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CASE REPORT

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Arteriovenous malformation after punch biopsy clinically mimicking a basal cell carcinoma: Case report and review of literature

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Key Clinical Message

Arteriovenous malformations (AVM's) of the skin can be acquired post blunt or penetrating trauma. They may clinically mimic basal cell carcinomas and other lesions with overlying telangiectasia. Specific clinical, dermoscopic, and histological clues differentiate these conditions. AVM's may progress to destructive lesions and early surgical intervention is key.

1 | INTRODUCTION

We report on a case of an arteriovenous malformation (AVM) that had its onset after a punch biopsy was performed on the bridge of the nose. The lesion was a clinical mimic of a basal cell carcinoma (BCC) and remains a differential to consider. A 53-year-old lady sustained blunt trauma to the bridge of her nose and presented with a telangiectatic erythematous papule. The initial biopsy in 2011 showed a reactive scar. This lesion did not resolve, increased in size, and caused pain. She represented in 2017, when the lesion mimicked a BCC clinically, but on biopsy, it was diagnosed as an AVM. Surgical excision provided successful management. AVMs are rare but potentially dangerous. They tend to progress to a destructive lesion and will inevitably require surgical intervention. Considering differentials for BCC's remain of clinical importance. AVM's and BCC's may have overlapping clinical features but dermoscopy and histology aid in differentiating these disorders.

Mulliken and Glowacki classified vascular anomalies in 1982 into vascular tumors and vascular malformations.[1] This classification is currently accepted by the International Society for the Study of Vascular Anomalies, they further subdivide AVMs as fast-flowing vascular malformations.[2]

Head and neck AVM are reported to occur in 0.1% of the population, only 8.1% of these occur extracranially and post traumatically acquired lesions are rare.[3] The majority of existing literature focuses mainly on the congenital AVM; approximately 51% of these occur in the head and neck. In contrast, traumatic AVMs are quite rare in the head and neck area and are seen mostly in the extremities.[4]

2 | PATIENT INFORMATION

A 53-year-old female presented in with an erythematous telangiectatic nodule on the bridge of her nose. This lesion first occurred in 2007 when she sustained blunt trauma

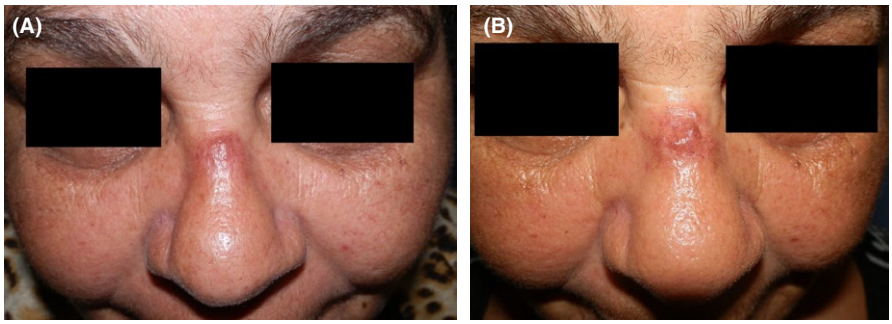


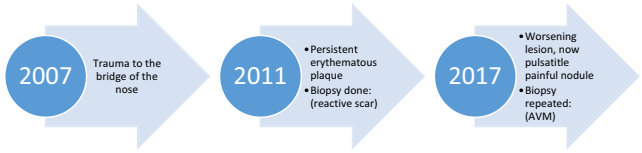
FIGURE 1 A, Erythematous plaque on the bridge of the nose in 2011. B, Erythematous pulsatile plaque with marked telangiectasia in 2017

from a plastic bottle to the bridge of the nose. Then in 2011 (Figure 1A), she presented to the Division of Dermatology where the lesion was biopsied and found to be a reactive scar. She was managed symptomatically and conservatively followed up. Now she presents again, in 2017, concerned that the lesion is increasing in size and became painful over the preceding year (Figure 1B).

