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Case Report

Unusual Vascular Malformation of Pinna

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ABSTRACT

Arterio-venous malformations of the pinna are a rare occurrence. They pose to the clinicians with an intriguing cosmetic defect, a feeling of unusual warmth and abnormal pulsations. In some situations, repeated episodes of bleeding become an issue of concern in some patients. The treatment however revolves around surgical excision with an addition of prior sclerotherapy or prior selective embolization. We report the case of a young female with a long-standing arteriovenous malformation involving the left ear pinna, which demonstrated rapid progression during pregnancy. The patient was successfully managed with complete surgical excision.

KEYWORDS: Arterio-Venous malformation, Macrotia, Pinna deformities, Vascular anomalies

INTRODUCTION

Arteriovenous malformations (AVMs) are congenital vascular anomalies characterized by direct communications between arteries and veins without an intervening capillary network. This abnormal shunting results in high-flow circulation, leading to progressive enlargement, tissue destruction, and potential life-threatening complications such as hemorrhage and cardiac overload. Arterio-Venous malformations present in head neck region with varied symptomatology ranging from cosmetic deformity, repeated bleeding episodes, unusually warmth in affected areas and abnormal pulsations. These lesions are usually congenital but may remain asymptomatic until triggered by hormonal changes or trauma. Arterio-Venous malformations of pinna are one of the uncommon occurrences in the head and neck region. The Arterio-Venous malformations of head and neck more commonly are high flow lesions and present with hemorrhage. These lesions always warrant careful surgical excision failing which there are issues of recurrence. Farmer et al reported a case of Arterio-Venous malformation of ear in 1956.

CASE REPORT

A 35-year-old female presented with a slowly enlarging mass over the left ear pinna, present since childhood [Figures 1 and 2]. The lesion remained small for many years but showed a noticeable increase in size during pregnancy. The patient reported multiple episodes of spontaneous bleeding from the lesion, which resolved spontaneously without medical intervention most of the time but sometimes needed treatment in the Emergency department. There was no family history of similar vascular anomalies.

On clinical examination, a soft, non-tender, irregularly shaped mass measuring approximately 7 × 3.5 cm was noted in its greatest dimension. The skin covering the lesion had a bluish red hue involving over the helix, crus, cavum and cymba conchae and posterior aspect of the left pinna. The lobule was spared. The left pinna was 1.5 cm long and 0.5 cm wide compared to the right pinna. The lesion exhibited multiple lobulations with areas of crusting and superficial ulceration. There was no extension into the preauricular, postauricular, or temporoparietal regions. Auscultation revealed an audible bruit over the swelling, suggestive of a high-flow vascular lesion. There was no cervical lymphadenopathy on neck examination. A contrast enhanced CT of head and neck showed areas of increased contrast uptake in the left pinna [Figure 3]. A Digital Subtraction Angiography (DSA) showed abnormal arteriovenous malformations in the left pinna [Figure 4]. The posterior auricular artery appears to be the feeder vessel for this AV malformation.

A full surgical work up was done. Blood grouping and cross matching was done and B positive blood was reserved for intra-surgical hemorrhage.

A skin flap was raised on posterior aspect of the pinna. Subcutaneous plane was entered and tortuous vessels were cauterized clearing both anteriorly and posteriorly to the pinna.

