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Case Report

Dentistry Section

Entangled Aetiologies: A Case Report on Post Extraction Arteriovenous Malformation in Pregnancy

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ABSTRACT

Arteriovenous Malformations (AVMs) are rare, high-flow vascular anomalies characterised by abnormal shunting between arteries and veins. These lesions present unique challenges in diagnosis and management due to their complex vascular anatomy, tendencies for recurrence, and potential complications. This case report discusses a 26-year-old postpartum female presenting with an AVM in the mandible, following a dental extraction during the second trimester of pregnancy. The lesion was initially managed conservatively with embolisation during pregnancy but showed significant progression postpartum, necessitating a multidisciplinary approach. Preoperative embolisation was performed using glue and Lipiodol, targeting branches of the right internal maxillary and facial arteries. This was followed by surgical resection and reconstruction using free fibula osteocutaneous grafts to restore function and aesthetics. Pathological examination confirmed the characteristics of an AVM, including fragmented elastic lamina and endothelial hyperplasia. This case underscores the importance of combining advanced diagnostic imaging, timely embolisation, and surgical intervention with aesthetic reconstruction to achieve optimal outcomes. Despite the unclear aetiology-whether trauma-induced, hormonally influenced, or congenital-the patient experienced favourable functional and aesthetic results with no recurrence at four months of follow-up. This report highlights the complexity of AVM management and reinforces the necessity of a multidisciplinary, multimodal approach for these challenging lesions.

Keywords: Embolisation, Extraction, Free fibula flap, Microvascular proliferation

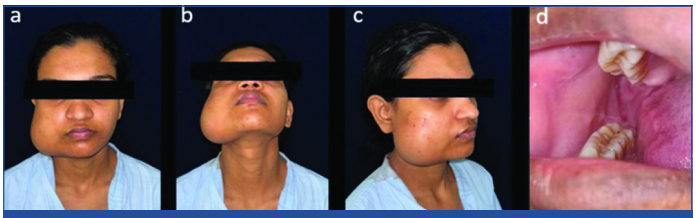
CASE REPORT

A 26-year-old female presented to the Oral and Maxillofacial Surgery outpatient department four months postpartum with a painless swelling on the right side of her face that had persisted for seven months. She reported a painful extraoral swelling that began after the surgical removal of her third molar during the second trimester of pregnancy, which did not resolve. She also described applying balm 3-4 times to the area and experiencing gradual onset of right-sided facial paresthesia 1-2 months after the extraction.

On examination, the diffuse extraoral swelling measured approximately 4.5×4 cm, was non tender, and had a soft to firm consistency, with minimal thrill. There was no skin discoloration or fixation to underlying structures. The swelling extended supero-inferiorly from the malar region of the right side to the inferior border of the mandible, and anteroposteriorly 2 cm from the corner of the mouth to the posterior border of the mandible on the right side, with ill-defined borders. Intraoral examination did not reveal any significant abnormalities. An ultrasound confirmed a high-flow vascular lesion.

Based on the anatomical location, history of trauma (dental extraction), and pregnancy-associated vascular changes, the differential diagnoses included: 1) AVM; 2) Hemangioma; 3) Vascular varix or venous malformation; 4) Infectious/inflammatory mass; 5) Central giant cell granuloma. Due to her advanced pregnancy (third trimester), she underwent embolisation at the treating center as a conservative approach. Embolisation helped temporarily reduce the lesion's vascularity, prevent potential hemorrhagic complications, and buy time until a safer postpartum surgical window could be utilised.

Postpartum, as the swelling persisted and increased in size, she returned four months after delivery for further evaluation with MRI (details of which were not available). The lesion had progressed to approximately 8×6 cm, extending horizontally from the corner of the mouth to the right preauricular region and vertically from the malar region to the inferior mandibular border, with no intraoral findings [Table/Fig-1]. A treatment plan consisting of preoperative embolisation followed by surgery within two days was finalised.



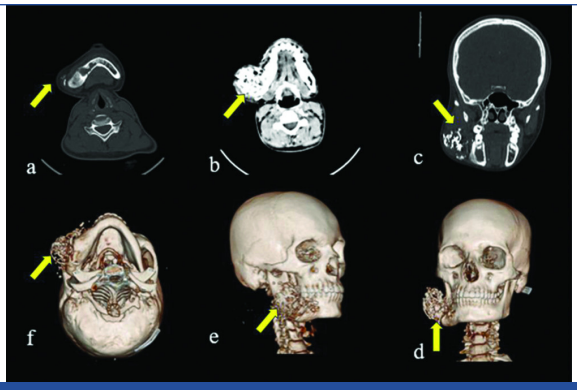
[Table/Fig-1]: Preoperative images- (right to left): a-frontal; b- worms view; c-quarter lateral showing the location & extent of swelling; d- Intraoral image showing no significant change.

