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Sinonasal Tract Angiofibroma – Revisited

Jay Sindino N. Valera, MD[1]
Jose M. Carnate, Jr, MD[2]

[1]*Department of Laboratories*
Philippine General Hospital
University of the Philippines Manila

[2]*Department of Pathology, College of Medicine*
University of the Philippines Manila

A 16-year-old boy presented with a two-year history of nasal obstruction. Further work-up revealed a right nasopharyngeal mass. The mass was excised through transoral approach revealing a tan, nodular, rubbery, irregularly shaped mass measuring 8.2 x 6.6 x 3.2 cm. Cut sections of the mass show cream white nodular surfaces interspersed with tan fibrous tissues.

Microscopically, the mass consists of variably sized vascular spaces interspersed within a collagenized fibrous stroma with fibroblasts. Some of the vascular spaces are slit-like, while others are dilated or branching. On higher magnification, the spindle to stellate cells interspersed within the stroma have bland oval nuclei. No mitoses or necrosis are seen. *(Figure 1)* Immunohistochemistry studies for Smooth Muscle Actin, beta-catenin, S100, and Androgen Receptor were subsequently performed. The stromal fibroblasts show strong diffuse nuclear staining with beta-catenin and androgen receptors, while SMA and S100 are both negative. *(Figure 2)* Given the histomorphology features and the immunohistochemistry results, the case was signed out as sinonasal tract angiofibroma.

Sinonasal tract angiofibroma is a benign mesenchymal tumor with a tendency to be locally aggressive. The tumor commonly affects adolescent males and typically arises in the posterolateral roof of the nasal cavity or the lateral wall of the nasopharynx. Patients usually present with progressive nasal obstruction, a visible nasal mass, and recurrent episodes of epistaxis. Computed tomography and magnetic resonance imaging are the imaging modalities of choice for evaluation. A characteristic radiologic feature is the Holman–Miller sign which refers to anterior bowing of the posterior wall of the maxillary sinus caused by tumor expansion. Grossly, angiofibromas often appear as firm, polypoid to lobulated masses.[1-3]

For the pathogenesis, androgens may play an important role for this neoplasm due to the expression of androgen receptors in the tumor. The involvement of androgens in this tumor may explain the strong predilection for adolescent males. In the majority of cases of sinonasal tract angiofibroma, mutations in *CTTNB1* gene are observed.[1] Aberrance in *WNT* signaling is involved in cases of sinonasal angiofibroma that are occasionally linked in patients with Familial Adenomatous Polyposis.[1,3,4]

The microscopic features of sinonasal tract angiofibroma consist of a loose to fibrous stroma with vascular spaces of different sizes. The bland fibroblasts within the stroma are usually stellate or bipolar in morphology. Occasional multinucleated giant cells may be seen in the stroma.[1,3] For immunohistochemistry, the tumor shows positivity for Androgen Receptor and B-catenin.[1]

The morphologic differential diagnosis for sinonasal tract angiofibroma includes hemangioma, inflammatory sinonasal polyp and solitary fibrous tumor. Hemangiomas are distinguished by their relatively uniform vessel caliber and less prominent stromal components. Solitary fibrous tumors typically exhibit characteristic staghorn-shaped vessels which are not

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Correspondence: Prof. Dr. Jose M. Carnate, Jr.
Department of Pathology
College of Medicine
University of the Philippines Manila
547 Pedro Gil St., Ermita, Manila 1000
Philippines
Phone: +63 2 8404 7808
Email: jmcarnate@up.edu.ph

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a feature of angiofibroma. Inflammatory sinonasal polyps usually show prominent inflammatory infiltrates and lack the marked vascular proliferation seen in angiofibroma. Lastly, sinonasal tract angiofibroma shows positivity in Androgen Receptor immunohistochemistry, which is negative for the other entities stated.[5,6]

The main treatment modality for sinonasal tract angiofibroma is surgical resection. Other treatment modalities for this neoplasm consist of antiandrogen medications, embolization and chemotherapy. The prognosis of this tumor is generally favorable, however, due to its locally aggressive behavior and high risk of recurrence it is important to be familiar with the clinical and histopathologic and immunologic features of this tumor in order to render the correct diagnosis.[1,2,6]

