Management of an Extensive Intraparotid and Auricular Arteriovenous Malformation

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: **Female, 40-year-old**
Final Diagnosis: **Arterio-venous malformation**
Symptoms: **Neck swelling**
Medication: **—**
Clinical Procedure: **Coil embolization • surgical resection**
Specialty: **Radiology**

Objective: **Rare disease**
Background: Extracranial arteriovenous malformations (AVMs) of the parotid gland and auricle are rarely encountered. Management of these AVMs depends on the Schobinger stage and their flow characteristics. We present a rare case of an AVM involving the parotid and auricle concurrently. The clinical and imaging features of these high-flow vascular malformations and their treatment options are discussed and we provide a review of the literature.
Case Report: A 40-year-old woman presented with a large 6.4×6.0×13.0 cm high-flow Schobinger stage II high-flow AVM of the parotid gland and auricle. Diagnostic imaging included magnetic resonance imaging (MRI) and conventional catheter angiogram, which defined the vascular anatomy and flow characteristics of the AVM. She was treated with preoperative endovascular embolization followed by surgical excision and free-tissue transfer reconstruction on the next day. The results were excellent, with no recurrence over 3.5 years of follow-up.
Conclusions: This is the second case reported in the literature of high-flow AVM concurrently involving the parotid gland and auricle, treated with perioperative embolization followed by surgical excision and grafting. Management of AVMs requires a multidisciplinary team approach and understanding of the natural history of the lesion. Although total surgical resection is the criterion standard for these AVMs, endovascular embolization is an alternative treatment that can be used as an adjunct to surgery. Furthermore, perioperative embolization can decrease the vascularity of the lesion and effectively reduce blood loss during AVM surgery.

Keywords: **Arteriovenous Malformations • Ear Auricle • Embolization, Therapeutic • Endovascular Procedures • Parotid Gland**

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Background

According to the International Society for Study of Vascular Anomalies (ISSVA), vascular anomalies can be categorized into vascular tumors and malformations [1]. Vascular tumors are soft-tissue growths that can be either benign or malignant, formed from blood vessels or lymph vessels (eg, hemangio-endotheliomas, Kaposi's sarcomas, and angiosarcoma) [1]. Vascular malformations are congenital vascular anomalies due to developmental error. They are further classified according to their vascular flow characteristics into low-flow (eg, hemangiomas, capillary, venous, and lymphatic malformations) and high-flow lesions, including arteriovenous malformation (AVM) and arteriovenous fistula (AVF) [1]. The fistulas

are functionally-open embryological residues between the arteries and veins and are present at birth. Local trauma, partial surgery, psychological stress, and endocrine events (eg, puberty, pregnancy, and birth) can cause the fistulas to grow and form malformations [2].

The extracranial AVMs are classified according to Schobinger (**Table 1**) into 4 stages according to the patient's symptoms: stage I (warm and discolored skin), II (pulsation, thrill, and swelling), III (pain, dystrophic skin, ulceration, bleeding), and IV (decompensation). These AVM can be further differentiated according to their flow rates into fast-flowing and slow-flowing types [4]. Fast-flowing in an existing vascular malformation can induce arteriovenous shunting, increasing flow demand

Table 1. Schobinger staging of arteriovenous malformation.