Embolisation was performed using glue and Lipiodol in branches of the right internal maxillary and facial arteries, followed by Computed Tomography (CT) imaging. The report suggested a soft-tissue density lesion with multiple hyperdense foci (possibly the embolising agent) within the right gingivobuccal sulcus, measuring approximately 58×40×69 mm. The lesion involved the masticator space and the buccinator, platysma, and masseter muscles, causing erosive destruction of the ramus, angle, and body of the right hemimandible, with involvement of both the outer and inner cortex [Table/Fig-2].

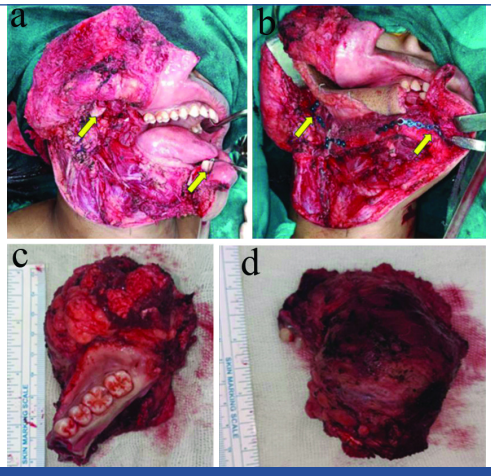
After reviewing the blood workup and other reports (including Complete Blood count (CBC), Liver Function test(LFT), Kidney Function Test (KFT), coagulation profile, chest X-ray, and ECG, all of which were within normal limits), the treatment plan was conveyed to the patient. Consent and documentation were completed for the resection of the lesion and microvascular reconstruction using a free fibula osteocutaneous graft under general anaesthesia [Table/Fig-3]. [Table/Fig-4] demonstrates the histopathological findings.

A postoperative MRI of the head revealed no intracranial or extracranial vascular abnormalities. The postoperative OPG showed satisfactory anatomical reconstruction with no new abnormalities.

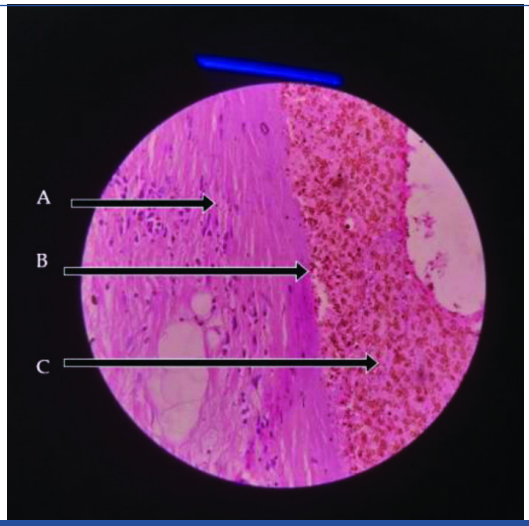
Regardless of the aetiology, the patient achieved favourable functional and aesthetic outcomes, with no recurrence observed



[Table/Fig-2]: Spiral CT cuts clockwise: a) axial hard tissue – erosive destruction of mandibular cortex; b) axial soft-tissue- involvement of massster and buccinator muscle seen c. coronal hard tissue- multiple hyperdense focci within the lesion; d) frontal 3D; e) right oblique 3D; f) Worms 3D view.



[Table/Fig-3]: Intraoperative photos: a) Surgical defect extends from lateral incisor on left side to condyle on right side; b) reconstruction with free fibula graft - osteotomised segments of free fibula graft fixed with titanium plates and screws to reconstruct lost tissue; c) resected specimen after segmental mandibulectomy (occlusal view); d- resected specimen (lateral view) showing the lesion.