3 | CLINICAL FINDINGS

Clinically, there was a tender pulsatile nodule on the bridge of her nose with marked telangiectasia and no surface changes (Figure 1B). The lesion now clinically mimicked a BCC.

4 | TIMELINE



5 | DIAGNOSTIC ASSESSMENT

On first presentation in 2011, a skin biopsy was done that showed mild chronic inflammatory infiltrate in the superficial dermis. There were some dilated capillaries in the superficial dermis, but no discrete thick-walled veins or arteries (Figure 2A). This was diagnosed as a post traumatic reactive scar.

In 2017, after worsening of the symptoms, a re-biopsy was done that showed the lesion to be an AVM (Figures 2B and 3). Sections of the skin punch biopsy showed a well-defined proliferation of small veins and small-to-medium sized arteries within a fibrotic superficial dermis. Dermal solar elastosis was evident. The overlying epidermis showed mild spongiosis and epidermal atrophy.

6 | INTERVENTION AND FOLLOW-UP

The patient was referred to plastic surgery where the lesion was successfully removed surgically.

7 | DISCUSSION

Arteriovenous malformations consist of dysmorphic arterial and venous vessels connected directly to one another without an intervening capillary bed and progress through 4 clinical stages according to the Schobinger clinical classification.[1] They start as erythematous plaques or macules (stage 1, dormant stage), then progress to the other stages. This progression is usually precipitated by trauma, pregnancy, or puberty. Progression to stage 2 is marked by expansion of the lesion. In stage 3, destruction of the lesion or the underlying structures occurs. The final stage 4 is associated with cardiac decompensation due to high output cardiac failure.[1,2,5-7] Traumatic AVMs are uncommon and occur in the setting of penetrating, blunt or postsurgical trauma. It appears that after receiving her first biopsy, the lesion progressed through stage 1 and 2.

In this case, the lesion mimicked a BCC, the most common malignant neoplasm of the skin.[8] Differentiating an AVM from a BCC is important as they require different interventions and if left untreated they can lead to destruction. Feinmesser et al[9] described these two disorders occurring concurrently where BCC's develop on top of an underlying AVM, distinguishing these disorders based on clinical, dermoscopic, and histology remains of importance.

Clinically, BCC's and AVM's may appear similar. Our patient's stage 2 AVM appeared to be a pearly nodule with overlying telangiectasia, a very similar presentation to a nodular BCC. At stage 1, AVM appears as an erythematous macule whereas a superficial BCC will appear as an erythematous patch that might have overlying scale. There may be clinical overlap between a stage 3 AVM and an ulcerated BCC where both these lesions will have ulcerated centers with rolled edges. An unique clue to an AVM is the palpable thrill or audible bruit caused by the fast-flowing blood. Doppler ultrasound will also highlight the fast-flowing vascular malformation. AVM's although predominantly occurring on extremities, are reported to also occur in the head and neck region. BCC's also present on the head and neck due to sun exposure in this region.[8] Other clinical diagnoses that manifest with superficial telangiectasia includes rosacea, primary telangiectasia, secondary telangiectasia due to sun damage, hormonal imbalances, and systemic

