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Arteriovenous Malformation of Maxilla: A Case Report and Literature Review

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Abstract

This is a rare case report of Arteriovenous malformation (AVM) in jaw which was confused as a periapical cyst in primary investigations and extraction of the painful tooth was done leading to uncontrolled bleeding and a life-threatening condition. Importance of thorough history taking and diagnosis of such cases is highlighted in this case report.

Keywords: Arteriovenous malformation, Periapical cyst, Embolization, Digital Subtraction Angiography

Introduction

AVMs in head and neck regions are types of high or low flow vascular anomalies that consists of a complex system of vessels resulting in a direct connection between feeding arteries and draining veins, forming a nidus.[1] They, are one of the most dangerous subtypes of vascular malformation because of their potentially life-threatening nature.[2] The current gold standard for defining the morphology, nidus, and supportive vasculature of AVM is Digital Subtraction Angiography (DSA) because it provides best spatial and temporal resolution for the study of vascular anatomy and also it is less invasive procedure when compared to conventional angiography and other imaging modalities.[1]

Case Report

Clinical Findings

Patient reported to the Department of Oral Medicine and Radiology with the complaint of a swelling and pus discharge from the left side of her face near the nose. The swelling was present since, 15 days and was associated with mild to moderate, dull aching, intermittent pain that spontaneously aggravated and subsided on taking over the counter analgesics.

On general examination her health state was good. A solitary swelling measuring 3 × 1.5 cm was noted on the left side of nose with obliteration of nasolabial fold. On palpation mild tenderness was elicited and it was firm. Sinus opening was observed with mixed blood and pus exudation during palpation. The swelling was non-pulsatile (Figure-1a).

On intraoral examination solitary diffuse swelling was noted involving left palate extending antero-posteriorly from the mesial surface of 22 to the distal surface of 27 measuring mediolaterally from gingival margin to mid palatal region (raphe) measuring 5×2.5 cm. Overlying mucosa was erythematous, smooth, and glistening. Socket of 26 was unhealed with evidence of pus exudation. Swelling was tender and soft in consistency with slight compressibility on palpation. 22, 23, 24 and 25 teeth showed evidence of RCT attempt. On percussion the above teeth were tender with a dull note (Figure 1b, c).

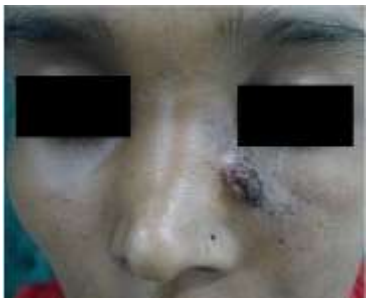


Figure 1 a:

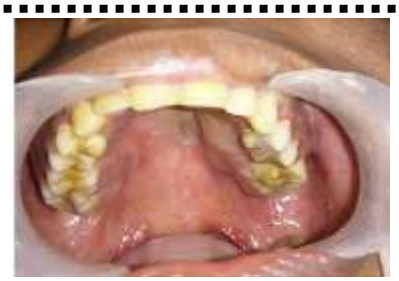


Figure 1 b:

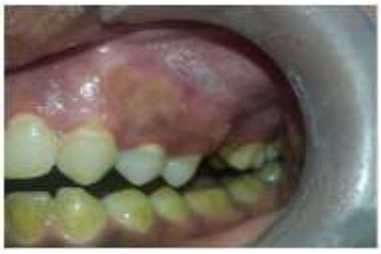


Figure 1 c:

Figure 1 d:

Patient gave history of extraction of upper left back tooth in a private clinic two months ago due to misdiagnosis of periapical cyst in CBCT findings (Figure- 2 a, b, c, d) leading to uncontrolled bleeding.

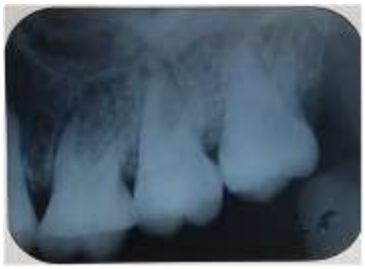
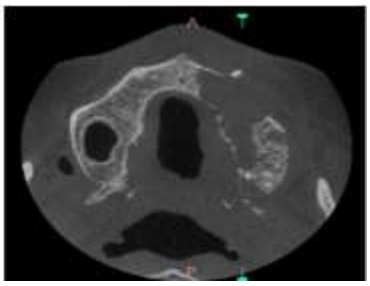


Figure 2 a:



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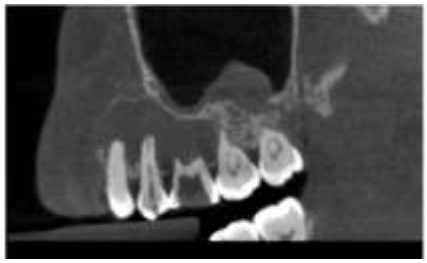

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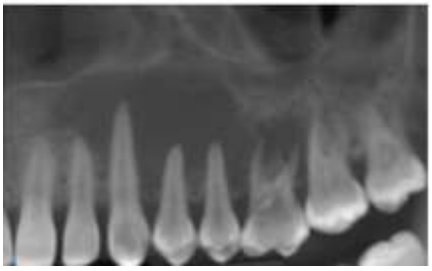

Figure 2 d:

An emergency embolization procedure involving left internal maxillary and facial artery was done under DSA using N- butyl-2-cyanoacrylate in a tertiary health center (Figure- 3a).

