Petrous internal carotid artery aneurysm: A cause of chronic otitis

Petrous Internal Carotid Artery aneurysm:

a cause of chronic otitis

Hélène Maréchal[a,b](*), Guillaume Dannhoff[a](*), Julien Todeschi[a], Thibault Dedieu[b], Raoul Pop[c], Salvatore Chibbaro[a]

[a] Department of Neurosurgery, Strasbourg University Hospital, 1 Avenue Molière, Strasbourg, France

[b] Department of ENT, Strasbourg University Hospital, 1 Avenue Molière, Strasbourg, France

[c] Interventional Neuroradiology Department, Strasbourg University Hospital, 1 Avenue Molière, Strasbourg, France

(*) The first 2 authors contributed equally to the paper.

Corresponding author:

Hélène Maréchal MD

Address: Service de Neurochirurgie - Hôpital de Hautepierre, 1 Avenue Molière, 67000 Strasbourg, France

Telephone: 0033 388 127 624

E-mail: helene.marechal@chu-strasbourg.fr

Author contributions:

Protocol/project development: Maréchal, Dannhoff, Chibbaro

Data collection or management: Maréchal, Dannhoff, Dedieu, Todeschi

Manuscript writing/editing: Maréchal, Dannhoff, Chibbaro

Illustrations and figures: Dannhoff, Maréchal, Pop

Critically revision: All authors.

Abstract

***Background* -** Aneurysm of the petrous segment of the internal carotid artery (pICA) is a rare pathology presenting with extracranial and especially oto-rhinological symptoms that can be misleading and delay diagnosis.

***Methods* –** We report the case of a giant pICA aneurysm compressing the Eustachian tube (ET), presenting with hearing loss due to chronic serous otitis. A PRISMA review of the literature was performed to find similar cases. In addition, relevant anatomical sources were screened.

***Results* -** Five reports about 7 cases of middle-ear effusion caused by pICA aneurysm compressing the ET were identified. Median age at diagnosis was 18.5 years. After endovascular treatment, overall outcome was favorable, with no mortality, although outcome was sometimes impaired by neurological comorbidities and unclear prognosis of hearing-loss recovery.

Discussion - These reports, though rare, offer relevant insights into the poorly known regional anatomy of the pICA, in the borderland between neurosurgery and ENT. Within the petrous bone, the osseous separation between the ET and the pICA is narrow, when not dehiscent. This leads to a risk of any pathological process in either the pICA or the ET impinging on the other.

***Conclusion* -** Giant pICA aneurysm is a rare cause of hearing loss, due to compression of the ET, leading to chronic serous otitis. This co-dependency between pICA and ET should be kept in mind, as it underlines the necessity of multidisciplinary management and could facilitate earlier diagnosis and therapeutic management when facing atypical clinical situations.

Keywords: aneurysm, Eustachian tube, internal carotid artery, conductive hearing loss

Abbreviations: DSA, digital subtraction angiography; ENT, ear, nose and throat; ET, Eustachian tube;

MCA, middle cerebral artery: pICA, petrous segment of the internal carotid artery.

1. Introduction

The petrous segment of the Internal Carotid Artery (pICA) runs within the carotid canal in the temporal bone, surrounded by functional structures that are underknown by neurosurgeons. Although the ICA, lying in its bony canal, is well protected in its petrous segment, it remains exposed to dissection and other pathological entities like infections, radiation and congenital malformations that could lead to the formation of aneurysms [1–3].

In the international English literature, only few case reports of extracranial ICA aneurysms - ie. affecting its cervical or petrous segments - are available. When symptomatic, petrous aneurysms usually present with neurological or oto-rhinological signs due to mass effect or hemorrhage [4–6]. The ear, nose and throat (ENT) presentation can be characterized by abrupt massive epistaxis/otorrhagia [4] or chronic symptoms such as hearing loss. The latter presentation is rarer and thus constitutes a particularly challenging diagnostic issue, as emphasized by the case here reported along with the literature review.

The present study is about the rare and unusual clinical presentation of a patient suffering from direct compression of the Eustachian Tube (ET) by a pICA aneurysm, hence revealed by middle ear chronic otitis. We performed a literature review according to the PRISMA statement in order to identify all cases of analogous presentation to depict common as well as peculiar features [7].

Such a rare and unusual presentation appears particularly relevant to neurosurgeons as it points out the need for a thorough understanding of anatomical structures surrounding the pICA. Particularly, the authors would like to emphasize how ET and pICA course side-by-side, as this counterintuitive fact might guide the understanding of all clinical manifestations as well as the management strategy of this rare pathology.

2. Methods

We report the case of a woman harboring a pICA aneurysm causing direct compression of the ET focusing on its initial diagnosis, clinical evolution and management. A literature review was also conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) statement [7] in order to identify similar cases. A literature search was performed in Pubmed and Ovid MEDLINE in August 2022, with no restriction regarding publication date, using Mesh terms [Aneurysm] AND [Hearing disorders] OR [Otitis]. Non-English or unavailable full-text records were excluded.

Two authors were implicated in the review process (HM, GD): one ENT surgeon and one neurosurgeon. Initial selection, screening of the abstracts and full-text readings were performed independently. No mismatch in articles selections was to be noted between both authors at the end of the process. References of previously selected articles were carefully analyzed, leading to two additional inclusions. Data were then extracted commonly by the two aforementioned authors; data accuracy was checked independently by a third author (TD). Finally in order to enrich the anatomical discussion, the authors manually selected sources with strong anatomical and historical relevance.

3. Results

3.1. Illustrative case

A 37-year-old woman, previously healthy, presented to our observation with a 2-month history of progressive right hearing loss associated with otalgia and intermittent tinnitus. The patient was initially referred to an ENT specialist who diagnosed a right chronic serous otitis. An audiogram investigation showed a conductive hearing loss, with a Rinne test of 40 dB.