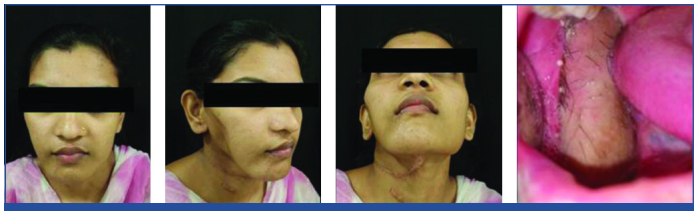


[Table/Fig-4]: High-power microscope (10x) with haematoxylin and eosin stain: (a) Muscle with arteries with disrupted or fragmented internal elastic lamina, with evidence of hypertrophy and hyperplasia in tunica media; (b) Enlarged endothelial cells with reactive changes and extravasated RBCs; (c) In focal area of endothelial damage.

during the four months of follow-up [Table/Fig-5], demonstrating the efficacy of the chosen multimodal therapeutic approach.

DISCUSSION

Vascular malformations are benign lesions that constitute approximately 7% of all vascular tumours. While the head and neck region represents less than 14% of the body's surface area, it accounts for nearly half of all vascular malformations [1]. Among high-flow vascular anomalies, AVMs are the most prevalent,



[Table/Fig-5]: Fourth month postoperative follow-up photos - shows satisfactory aesthetic outcome (frontal, quarter lateral and worms view - from left to right) with healthy intraoral flap (first image from right).

commonly affecting the head, neck, limbs, and lungs. AVMs of the face and oral cavity are particularly rare, with mandibular involvement reported more frequently than maxillary, and fewer than 200 cases documented in the literature [2]. These lesions are characterised by direct arteriovenous shunts that bypass the capillary network [3]. AVMs are predominantly diagnosed during adolescence, but they can occur across a wide age range, from infancy to late adulthood. The gender distribution is inconsistent, with some studies reporting a female predilection of 2:1, while others show no significant differences [4]. A 20-year analysis by Kohout MP et al., found that 69% of AVMs were located in the midface, 17% in the lower third, and 14% in the upper third [5]. AVMs constitute 1.5% of all vascular malformations, with 90% occurring intracranially. Complications depend on the anatomical location and developmental stage, including stroke, brain abscess, hypoxemia, and local rupture, leading to life-threatening hemorrhage [3].

Schobinger's clinical classification describes the stages of AVM progression [6]. The lesion in this report can be placed in the progression from stage 1 to stage 2. Approximately 6.4% of extracranial AVMs occur in the face and oral cavity, with bony AVMs in the head and neck most commonly involving the mandible in two-thirds of cases [6]. These lesions are characterised by progressive bone enlargement, tooth mobility, and gingival bleeding, which, when accompanied by radiographic evidence of a hypodense mass, form a pathognomonic triad for diagnosis [7]. A sub-classification by Nair SC et al., (2011) categorises AVMs based on anatomical site and depth [8]. This case, although it did not present with the pathognomonic triad, had other positive findings on imaging and clinical examination. It can be considered a mixture of types II through V according to Nair SC et al.,'s classification. Facial and oral AVMs are rare but can cause significant aesthetic and functional impairments. Extracranial AVMs are among the most challenging vascular malformations to manage, with curative treatment often being elusive [9]. The development of AVMs is multifactorial, involving congenital, environmental, and genetic factors. During embryonic vascular development, vasculogenesis and angiogenesis form a capillary plexus that differentiates into arteries, veins, and lymphatics. Errors in this process can result in vascular malformations, as incomplete differentiation of plexus cells leads to persistent abnormalities [10]. Hemodynamic stress, such as trauma or surgical interventions (e.g., dental extractions), has been implicated in the development of AVMs. This stress may trigger abnormal vascular remodeling, resulting in direct arteriovenous connections. In Hereditary Hemorrhagic Telangiectasia (HHT), AVMs develop in postcapillary venules through a sequence involving venular dilation, arteriole-venule connections, and the disappearance of capillary beds, culminating in direct arterial-venous shunting [1,3]. Trauma or iatrogenic factors, including surgical and dental procedures, may also induce abnormal vascular connections, contributing to AVM formation.

