



INFARCTED ANGIECTATIC SINONASAL POLYP MASAQUERADING MALIGNANCY

Pathology

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ABSTRACT

A 44 years female presented with nasal obstruction, headache, left nostril bleeding since 8 months. History of difficulty in breathing was present. Clinically a polypoidal mass seen in left nostril at the level of middle turbinate arising from middle meatus. Mass was friable posteriorly and was bleeding on touch. CECT showed a large lobulated soft tissue mass of size 4.7 x 3.6 cm in left maxillary sinus with significant deviation and erosion of nasal septum involving turbinates and extending in ethmoid sinus, destructing maxillary sinus wall along with mucosal thickening of all sinuses. After necessary base line investigation a biopsy was taken. Biopsy revealed only necrotic material. There after two successive Biopsies also showed the only necrotic material. Owing to inconclusive biopsy report. Complete excision of mass through Caldwell Lucs approach was done and specimen sent for histopathological examination. Specimen was sent in toto and total 61 blocks were prepared. None of the slides from 61 blocks showed evidence of malignancy. Final diagnosis was offered as Infarcted angiectatic inflammatory sinonasal polyp.

KEYWORDS

Angiectatic, Sinonasal Polyp, Inflammatory

INTRODUCTION

Inflammatory Sinonasal polyps are choanal polyps that originate from the nasal cavity, paranasal sinuses and may extend to the nasopharynx. On the basis of histopathology sinonasal polyps are classified into five types viz glandular, fibrous, edematous, cystic and angiectatic or angiomatous polyps. The sinonasal angiomatous polyp is a benign, pseudoneoplastic lesion. Angiectatic polyps account for only 4-5% of inflammatory nasal polyps¹. Infarcted angiectatic polyp presents in myriad ways and often simulate malignancy. Initial biopsy may yield necrotic material. Hence a detailed histomorphological study of a viable piece of biopsy material from repeat biopsy is crucial for definitive diagnosis.²

CASE REPORT

A 44 years female, presented with complaints of Nasal obstruction, headache and bleeding through the left nostril since 8 months. She was also experiencing difficulty in breathing and blood stained sticky discharge through the left nostril. She got medications for it at her native place. There was no history of cold, sneezing, fever, anosmia, weight loss or addiction. On clinical examination deviated nasal septum towards the right was noticed. A soft polypoidal mass was found at the level of middle turbinate arising from middle meatus with left inferior turbinate hypertrophy. Mass was friable and was bleeding on touch. CBC, renal function tests, liver function tests, coagulation profile viz PT, aPTT, INR were within normal limits.

CT Scan revealed a large lobulated soft tissue mass measuring 4.7 x 3.6 cm in left maxillary sinus with significant deviation and erosion of nasal septum. Mass was involving turbinates and extending in ethmoid sinus, destructing maxillary sinus wall suggestive of neoplastic etiology. Along with this mucosal thickening of all sinuses were seen.

Nasal endoscopy and biopsy were done which showed only non-viable necrotic tissue along with polypoidal mucosa and dense chronic inflammation. Repeat biopsy was advised which also showed nonviable, necrotic tissue along with blood and unremarkable mucosa. Due to strong clinical and radiological suspicion of malignancy coupled with necrotic tissue seen on both the biopsies, the biopsy of the bigger viable chunk of tissue by Caldwell Lucs approach was contemplated. The biopsy revealed abundant necrotic material, acute and chronic inflammatory cells. No malignancy, atypia or dysplasia was seen. During all the three biopsy procedures lesion bled copiously. Later hemostasis was achieved. In view of a Clinicopathological

discrepancy, the case was discussed with treating surgeon and excision of complete mass through Caldwell Luc approach was planned.