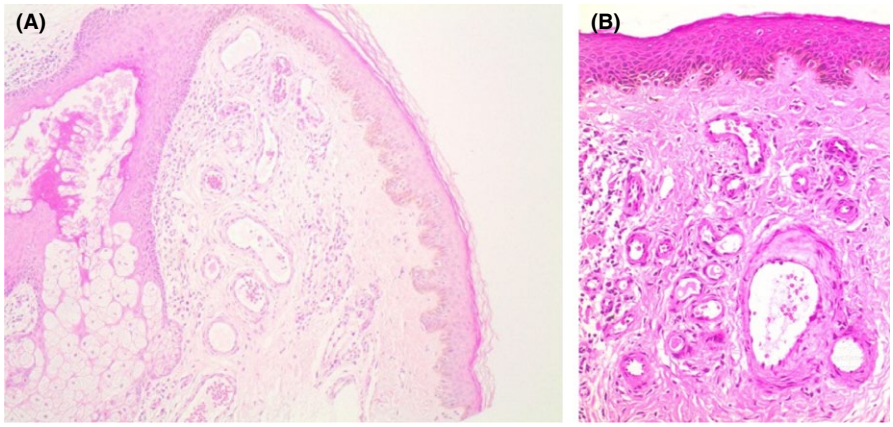


FIGURE 2 A, Haematoxylin and eosin (H&E) stain (2011)—20× objective magnification. Dilated capillaries in the superficial dermis, but no discrete thick-walled veins or arteries. B, Haematoxylin and eosin (H&E) stain (2017)—100× objective magnification. Showing the blood vessels to be arteries and small veins

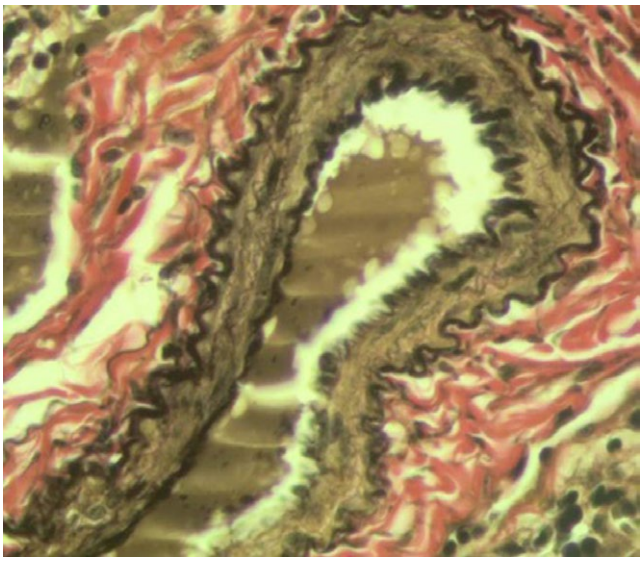


FIGURE 3 Verhoef elastic von Gieson (2017)—400× objective magnification. Highlighting the elastic lamina of the arteries

conditions (eg, angiolupoid sarcoid and autoimmune connective tissue diseases). Although these differentials often have other clinical clues distinguishing them from AVM's and BCC's.

Dermoscopically AVMs are not well described in the literature. One case report describes blue-red lacunae, milky red areas, red ovoid areas, winding red vessels of several thicknesses, and some dotted vessels.[10] Whereas BCC's are well described in the literature as arborizing vessels (94.1% positive predictive value) with shiny red-white structureless areas.[11,12] Although differentials exist, identifying these vascular patterns will aid in the diagnosis.

Histologically, these two entities are distinct. BCC's manifest as a proliferation of atypical basaloid cells with hyperchromatic ovoid nuclei and minimal cytoplasm, often displaying peripheral nuclear palisading, and peritumoral clefting. Other tumors that also present with a basaloid collection includes follicular tumors, sebaceous tumors, sweat gland tumors, and seborrheic keratosis.[13] AVM's shows proliferations of thick and thin-walled vessels that include arteries and veins, elastin stains may aid in highlighting the elastic lamina in arteries. Differential diagnoses for AVM's include other vascular malformations and vasoformative tumors.[5]

In conclusion, there is a paucity of data on adult-onset AVMs; as well as post traumatic AVMs, these are limited to case reports and series. A punch biopsy might have led to progression of the lesion, and to our best knowledge, this has not been described. This lesion also clinically mimicked a BCC and remains an important differential to exclude. AVM's and BCC's manifests with clinical overlap but can be distinguished by dermoscopy and histological assessment. An early accurate diagnosis of AVM's is important since patients receiving surgical intervention sooner will have a better outcome.[5,14]

CONFLICT OF INTEREST

None declared.

AUTHORSHIP

LW: managed the case and compiled the manuscript. RN: co-authored manuscript also assisted with editing and corrections. TI: assisted with editing and corrections of manuscript. RR: assisted with histological interpretation and editing.

INFORMED CONSENT

The patient kindly agreed on the use of her clinical information, photographs, and histology slides. She supports academic learning and development.

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