

Angiomatous-Angiectatic Nasal Polyps: A Varied Clinical, Radiological and Histomorphological Spectrum.

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ABSTRACT

Background: Angiomatous nasal polyp (ANP) is a rare interesting clinical entity cause diagnostic dilemma by presenting either gradually progressive or sometimes in acute fashion with sign and symptoms suggestive of malignancy. **Objective:** To study the varied clinical symptoms, growth pattern and spectrum of radiological, histomorphological features. **Methods:** Here we studied 10 cases diagnosed with this entity and characterized the histomorphological features. We have also discussed clinical presentations and radiological features of this disease as described in the literature. **Results:** ANP have a prominent component of dilated capillary-type blood vessels with intraluminal thrombosis- infarction with compromise of their blood supply and extensive accumulation of extravascular stromal, perivascular pools of eosinophilic, congo red -negative pseudoamyloid- like material. **Conclusion:** Angiomatous nasal polyp is although benign does at times mimics malignancy causing radiologically evident surrounding bone erosion / destruction. Preoperative radiological diagnosis helpful in avoiding extensive surgery but final diagnosis is usually made by histopathological examination.

Keywords: Angiomatous, Angiectatic polyp, Nose, Paranasal sinus, Histomorphology, Clinico- radiological features.

INTRODUCTION

Inflammatory sinonasal polyps are histomorphologically divided into five main groups based on prominent stromal component: oedematous, glandular, fibrous, cystic and angiomatous / angiectatic (ANP). Since ANP is fragile, is difficult to remove en block hence has a number of alternative names within the literature. It is also known as nasal polyp with haemorrhage and necrosis, sinonasal organized haematoma, inflammatory granuloma telangiectaticum, vascular granuloma, pseudo-angioma etc. reflecting a large variation in pathologic description. ANPs have a prominent component of dilated capillary-type blood vessels, as compared to non-angiomatous polyp which have decreased density of blood vessels than normal mucosa.^[1] Intraluminal thrombosis is rarely widespread as compared to deposition of eosinophilic hyaline pseudoamyloid – like material, but necrosis leads to infarction of the polyp.^[2,3]

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MATERIALS AND METHODS

This is a retrospective study of 10 cases of angiomatous polyp who presented in ENT clinics and were treated surgically after proper work up. Out of total 10 cases eight were male and two were female. Male to female ratio was 4:1. Most common complaint was nasal obstruction followed by nasal bleeding. It was interesting to note that mostly left side of nose was involved. CT scan also showed unilateral sinus involvement. No obvious cause could be detected for the above predilection. In two cases there was bony erosion so preoperative diagnosis of malignancy was made. Preoperative biopsy helps us in deciding the protocol management in doubtful cases. Polyp was removed in toto in every case and there was no recurrence. All the details of the cases were tabulated as shown in table no.1.

Final diagnosis was made after histopathological examination based on some special stains (Periodic Acid Schiff, Congo red, Masson's Trichrome, Reticulin). In our cases the predominant histomorphological features of ANPs are multiple proliferated dilated / ectatic blood vessels [Figure 1-3] with deposition of perivascular and extravascular abundant fibrin-like eosinophilic congo red negative pseudoamyloid material deposited in either

lamellated [Figure 1], nodular pattern [Figure 4] or diffusely in the stroma [Figure 2,3] and superimposed fibrinoid necrosis, luminal thrombosis

undergoing fibrinoid change in the ectatic blood vessels. [Figure 2,3,5]

Table 1: Showing clinical, radiological and pathological details of patients included in the study.