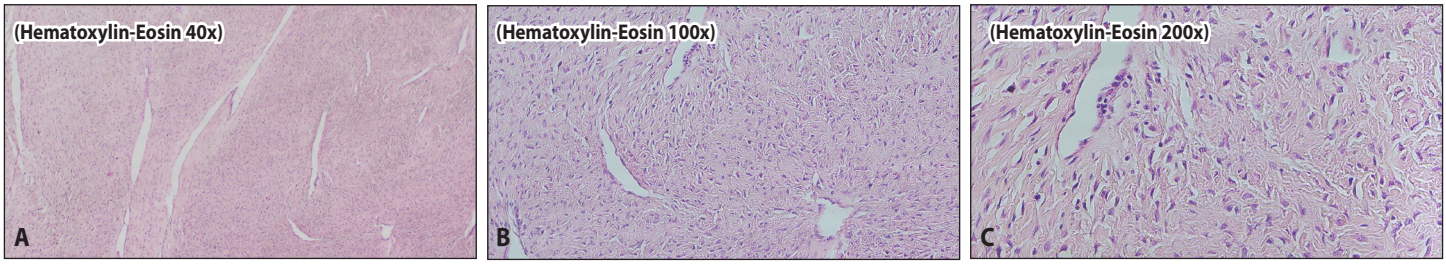

Figure 1. Histopathologic slides (Hematoxylin and Eosin stain): **A.** 40X magnification; **B.** 100X magnification; and **C.** 200X magnification. The tumor is composed of vascular spaces of varying caliber interspersed in a fibrous stroma containing spindly to stellate fibroblastic cells with ovoid bland nuclei

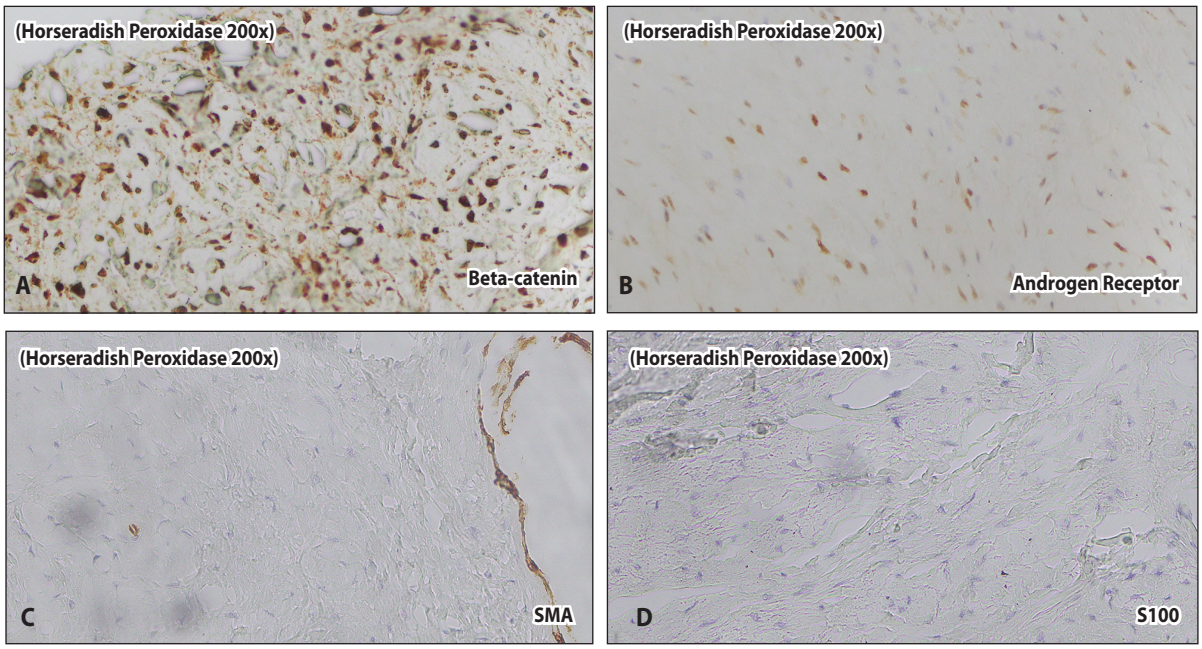

Figure 2. Immunohistochemistry, (Horseradish Peroxidase method, 200X magnification): **A.** Beta-catenin shows nuclear staining in stromal cells; **B.** Androgen Receptor shows nuclear staining in stromal cells; **C.** Negative staining with SMA; and **D.** Negative staining with S100

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