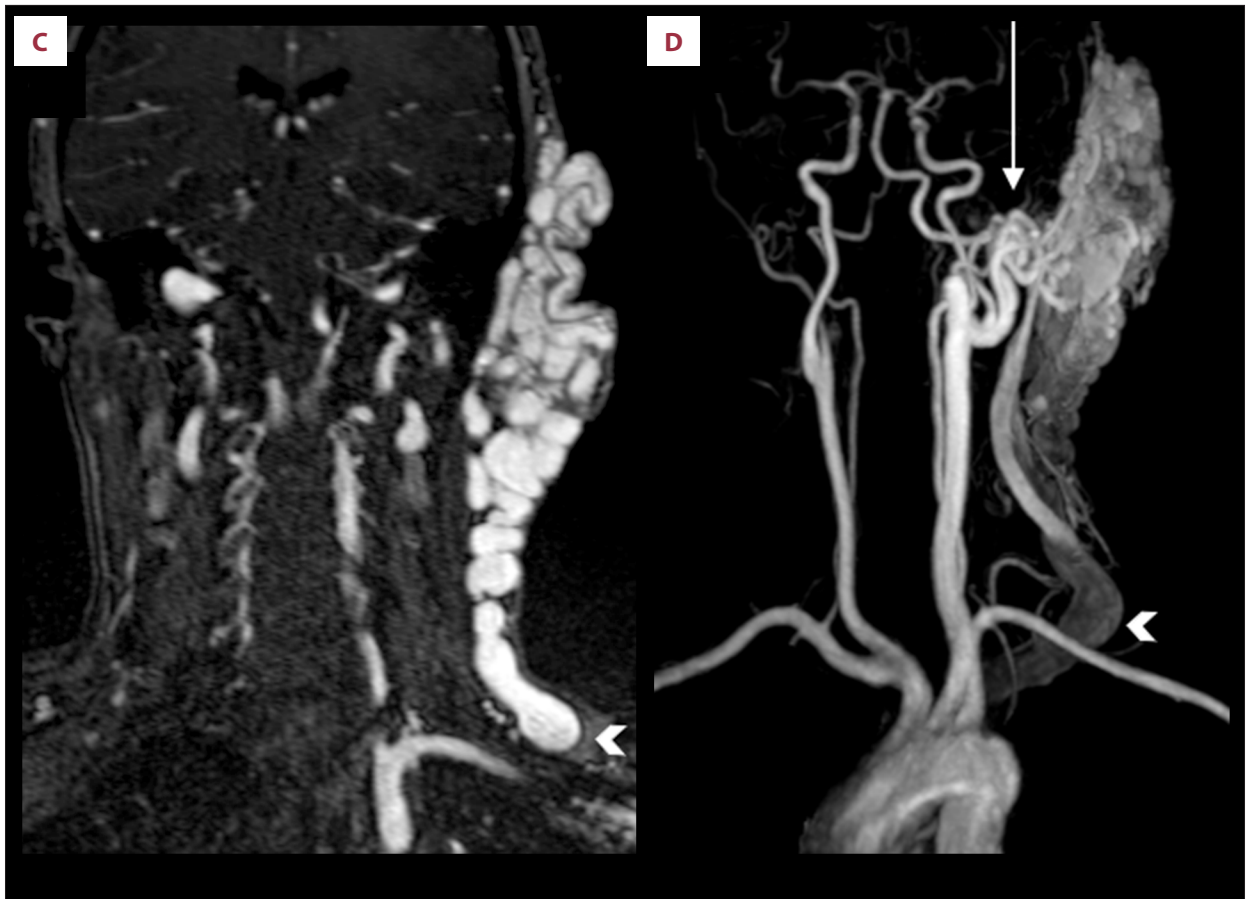| Stage	Nature	Clinical findings	Intervention [3]
I	Quiescent	Warm and discolored skin	No, observation
II	Expansile	Bruit/thrill, pulsation and swelling	Yes, based on symptoms
III	Destructive	Pain, dystrophic skin, ulceration, bleeding	Yes
IV	Decompensation	Cardiac failure	Yes
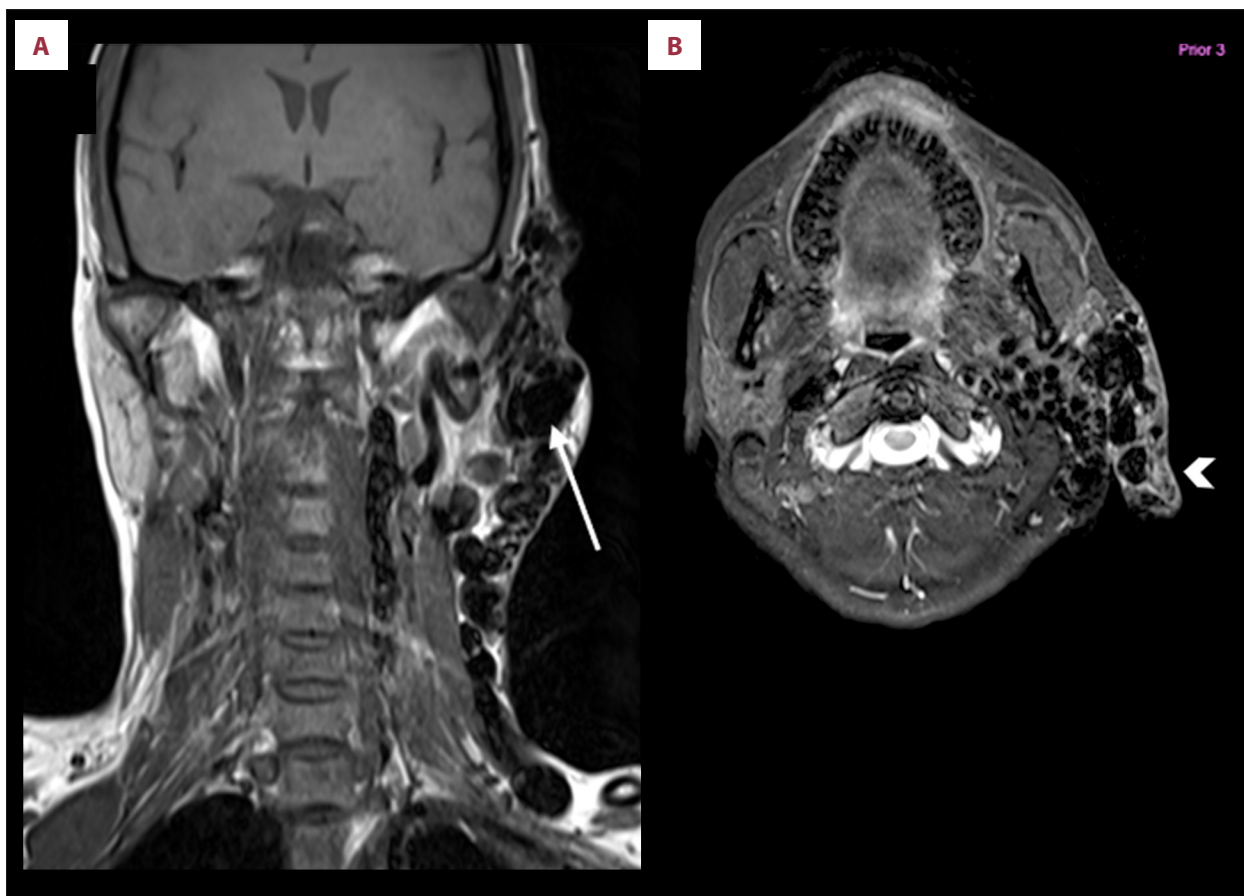
Figure 1. Coronal T1-weighted (**A**) and axial T2-weighted (**B**) MRI neck images of a 40-year-old woman show "bag of worms" or tangle of serpiginous "honeycomb" flow voids in the left parotid gland and left auricle (arrowhead, **B**) indicating an extensive high-flow arteriovenous malformation (AVM). An intranidal aneurysm (arrow, **A**) is present within the AVM. Coronal contrast-enhanced (CE) MRA image (**C**) shows the dilated and serpiginous vessels within the left parotid and auricular AVM and dilated veins draining blood into the left external jugular vein (arrowhead). Maximum-intensity projection of CE-MRA image (**D**) shows the left posterior auricular artery (arrow), one of the main arterial blood supplies to the AVM, along with the superficial temporal artery. These 2 are branches of the left external carotid artery (arrow). The AVM drains predominantly into a dilated and serpiginous left external jugular vein (arrowhead).