Initial brain and internal auditory canal MRI revealed an expanding intra-petrous lesion developing from the right ICA and compressing the ET. Subsequent Angio-CT and angio-MRI demonstrated a partially thrombosed giant (25 mm of diameter) pICA aneurysm, with a large circulating portion of 16 mm (Fig. 1 A, B). After discussion during our weekly multidisciplinary meeting, an endovascular management was decided and scheduled.

However, before treatment could be performed, the patient presented to the emergency department 3 weeks later, with an abrupt left body sensorimotor deficit and slurred speech. MRI showed an acute right sylvian ischemic stroke with thrombotic stenosis of the right M1 terminal segment (Fig. 2 A, C).

Urgent Digital Subtraction Angiography (DSA) showed a pseudoaneurysm aspect of the pICA. The patient thus underwent a combination of the following strategies:

- Urgent mechanical thrombectomy of the right MCA, allowing complete recanalization (Fig. 2B)
- Stenting of the pICA with a 7/30 mm Wallstent (Boston Scientific, Marlborough, Massachusetts, USA), at the level of the aneurysm neck in order to restore the narrowed lumen (Fig. 2D)
- Coiling of the remaining circulating aneurysm portion (Fig. 2 E).

In the post-procedural stage, the patient showed a partial Horner's syndrome probably caused by sympathetic irritation due to stent positioning. Symptoms went remarkably improving within 7 days, with only persistence of a mild narrowing of the right eyelid rim and complete recovery of the sensorimotor deficit. Six weeks later, due to a persistent conductive hearing loss, a trans-tympanic drain was inserted. At 3, 6 and 12-month clinical follow-up the patient showed full symptoms recovery and reported a generalized well-being.

Further etiological investigations did only identify moderate tobacco use (10 cigarettes/day for a few years). The BMI was 24, lipid and glycemic evaluations were normal. All advanced explorations did not show any other factor favoring the occurrence of an ischemic event: electrocardiogram showed no atrial fibrillation, transthoracic ultrasound showed an isolated small-sized patent foramen ovale, doppler ultrasound of supra-aortic trunks as well as aortic, leg and renal arteries angio-MRI showed no abnormality. No signs for either connective tissue disease or fibromuscular dysplasia were found.

3.2. Review of literature

A detailed literature search revealed 5 reports gathering 7 analogous clinical cases of chronic otitis media caused by direct compression of the Eustachian tube by a pICA aneurysm. The detailed clinical outcomes of each case included in our literature review are presented in Table 1.

Leonetti et al study [8] included 79 patients with occult skull base lesions causing middle ear effusion by obstructing or invading the ET. Beside a predominance of tumoral lesions, 3 patients (3.8%) corresponded to a giant compressive aneurysm of the pICA.

The median age of presentation was 18.5 years old (ranging from 13 to 37 years) with a sex ratio of 3M/1W. Association with neurological signs was reported, under the form of Horner’s syndrome for 2 patients and retro-orbital headache homolateral to the aneurysm for 1 patient. All patients underwent an endovascular treatment. Level of hearing loss recovery remained unclear, with a partial recovery documented in 2 patients. Horner’s syndrome persisted in 1 patient.

No thromboembolic complications or hemorrhage have been reported in these 7 cases. However, in Cooper et al study [9], the petro-cavernous junction of the aneurysm was noted to indent the posterolateral wall of the sphenoid sinus with erosion of this part of the sphenoid sinus. This has been described as a potential site of aneurysm rupture into the sphenoid sinus by the authors.

4. Discussion

Anatomical knowledge is a well-known basis of surgical training [10,11] but should not be limited to focal areas of most surgical interest, as it can unravel diagnostic issues in lesser known regions. Anatomy of the pICA, encased in its osseous box, is on the borders of ENT and neurosurgery and can easily be disregarded. The narrow boundaries between pICA and ET can thus come as counterintuitive to the clinician but bears strong clinical significance. As these two structures walk side-by-side within the petrous bone, possible intermingling of their pathological processes should be kept in mind [12].

Hence, this discussion section focuses in its first part on anatomical overview of regional ICA anatomy and its relationships with surrounding structures. The second part addresses the diagnostic and therapeutic issues raised by the present cases along with their anatomical background.

4.1. Anatomy

Various nomenclatures and ICA segmentation classifications have been proposed in the literature. For our report, we refer to Bouthillier et al. classification because of its peculiar neurosurgical relevance. ICA is divided in seven segments defined by surrounding anatomical structures at each level (C1, cervical; C2, petrous; C3, lacerum; C4, cavernous; C5, clinoidal; C6, ophthalmic; and C7, communicating) [13]. The cervical segment (C1) is of great mobility. It becomes the petrous segment (C2), fixed in an osseous compartment, when entering the base of the skull into the carotid canal. This transition site constitutes a preferential site of arterial dissection (Fig. 4 A).

The petrous segment can be divided into a vertical portion, a genu, and a horizontal portion.

- The vertical portion enters the carotid canal anteriorly and internally to the internal jugular vein. It is anteromedial to the tympanic cavity.
- The genu is the turn between the vertical portion and the horizontal portion. It runs medially to the osseous part of ET [14].
- The horizontal portion is longer and runs anteromedially toward the petrous apex, it finally passes above (and not through) the foramen lacerum (which is filled by fibrocartilaginous tissue) and there turns upward to become the lacerum segment (C3) [10,13].

pICA may give 2 inconstant types of branches: the artery of the pterygoid canal (or vidian artery) and the carotico-tympanic arteries. The artery of the pterygoid canal arises from the horizontal portion and passes through the pterygoid canal with the corresponding nerve. The carotico-tympanic arteries arise from the vertical portion or genu of pICA and enter the tympanic cavity [10].