Intraoperatively anterior wall of the maxillary sinus and whole cavity showed polypoidal mass along with friable necrotic mass which was bleeding on touch. Total excision of the mass was performed. In Histopathology section received multiple polypoidal grey-white tissue bits along with few bony fragments and blood clots all together measuring 10.5 x 7 x 2 cm. Few tissue bits had a glistening appearance. As malignancy was not seen in any of the sections of consecutive three biopsies, complete tissue was exhausted for block making and total 61 blocks were prepared. Microscopically multiple sections studied showed a polyp covered by respiratory epithelium with ulcerations at places, chronic inflammatory cells and granulation tissue on the surface. The superficial portion of core showed numerous thin-walled irregular ectatic vessels in aggregates lined by flattened epithelium, devoid of the muscular layer. The main core of polyp showed necrosis, infarction, haemorrhages, oedema and fibrin deposition. Focal aggregates of acute inflammatory cells and luminal thrombosis of vessels were also seen. Multiple necrotic bony fragments with surrounding fibrous tissue were seen in few areas. There was no evidence of malignancy, dysplasia, atypia or fungus in any of the multiple serial sections of 61 blocks prepared from totally submitted tissue. All morphological features were in favour of inflammatory pathology with massive infarction. Finally, a diagnosis of infarcted angiectatic inflammatory sinonasal polyp was signed out.



Figure 1 showing swelling over left side of face

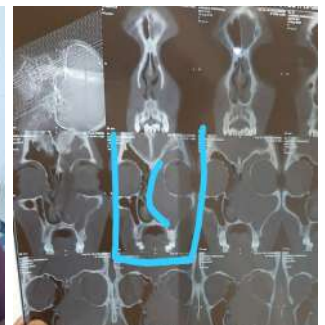


Figure 2 coronal bone window showing deviation of nasal septum

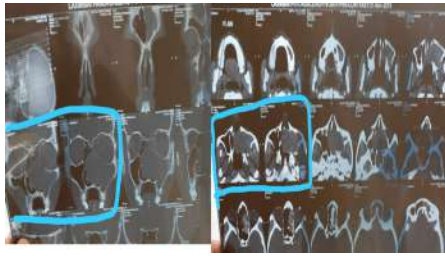


Figure 3 Heterogeneously enhancing mass in left maxillary sinus with thinning of walls



Figure 4 Intraoperative image showing polypoidal mass in left maxillary sinus .



Figure 5 Intraoperative image after total excision of polypoidal mass from maxillary sinus.



Figure 6 Multiple polypoidal grey white masses with blood clots.

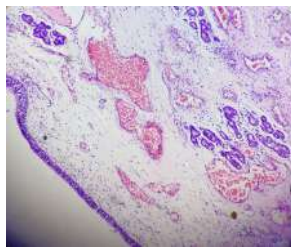


Figure 7 Lining respiratory epithelium. superficial core shows many thin walled ectatic vessels.

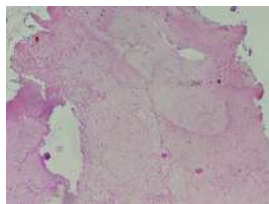


Figure 8 Areas of infarction

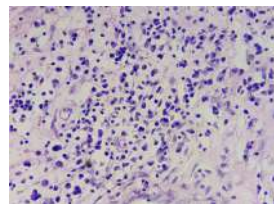


Figure 9 Aggregates of chronic inflammatory cells showing plasma cells..

DISCUSSION:

Inflammatory sinonasal polyp is a day to day pathology encountered by ENT surgeons and hence by pathologists. They are histologically divided into five main types, viz oedematous, glandular, fibrous, cystic and angioectatic or angiomatous. Angiectatic nasal polyps (ANP) are very rare and account only for 4 to 5% of all nasal polyps.^{2,3} Various terms have been used for angiectatic polyps in literature, but the present study preferred to use the same. The name angiectatic correlates with the cardinal component of vessels seen on microscopy. Yuan Yun Tam et al in his study of 13 cases of sinonasal angiomatous polyps found few unique features common to all cases. They found that angiectatic polyp in all patients was unilaterally originating in the