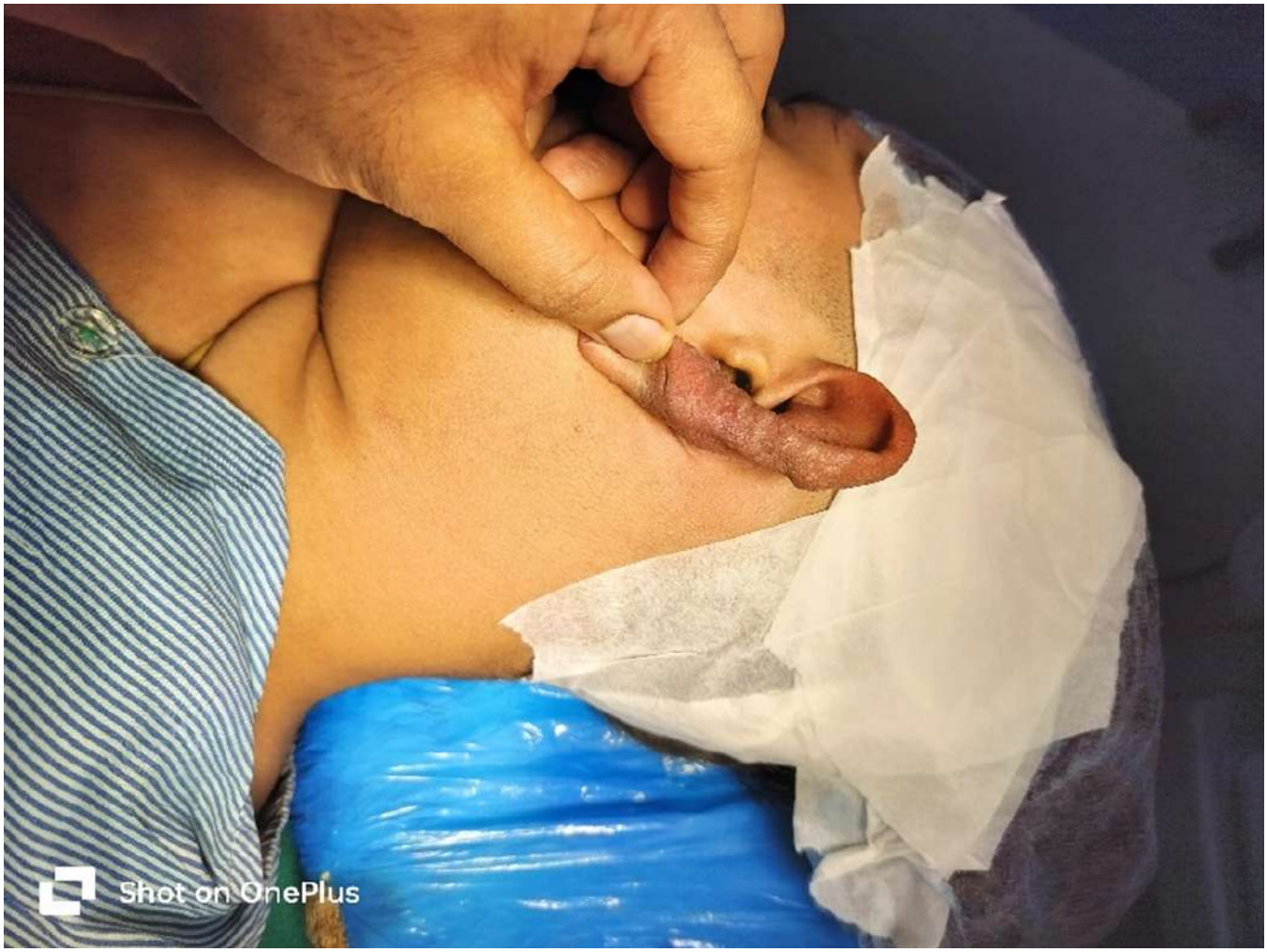

Figure 1: Pre-operative Photograph of the Left Pinna

A superior based flap also was developed to address the tortuous vessels on the superior pocket of the helix. Care was taken to avoid injury to the cartilage. Wound was closed after ensuring proper hemostasis [Figures 5 and 6].

Histopathology showed features of ectatic arterioles and other features of AVMs. Patient was advised to follow-up regularly as advised. On first follow-up sutures were removed after ensuring wound closure was achieved. Subsequent follow-ups were done to assess the operative site.

The histopathology report suggested vascular malformation and the patient was followed up for 3 months following which no deformity was noted [Figures 8 and 9].