The ET links the middle ear to the nasopharynx to regulate the air pressure gradient between the middle ear and the atmospheric compartment. It runs in an anterior, medial and inferior way from the middle ear to the nasopharynx. ET can be divided in two parts: the posterolateral osseous part and the anteromedial fibrocartilaginous part [15]. Attachment of the *levator veli palatini* muscle, that contributes to opening and closing of the ET, is located just inferiorly to the junction of these two portions.

The osseous part begins at the anterior wall of the middle ear and ends with its medial opening just medially to the sphenoid spine where it continues with the cartilaginous part (Fig. 4 C). The osseous part of the ET is lateral to the superior portion of the vertical segment and genu of the pICA. Anatomical studies unanimously describe the medial wall of the osseous part as a very thin plate of bone [4,5,10,12,16,17], with an average thickness of 0.8 mm [12]. In 56% of cases, this bone wall separating the pICA and the ET would be even thinner, measuring 0.1 mm to 0.3 mm in thickness. This bony separation can be found dehiscent in cadaveric studies in 6% to 40% of cases [12,16].

The cartilaginous part of the ET can be subdivided into 3 parts [15]. The posterolateral part runs in the tubae sulcus, which is a groove, under the junction between the sphenoid bone greater wing and the petrous apex. The tubae sulcus is medial to foramen ovale and foramen spinosum. It is anterior to the horizontal segment of pICA [10]. Then, the middle part of cartilaginous ET runs anterolaterally to the foramen lacerum. Finally, the anteromedial part of cartilaginous ET runs inferior to the scaphoid fossa, it is attached to the medial pterygoid plate, runs inferiorly to the vidian canal (pterygoid canal) and finally gets through on the lateral wall of nasopharynx, anterior and inferior to Rosenmüller's fossa [18,19]. The cartilaginous part of ET runs infero-laterally, almost parallel to the horizontal pICA. The internal carotid artery is located superiorly to the vidian canal.

4.2. Pathology

In most cases, pICA aneurysms are diagnosed in young patients [5,6]. They are often related to trauma, iatrogenic causes (such as radiation therapy or middle ear surgery), otological infection [1–3] or congenital malformation. No evidence of these etiologies was found in our patient. Delayed formation of a pseudoaneurysm secondary to asymptomatic arterial dissection is strongly suggested in our case by the narrowing of the lumen at the level of the aneurysm. Though most dissections heal spontaneously with ad integrum recovery, it may sometimes lead to the formation of pseudoaneurysms [20–22].

Mesh term “hearing disorders” gathers hearing losses and tinnitus. Through our literature review, we found some cases of extracranial ICA dissection, extending just proximal to the petrous portion, with or without pseudoaneurysm, revealed by tinnitus [21,23,24]. Hearing loss associated with pICA aneurysm has been reported due to cochlear erosion, chronic otitis media, hemotympanum or extension of the aneurysm into the middle ear [3,4,9,25–29]. Reciprocally, infratemporal carotid pseudoaneurysms have been described as complications of chronic otitis media. Indeed, chronic infection can lead to weakening of arterial wall and aneurysmal formation/dilatation (i.e. mycotic aneurysm) [30]. Moreover, several cases of malignant otitis externa causing intrapetrous or cervical ICA pseudoaneurysm have been reported [31–33].

Among skull base lesions causing ET obstruction, pICA aneurysms are rare (3.8% in Leonetti et al) but remind us that physical examination and nasopharyngoscopy are not sufficient to rule out all etiologies. As suggested in Leonetti et al study [8], MRI or CT with contrast must be carried out before trans-tympanic drain insertion in patients with persistent unilateral chronic otitis media and normal findings on physical examination (including nasopharyngoscopy).

Clinical presentations of pICA aneurysms are characterized by a large range of symptoms going from headaches, cranial nerve palsies (especially VIII, VII and V nerves) to Horner syndrome, epistaxis, tinnitus, and hearing loss [5,6,34,35]. Overall, symptoms and clinical signs at diagnosis of a pICA aneurysm depend mostly on the aneurysm's location and its growth direction, leading to impairment of various structures.

In our patient, compression on the osseous part of the ET was caused by the superior portion of vertical pICA aneurysm. Medial wall of the ET was dehiscent at the level of the aneurysm (Fig 3 B and D). This explains the unusual manifestation as a chronic serous otitis causing hearing loss. The 7 cases here reported illustrate how pICA aneurysms tend to impinge on the ET, causing middle ear disorders. In the literature, cases of epistaxis by pICA aneurysm rupture have been reported, bleeding through the ET [34,35]. Added to the aforementioned pathologies spreading the other way around from the middle ear to the pICA, this illustrates the reciprocal relation between pathologies of the pICA and pathologies of the ET. Independently from the etiology, one should keep in mind that any evolving pathological process affecting either the pICA or the ET could endanger the other.

Outcome of pICA presenting at diagnosis with serous chronic otitis appears globally favorable. However in our case, evolution has been unexpectedly hindered by an ischemic stroke occurring shortly after diagnosis. Among the cases identified in the literature, our case is the first ischemic complication reported. Given the sequence of events, with rapid thrombosis of the dissecting aneurysm occurring in the three weeks between initial diagnosis and the ischemic stroke, the most likely stroke etiology was distal embolization in the M1 segment originating from the thrombosed aneurysmal sac. As already discussed in the results, no alternative stroke etiology was detected upon systematic screening in the neurovascular unit. The causal relationship between cervical ICA dissections and distal intracranial embolization is well documented. Tandem lesions (defined as cervical ICA occlusion / stenosis in addition to an intracranial arterial occlusion) are encountered in around 15% of acute stroke thrombectomies in the anterior circulation [36] and dissections account for approximately 20% of tandem lesion thrombectomies.