Based on presenting history and clinical features a provisional diagnosis of osteomyelitis of left maxilla was made. Chronic suppurative maxillary sinusitis, chronic periapical abscess, mucormycosis and cervicofacial actinomycosis were considered under differential diagnosis.

Intraoral periapical radiograph showed well-defined radiolucent ovoid regions in the occlusal surface of 24, 25 suggestive of access cavity preparation. Ill-defined radiolucency seen from the alveolar crest of 23 24 25 till the floor of maxillary sinus with multiple ovoid well defined radiopacities (Figure- 3b).

OPG showed well defined radiopaque structures as a result of introduction of N-butyl 2 cyanoacrylate used for embolization. Radiopaque masses were located in the periapical region of 24 25 and little above the periapical region of 27 28 and were interconnected (Figure-3 c, d).

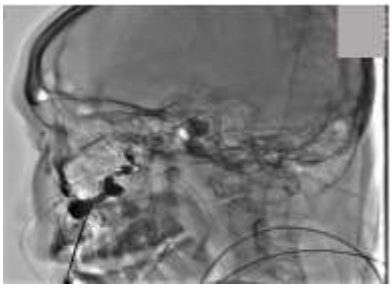

Figure 3 a:

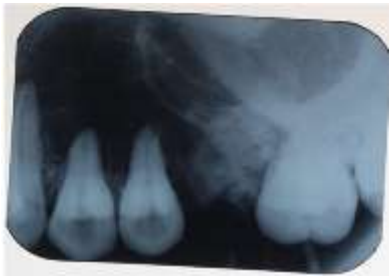

Figure 3 b:

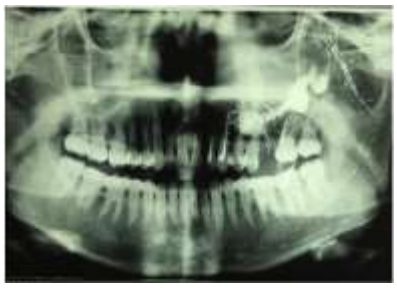

Figure 3 c:

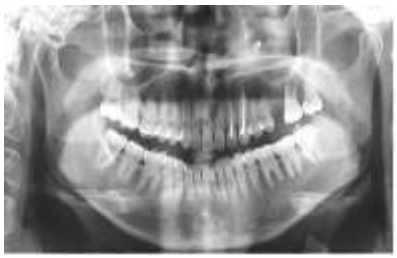

Figure 3 d:

Therapeutic Intervention

Patient was conservatively managed with high dose of parentral antibiotics Imipenem 1 g IV every 8 hours, plus gentamicin 1 mg/kg IV infusion every 8 hours for 7 days. The lesion healed completely after the course of medication (Figure-1 d). The patient later underwent completion of RCT, but refused for prosthetic rehabilitation by crowns and bridges.

Discussion

AVMs are fast-flow vascular lesions composed of dysmorphic arterial and venous vessels that are connected directly to one another without an intervening capillary bed. The most common facial sites of AVMs are the cheeks and ears, as they have higher surface area to volume ratio than other facial structures during early embryonic development.[3]

Mulliken and Glowacki in the year 1982 were the first to propose classification of vascular anomalies based on histology. It was later accepted by the International Society for the Study of Vascular Anomalies (ISSVA) in the year 1992. These vascular anomalies were divided into tumors, such as hemangiomas and vascular malformations.[2]

Majority of cases of AVMs are sporadic, but there are also few inherited syndromes reported whose molecular genetics have been recently elucidated. RASA1 (Ras GTPase-activating protein 1) gene mutation on chromosome 5q, expressing p120-Ras GAP has been identified in families with congenital malformations in arteriovenous malformations.[3]

In a study done by Kohout MP et al of AVM of head and neck, AVM was present at birth in 59% of cases.[4]

AVMs account for (0.5–1%) of all lesions which are found in jaws.[7] AVM may present clinically with mild bleeding from gingiva, swelling, loosening of teeth, paresthesia, and asymmetry of face.[5]

Radiological examination helps in confirming the diagnosis mapping the lesion, showing the possibilities and route of endovascular treatment, identifying associated anomalies and providing surveillance of disease and response to therapy. Ultrasonography with color doppler, Computed tomography angiography (CTA) and magnetic resonance angiography (MRA) are the modalities to detect AVMs.[2]

Radiographic features include parallel radio-opaque striations, altered radiodensity with osteolytic areas and trabecular pattern which are altered. Honeycomb appearance because of osteolytic feature of AVM is a strong radiological finding. Disruption of bony surface can appear with scalloped areas and both unilocular or multilocular appearances as well as ‘sunburst’ effects. It can create a confusion with other pathologies like osteosarcoma, myxomas, fibrous dysplasia or ameloblastoma.[7]

DSA is the gold standard for diagnosing and characterizing AVMs. Classic appearance of an AVM in DSA shows multiple enlarged feeding arteries that rapidly shunts into a nidus and then into dilated draining veins. Delineating the feeding vessels and the nidus can be done by super-selective catheterization.[2]

Multiple options of treatment are available for AVMs, including endovascular or percutaneous embolization, sclerotherapy, and surgical excision. Embolization can be done as a preoperative adjunct either to reduce the blood flow within the nidus or to embolize deeply, situated feeder arteries which are surgically inaccessible.[6]

Conclusion

AVMs presents a diagnostic challenge and they become life threatening in lack of proper diagnosis and management. Before any invasive procedure is done for these types of cases a thorough clinical examination and a detailed investigations should be done to prevent misdiagnosis and any grave consequences because of these vascular lesions.

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