S. no.	Age/sex	Complaints.	Diagnostic Nasal endoscopy.	CT Findings.	Clinical Diagnosis.	Surgery.	Histopathology Diagnosis.
1	48/M	Nasal obstruction, Nasal discharge, headache.	Fragile mass in left middle meatal area with mucopus, bleed on touch.	Left maxillary sinus with extension to nasopharynx.	Antrochoanal polyp left with DNS.	Functional endoscopic sinus surgery(FESS).	Angiomatous polyp.
2	45/M	Growth left nose with external deformity, left sided hard palate swelling.	Rt. sided DNS and polypoidal growth nasal cavity left side.	Left sided maxillary, ethmoid, frontal and sphenoid sinus involved.	Benign nasal tumour.	Excision of mass under GA.	Angiomatous polyp.
3	18/M	Bilateral nasal obstruction, occasional nasal bleeding.	Mass in left middle meatus.	Left maxillary and ethmoid sinus. involved	Inflammatory nasal polyp.	FESS	Angiomatous polyp.
	27/M	Bleeding left nose for 2-3 months.	Left nasal cavity filled with polypoidal mass.	Left side maxillary involvement with nasopharynx clear.	Vascular nasal mass.	FESS.	Angiomatous polyp.
5.	18/M	Nasal obstruction (Left).	Left nasal cavity filled with polypoidal mass.	Left nasal cavity, maxillary sinus.	Inflammatory antrochoanal polyp.	FESS.	Angiomatous polyp.
6	20/F	Nasal obstruction, (left) external deformity.	Left nasal cavity filled with polypoidal mass causing bony destruction resulting in external Deformity.	Left sided maxillary, ethmoid, frontal and sphenoid sinus involved.	Nasal mass? Malignancy.	Excision.	Angiomatous polyp.
7	34/M	Nasal obstruction (Left), occasional blood stained discharge.	Dirty white polypoidal mass occupying Lt. nasal cavity.	Left sided maxillary, ethmoid sinus involvement with bony remodeling.	Benign vascular lesion/ Inflammatory nasal polyp.	FESS	Angiomatous polyp.
8	60/M	Nasal obstruction (Rt.), facial numbness, cheek deformity (Rt.).	Polypoidal mass occupying Rt. nasal cavity with bony breach in anterior wall of maxilla.	Expansion of Rt. sinus with erosion of roof and anterior wall widening of osteomeatal complex on Rt. side.	Sinonasal malignancy.	Biopsy-polypoidal mass; FESS.	Angiomatous polyp.
9	25/M	Nasal bleed Lt. side.	Polypoidal mass occupying Lt. nasal cavity, bleeding on manipulation.	Left side maxillary involvement with nasopharynx clear.	Nasal malignancy.	Removal under GA.	Angiomatous polyp.
10.	18/M	Nasal obstruction (Left), occasional bleed.	Polypoidal mass occupying Lt. nasal cavity, bleeding on manipulation. H/O previous surgery.	Left side maxillary sinus involvement.	Vascular tumour.	Excision under GA.	Angiomatous polyp.

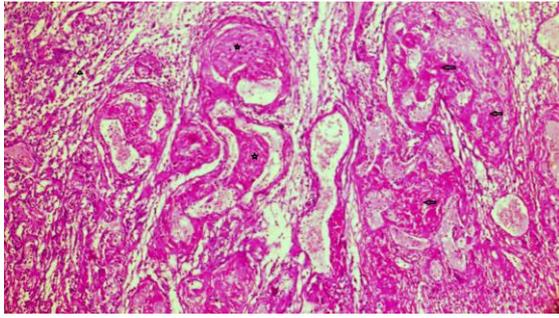


Figure 1: Microphotograph shows edematous stroma (star) containing multiple thin walled ectatic blood vessels with perivascular lamellated (arrow) fibrinoid material deposition. (H & E X 100)

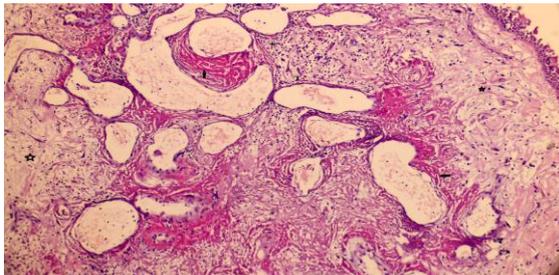


Figure 2: PAS stain highlights multiple ectatic blood vessels with extravascular fibrinoid PAS positive somewhat lamellated pseudoamyloid-like material deposition in the stroma (arrow) and intraluminal fibrin thrombi (star) along with mild chronic inflammatory cell infiltrate (triangle). (PAS X100)

