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# Microspheres embolization of juvenile nasopharyngeal angiofibroma in an adult

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ABSTRACT

*INTRODUCTION:* Juvenile nasopharyngeal angiofibroma (JNA) is a benign though locally aggressive, highly vascular tumor primarily affecting adolescent males which has traditionally posed a significant intraoperative challenge during its resection due to the high risk of uncontrollable hemorrhage. Pre-operative angiographic embolization of the major feeding vessels to the tumor has become a valuable, even necessary, tool in the surgical treatment of these lesions.
*PRESENTATION OF CASE:* Our patient was a 32-year-old man with a chief complaint of recurrent left-sided epistaxis for one year, brisk and continuous for ten days prior to presentation, subsequently found to have a 4 cm vascular skull base tumor causing mild expansion of the pterygopalatine fossa. The patient underwent pre-operative embolization utilizing 300–500 micrometer microspheres injected into the ipsilateral maxillary artery. The following day, the patient underwent definite Stereotactical surgical resection of his JNA tumor. Estimated blood loss during the operation was 50 mL, and the patient was discharged the same day.
*DISCUSSION:* Juvenile nasopharyngeal angiofibromas pose a significant bleeding risk for the surgeon due to their highly vascular nature. Pre-operative embolization of juvenile nasopharyngeal angiofibromas can reduce intraoperative blood loss while lessening the risk of massive hemorrhage, shortening operation times, increasing intra-operative visibility, and allowing for easier resection of lesions.
*CONCLUSION:* Pre-operative embolization of JNA is a safe, effective method to prevent against the risk of massive, sometimes fatal, hemorrhage that occurs with these highly vascular tumors.


## 1. Introduction

Juvenile nasopharyngeal angiofibroma is a relatively uncommon, benign tumor of children which accounts for approximately 0.05% of all head and neck tumors.[1] The lesion almost exclusively affects males, usually around the age of adolescence with a range involving boys from ages 7 to 29.[1] Initial symptoms upon presentation include recurrent unilateral epistaxis, nasal obstruction, nasal drainage and a nasopharyngeal mass. Although a benign tumor, lesions may be locally aggressive and erode into the surrounding osseous and soft tissue structures, arising from their usual location in the lateral nasopharynx near the superior border of the sphenopalatine foramen. Histopathologically, the tumor is comprised of haphazardly arranged vascular channels surrounded by dense paucicellular fibroblastic stroma, with the myofibroblast being the principal cell.[2,3] The bleeding propensity of these tumors is due to the findings that the smaller vessels in the center of the lesion tend to lack muscular elastic lamina, predisposing these vessels without a muscular surrounding layer that may otherwise assist in vasoconstriction to uncontrolled bleeding.[2] The blood supply to these lesions is derived from the internal maxillary artery,[8,9] a branch off the external carotid artery.

Diagnosis of JNA may originally be made on computed tomography (CT) examination or even suspected on the basis of plain radiographs. CT has the benefit of better detailing osseous involvement of tumor. Definitive diagnosis, however, is made on magnetic resonance imaging (MRI) examination. The optimal sequence to identify the anatomy and extent of intracranial extension of JNA involves T1 weighted imaging (T1WI) with gadolinium contrast, utilizing fat-suppression. An MR-angiogram can additionally be valuable in defining the arterial supply to the tumor, of primary importance for pre-embolization planning. Due to the highly vascular nature of JNA, biopsy is contraindicated.[10] Furthermore, careful attention must be paid to the surrounding vascular structures during time of resection accounting for the high risk of potentially fatal bleeding. Specifically, feeding vessels from the internal carotid artery, as well as any arterial supply from the contralateral external carotid artery must be identified prior to attempted resection. Although surgical resection remains definite treatment for these lesions, pre-operative angioembolization is increasingly recommended to minimize the risk of uncontrolled intraoperative

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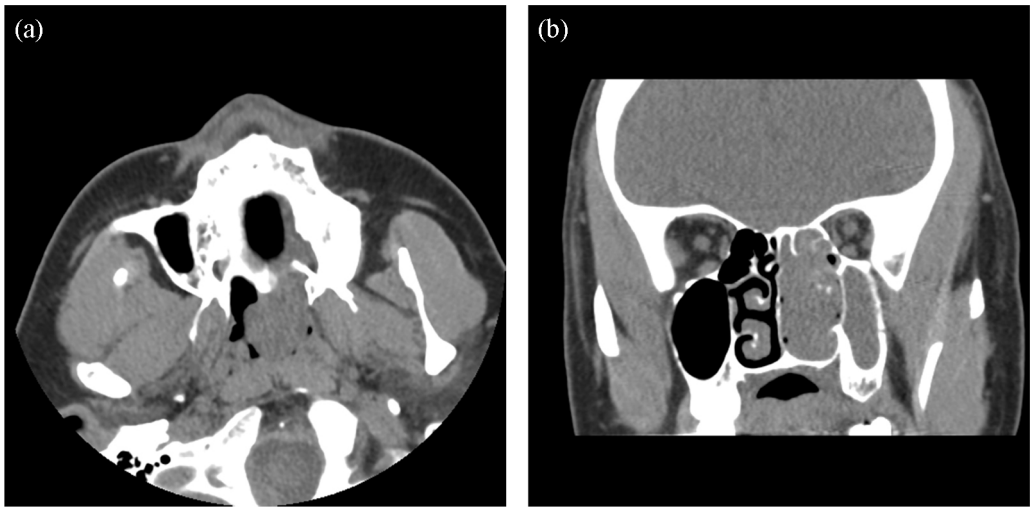


