Arteriovenous malformation of the external ear

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Abstract Arteriovenous malformation (AVM) involving the external ears is relatively uncommon in adults. Arteriovenous malformations typically present during childhood and rarely manifest during adulthood in the head and neck region. Symptomatic arteriovenous malformations often pose difficult therapeutic challenges. We report a case of large arteriovenous malformation of the external ear in a 21-year-old female.

Keywords Arteriovenous malformation ·
Vascular lesions · Pulsatile swelling

Introduction

Vascular lesions have been classified by Mulliken and Glowacki on the basis of cellular characteristics and correlation of the cellular behavior to the clinical progression of the lesion [1, 2]. According to this classification, vascular anomalies are divided into two main categories; hemangioma and vascular malformation. Hemangioma is a lesion characterized by accelerated endothelial proliferation and eventually followed by regression. Its clinical history is marked by rapid growth during the postnatal period (proliferative phase) [3, 4]. In vascular malformations, the endothelial cells have a normal rate of cellular division and do not regress. Vascular malformations are morphogenic anomalies of the vascular structures that are present at birth and tend to grow with age.

Malformations are subdivided according to the vascular flow dynamics into slow flow and fast flow types [5, 6]. A vascular malformation that exhibits slow flow type is either a capillary, venous, lymphatic, or combined malformation, whereas a fast flow vascular malformation is predominantly an arteriovenous malformation or a fistula. Clinical manifestations of arteriovenous malformation are related to abnormal blood perfusion and mass effect.

Case history

A 21-year-old female was evaluated for a massive pulsatile swelling of right external ear and continuous buzzing sensation for two years. There was no history of ear discharge, deafness, or vertigo. There was no history of trauma or surgery. Examination showed two pulsatile swellings; 3 × 3 cm pulsatile cystic mass on the medial surface of the pinna with contiguous extension and 1 × 1 cm swelling in the post aural region (Fig. 1). On pressing the temporal artery, pulsation of the AVM stopped which confirmed that temporal artery was the feeding vessel. Overlying skin showed reddish

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pigmentation. Thrill was felt on palpation and continuous bruit on auscultation. Angiography showed diffuse presence of shunts with the temporal artery as the primary feeder (Fig. 2). Preoperative embolization was attempted but failed. Ligation of temporal artery was done early in the surgery. As soon as temporal artery (feeding vessel) was ligated, the pulsation stopped and excision of AVM became easier. Her postoperative course was uneventful. She is asymptomatic without recurrence at three year follow up.

Fig. 1 Arteriovenous malformation producing the swelling on medial surface of the right pinna and post aural region

Fig. 2 Angiography showed diffuse presence of shunts with the temporal artery as the primary feeder

Discussion and conclusion

Most vascular malformations manifest clinically during childhood and grow commensurately with the child's age. However, some malformations, especially an AVM, may remain quiescent until adolescence and in some cases into adulthood. Enlargement of the AVM may be triggered by trauma, infection, or hormonal influences. Clinical manifestations of AVM are related to abnormal blood perfusion and mass effect. A high rate of blood flow to the AVM may result in increased warmth, erythema, and edema of the affected tissues. Arteriovenous shunting may be marked by pulsation and thrill on palpation and bruit on auscultation.

History and physical examination are often sufficient to determine a clinical diagnosis of AVM especially in cases that involve the superficial tissues of the head and neck region. Plain x-ray film and computed tomography (CT) scan usually show a soft tissue mass. Angiography is the most useful for the investigation of AVM, to identify the arterial supply and a portal for embolization. If the AVM is small and asymptomatic, treatment is unnecessary. Ligation of arterial vessels alone should be avoided because new collateral circulation will form, causing further enlargement [5, 6]. Optimal treatment is based on a combined approach of a super selective embolization and a complete surgical excision [7].

References

1. Mulliken JB, Glowacki J (1982) Hemangiomas and vascular malformations in infants and children; a classification based on endothelial characteristics. *Plast Reconstr Surg* 69: 412–422
2. Finn MC, Mulliken JB, Glowacki J (1983) Congenital vascular lesions: Clinical application of a new classification. *J Pediatr Surg* 18:894–900
3. Hurmitz DJ, Kerber CW (1981) Hemodynamic considerations in the treatment of arteriovenous malformations of the face and scalp. *Plast Reconstr Surg* 67:421–434
4. Jackson IT, Carreno R, Potparic Z, Hussain K (1993) Hemangiomas, vascular malformations, and lymphovenous malformations classification and methods of treatment. *Plast Reconstr Surg* 91:1216–1230
5. DesPrez JD, Kiehn CL, Vlastau C, Bonstelle C (1978) Congenital arteriovenous malformation of the head and neck. *Am J Surg* 136:424–429
6. Coleman CC (1973) Diagnosis and treatment of congenital arteriovenous fistulas of the head and neck. *Am J Surg* 126: 557–565
7. Biller HF, Krespi YP, Som PM (1982) Combined therapy for vascular lesions of the head and neck with intra-arterial embolization and surgical excision. *Otolaryngol head and neck surg* 90:37–47