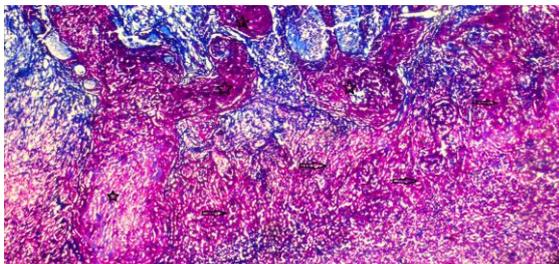


Figure 3: Masson's Trichrome stain highlights intraluminal lamellated fibrin thrombi (star) in ectatic blood vessels with diffusely deposited extravascular pseudoamyloid-like fibrinoid material (arrow) in the stroma. (MT X 100)

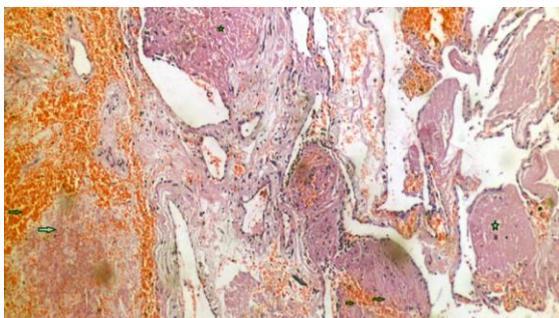


Figure 4: Shows extravascular hemorrhage with transformation into fibrinoid material in the stroma (arrow), dilated blood vessels with nodular fibrinous-pseudoamyloid material (star) in the stroma. (H&E X 100)

In other cases are extensive extravasation of blood components (platelets & fibrin) - hemorrhage,

thrombosis undergoing fibrinoid change [Figure 4] and diffuse deposition of eosinophilic congo red negative pseudoamyloid material [Figure 6a, 6b].

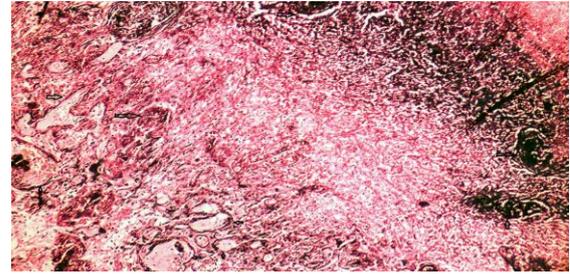


Figure 5: Microphotograph show well delineated ectatic / dilated thin walled blood vessels (arrow) with intraluminal thrombi (arrow). [Reticulin stain X 200]

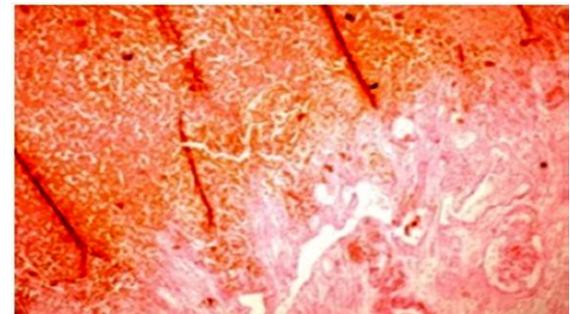


Figure 6a:

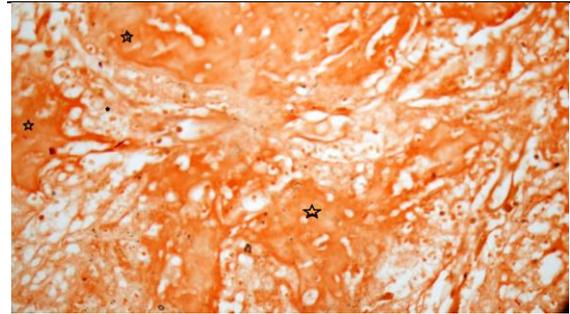


Figure 6b: 6a, b- Microphotograph shows ectatic blood vessels (5a) and diffusely deposited extravascular Congo red negative pseudoamyloid-like material (star) in the stroma on polarizing microscopy. (Congo red X 100)

Scattered sparse to mild chronic inflammatory cell infiltrate also seen along with few scattered atypical pleomorphic spindle cells (Myofibroblasts) in the stroma- a pseudosarcomatous change [Figure 7].