Figure 2: Pre-operative Photograph of the Left Pinna

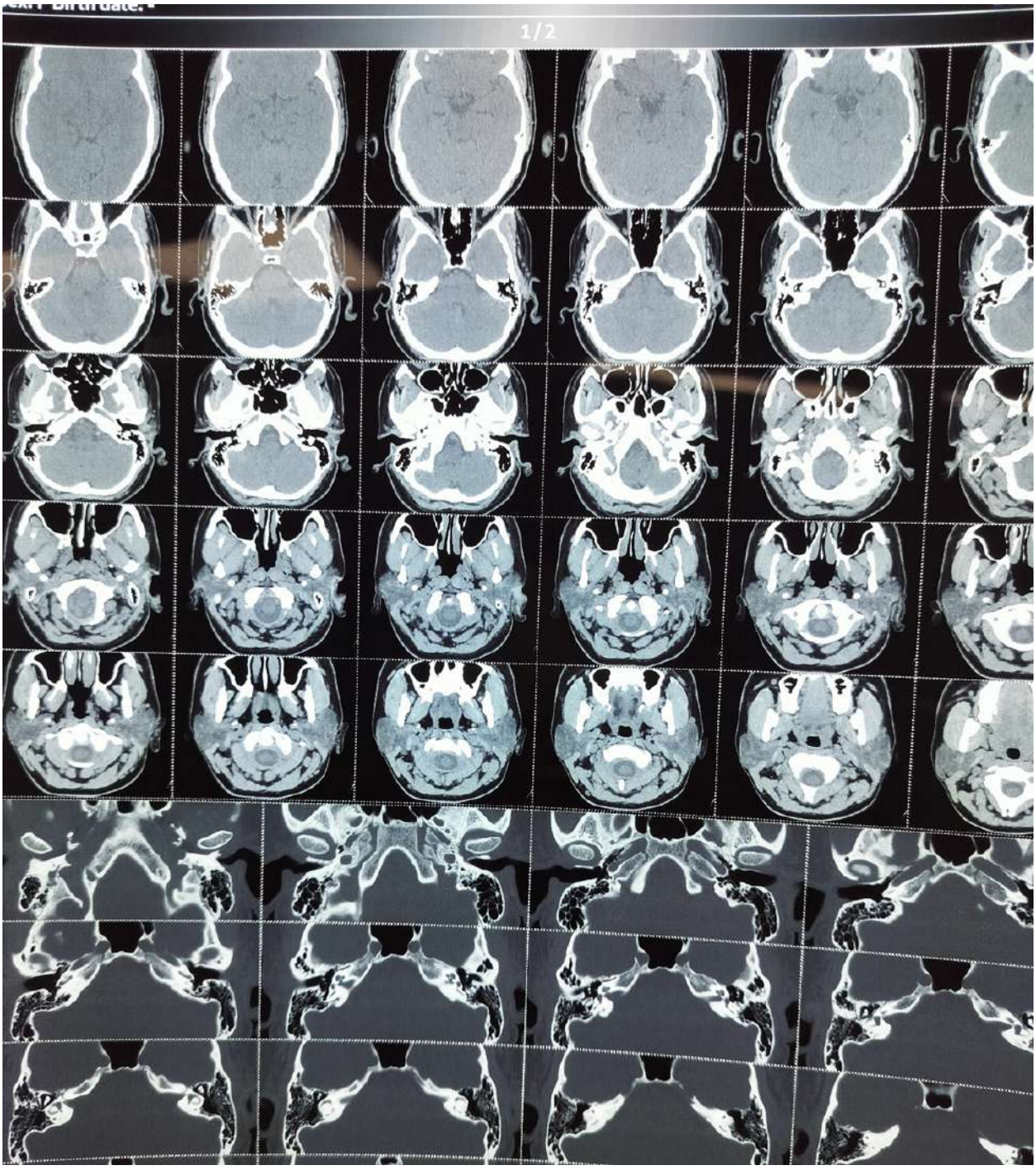

Figure 3: Contrast Enhanced CT of Head and Neck Showing Contrast Enhancement in the Left Pinna