Because of their deep localisation, pICA aneurysms are preferentially managed endovascularly with excellent results [37,38]. A dissecting aspect of the ICA was observed during endovascular management, that was retained as the probable occult cause underlying pseudoaneurysm formation, as suggested in the literature [3]. This justified a combination of stenting to treat the dissection and re-establish normal caliber of the ICA associated with coiling to occlude the residual aneurysm. The treatment strategy should be tailored on a case-by-case basis [39] depending on aneurysm morphology and location. Fusiform aneurysms are more technically challenging; endovascular management for these cases can be performed either by parent artery occlusion [39] or using single or multiple telescoping flow diverter stents [40].

4.3. Limitations

The present study is relatively limited by the paucity of data available in the literature. As the aforementioned pathology is a rare cause of middle ear obstruction, a few cases might have gone unnoticed as they might be hidden in larger series including a wide range of compressive etiologies. Nevertheless, the initial research equation was designed to widely address the subject and minimize the risk of missing pertinent reports. In a will of completeness of the present work, some cases were included despite lack of clinical data, such as Leonetti's et al report [8].

Moreover, it is to be noted that in the 7 cases encountered through our literature review, none of the authors did precisely report the location of the Eustachian tube compression. The detailed anatomical background depicted here aimed to fulfill that gap. As demonstrated tangentially in the present work, anatomy provides strong reasoning tools to approach the pathology at stake and thenceforth to assess the likely site of compression and clinical outcomes.

5. Conclusion

pICA aneurysms represent a rare pathology presenting clinically by extra-cranial and especially oto-rhinological symptoms, involving a multidisciplinary team of ENT, interventional neuroradiologists and neurosurgeons. Though rare, these reports are crucial as they offer relevant insights into the anatomy of this underknown region, illustrating the close anatomical relationship between pICA and ET. As these two structures navigate side-by-side within the petrous bone, possible extension of any pathological process from one structure to the other should be kept in mind. More systematic consideration of this regional anatomy could lead to early diagnosis and therapeutic management, avoiding late complications such as ischemic stroke.

Figures

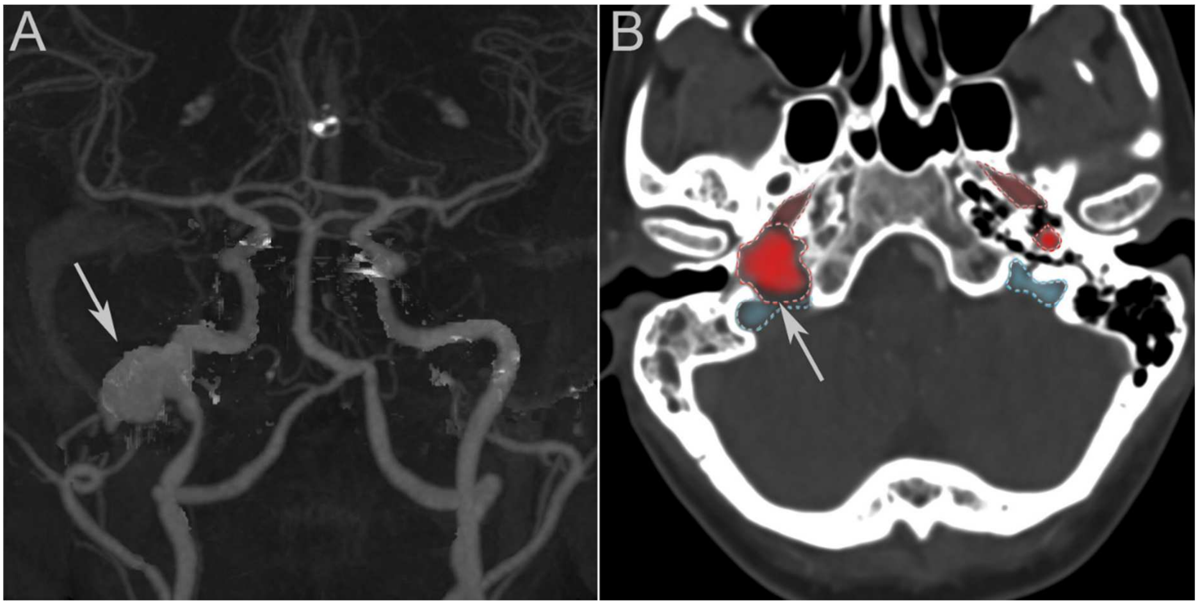

Figure 1. Initial imaging

A – Cerebral CT angiography, maximum intensity projection (MIP) reconstruction, depicting a 16 mm irregular shaped dissecting aneurysm (white arrow) on the lateral aspect of the subpetrosal segment of the right internal carotid artery (ICA). **B** – Axial images showing anterior extension of the aneurysmal sac causing compression of the Eustachian tube, as well as posterior extension into the jugular foramen.