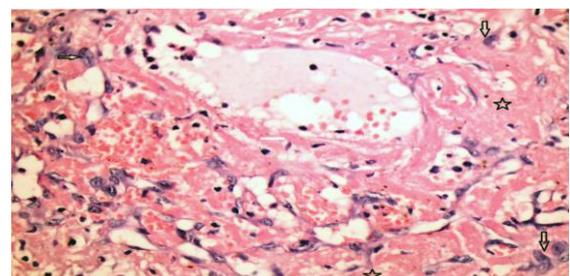


Figure 7: Microphotograph shows scattered fibroblast-like atypical spindle stromal cells (arrow) and extravascular pseudoamyloid-like material (star) along with sparse inflammatory cells. (H&E X 400)

RESULTS & DISCUSSION

ANPs are reported to be a derivative of antrochoanal polyps commonly, but may be a variant of sinonasal polyps of any location. The true pathogenesis of ANPs is still not clear, although many hypotheses have been proposed. One is that inflammation and/or allergy of the maxillary sinus and nasal cavity causing proliferation, dilation, rupture, and hemorrhage of mucosal blood vessels, leading to mucosal edema and polypoid changes.^[1] Most probable theory hypothesized by Batsakis JG and Sneige N is that the polyp pedicle is vulnerable to compression and vascular compromise at the following extra antral sites: the ostial exit site, the posterior end of inferior turbinate, the posterior choana and at the most dependent part within the nasopharynx,^[2,3] leading to initial vascular dilatation / ectasia, extravascular edema. Resulting hemodynamic condition predisposes to extensive extravasation of blood components (platelets & fibrin)- hemorrhage, accumulation of large perivascular pools of eosinophilic, congo red - negative pseudoamyloid- like material and possibly infarction followed by reactive and reparative changes with neo-vascularisation,^[1,2,4] setting the stage for continuing development of polyp- repeated vascular occlusion and further infarction. Clinically, to start with ANPs present as soft, gelatinous translucent polypoidal, painless mass with nasal discharge often resulting in gradual obstruction of the nasal cavity, loss of smell sensation and recurrent epistaxis. Most ANPs arise in maxillary sinus and extend towards the choana and into the nasopharynx.^[2,5] there may be gradual enlargement of the lesion or fast growth which may display aggressive clinical behavior causing widespread erosion of the adjacent bony structures resulting in cheek swelling, exophthalmos etc. thereby simulating malignancy.^[6]

Li-Bo Dai et al studied 31 ANPs cases and observed that 24 masses out of 31 ANPs were located in the maxillary sinus (based on CT findings) and 21 involved the ipsilateral nasal cavity.^[7] Of these 21 patients, two extended into the orbit, 16 involved the ipsilateral ethmoid sinus and three cases involved the choana extending in to contra lateral nasal cavity. None of the cases showed invasion into the sphenoid sinus. All of these lesions caused changes in the adjacent bone, including expansile remodeling, hyperemic demineralization / resorption, frank osseous erosion, and hyperostosis of the maxillary walls. The most common site of maxillary wall erosion was the medial wall (21/31) followed by the posterior lateral wall (3/31), upper wall (2/31). Bony sclerosis was most evident along the posterior lateral wall (20/31), followed by the upper wall (5/31). Jing Zou studied 15 ANPs using combination of CT and MRI and concluded that most lesions caused changes in the adjacent bone,^[8] including expansile

remodeling (n = 8), defect or destruction (n = 7), and hyperostosis (n = 6).