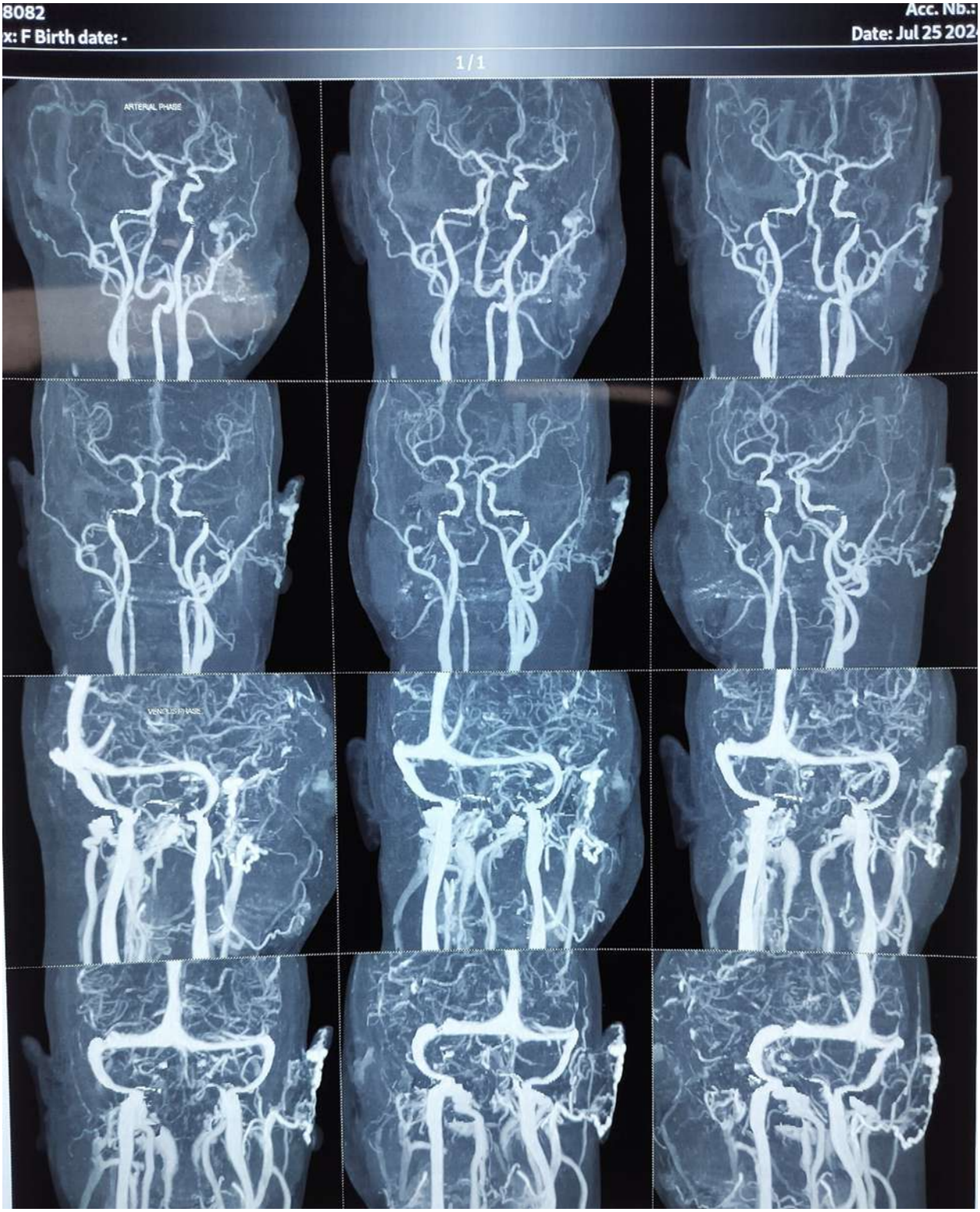

Figure 4: DSA Showing Abnormal AVMs in the Left Pinna

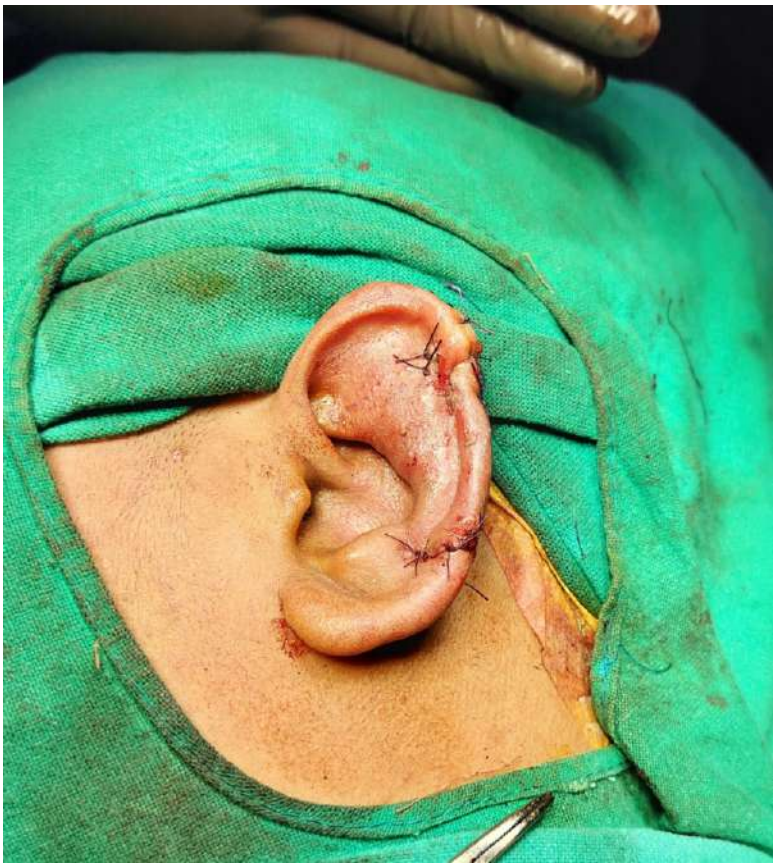

Figure 5: Immediate Post-operative Antero-lateral View

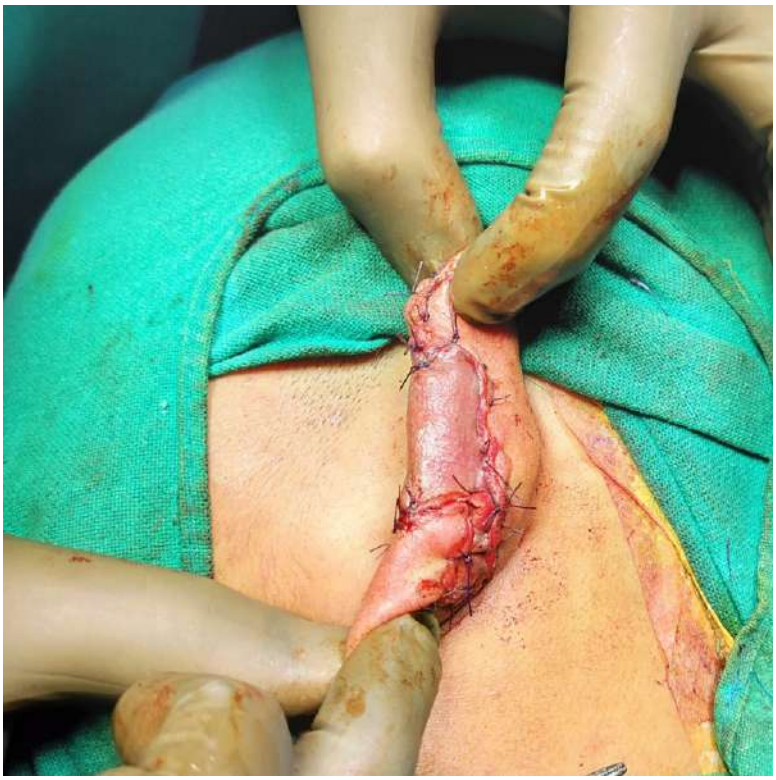