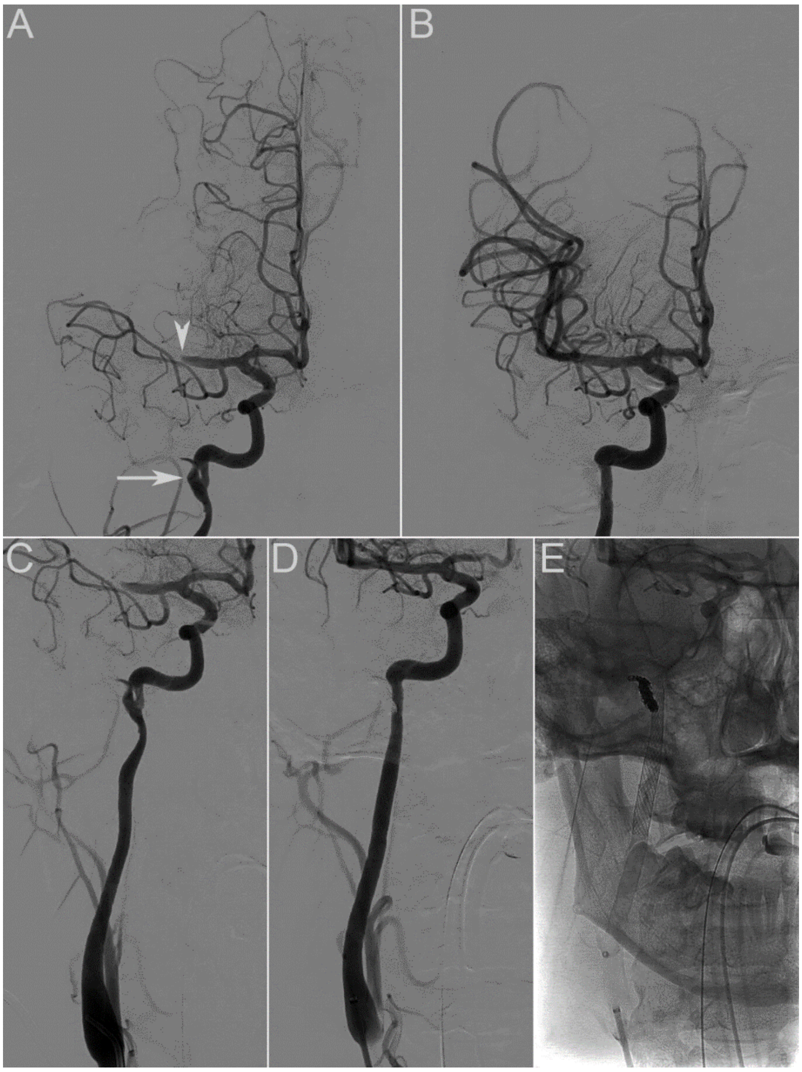

Figure 2. Mechanical thrombectomy and aneurysm embolization

Following acute presentation with acute ischemic stroke in the territory of the right middle cerebral artery (MCA), emergency endovascular thrombectomy was performed. On initial antero-posterior selective angiographic runs of the right ICA (**A and C**), spontaneous thrombosis of the aneurysmal sac was observed, with a small residual circulating pouch at the neck (white arrow). The neck of the aneurysm represents the entry point into the false lumen of an ICA dissection. A distal intracranial embolic occlusion is observed in the M1 segment of the right MCA (arrowhead). First, the MCA was completely recanalized by thromboaspiration (**B**). Subsequently, the ICA dissection was treated using a 7x30 mm Wallstent (Boston Scientific, Marlborough, Massachusetts, USA) and the residual aneurysm was coiled. (**D and E**).