and cascading enlargement of the malformation [5]. Early intervention can arrest or even reverse such changes [5]. Stage I AVMs are usually just observed, while stage II may need intervention based on symptoms and location; stage III and IV often require intervention [3].

AVMs of the parotid and auricle are rare malformations and are exceedingly rare when they occur concurrently in the same individual. Here, we report an exceedingly rare case of a Schobinger stage II fast-flowing AVM of the parotid gland and the left auricle, which were treated by endovascular embolization and surgical resection. The clinical and imaging features of these high-flow vascular malformations and their treatment options are discussed and we provide a review of the pertinent literature.

Case Report

A 40-year-old woman presented with progressive swelling on the left side of the neck for more than 6 months. She has no significant past medical history. Physical findings showed a swelling over the left parotid gland and auricle, with skin discoloration. There was a palpable thrill and vascular murmur detected on auscultation. Magnetic resonance imaging (MRI) showed serpiginous vessels with "honeycomb" flow voids in the left parotid gland and the left auricle, indicating a large high-flow AVM (**Figure 1**). The large AVM measured approximately 6.4×6.0×13.0 cm (anteroposterior×transverse×craniocaudal dimensions). The presenting signs and symptoms of the patient correlated with the Schobinger stage II AVM. The AVM was primarily supplied by branches of the external carotid artery and drained into the external jugular vein (**Figure 1**). Preoperative embolization was
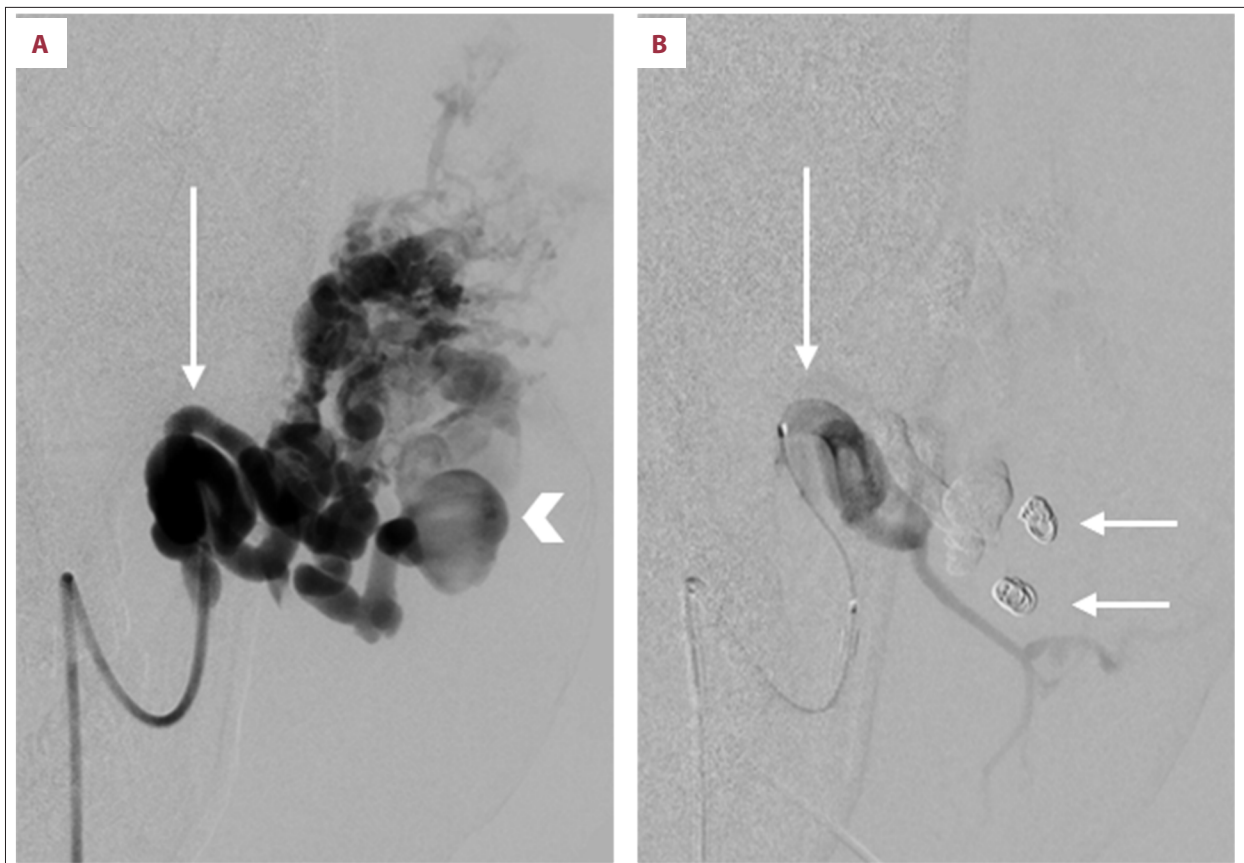
Figure 2. Conventional catheter angiogram (**A**) with the catheter tip within the posterior auricular branch (arrow) of the left external carotid artery. There is opacification of the dilated vessels and intranidal aneurysm (arrowhead) within the AVM. The post-embolization angiogram (**B**) shows successful embolization of the AVM with a combination of coils (short arrows) and diluted mixture of (25%) Histoacryl in Ethiodol. There is a marked decrease in vascularity of the AVM and occlusion of the intranidal aneurysm.