Figure 6: Immediate Post-operative Posterior View

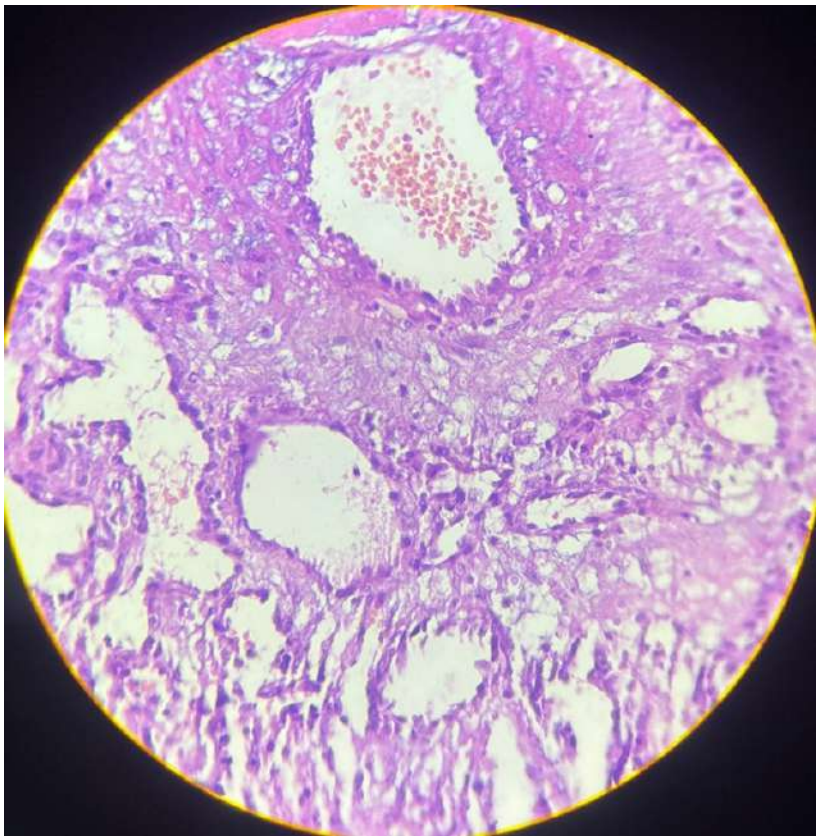

Figure 7: HPE Shows Ectasia of Arterioles with Thin Endothelial Lining

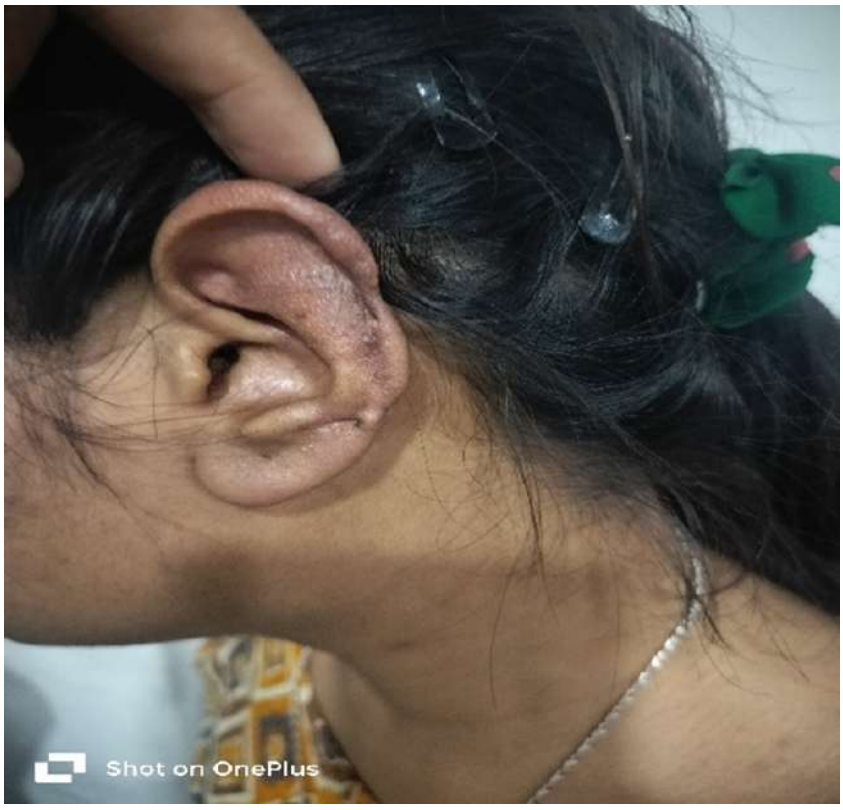

