**Scholars Journal of Medical Case Reports**
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

**Radiology**

# An Arteriovenous Malformation of the External Ear in the Pediatric Population: A Case Report

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**DOI:** 10.36347/sjmcr.2023.v11i11.022 | **Received:** 02.09.2023 | **Accepted:** 09.10.2023 | **Published:** 20.11.2023

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**Abstract** **Case Report**

Arteriovenous malformation (AVM) results from errors in vascular development during embryogenesis; absent capillary beds lead to shunting directly from the arterial to venous circulation. Although it is common in the head and neck region, Arteriovenous malformations (AVM) of the external ear are relatively uncommon lesions. They typically present during childhood. We report a case of giant AVM of the right pinna in 8 years child, which are very rarely seen. We discussing the definition, clinical findings, diagnostic approaches of arteriovenous malformations.

**Keywords:** Arteriovenous malformation (AVM), CT, MRI, head and neck.



## INTRODUCTION

An arteriovenous malformation (AVM) is a direct communication between an artery and vein without capillary connections, and is mainly located in the intracranial region, lung and kidney [1]. An AVM outside the cranium is uncommon. However, the most common sites are cheek, ear, nose and forehead in the extracranium [2].

Arteriovenous malformations (AVM) of the external ear are relatively uncommon lesions. They typically present during childhood. Some of them may remain quiescent until adolescence and in some cases into adulthood. Enlargement of the AVM may be triggered by trauma, infection, or hormonal influences [3].

We experienced a case of an AVM in the external ear and present this case with a brief literature review.

## CASE

A 6 -year-old male patient was evaluated for a reddish and pulsatile mass of the right external ear in the Otolaryngology Department of our medical center. The patient presented with pulsatile tinnitus over the past 9 months and a reddish and pulsatile mass of theright external ear. He did not have any specific medical history. There was no history of trauma or surger.

Physical examination showed a soft, compressible, nontender, pulsatile, well-defined mass in the right temporal region and external ear. A bruit was heard on auscultation of the mass and the tissue adjacent to the mass was erythematous and edematous.

The patient was referring to our radiology department for a CT scan angiography of head and neck. Spirally acquires CT study performed in arterial and veinous phases with axial, coronal and sagittal reconstruction revealed large arteriovenous malformation in right pinna extends to the external part of the external auditory canal and parotid region. The MAV was fed by a branch of the external carotid. It was draining into external and internal jugular vein.

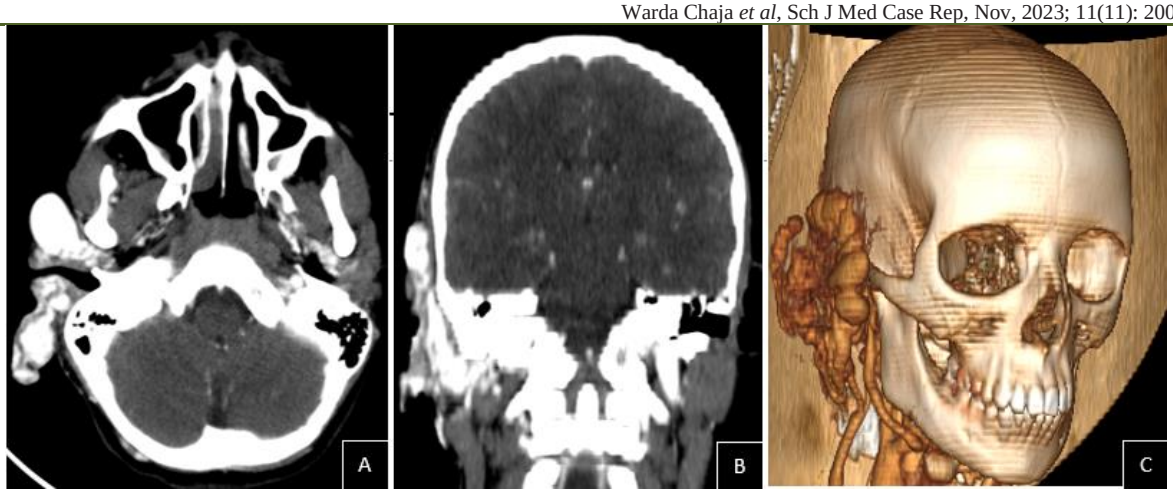


**Figure 1: Facial CT scan with contrast: axial (A), coronal (B) and in 3D reconstruction (C): Thickening of the right pinna extending into the external auditory canal with individualization of several serpiginous vascular structures within it after injection of contrast agent**

## DISCUSSION

An AVM is the result of failure of regression of arteriovenous channels in the primitive retiform plexus between the fourth and sixth weeks of gestation. An AVM is present at birth but usually is not clinically significant, except for a cosmetic problem [1, 2]. Over the course of time, an AVM can expand due to increased blood flow and collateral vessel formation. For a histopathological aspect, unlike a hemangioma, an AVM does not demonstrate cellular hyperplasia but displays progressive ectasia of abnormal vessels [3].

Vascular malformations are subdivided into 'slow-flow' or 'fast-flow' types, based on flow dynamics – 'slow-flow' malformations may be of capillary, venous, lymphatic type or combination, whereas 'fast-flow'ones would be of AVMs or fistulas. In the head and neck region, 'fast-flow' AVMs are less common as compared with the 'slow-flow' vascular malformations, and when present, they are most commonly located in the brain followed by perioral, parotid and neck regions [4-6].

The exact pathogenesis of an AVM has not been clearly defined. One theory states that these malformations arise during fetal development as a result of the failure of regression of arteriovenous channels in the primitive retiform plexus [7].