Predominant histomorphological features of ANPs are clusters of ectatic blood vessels to begin with, surrounded by abundant fibrin-like eosinophilic pseudoamyloid material both lamellated and diffusely deposited extravascularly and superimposed fibrinoid necrosis. Later on there may be extravascular hemorrhage, luminal thrombosis in the ectatic blood vessels which may undergo fibrinoid change. There may be fibrinoid material deposition in the extravascular stroma.^[1-3,6] Scattered atypical pleomorphic spindle cells (Myofibroblasts) in the stroma are a part of reactive secondary changes, seen occasionally in sinonasal polyp, but are quite common in angiectatic polyps- a pseudosarcomatous change. However, other vascularised fibromatous polyps do not show deposition of pseudoamyloid- like eosinophilic extracellular material, superimposed fibrinoid thrombosis and necrosis of blood vessels wall, or pseudosarcomatous stroma.^[1,5]

Histologically and radiologically the differential diagnosis includes juvenile angiofibroma, hemangioma and inverted papilloma. Angiofibroma occur almost exclusively young adolescent males, thus ANP should always be considered in elderly or female patients presenting with similar features. The hallmark feature differentiating ANPs from a JNA is lack of PPF involvement despite nasopharyngeal extension. Angiography may be used for early diagnosis and to differentiate ANP from juvenile angiofibroma.^[3] ANPs show hypovascular or avascular appearance on angiography due to their irregular racemose arrangements of dilated capillary-type vessels, in contrast to normal arborizing pattern of hyper vascularity associated with an angiofibroma.^[9,10] Haemangiomas can usually be distinguished from ANPs as they classically arise from the nasal septum or vestibule, and are of capillary type while few arise from the lateral wall of the nose, and are usually cavernous. Hemangiomas are even rarer in paranasal sinuses.^[11] On CT scanning they show greater contrast enhancement on contrast CT than ANPs. Inverted papillomas are in the paranasal sinuses, difficult to distinguish from ANP on clinical and radiological grounds. On CT, an inverted papilloma is homogeneous; it has a density like that of soft tissue and heterogeneous enhancement after injection of contrast material.^[12] However, the histological features of ANPs often show more prominent vascular changes, which when present should alert further histological and radiological examination, enabling an accurate diagnosis to be made. CT is the gold standard for diagnosing angiomatous antrochoanal Polyps, which radiographically, are very similar to non-angiomatous antrochoanal polyps. In general ANP are a minimally enhancing lesion and originate in the maxillary sinus, extend through the maxillary

ostium, and continue posteriorly toward the choanae.^[13] On CT, the mass shows heterogeneous density: the inflammatory, necrotic, and cystic tissues are responsible for the low density of the mass while high density of the mass is due to hemorrhagic lesions. The central part of the polyp shows no enhancement while the peripheral area of the mass has inflammation and vascular proliferation, leading to enhancement.^[9] MRI is now the investigation of choice for soft-tissue lesions of the nasal cavity, including ANPs.^[9,13] Magnetic resonance imaging can also demonstrate certain characteristic features including hypointensity on T1 weighted images and areas of mixed signal intensity with peripheral hypointense rim on T2 weighted images. A strong nodular and patchy enhancement can be seen in postcontrast MRI images.^[3,9,13] Moreover, progressive enhancement on DCE MRI is very important diagnostic clue.^[13] Areas of mixed signal intensity on T2 weighted images are supposed to be caused by the extensive areas of organized thrombus and necrosis in that part of polyp and the peripheral hypointense rim on T2 weighted images due to old microhemorrhage with hemosiderin deposition on the surface of the polyp.^[3,14] Post contrast strong enhancement of nasochoanal portion of ANP suggest extensive vascular proliferation and ectasis.^[3,7]

Transnasal endoscopic surgical excision is the treatment of choice without concern for significant blood loss and ANPs rarely recur.^[1,10]

CONCLUSION

ANPs do not invade peripheral soft tissue but can cause significant benign bony changes hence for establishing an early definitive diagnosis and guiding management awareness of different clinical presentations & distinct histomorphological features is important.

Limitations of the Present Study

Our findings lacked MRI information as it is costlier than CT. Although MRI is now the investigation of choice for soft-tissue lesions of the nasal cavity CT may aid the diagnosis of ANPS.

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