Figure 8: Post-operative Lateral View

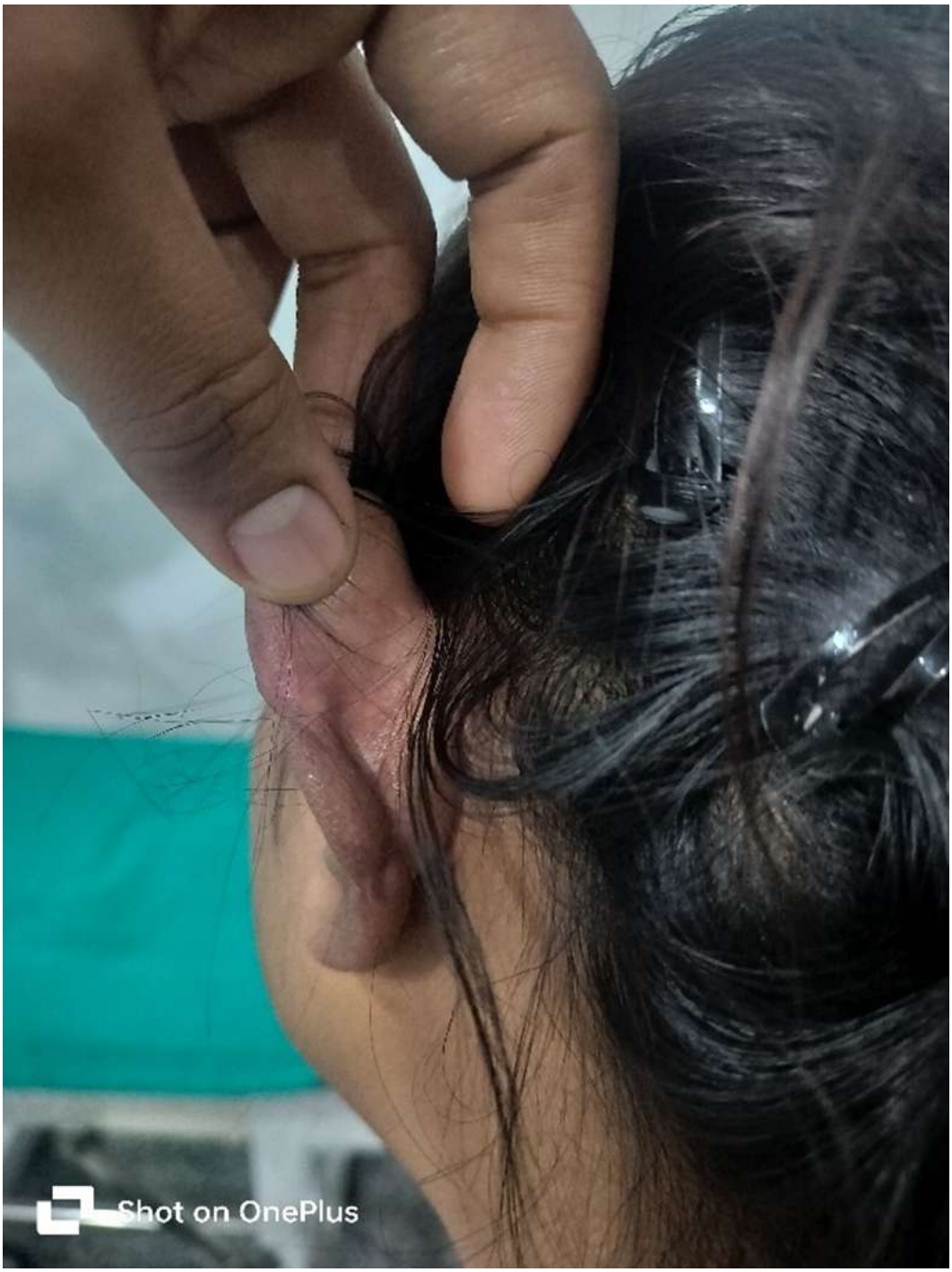

Figure 9: Post-operative Posterior View of the Left Pinna

DISCUSSION

AV Malformations of the external ear are rare. Most of them are fast flow lesions consisting of arterio-venous anastomosis. These give an audible bruit. The rest of them are slow flowing lesions involving the anastomosis between venous, capillary and lymphatic system[1].