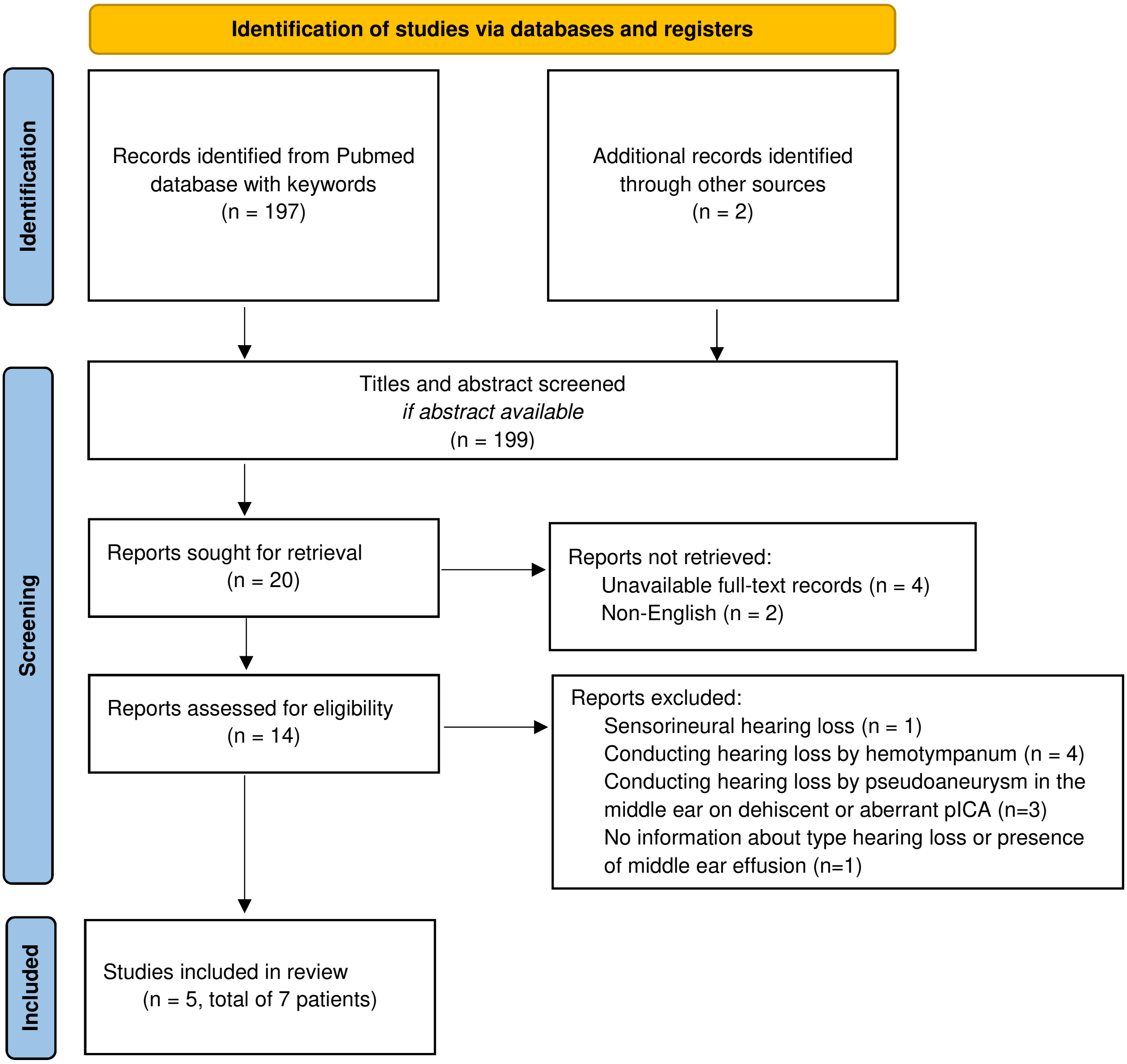

Figure 3. PRISMA flow diagram

Table 1. Characteristics of pICA aneurysm causing Eustachian tube compression retrieved in the literature: epidemiological characteristics, clinical and radiological findings, treatment and outcome

Reference	Number of patients	Sex / age (years)	Medical history	Symptoms duration before diagnosis	Clinical presentation	Particular features of the aneurysm	Treatment	Follow-up	Outcome
Goodman et al, 1996 [5]	1	M / 22	Unknown	3 years	Conductive hearing loss and middle ear effusion treated by tympanotomy and tube placement Otorrhagia after myringotomy Partial Horner's syndrome	Giant aneurysm Fusiform	Endovascular (balloon occlusion)	Unknown	Partial hearing recovery Regressive Horner's syndrome
Coley et al, 1998 [41]	1	M / 15	Minor head injury 3-years previously (fall from a bicycle)	3 months	Conductive hearing loss and middle ear effusion Otalgia Homolateral retroorbital headache Partial Horner's syndrome (ptosis)	Giant aneurysm Fusiform Partially thrombosed	Endovascular (balloon occlusion)	48 hours	Unknown hearing recovery Improved otalgia Improved retroorbital headache Persistant Horner's syndrome
Leonetti et al, 2013 [8]	3	Unknown	Unknown	5 to 51 months	Middle ear effusion	Giant aneurysm	Endovascular (stenting)	9 to 19,5 months	Uknown hearing recovery
Cooper et al, 2014 [9]	1	M / 37	No medical history	1 year	Conductive hearing loss (40 dB) Serous otorrhea Retracted tympanic membrane	Giant aneurysm Fusiform	Endovascular (balloon occlusion)	Several days	No hearing recovery
Wang et al, 2015 [17]	1	W / 13	No medical history	6 months	Blocked ear sensation Conductive hearing loss (35 dB) and middle ear effusion treated by tympanotomy, tube placement and adenoidectomy Relapse of serous otitis media 15 days after tube removal	Giant aneurysm (25 x 16 mm) Compressing the Internal Jugular Vein	Endovascular (coil embolisation)	1 year	Complete hearing recovery
Present Case	1	W / 37	No medical history	2 months	Conductive hearing loss (40 dB) and middle ear effusion Otalgia Intermittent tinnitus Acute sylvian ischemic stroke	Giant aneurysm (25 mm) Partially thrombosed	Endovascular (mechanical thrombectomy, pICA stenting and coil embolisation)	1 year	Complete hearing recovery after tympanotomy and tube placement Complete neurological recovery

Legends: pICA: petrous Internal Carotid Artery

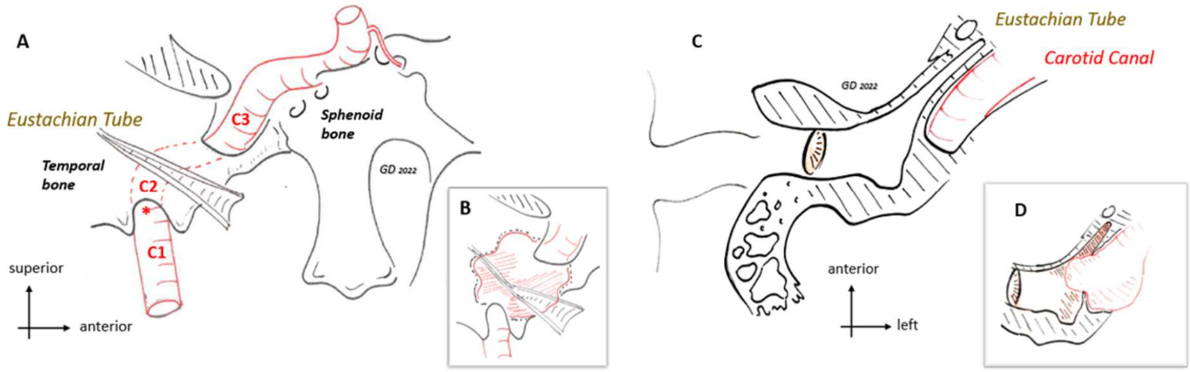

Figure 4. Anatomical relationships between pICA and ET.

A – Schematic drawing of a right lateral view, showing the ET lateral to the ICA. ICA is shown in its cervical (C1), petrous (C2) and lacerum (C3) segments. The transition C1-C2 (*) is a preferential site of arterial dissection, just below the genu of C2 segment that runs medially to the osseous part of the ET. The horizontal portion of C2 runs toward the petrous apex of the temporal bone. The cartilaginous part of the ET runs anteriorly, medially and inferiorly. It is attached to the medial pterygoid plate (sphenoid bone) and finally gets through the lateral wall of the nasopharynx. B – Schematic drawing showing this anatomy disrupted by a giant pICA aneurysm compressing the ET. C – Schematic drawing of an axial section showing the right middle ear and its relation with the ICA. The osseous part of the ET runs from the anterior wall of the middle ear (temporal bone) to the cartilaginous part of the ET, medially to the sphenoid spine and foramen spinosum (sphenoid bone). D – Schematic drawing showing this anatomy disrupted by a pICA aneurysm compressing the osseous part of the ET. Medial wall of the ET is dehiscent at this level.”