performed with vascular access via the right femoral artery and insertion of a guiding catheter in the left external carotid artery. A conventional catheter angiogram showed a dilated intranidal aneurysm within the left parotid AVM with the main blood supply from the posterior auricular and superficial temporal branches of the left external carotid artery (**Figure 2**). A high-flow shunt with venous drainage into the dilated left external jugular vein was identified. Blood flow control was achieved using the proximal balloon occlusion technique. Coil embolization of the feeding arteries and shunts was performed under general anesthesia. As a result, there was a significant reduction in the vascularity of the AVM. The patient underwent surgical excision of the AVM on the next day, including left total parotidectomy and left total auriculectomy. Careful dissection allowed for retrograde identification and preservation of the left facial nerve with branches. There was further extension of the dissection down into the neck, allowing for surgical closure with a musculocutaneous flap with end-to-end vessel anastomosis. The postoperative course was uneventful, with no complications related to either the preoperative embolization or surgery. No facial

palsy was detected. Histology confirmed an AVM of the parotid gland and auricular subcutaneous tissue with no evidence of atypical cells or malignancy. The patient was discharged on the fifth postoperative day. Follow-up MRI at 6 months after surgery showed no remnant or recurrent AVM (**Figure 3**). There was no clinical or radiological evidence of AVM recurrence over 3.5 years of follow-up after surgery.