The Arteriovenous malformations generally are found to be comprising of a nidus typically surrounded by abnormal anastomotic network between AV shunts. The clinical manifestations of the AVM scan be attributed to the absence of capillary system which interpose between the arterioles and the venous system. This shifts the high pressure from the arterial system to the venous system[2].

Kohout et al presented a large retrospective study of 81 patients with head and neck vascular malformations at Vascular anomalies program in Boston in 1998. Out of these 16 percent of patients had vascular malformations of the pinna. In this study 8 patients noted AVMs in the pubertal period, six patients noted exacerbation during puberty. According to the Clinical Staging System by Schobinger for Vascular Malformations: Stage I (Quiescence) - cutaneous blush/ warmth; Stage II (Expansion) - bruit, audible pulsation, expending lesions; Stage III (Decompensation) Cardiac failure[3]. These lesions can be classified into four stages as described by Schobinger, I: Cutaneous blush/ warmth; II: Bruit, audible pulsations, expanding lesion; III: Pain, ulceration, bleeding and infection; and IV: Cardiac failure[3].

The AVMs typically display ectasia of abnormal vessels leading on to collateral formation. This diverts the blood from the regional circulation. Histological, the arteries turn out to be lengthened, tortuous and thin walled. The veins however tend to have a thicker muscular coat because of increased pressure[4].

The Arteriovenous malformations of the external ear typically derive their blood supply from branches of the external carotid artery, including the superficial temporal, posterior auricular, and occipital arteries. Although congenital in origin, these lesions may remain clinically silent until precipitated by hormonal influences such as puberty or pregnancy, or by trauma[5].

The exact pathogenesis of AVMs remains unclear, though most theories attribute their development to errors in late embryonic angiogenesis, resulting in persistent arteriovenous connections. Clinically, AVMs may progress through stages ranging from localized warmth and discoloration to ulceration, bleeding, and, in severe cases, high-output cardiac failure.

Imaging plays a crucial role in diagnosis and treatment planning. Doppler ultrasonography helps identify high-flow vascularity, while MRI delineates soft tissue involvement. Angiography remains the gold standard, as it defines the vascular anatomy and enables therapeutic embolization. Characteristic angiography findings are marked hypertrophy and tortuosity in the feeding vessels. The appearance of the nidus (center) of the lesion varies from large tortuous vessels to innumerable small vessels appearing as an intense blush. Collateral vessels usually have a 'cork screw' appearance[6,7].

Management of auricular AVMs is challenging due to the risk of excessive bleeding, cosmetic deformity, and recurrence. If the AVM is small and asymptomatic, no treatment is required, especially in children. Embolisation alone may provide temporary control but is associated with high recurrence rates. Therefore, combined therapy involving embolisation followed by complete surgical excision within a short interval is considered the most effective approach. Incomplete resection remains the most common cause of recurrence, emphasizing the need for meticulous surgical planning and long-term follow-up. For a symptomatic AVM, complete excision with prior embolisation is the treatment of choice[8]. Only surgical ligation or only proximal embolisation may lead to not only aggravating the lesion by establishing new collaterals but also precludes later embolisation hence leading to failure for the same reasons[9,10]. Embolisation alone rarely has to be done in deeper located lesions where surgical approach is very challenging and areas close to highly important structures[10]. However, in such cases, complications like stroke, cranial nerve palsy, and blindness can occur[11]. Total resection which requires a wide-field resection of all the involved tissue is necessary to prevent recurrence, however, cosmetic and functional issues might limit the extent to which tissue can be removed. Partial excision usually leads to rapid recurrence so in these cases the remaining AVM tissue must be obliterated using intravascular embolisation.

CONFLICT OF INTEREST: None

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