Schobinger classified AVM into four stages. The symptoms of stage I (quiescence) are warm and discolored skin; those of stage II (expansion) are bruit, pulsation, and swelling. Stage III (destruction) is characterized by pain, ulceration, and bleeding; whereas stage IV (decompensation) features cardiac failure [8, 9].

The presenting signs and symptoms correlate with the stage of AVM. Although many AVMs are asymptomatic, they may alternatively trigger severe pain and/or bleeding. The most common symptoms are pulsation (51.2%), bleeding (41.5%), and pain (29.3%) [7]. Hearing can also deteriorate, presumably because the bruit is audible [10, 11].

Radiological evaluation of these lesions can be accomplished via various modalities. Magnetic resonance imaging can be used to define the extent of soft tissue involvement, as well as display flow dynamics of the lesion. Computed tomography may determine skeletal involvement [11, 13].

Angiography is the most vital tool in evaluating the AVM because it identifies the vascular supply and allows selective embolization of these collateral vessels before operative resection [1, 2]. Although superselective embolization may limit intraoperative bleeding, it is essential that the procedure is not delayed more than 48 h after the embolization because of the development of extensive collaterals [12, 13].

Angiography is useful in residual lesions after surgery to show the major vessel and embolize if necessary. The angiographic features of AVMs are dilatation and lengthening of afferent arteries with early opacification (shunting) of enlarged efferent veins [12, 13].

## CONCLUSION

AVMs of the external ear are an uncommon entity, often presenting as an enlarging pulsatile mass. The exact pathogenesis of these lesions has not been clearly defined, but are thought to arise during fetal development as a result of the failure of regression of arteriovenous channels in the primitive retiform plexus. It has also been postulated that local ischemia plays a role in the development of AVMs. Depending upon the extend of the malformation, the work-up may be limited to a physical examination or include angiography, magnetic resonance imagine and computed tomography to assist in the delineating the extent of the lesion.

Treatment may be limited to surgical excision alone, or may include preoperative embolization with a variety of agents.

## REFERENCES


1. Kohout, M. P., Hansen, M., Pribaz, J. J., & Mulliken, J. B. (1998). Arteriovenous malformations of the head and neck: natural history and management. *Plastic and reconstructive surgery*, *102*(3), 643-654.
2. Wu, J. K., Bisdorff, A., Gelbert, F., Enjolras, O., Burrows, P. E., & Mulliken, J. B. (2005). Auricular arteriovenous malformation: evaluation, management, and outcome. *Plastic and reconstructive surgery*, *115*(4), 985-995.
3. Yamamoto, Y., Ohura, T., Minakawa, H., Sugihara, T., Yoshida, T., Nohira, K., & Shintomi, Y. (1994). Experience with arteriovenous malformations treated with flap coverage. *Plastic and reconstructive surgery*, *94*(3), 476-482.
4. Seccia, A., Salgarello, M., Farallo, E., & Falappa, P. G. (1999). Combined radiological and surgical treatment of arteriovenous malformations of the head and neck. *Annals of plastic surgery*, *43*(4), 359-366.
5. Song, J. J., An, G. S., Choi, I., De Ridder, D., Kim, S. Y., Choi, H. S., ... & Lee, K. (2016). Objectification and differential diagnosis of vascular pulsatile tinnitus by transcanal sound recording and spectrotemporal analysis: a preliminary study. *Otology & neurotology*, *37*(6), 613-620.
6. Kim, S. H., An, G. S., Choi, I., Koo, J. W., Lee, K., & Song, J. J. (2016). Pre-treatment objective diagnosis and post-treatment outcome evaluation in patients with vascular pulsatile tinnitus using transcanal recording and spectro-temporal analysis. *PLoS One*, *11*(6), e0157722.
7. Kim, C. S., Kim, S. Y., Choi, H., Koo, J. W., Yoo, S. Y., An, G. S., ... & Song, J. J. (2016). Transmastoid reshaping of the sigmoid sinus: preliminary study of a novel surgical method to quiet pulsatile tinnitus of an unrecognized vascular origin. *Journal of neurosurgery*, *125*(2), 441-449.
8. Song, J. J., Kim, Y. J., Kim, S. Y., An, Y. S., Kim, K., Lee, S. Y., & Koo, J. W. (2015). Sinus wall resurfacing for patients with temporal bone venous sinus diverticulum and ipsilateral pulsatile tinnitus. *Neurosurgery*, *77*(5), 709-717.
9. Pham, T. H., Wong, B. J., & Allison, G. (2001). A large arteriovenous malformation of the external ear in an adult: report of a case and approach to management. *The Laryngoscope*, *111*(8), 1390-1394.
10. Tark, K. C., & Chung, S. (2000). Histologic change of arteriovenous malformations of the face and scalp after free flap transfer. *Plastic and reconstructive surgery*, *106*(1), 87-93.
11. Thornton, B. P., Sloan, D., & Rinker, B. (2006). Squamous cell carcinoma arising from an arteriovenous malformation of the scalp. *Journal of Craniofacial Surgery*, *17*(4), 805-809.
12. Wu, J. K., Bisdorff, A., Gelbert, F., Enjolras, O., Burrows, P. E., & Mulliken, J. B. (2005). Auricular arteriovenous malformation: evaluation, management, and outcome. *Plastic and reconstructive surgery*, *115*(4), 985-995.
13. Yoshida, Y., Ishizawa, T., Mitsuhashi, Y., Kondo, S., Ohta, N., & Sakai, N. (2006). A patient with subaural arteriovenous malformation complicated by macrotia. *Clinical and Experimental Dermatology: Viewpoints in dermatology*, *31*(1), 151-152.