**Fig. 1.** (a) Axial unenhanced CT image of the face demonstrates a soft tissue mass in the left sphenoid sinus. (b) Coronal CT image of the face demonstrates opacification with a soft tissue mass within the left maxillary sinus and osteomeatal units.

hemorrhage. Previously described treatments including radiation, cryotherapy, electrocoagulation, or hormonal therapy have proven less effective with increased recurrence rate and a greater incidence of side effects.[3,4]

## 2. Case report

Our patient was a 32-year-old man with a chief complaint of recurrent left-sided epistaxis for one year, brisk and continuous for ten days prior to presentation. The Otolaryngology (ENT) service was consulted due to uncontrollable epistaxis, and imaging work-up with computed tomography (Fig. 1) followed by contrast enhanced MRI (Fig. 2) revealed a 3–4 cm vascular skull base tumor with mild proximal expansion of the pterygopalatine fossa and extension into the nasopharynx and left pterygoid plate, most consistent with a Radkowski IIA lesion.[11] Plans were made for surgical resection, and the Interventional Radiology service was subsequently consulted by ENT for pre-operative embolization to take place on the day prior to surgical resection.

In the Interventional Suite, the right femoral artery was accessed, and a 5-French Berenstein (Cordis Endovascular, Johnson and Johnson Corp., Miami, FL) was eventually placed to access the common carotid artery. An angiogram of the right common carotid and selective angiogram of the right external carotid artery revealed normal arterial anatomy with no significant tumor blush. Subsequently, an angiogram of the left common carotid artery was performed revealing normal branching of the internal and external carotid arteries with significant tumor blush in the skull base (Fig. 3). A normal left sphenopalatine artery could not be identified, as numerous abnormal vessels had replaced the sphenopalatine artery due to neovascularization (Fig. 4). Selective injections evaluating the occipital and facial

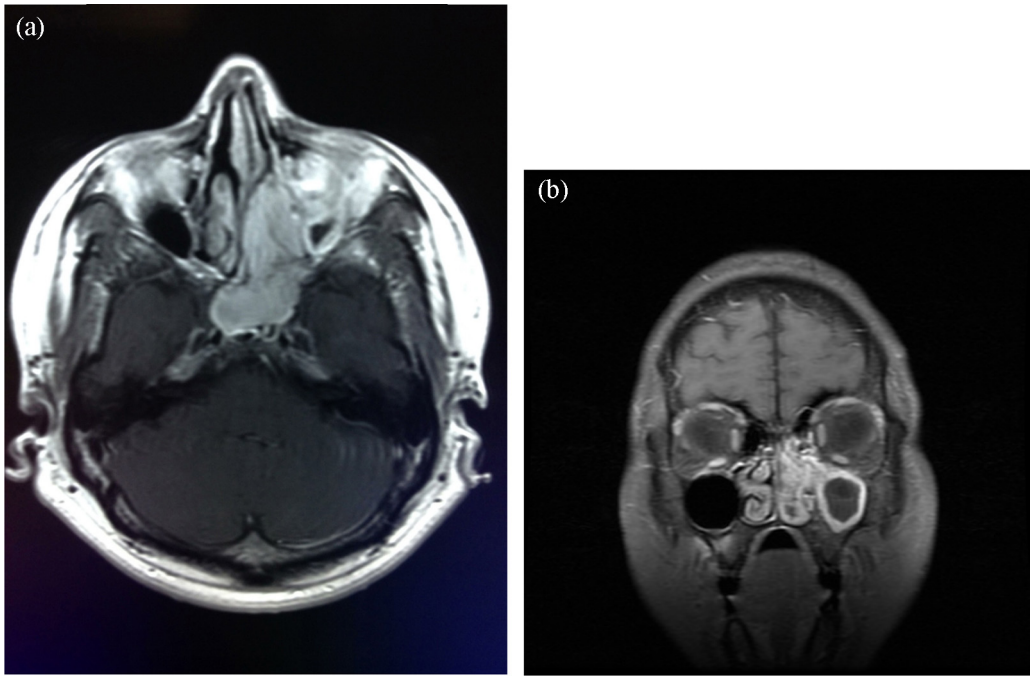


**Fig. 2.** (a) Axial contrast enhanced MR T1 weighted image demonstrates an enhancing soft tissue mass arising from the skull base and left nasopharynx into the sphenoid sinus, crossing midline. (b) Coronal contrast enhanced MR T1 weighted image demonstrates an enhancing soft tissue mass within the maxillary sinus and osteomeatal units.