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Angiofibromas with visual acuity impairment

Abstract

Three patients with extensive angiofibromas who experienced visual acuity impairment due to orbital involvement are described in this article. The appropriate treatment is described based on the patient's history and the extent of the angiofibroma. We establish a therapeutic algorithm and outline the most appropriate endoscope-assisted surgical technique for resecting intraorbital extensions of the angiofibroma

Keywords: angiofibroma, visual acuity, endoscopic surgery, orbit

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Introduction

Nasopharyngeal angiofibroma (NA) is a benign tumor with locally aggressive behavior due to its capacity to erode bone and extend into adjacent regions, including the endocranium. Orbital extension may occur through the infraorbital fissure, the superior orbital fissure, or by bone erosion of the orbital walls. One study reported that the most frequent route of invasion was the infraorbital fissure.[1]

The neoplasm may compress the globe, the optic nerve, or the optic chiasm in the middle cranial fossa, resulting in visual disturbances. Ocular signs and symptoms are uncommon. In a review of 208 patients with NA, Stern et al.[2] reported exophthalmos in 14% of cases, intraorbital extension in 12%, decreased vision in 5%, and partial ophthalmoplegia in 2%. We describe three clinical cases of angiofibromas producing visual acuity impairment and propose an algorithm for the management of NA with intraorbital extension.

Clinical cases

Case 1

A 12-year-old boy presented to the emergency ENT department with right-sided epistaxis, complete bilateral nasal obstruction, and progressive decline in visual acuity over three months.

Anterior rhinoscopy and nasal endoscopy revealed a vascular-appearing tumor occupying both nasal cavities, and examination showed left-sided proptosis.

Computed tomography (CT) of the face identified a tumor occupying the nasopharynx and both nasal cavities, with extension into the left pterygopalatine and infratemporal fossae, skull base erosion, and intracranial extension into the middle cranial fossa. It extended into the sphenoid sinus, sellar and suprasellar regions, and the left cavernous sinus, encasing the paraclival and intracranial segments of the internal carotid artery.

The NA extended into the left orbit via the inferior and superior orbital fissures.

Magnetic resonance (MRI) demonstrated a heterogeneous lesion with intense enhancement after intravenous contrast, with intraorbital extension to the apex. The left internal carotid artery was encased without luminal narrowing.

According to the Radkowski classification, the angiofibroma was staged as III-B (Table 1). Ophthalmologic evaluation demonstrated left amaurosis, exotropia on supraversion and supradextroversion, and proptosis (Figure 1).

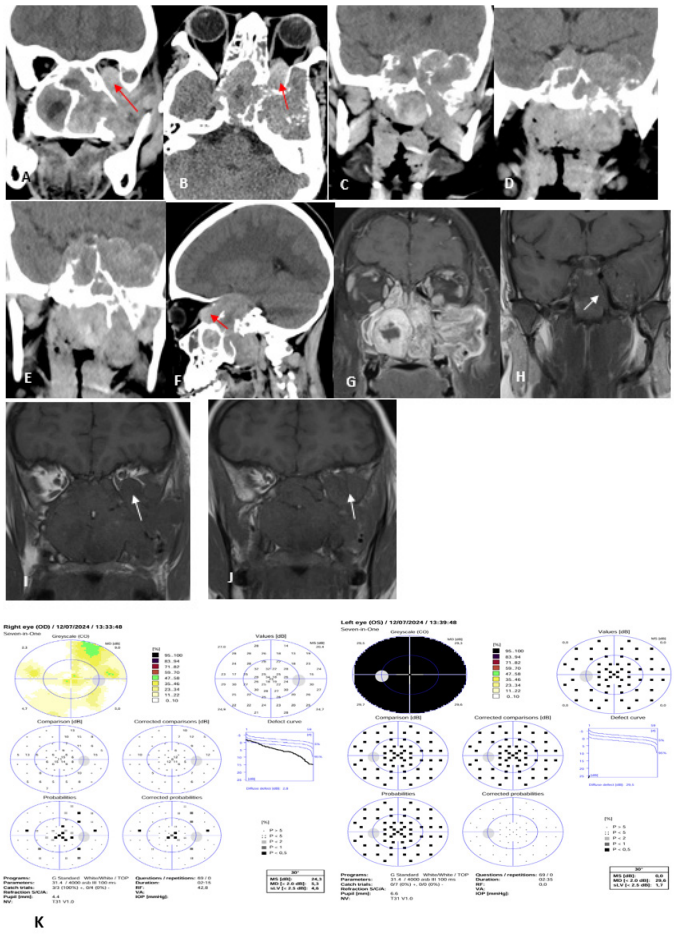

Figure 1 Angiofibroma with orbital and intracranial extension.

A and B) CT, coronal and axial views: orbital extension of the AF is observed through the inferior and superior orbital fissures, respectively (red arrows). C, D, E) CT, coronal views: bone destruction of the skull base and intracranial extension of the AF toward the sellar region and middle cranial fossa.

F) Lateral CT: bone erosion of the skull base and tumor extension into the orbit (red arrow).

G) MRI: pterygopalatine and infratemporal extension.

