \text{ mm} \pm 1.5 \text{ mm}$,⁵ which corresponds roughly to the cisternal segment of AICA and also the adjacent part of its meatal segment. CM portion together with its transitional zone (TZ) have been hypothesized to be a more vulnerable and "excitable" structure.^{2, 5, 6, 11, 13} McCabe and Harker reported 8 cases of vestibulocochlear nerve compression by an AICA intrameatal loop. Was the loop inside the meatus the cause of vertigo? The authors did not carry out AICA decompression because they estimated that manipulation of the loop would have entailed excessive risk for the labyrinthine artery. They performed vestibular neurectomy.¹⁰ In Moller et al.¹¹ and Brackmann et al.¹ series authors mentioned intrameatal AICA compression but did not give any details on the number, surgery and results in these particular cases. Wuertenberger and Rosahl reported one patient with disabling vertigo and tinnitus in relation with a vestibulocochlear vein compression in coexistence with a small schwannoma at fundus of IAC. They only decompressed the nerves by resecting the IAC posterior wall without removing the schwannoma, and interposed teflon between nerve and vein.¹⁵ It is not clear whether the main compressive factor was the tumor or the vein. Nevertheless the patient had a good outcome.

MRI evaluation to determine the characteristics of the vascular compression is important. According to Leal et al., association of the three high-resolution MRI sequences: 3D-T2, 3D-TOF and 3D-T1 with Gadolinium provides a good sensitivity and specificity to depict neurovascular conflicts.⁷ 3D-T2 sequence allows a fine anatomical analysis of

the pontocerebellar structures with good contrast between cerebrospinal fluid (CSF) and vasculo-nervous cisternal structures, but the limits are the absence of differentiation of signal not only between arteries and veins but also between vessels and nerves. 3D-TOF-angio sequence allows visualization of the arteries, in hyper signal. 3D-T1 with Gadolinium sequence shows nerves in intermediate signal in relation to CSF (in hypo signal) and arteries and veins (in hyper signal). Comparison of 3D-TOF and 3D-T1-gado allows differentiating arteries from veins.

Cure rates of MVD for disabling vertigo are not as high as for trigeminal neuralgia, hemifacial spasm or vago-glossopharyngeal neuralgia, which are in the order of 85%¹⁴. Despite the efforts to establish proper selection in patients presenting with disabling vertigo, criteria have not yet been completely established. Good candidates are the patients with drug-resistant incapacitating vertigo and evidence of vascular vestibular nerve compression.¹² When there is no clear-cut compression at brainstem / REZ, an image of intrameatal AICA loop may be considered amenable for decompressive surgery. Logical approach is a retromastoid cerebellopontine exposure of the VIIIth nerve and AICA, followed by drilling of the IAC posterior wall. IAC opening was not frequently reported in the literature for vascular decompression; authors preferred to perform vestibular neurectomy.¹⁰ Experience with microsurgical removal of intracanalicular schwannomas with excellent hearing postoperative outcome,³ should incite to perform such approach in cases with likely responsible intrameatal loop of AICA, as in our reported patient.

Conclusión

Vestibular nerve decompression from an intrameatal loop of AICA may be the solution for disabling positional vertigo due to intrameatal neurovascular conflict evidenced by high-resolution imaging.

Declaración de conflictos de interés

Los autores declaran que en este estudio no existen conflictos de interés relevantes.

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