maxillary sinus, involving ostiomeatal complex having extension in the nasal cavity with or without the involvement of posterior choana and nasopharynx. Polyps in all patients were heterogeneous on imaging studies having sinus expansion, bony destruction, and bone remodeling.⁴ In the present case, the polyp was originating from the anterior wall of the maxillary sinus, with complete filling of the sinus and extension in the nasal cavity. The polyp also showed the friable, necrotic component. Literatures have described the hypothesis of ANP which has gained acceptance. ANP is derived from antrochoanal polyp, originates from maxillary sinus and descends in the nasal cavity through medial meatus. If the polyp grows, it may extend posteriorly to choana and sometimes nasopharynx. Rapidly growing Causes bony erosion.^{2,4,5,6} The growing polyp is susceptible to vascular compromise at many sites of anatomically narrowed structures. Compression of vessels may occur at the meatus, posterior choana, posterior end of inferior turbinate or nasopharynx.^{2,4,5,7} Pedicle of the polyp may undergo twist or torsion. The compression cause ischemia, infarction, necrosis, vascular dilatation, oedema, fibrin deposition, and thrombosis of vessels. This eventually leads to granulation tissue formation and fibrosis. Present study is in agreement with the hypothesis. However, the authors of the present study believe that the quantum of the infarct depends on severity, quickness, and duration of vascular compression. Sudden and severe compression results in massive infarction of the polyp. In the present case, a major chunk of polyp core showed infarction. Only the surface of the polyp was preserved which showed respiratory lining epithelium along with dense chronic inflammation and granulation tissue. Superficial core of polyp showed ectatic vessels, fibrinous material, and hemorrhage. Reactive atypical stromal cells are common in ANP, but the present case didn't find it in any of the serial sections prepared from 61 blocks.

Owing to bony erosion and necrosis seen in biopsies done on three occasions and the fact of heavy bleeding from the site during biopsy procedure clinically suggested the strong possibility of malignancy. But malignancy, cellular atypia, dysplasia was not seen in any of the sections. The copious bleeding during biopsy procedure was due to the surface granulation tissue and ectatic thin-walled vessels in the superficial core of polyp which Bled on trivial trauma.

Capillary hemangioma, cavernous hemangiomas, and angiofibroma are the closest differential diagnoses of ANP. But they were ruled out on histology and clinical features. The main concern in the present study was an intentional search for malignancy to avoid rigorous and disfiguring surgery.

The important microscopic features of ANP are 1) Polyp with a covering of respiratory epithelium. 2) The core having thin walled irregularly shaped ectatic vessels devoid of the muscular layer. 3) Dense chronic inflammation with superimposed acute inflammation. 4) Hemorrhage, edema, necrosis, infarction, thrombosed vessels, and fibrin deposition if a vascular compromise has ensued. 4) Granulation tissue, variable degree of stromal cell proliferation with the presence of atypical cells. Atypical stromal cells may simulate malignancy.

The present case showed all the microscopic features. However stromal cell hyperplasia was not seen in the studied case. It can be hypothesized that massive infarction did not give the required time for healing, fibrosis and stromal cell proliferation.

The literature review suggests that CT findings Are not specific for ANP. It shows an expansile soft tissue mass, sometimes heterogeneous, arising and packing the sinus. Growing polyps cause erosion, destruction, and remodeling of adjoining bony margins. This also causes deviation of structures to the opposite side. Hemorrhage or oedema causes hypodense shadows. These findings are also seen in angiofibroma, inverted papilloma, transitional cell carcinoma, and other well-differentiated malignancies. The present case had similar findings on CT scan.^{8,9}

CONCLUSION:

Angiectatic sinonasal inflammatory polyp is rare variant of an inflammatory polyps. Infarcted polyps clinically and radiologically simulate malignancy and even remain inconclusive on initial biopsy. Preoperative incisional biopsy from viable portions or intraoperative biopsy for frozen section will be helpful in managing

such patients. will be helpful in managing such patients.

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