References

[1] Lau WYS, Chow CK. Radiation-induced petrous internal carotid artery aneurysm. Ann Otol Rhinol Laryngol 2005;114:939–40. https://doi.org/10.1177/000348940511401208.

[2] Mori R, Murayama Y, Irie K, Takao H, Ebara M, Ishibashi T, et al. [Endovascular treatment of large petrous internal carotid artery aneurysm associated with chronic otitis media: case report]. No Shinkei Geka 2006;34:415–9.

[3] Saylam G, Tulgar M, Saatci I, Korkmaz H. Iatrogenic carotid artery pseudoaneurysm presenting with conductive hearing loss. Am J Otolaryngol 2009;30:141–4. https://doi.org/10.1016/j.amjoto.2008.08.004.

[4] Moonis G, Hwang CJ, Ahmed T, Weigele JB, Hurst RW. Otologic manifestations of petrous carotid aneurysms. AJNR Am J Neuroradiol 2005;26:1324–7.

[5] Goodman TR, Renowden S, Byrne JV. Case report: petrous internal carotid artery aneurysm: an unusual cause of eustachian tube dysfunction. Clin Radiol 1996;51:658–60. https://doi.org/10.1016/s0009-9260(96)80065-1.

[6] Rawlinson J, Colquhoun IR. Aneurysms involving the intrapetrous internal carotid artery: a rare cause of Horner’s syndrome. Br J Radiol 1990;63:69–72. https://doi.org/10.1259/0007-1285-63-745-69.

[7] Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. https://doi.org/10.1136/bmj.n71.

[8] Leonetti JP. A study of persistent unilateral middle ear effusion caused by occult skull base lesions. Ear Nose Throat J 2013;92:195–200. https://doi.org/10.1177/014556131309200411.

[9] Cooper L, Ford K, Talwar R, Wareing M. An unusual cause of chronic otitis media with effusion. J Laryngol Otol 2014;128:179–81. https://doi.org/10.1017/S0022215113003563.

[10] Osawa S, Rhoton AL, Tanriover N, Shimizu S, Fujii K. Microsurgical anatomy and surgical exposure of the petrous segment of the internal carotid artery. Neurosurgery 2008;63:210–38; discussion 239. https://doi.org/10.1227/01.NEU.0000327037.75571.10.

[11] Matsushima T, Kobayashi S, Inoue T, Rhoton AS, Vlasak AL, Oliveira E de. Albert L. Rhoton Jr., MD: His Philosophy and Education of Neurosurgeons. Neurol Med Chir (Tokyo) 2018;58:279–89. https://doi.org/10.2176/nmc.ra.2018-0082.

[12] Paullus WS, Pait TG, Rhoton AI. Microsurgical exposure of the petrous portion of the carotid artery. J Neurosurg 1977;47:713–26. https://doi.org/10.3171/jns.1977.47.5.0713.

[13] Liu JK, Gottfried ON, Amini A, Couldwell WT. Aneurysms of the petrous internal carotid artery: anatomy, origins, and treatment. Neurosurg Focus 2004;17:E13. https://doi.org/10.3171/foc.2004.17.5.13.

[14] Liu J, Pinheiro-Neto CD, Fernandez-Miranda JC, Snyderman CH, Gardner PA, Hirsch BE, et al. Eustachian tube and internal carotid artery in skull base surgery: an anatomical study. Laryngoscope 2014;124:2655–64. https://doi.org/10.1002/lary.24808.

[15] Komune N, Matsuo S, Miki K, Akagi Y, Kurogi R, Iihara K, et al. Surgical Anatomy of the Eustachian Tube for Endoscopic Transnasal Skull Base Surgery: A Cadaveric and Radiologic Study. World Neurosurg 2018;112:e172–81. https://doi.org/10.1016/j.wneu.2018.01.003.

[16] Leonetti JP, Smith PG, Linthicum FH. The petrous carotid artery: anatomic relationships in skull base surgery. Otolaryngol Head Neck Surg 1990;102:3–12. https://doi.org/10.1177/019459989010200102.

[17] Wang Y, Guan B, Tian T, Peng X, Xu LI. Intrapetrous internal carotid artery aneurysm diagnosed on the basis of middle ear effusion: A case report. Oncol Lett 2015;10:283–6. https://doi.org/10.3892/ol.2015.3178.

[18] Mudry A. Pharyngeal recess or Rosenmüller’s fossa: Its first description revisited. Head Neck 2021;43:2295–6. https://doi.org/10.1002/hed.26766.

[19] Amene C, Cosetti M, Ambekar S, Guthikonda B, Nanda A. Johann Christian Rosenmüller (1771-1820): A Historical Perspective on the Man behind the Fossa. J Neurol Surg B Skull Base 2013;74:187–93. https://doi.org/10.1055/s-0033-1342927.