Discussion

Having reviewed the PubMed database literature from 1 January 2000 to 31 December 2021, we found a total of 82 and 21 reported cases of auricular (**Table 2**) and parotid (**Table 3**) AVMs, respectively. These high-flow AVMs often develop in early childhood and can enlarge secondary to various factors such as increased blood flow, arterial or venous thrombosis, and trauma [5]. Other factors include infection and iatrogenic insults such as incomplete surgery and proximal embolization [28,29].
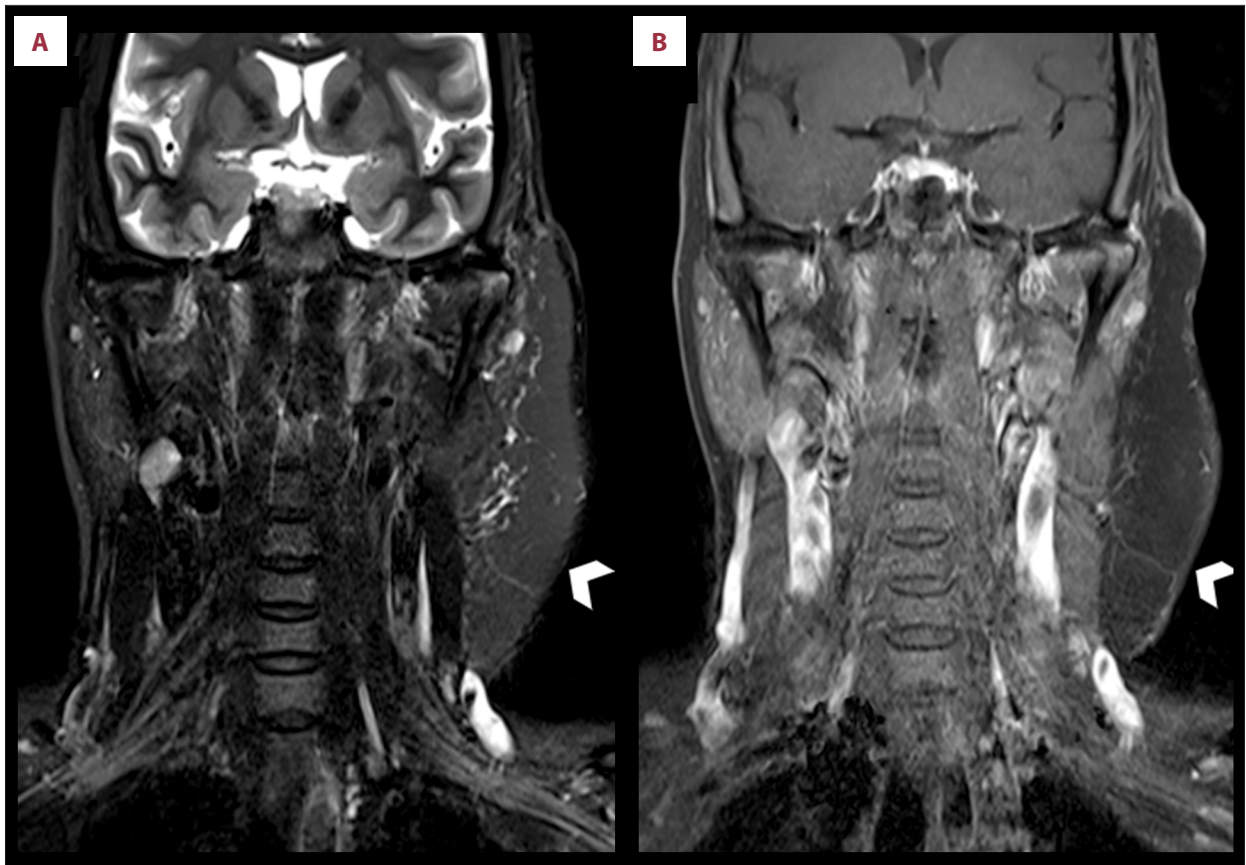
Figure 3. Coronal T2-weighted (**A**) and T1-weighted post-contrast (**B**) MRI neck images of the patient about 6 months after left parotidectomy and left total auriculectomy. An intact musculocutaneous flap is present in situ (arrowhead). There is no remnant or recurrent AVM.

Pathophysiology of AVM

Enlargement of AVM can be caused by increased blood flow secondary to collateralization, dilatation, and thickening of adjacent vessels [3]. Latent arteriovenous (AV) shunts can open and stimulate hypertrophy of surrounding vessels and even cause aneurysm formation, which further increases the size of the AVM [30]. Expansion of AVMs can be due to angiogenesis (growth of new blood vessels from pre-existing vasculature) or vasculogenesis (de novo formation of new vasculature) [3]. There are endothelial proliferation and elevated vascular endothelial growth factor (VEGF) in AVM tissue [31]. Increased tissue VEGF expression correlates with AVM recurrence [31]. Endothelial cells of AVMs can express high levels of VEG, which results in proliferation and rapid migration of cells, forming anomalous vascular channels [32]. Matrix metalloproteinases (MMPs) are elevated in AVM; conversely, MMPs inhibitors control the progression of MMPs by reducing their expression [33].

Another stimulus for AVM expansion is tissue hypoxia or ischemia, a potent stimulator of angiogenesis, particularly after proximal arterial ligation or trauma [8]. Increased blood flow caused by AV shunting can promote angiogenesis and stimulate VEGF production and endothelial proliferation [34]. There is a higher risk of AVM progression in puberty due to increased hormonal activity during this period, thus stimulating AVM growth by promoting angiogenesis [35]. Estrogen and testosterone stimulate VEGF production, endothelial proliferation, and angiogenesis [36], while growth hormone has pro-angiogenic activity [37].

In adults, the diagnosis of AVM is often made around the fourth decade of life, with a female predominance [4]. Pregnancy, like puberty, is thought to increase the risk of AVM expansion [2]. Approximately 60% of AVMs occur in the superficial lobe of the parotid gland, 20% in the deep lobe, and 20% within the masseter muscle, with no dominant side [4,29].

The most common clinical manifestation of the AVM is the presence of a pulsatile mass [29].

MRI and conventional angiogram are the preferred imaging modalities for diagnosis and surveillance of vascular malformations. These imaging modalities are paramount for understanding both the extent of the disease and the flow characteristics

Table 2. Summary of the reported cases of arteriovenous malformation of the auricle in the literature.