Genetic mutations, particularly in pathways regulating angiogenesis (e.g., RAS-MAPK), are recognised in hereditary conditions such as HHT and Capillary Malformation-Arteriovenous Malformation Syndrome (CM-AVM). Pathogenic variants in genes like RASA1, EPHB4, and PIK3CA are associated with AVMs in syndromic contexts, including overgrowth syndromes [5]. Hormonal changes, particularly during pregnancy, may influence the development of AVMs. Hormonal modulation of vascular endothelial growth factor

expression is hypothesised to promote vascular remodeling and lesion growth, though this remains speculative. Collectively, these factors underscore the complexity of AVM pathogenesis, involving congenital anomalies, genetic predisposition, environmental triggers, and potential hormonal influences [11,12]. This case highlights the critical interplay between hormonal, traumatic, and possibly congenital factors in the progression of AVMs. The emergence of the lesion following a second trimester dental extraction and its progression during pregnancy suggests a potentiating role of both localised trauma and systemic hormonal surges. Recent studies support this association; for instance, Schimmel K et al., and Utami AM et al., in their respective publications, demonstrated increased microvascular proliferation in AVMs during pregnancy, suggesting that hormonal upregulation of Vascular Endothelial Growth Factor(VEGF) may accelerate lesion growth [3,11]. Moreover, Sasaki R et al., and Azis KA et al., reported AVM progression or rupture during late gestation, reinforcing the link between elevated haemodynamic load and AVM instability [13,14]. A high-flow vascular lesion was identified on Doppler ultrasound, consistent with a probable AVM. While AVMs are typically congenital, the emergence of clinical symptoms after the extraction suggests a "second-hit" model, where local trauma acts as a trigger in a genetically predisposed vascular bed [3,15-17]. Trauma from the surgical extraction may have disrupted existing vascular channels, while hormonal changes during pregnancy likely promoted VEGF-driven angiogenesis, exacerbating lesion growth.

The current case exemplifies the management principles outlined by Kohout MP et al., with resection performed within 48 hours of embolisation to minimise neovascularisation and the risk of bleeding [5,16]. Proximal ligation was consciously avoided to prevent collateral recruitment, and a multidisciplinary strategy that incorporated diagnostic imaging, selective embolisation, and radical resection with immediate microvascular reconstruction was implemented, aligning with current best practices [17]. Embolisation was performed during the second trimester as a conservative measure to reduce vascularity, prevent haemorrhagic complications, and delay definitive surgery until a safer postpartum window. For similar cases, we recommend early referral to a vascular anomalies team, preoperative angiography with high-flow mapping, timely embolisation (within 24-48 hours of surgery), and the application of Schobinger staging to guide the timing and aggressiveness of intervention. Long term surveillance with Magnetic Resonance Imaging(MRI) and clinical monitoring is crucial due to the potential for recurrence, particularly in hormonally active or trauma-exposed patients. The management of high-flow AVMs primarily involves surgery, catheter-guided interventions, or stereotactic radiosurgery, with the aim of embolising, resecting, or irradiating lesions. The primary goal is the eradication of the low-pressure nidus, which drives recurrence by recruiting collateral vessels. Treatment is challenging due to the complexity of arterial supply. Proximal vessel ligation is ineffective, as it promotes collateral recruitment and recurrence. Similarly, proximal embolisation with coils or devices often fails and is best avoided. Effective management focuses on directly occluding the nidus to achieve long-term control. Recurrence rates for AVMs range widely, from 8% to 93% [6].

Principles derived from multiple studies that remain valid include: (1) the importance of performing resection within 24 to 48 hours of embolisation; (2) the ineffectiveness of laser treatments, steroids, and irradiation; and (3) the contraindication of proximal ligation of vessels, which results in recruitment of vessels from adjacent vascular territories and compromises later attempts at highly selective embolisation [5,18-20]. Similar cases from various studies have been listed in [Table/Fig-6] [11,13,14,21-25].

Recent developments in AVM management reflect a paradigm shift toward precision diagnostics, targeted therapies, and multidisciplinary care. The ARISE I consensus, led by Samaniego EA et al., (2024),