[20] Touzé E, Randoux B, Méary E, Arquizan C, Meder JF, Mas JL. Aneurysmal forms of cervical artery dissection : associated factors and outcome. Stroke 2001;32:418–23. https://doi.org/10.1161/01.str.32.2.418.
[21] Chen SY, Zipfel GJ, Wick CC. Internal carotid artery dissection causing pulsatile tinnitus. Am J Otolaryngol 2019;40:121–3. https://doi.org/10.1016/j.amjoto.2018.09.008.
[22] Patel RR, Adam R, Maldjian C, Lincoln CM, Yuen A, Arneja A. Cervical carotid artery dissection: current review of diagnosis and treatment. Cardiol Rev 2012;20:145–52. https://doi.org/10.1097/CRD.0b013e318247cd15.
[23] Agud JL, Ferreiro-Argüelles C, Casqueiro JC. Listening to the diagnosis: a physician's account of spontaneous carotid artery dissection. Eur Arch Otorhinolaryngol 2011;268:319–21. https://doi.org/10.1007/s00405-010-1430-9.
[24] Liess BD, Lollar KW, Christiansen SG, Vaslow D. Pulsatile tinnitus: a harbinger of a greater ill? Head Neck 2009;31:269–73. https://doi.org/10.1002/hed.20851.
[25] Hwang CJ, Moonis G, Hurst RW, Hockstein N, Bigelow D. Bilateral petrous internal carotid artery pseudoaneurysms presenting with sensorineural hearing loss. AJNR Am J Neuroradiol 2003;24:1139–41.
[26] Bonnard D, de Monès E, Sagardoy T, Franco-Vidal V, Darrouzet V, Fierens S. Transtympanic pseudoaneurysm of the internal carotid artery complicating a myringotomy in a four-year old child: Case report and literature review. Am J Otolaryngol 2017;38:713–7. https://doi.org/10.1016/j.amjoto.2017.08.003.
[27] Kawamura Y, Sayama T, Maehara N, Nishimura A, Iihara K. Ruptured Aneurysm of an Aberrant Internal Carotid Artery Successfully Treated with Simultaneous Intervention and Surgery in a Hybrid Operating Room. World Neurosurg 2017;102:695.e1-695.e5. https://doi.org/10.1016/j.wneu.2017.03.133.
[28] Reece PH, Higgins N, Hardy DG, Moffat DA. An aneurysm of the petrous internal carotid artery. J Laryngol Otol 1999;113:55–7. https://doi.org/10.1017/s0022215100143142.
[29] Güneş S, Yazici ZM, Selçuk H, Şimşek BM, Çelik M, Kayhan FT. Carotid Artery Aneurysm: A Rare Cause of Hemotympanum. J Craniofac Surg 2017;28:e74–5. https://doi.org/10.1097/SCS.0000000000003237.
[30] Kimmelman CP, Grossman R. Intratemporal carotid aneurysm as a complication of chronic otitis media: treatment with balloon catheter obliteration. Otolaryngol Head Neck Surg 1983;91:306–8. https://doi.org/10.1177/019459988309100320.
[31] Baker A, Rizk H, Carroll W, Lambert P. Cervical internal carotid artery pseudoaneurysm complicating malignant otitis externa: first case report. Laryngoscope 2015;125:733–5. https://doi.org/10.1002/lary.24877.
[32] Lau JSK, Wong JCY, Ng RYT, Pang VKY, Wong CK. Malignant otitis externa complicated by multiple cervical-petrous internal carotid artery pseudoaneurysms: a case report. Hong Kong Med J 2019;25:152–5. https://doi.org/10.12809/hkmj165016.
[33] Al Maghraoui O, Ezzahraoui R, Hanine MA, En-Nouali A, Darouassi Y, Chaara F, et al. Pseudoaneurysm of the intra-petrous internal carotid artery secondary to external malignant otitis. J Med Vasc 2021;46:148–51. https://doi.org/10.1016/j.jdmv.2021.03.010.
[34] Willinsky R, Lasjaunias P, Pruvost P, Boucherat M. Petrous internal carotid aneurysm causing epistaxis: balloon embolization with preservation of the parent vessel. Neuroradiology 1987;29:570–2. https://doi.org/10.1007/BF00350444.
[35] Tamada T, Mikami T, Akiyama Y, Kimura Y, Wanibuchi M, Mikuni N. Giant petrous internal carotid aneurysm causing epistaxis: A case report. J Clin Neurosci 2018;58:221–3. https://doi.org/10.1016/j.jocn.2018.10.007.
[36] Rubiera M, Ribo M, Delgado-Mederos R, Santamarina E, Delgado P, Montaner J, et al. Tandem internal carotid artery/middle cerebral artery occlusion: an independent predictor of poor outcome after systemic thrombolysis. Stroke 2006;37:2301–5. https://doi.org/10.1161/01.STR.0000237070.80133.1d.
[37] Halbach VV, Higashida RT, Hieshima GB, Dowd CF, Barnwell SL, Edwards MS, et al. Aneurysms of the petrous portion of the internal carotid artery: results of treatment with endovascular or surgical occlusion. AJNR Am J Neuroradiol 1990;11:253–7.

[38] Ghali MGZ, Binning M. Flow Diversion for the Treatment of Petrous Internal Carotid Artery Aneurysms. Asian J Neurosurg 2019;14:1058–62. https://doi.org/10.4103/ajns.AJNS_119_18.
[39] Borha A, Patron V, Huet H, Emery E, Barbier C. Endovascular management of a giant petrous internal carotid artery aneurysm in a child. Case report and literature review. Childs Nerv Syst 2019;35:183–6. https://doi.org/10.1007/s00381-018-3941-4.
[40] Sun L, Ren J, Ye M, Zhang H. Multiple Telescoping Flow Diverter Technique in Endovascular Treatment of a Giant Petrous and Cavernous Carotid Aneurysm. World Neurosurg 2022;167:7. https://doi.org/10.1016/j.wneu.2022.08.012.
[41] Coley SC, Clifton A, Britton J. Giant aneurysm of the petrous internal carotid artery: diagnosis and treatment. J Laryngol Otol 1998;112:196–8. https://doi.org/10.1017/s0022215100140307.