Authors	Nr pt	Age (y), gender	Lesion site	Symptoms, duration (Schobinger clinical stage)	Key Imaging modalities	Main feeding arteries	Treatment	Follow up (FU) duration; outcome
Ramadass T (2000) [6]	1	25 y, M	R auricle	Swelling and bleeding 7 y (Stage III)	Angio: enlarged and tortuous vessels	PAA, OA	Staged surgery: Exc+STSG >> Ear elevation	4 m; lost to FU after last surgery
Pham TH (2001) [7]	1	41 y, M	L auricle	Swelling, PT, intermittent bleeding, 6 y (Stage III)	Angio: diffuse network of shunts	PAA, STA, OA	Embo >> Exc+STSG	2 y; no recurrence
Wu JK (2005) [8]	41	26 y (range 1-55)	Auricle and extraauricular involvement (Retroauricular: 46.3%, Neck: 22%, None: 22%)	PT, bleeding, pain, bruit/thrill (Stage II and III)	Angio and MRI	PAA, STA, OA	Observation (nr: 12, 29.3%), Embo (nr: 9, 21.9%), Exc+Embo (nr: 20, 48.8%)	5y FU in 20 pts with amputation: Controlled (n: 16) Improved (n: 3) Persistent (n: 1)
Meher R (2008) [9]	2	pt 1: 16 y, M; pt 2: 22y, F.	pt 1: L auricle; pt 2: R auricle.	pt 1: Intermittent pain; pt 2: swelling, PT, bleeding 10 y (both Stage III)	pt 1: DS: Multiple dilated anechoic areas; pt 2: MRA: dilated serpiginous structures.	pt 2: PAA, STA	Both had Exc+STSG	NA
Saxena SK (2008) [10]	1	21 y, F	R auricle	Swelling+PT (Stage II)	Angio: diffuse shunts with STA	STA	Failed Embo >> Exc+PAL	3 y, no recurrence
Wu HJ (2008) [11]	1	20 y, M	L auricle	PT 7m (Stage II)	MRI: abnormal signal voiding intensity of the mass	PAA, STA	Failed Embo >> Exc	2 y, no recurrence
Whitty LA (2009) [12]	1	15 y, M	L auricle	Swelling, bruit+intermittent pain 2 y (Stage III)	NA	NA	Exc	1m, no recurrence
Zheng LZ (2009) [13]	17	25.4 y (range 3-47), 11 M, 6 F	Auricle	Ear swelling, redness, thrill/bruit, ulceration, hemorrhage and infection (majority in Stage II and III). All were present at birth	Angio	PAA, STA, OA, FA, MA, branches of ICA and contralateral ECA	Embo (nr: 17, 100%); prior PAL, incomplete resection, and/or Embo (nr: 10, 58.8%)	3-4 m; AVMs were devascularized 100% (nr: 3, 17.6%), ≥50% (nr: 11, 64.8%), <50% (nr: 3, 17.6%)
Jin YB (2009) [14]	8	31.5y (range 10-59), 5 M, 3 F	Auricle	Bleeding, ulceration, disfiguring (Stages I, II and III)	MRI	NA	Embo (nr: 8, 100%); prior PAL (nr: 1, 12.5%).	12.6 m (range, 5-27 m). AVMs were devascularized 100% (nr: 6, 75%), ≥50% (nr: 2, 25%)
Prasad KC (2011) [15]	1	45 y, M	Auricle	Swelling, 2-3 y (Stage II)	Angio: AVM and PAA aneurysm	PAA	PAL+Exc	2 y, no recurrence
Goel A (2011) [16]	1	22 y, F	R auricle	Swelling, pulsatile tinnitus bleeding, 4 y (Stage III)	DS: AVM	NA	Exc+STSG	NA

Table 2 continued. Summary of the reported cases of arteriovenous malformation of the auricle in the literature.

Authors	Nr pt	Age (y), gender	Lesion site	Symptoms, duration (Schobinger clinical stage)	Key Imaging modalities	Main feeding arteries	Treatment	Follow up (FU) duration; outcome
Meena BK (2013) [17]	1	21 y, F	L auricle	Swelling, pulsatile tinnitus bleeding 1 y (Stage III)	DS, CTA: Enlarged serpiginous structures	PAA, STA	Exc	NA
Dixit SG (2013) [18]	1	21 y, M	Auricle	Swelling since birth (Stage II)	MRA: Tortuous vessel	PAA	Embo	NA
Anesti K (2014) [5]	1	34 y, M	L auricle and L parotid gland	Progressive enlargement, bleeding, pain, skin tightness and pulsations at night (Stage III)	MRI: AVM of the left ECA supplying the scalp and the left ear	STA, OA	Embo >> Exc+MCF closure	No recurrence
In't Veld M (2016) [19]	1	30 y, M	L auricle	Pain, redness, pulsatile swelling >10 y (Stage III)	MRI: Vessels with prominent flow voids	PAA, STA	Embo	2 y; no recurrence
Kim SH (2017) [20]	1	60 y, M	R auricle	Swelling+bleeding with PT 3 y (Stage III)	CTA: Inner vascular tangles	PAA	Embo >> Exc	3 m; no recurrence
Ishikawa K (2021) [21]	1	46 y, M	Auricle	Swelling/macrotia (Stage III)	Angio	NA	Embo >> scleroth >> Exc	6 m; no recurrence
Yee & Ho (2022) [current study]	1	40 y, F	L parotid+ L auricle+ extra-auricular	Swelling+palpable thrills, 6 m (Stage II)	MRI: Serpiginous vessels with "honeycomb" flow voids	PAA, STA	Embo >> Exc+MCF closure	3.5 y; no recurrence

Angio – angiography; AVM – arteriovenous malformation; BWPC – bone wax packing and curettage; CTA – computed tomography angiography; CECT – contrast enhanced CT; Embo – embolization; DS – Doppler sonography; ECA – external carotid artery; Exc – excision; FA – facial artery; LA – lingual artery; IAA – inferior alveolar artery; ICA – internal carotid artery; MA – maxillary artery; MCF – musculocutaneous flap; MRI – magnetic resonance imaging; MRA – magnetic resonance angiography; NA – no available information; nr – number(s); OA ,– occipital artery; PAA – posterior auricular artery; PAL – proximal artery ligation; PT – pulsatile tinnitus; Scleroth – sclerotherapy; STA – superficial temporal artery; STSG – Split-Thickness Skin Graft; Symbols >> – followed by; + – and; F – female; M – male; m – month(s); pt – patient; R – right; L – left; y – year(s).