Author(s)	Age/ Gender	Lesion description and site	Treatment performed
Elliott JA et al., 1985 [21]	28/ Female	AVM of left upper limb with swelling, pain, and cyanosis at 18 weeks gestation	Conservative management; condition improved postpartum
McMahon MJ et al., 1997 [22]	32/ Female	Mandibular AVM with pulsatile mass at 35 weeks gestation; extensive oral bleeding	Selective arterial embolisation; uncomplicated vaginal delivery; surgical resection 1 week postpartum
Sasaki R et al., 2008 [13]	29/ Female	Mandibular gingival AVM with massive bleeding at 35 weeks gestation	Local compression, cauterisation; conservative postpartum; lesion regressed spontaneously
Martines F and Immordino V, 2009 [23]	32/ Female	AVM at base of tongue; swelling and mild airway symptoms during second trimester	Conservative; tracheotomy performed during caesarean section
Diep J et al., 2017 [24]	24/ Female	Multifocal AVMs of face, tongue, and larynx; airway involvement	Conservative; caesarean under neuraxial anaesthesia with airway precautions
Azis KA et al., 2022 [14]	35/ Female	Ruptured forehead AVM with prior growth during pregnancy	Angioembolisation followed by surgical excision
Ryan K et al., 2022 [25]	30/ Female	Mandibular AVM rupture with massive oral haemorrhage at 39 weeks gestation	Emergency caesarean section for fetal distress; selective embolisation; postpartum follow-up planned
Utami AM et al., 2023 [11]	24/ Female	AVM of left hand since birth; worsened during pregnancy with MVP and hormone receptor expression	Amputation due to recurrent pain and failed embolisation
Present case (current report)	26/ Female	High-flow AVM of left posterior mandible following dental extraction in pregnancy	Selective embolisation during third trimester; radical resection with free flap microvascular reconstruction postpartum with immediate preoperative embolisation

[Table/Fig-6]: Previous literature review of similar cases [11,13,14,21-25]

emphasised centralised care in high-volume centers, integration of stereotactic radiosurgery, advanced neuroimaging (e.g., 4D-DSA, fMRI, HYPRflow MRI), and the collection of multicenter clinical and genetic data to improve risk stratification and personalise treatment plans [26]. Concurrently, Coulie J et al., (2025) advocated for incorporating genetic profiling (e.g., KRAS and MAP2K1 mutations) into therapeutic decisions, suggesting that targeted inhibitors originally developed for oncology may complement embolisation and resection for difficult or recurrent AVMs [16].

Additionally, nanomedicine has emerged as a transformative tool, offering precise diagnostic contrast agents and nanoparticle-mediated drug or embolic delivery. At the clinical level, Roditis (2024) reported promising outcomes using laser ablation and direct percutaneous embolisation in scalp AVMs, stressing personalised treatment guided by lesion angioarchitecture, hormonal status, and cosmetic outcomes [27]. These approaches, when implemented within a multidisciplinary framework, are significantly improving long-term control and patient quality of life. In the presented case, the integration of preoperative embolisation and radical resection aligns with recent ARISE I consensus recommendations, emphasising timely, image-guided, multidisciplinary management. The use of targeted embolic agents and reconstruction mirrors advances in nanomedicine-enhanced precision therapy. This case exemplifies the evolving shift toward aetiology-based, patient-tailored AVM treatment strategies endorsed by recent literature.

Post-treatment surveillance is critical for detecting recurrent AVMs, particularly in pediatric and high-flow extracranial locations. Contrast-enhanced MRI and MRA are recommended three months postoperatively to establish a baseline, followed by surveillance every 6 to 12 months for at least 2 to 5 years. Digital Subtraction Angiography

(DSA) remains the gold standard when MRI is inconclusive or for small residual nidus detection [28]. The literature on extracranial head and neck AVMs underscores that early total resection reduces the risk of recurrence, and long-term clinical monitoring should focus on signs such as bruit, pulsation, swelling, or bleeding.

CONCLUSION(S)

This case exemplifies the complex interplay of trauma, hormonal influences, and potential congenital predisposition in the pathogenesis and progression of AVMs. The mandibular lesion developed following a second trimester dental extraction and progressed significantly during pregnancy, suggesting that hormonal modulation, particularly VEGF-driven angiogenesis, may have exacerbated a latent vascular anomaly triggered by local trauma. Consistent with literature, such as the findings of Utami AM et al., and Azis KA et al., this supports a "second-hit" model where genetic or congenital susceptibility is unmasked by external stimuli. Diagnostic imaging confirmed a high-flow AVM with extensive cortical destruction, and Doppler ultrasound aided early suspicion. The management-timely antenatal embolisation followed by postpartum radical resection and free fibula flap reconstruction-was aligned with Kohout MP et al.,'s principle of surgical intervention within 48 hours of embolisation to limit revascularisation. The avoidance of proximal ligation, multidisciplinary planning, and postoperative surveillance further strengthened outcomes. Despite an indeterminate aetiology, the patient's course demonstrates that with prompt diagnosis and a multimodal, interdisciplinary strategy, high risk AVMs in pregnancy can be effectively controlled. The four month follow-up showed excellent aesthetic and functional recovery with no recurrence, underscoring the efficacy of integrated embolisation and surgical reconstruction protocols for complex facial AVMs.

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