[20]. Typical MRI features of AVM includes "bag of worms" or "honeycomb" flow voids of the serpiginous vessels (**Figure 1**). Unlike the presence of a nidus in intracranial AVM, blood is shunted directly from the arteries into venous tributaries. The veins become engorged over time, resulting in skin ulceration and necrosis [4]. The blood-shunting effects of AVM can cause high-output heart failure (Schobinger stage IV) [28].

Treatment Options

Management of these AVMs can be challenging and is best achieved by understanding the natural history of the lesion and the patient's needs. Treatment is performed not only for aesthetic purposes but also to avoid complications such as skin ulceration/necrosis and post-traumatic hemorrhage [28]. The primary goal is to restore and preserve function, stop bleeding, and improve or restore cosmesis [5]. Total AVM excision is the criterion standard treatment because it allows a definitive diagnosis with histological confirmation [5]. There is little role for radiation therapy in the treatment of parotid and auricular AVMs [5]. The extent of the parotidectomy depends on the extent of involvement of the gland. The risks of surgery include neurovascular injuries, particularly involving the facial nerve.

Endovascular embolization is an adjunct or alternative treatment. By reducing the vascularity of the AVM, blood loss can

Table 3. Summary of the reported cases of arteriovenous malformation of the parotid gland in the literature.

Authors	Nr pt	Age (y), gender	Lesion site	Presenting symptoms, duration (Schobinger clinical stage)	Key diagnostic studies and findings	Main feeding arteries	Treatment	Follow up (FU) duration; outcome
Hamden AL (2001) [22]	1	20 y, F	L parotid	Enlarging left cheek mass with palpable thrills 1 y (Stage II)	MRI and Angio: AVM involving the superficial and deep lobes of the parotid	MA, LA, FA	Embo >> Exc	NA
Chen WL (2009) [23]	13	9.1 y (range 5-13); 10 M, 3 F	Parotid, cheek, mandible, maxilla, floor of mouth	Facial asymmetry, skin discoloration, pulsation, wind-blowing noise, gingival bleeding, intraoral hemorrhage after tooth extraction (Stage III)	CTA	IAA, LA, MA, FA, STA	Embo only, Embo >> Scleroth. Exc	13.5 m (range 6-22 m). 69.2% of AVMs involuted, 23.1% mostly involuted + partial involution in 7.7%. Reduction of AVM size and cure rates after Exc were 23.1% and 84.6%, respectively
Shailaja SR (2012) [24]	1	18 y, F	R parotid	R hemifacial swelling 1y and pain R lower back 6 m, with bruit (Stage III)	MRI+MRA: multiple flow voids in R parotid, ramus and condyle of the mandible	FA	NA	NA
Anesti K (2014) [5]	1	34 y, M	L parotid and auricle	Progressive enlargement, bleeding, pain, skin tightness and pulsations at night (Stage III)	MRI: vascular malformation of L ECA supplying scalp and L ear	STA, OA	Embo >> Exc+MCF closure	No recurrence
Bhatia C (2017) [25]	1	55 y, F	R parotid	Swelling in front and below R ear 1 y (Stage II)	MRI: R parotid enlargement with multiple tubular enhancing structures traversing the R parotid	NA	Exc	NA
John H (2020) [26]	1	47 y, F	L parotid	Pain and swelling in front of L ear, 4 m (Stage III)	MRI+MRA: AVM in the superficial parotid lobe	FA	Scleroth (prior Ex ?)	No recurrence
Gupta M (2021) [27]	2	pt 1: 32 y, F; pt 2: 43 y, M	pt 1: R parotid pt 2: L parotid	pt 1: Swelling below R ear, 8 y (Stage III); pt 2: Swelling below and front of L ear, 1.5 y (Stage II)	pt 1: CECT: Enhancing parotid with multiple pin head calcifications. pt 2: MRI: T2w hyperintensity with few lobulated hypointense foci within parotid	NA	Pt 1: Exc Pt 2: Exc	Pt 1: 1 m; no recurrence Pt 2: NA

Table 3 continued. Summary of the reported cases of arteriovenous malformation of the parotid gland in the literature.

Authors	Nr pt	Age (y), gender	Lesion site	Presenting symptoms, duration (Schobinger clinical stage)	Key diagnostic studies and findings	Main feeding arteries	Treatment	Follow up (FU) duration; outcome
Yee & Ho (2022) [current study]	1	40 y, F	L parotid and L auricle	Swelling with palpable thrills, 6 m (Stage II)	MRI: Serpiginous vessels with "honeycomb" flow voids	PAA, STA	Embo. >> Exc+MCF closure	3.5 y, no recurrence

Angio – angiography; AVM – arteriovenous malformation; BWPC – bone wax packing and curettage; CTA – computed tomography angiography; CECT – contrast enhanced CT; Embo – embolization; DS – Doppler sonography; ECA – external carotid artery; Exc – excision; FA – facial artery; LA – lingual artery; IAA – inferior alveolar artery; ICA – internal carotid artery; MA – maxillary artery; MCF – musculocutaneous flap; MRI – magnetic resonance imaging; MRA – magnetic resonance angiography; NA – no available information; nr – number(s); OA – occipital artery; PAA – posterior auricular artery; PAL – proximal artery ligation; PT – pulsatile tinnitus; Scleroth – sclerotherapy; STA – superficial temporal artery; STSG – Split-Thickness Skin Graft; Symbols >> – followed by; + – and; F – female; M – male; m – month(s); pt – patient; R – right; L – left; y – year(s).

be limited, and safe tissue planes can be identified during surgery, thus facilitating AVM excision. The selection of an embolization agent depends on the type of lesion, method of embolization, and the experience of the interventional radiologist [4]. Detachable balloons, coils, and embolic agents are some of the commonly used agents [29]. The use of embolic agents is, however, not advisable for high-flow AVMs, as migration of embolic agents in high-flow shunts can increase the risk of pulmonary embolism [28], and platinum coils can be employed in such cases [28]. Microcatheters can be placed distal to the shunt during the procedure, and blood flow within the feeding vessel can be controlled with a balloon-tip catheter. Platinum coils can then be used to occlude the feeding arteries and the AV shunts [4]. Accurate identification of these shunts is crucial during the procedure. Although less invasive than surgery, endovascular treatment may not completely occlude the AVM in a single attempt, and multiple attempts may be required [4].

Among the 82 patients reported with auricular AVMs (**Table 2**), the majority underwent endovascular embolization (37 patients) and embolization followed by excision (25 patients), while the minority were treated with excision (8 patients) and some had only observation without intervention (12 patients) [5-22]. Incomplete devascularization of the auricular AVMs has been reported in 16 patients (43%) following embolization [13,14]. There was no recurrence or re-expansion of auricular AVMs after treatment. Among the 21 patients with parotid AVMs (**Table 3**), the majority underwent surgical excision (8 patients) and embolization (5 patients), and embolization with sclerotherapy (4 patients), while a few patients underwent embolization followed by excision (3 patients), and only 1 had sclerotherapy [5,22-27]. Although no recurrence was detected in any of the AVM cases, there was incomplete involution in 3 cases (60%) of parotid AVMs following embolization [23].

Patient Outcomes

Recurrence and re-expansion of AVMs following embolization have been described in other extracranial AVMs within 5 years of treatment [3]. This is because the AVM was not removed and perhaps partially occluded following embolization [3]. Embolization can effectively reduce the size of the AVM by slowing its expansion and reducing blood loss during AVM excision. Postembolization ischemia subsequently can lead to angiogenesis and collateralization [3]. This is consistent with the natural cause of progression and re-expansion of AVM following treatment due to collateralization, dilation of vessels, and angiogenesis [3].

According to some authors, favorable long-term outcomes can be achieved by AVM resection with or without preoperative embolization [3]. Some authors believe that AVM recurrence can be stimulated by treatment, as both surgery and embolization can cause local tissue trauma, producing a pro-angiogenic milieu that incites AVM expansion [8]. These treatment modalities resulted in local tissue hypoxia and increased the release of hypoxia-inducible factor-1α, MMP-2, and MMP-9 from platelets, neutrophils, and macrophages [38]. Furthermore, interventions result in a reduction of the antiangiogenic tissue inhibitor of MMP [38]. Other authors advocate free-tissue transfer reconstruction following AVM resection to minimize the incidence of recurrence by reducing local tissue hypoxia [39].

In our patient's case, endovascular treatment was used to decrease the vascularity of the AVM. Surgical excision of the AVM followed by free-tissue transfer reconstruction was performed 24 h later, with an excellent outcome. To the best of our knowledge, this is the second case reported in the literature of a high-flow parotid and auricular AVM successfully treated by the aforementioned approach [5].

Conclusions

Management of these AVMs can be challenging and it is important to understand the natural history of the lesion. The pathophysiology and the natural progression, as well as the re-expansion of AVMs following treatment, involve collateralization, dilation of vessels and angiogenesis. Treatment requires a multidisciplinary team including physicians, surgeons, anesthetists, and diagnostic and interventional neuroradiologists. Diagnostic imaging is essential to define the AVMs' vascular anatomy, regions of involvement, and flow characteristics. Management of parotid and auricular AVMs often depends on the Schobinger stage and the flow rates of the lesion. Although total surgical resection is the criterion standard for these AVMs, endovascular embolization is an alternative treatment that can be used as an adjunct to surgery. Furthermore, perioperative embolization can decrease the vascularity of the lesion and effectively reduce blood loss during AVM surgery.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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