H) MRI: involvement of the cavernous sinus and medial portion of the middle cranial fossa (white arrows), encasing the left internal carotid artery.

I, J) MRI: massive intraorbital extension through the inferior orbital fissure (white arrow).

K) Visual field test showing severe reduction in left visual acuity.

Table 1 Radkowski Staging System for Angiofibroma.

Stage	Description
I-A	Tumor limited to the nasal cavity or nasopharynx
I-B	Extension into one or more paranasal sinuses
II-A	Minimal extension into the pterygopalatine fossa
II-B	Complete occupation of the pterygopalatine fossa with or without orbital bone erosion
II-C	Extension into the infratemporal fossa or pterygoid process
III-A	Skull base erosion with minimal intracranial extension
III-B	Skull base erosion with significant intracranial extension, with or without cavernous sinus invasion

Surgical treatment was indicated using a combined approach: endonasal endoscopic resection (Otolaryngology) and an external frontotemporal craniotomy (Neurosurgery).

Preoperative embolization was performed 48 hours before surgery. The NA received vascular supply from the left maxillary, sphenopalatine, ascending pharyngeal, facial, and meningeal arteries, and from the right sphenopalatine and ascending pharyngeal arteries. These vessels were successfully embolized with Variant spheres (400–500 µm), larger spheres (700–900 µm), and gelatin fragments.

The left internal carotid artery supplied the tumor through the carotid siphon, via the artery of the foramen rotundum and petrous branches. Intraorbital components received flow from ethmoidal artery branches.

According to Snyderman's endoscopic staging system, the tumor was classified as stage IV-A.[3]

Arteries originating from the internal carotid were not embolized.

Endonasal surgery began with cauterization of the left maxillary artery through a transoral paramaxillary approach to the infratemporal fossa, followed by cauterization of the left ethmoidal arteries through a medial transorbital incision.

The intraorbital extension was resected endonasally after removal of the central, infratemporal, and pterygopalatine components. The intraorbital portion was dissected and extracted through the inferior orbital fissure.

Dural infiltration was identified in the sellar, suprasellar, and temporal fossa regions (via craniotomy). The patient required transfusion of nine units of packed red blood cells.

Postoperative MRI at day 7 showed a marked reduction in tumor size, with residual disease in the cavernous sinus and along the left internal carotid artery (Figure 2).

Postoperative ophthalmologic evaluation showed no recovery of left visual acuity.

During one year of follow-up, the residual NA continued growing, and radiotherapy was indicated.

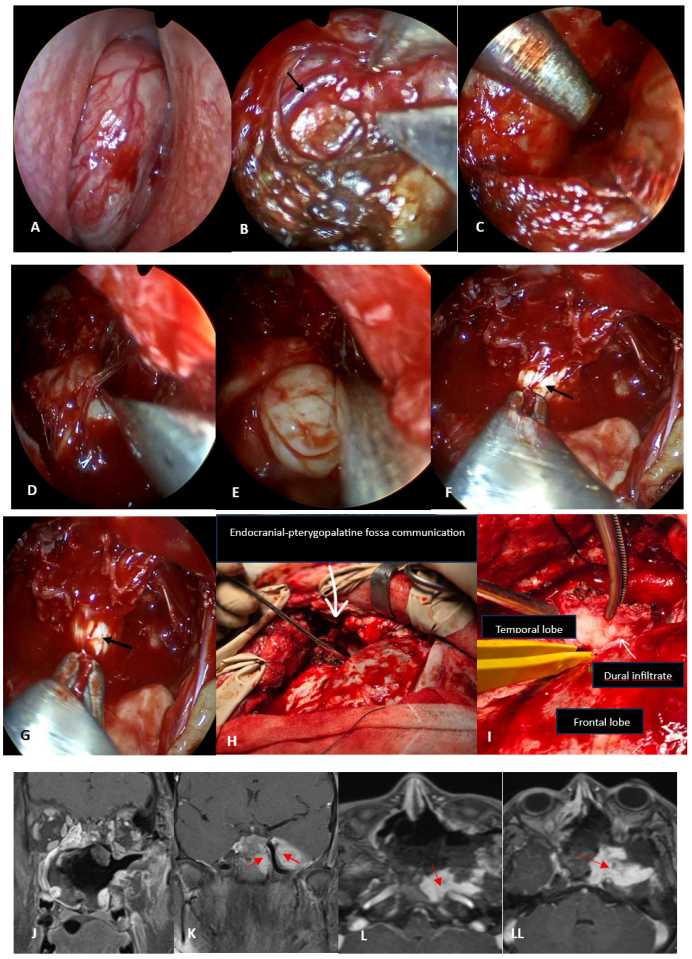

Figure 2 Endonasal endoscopic resection combined with frontotemporal craniotomy.

A) Endonasal view of the angiofibroma.

B) Cauterization of the maxillary artery in the infratemporal fossa through a paramaxillary transoral corridor (black arrow).

C) Cauterization of the anterior and posterior ethmoidal arteries via a medial transorbital approach.

D and E) Dissection and resection of the infratemporal and pterygopalatine extensions after removal of the nasal and nasopharyngeal components of the angiofibroma.

F and G) Dissection, traction, and excision of the intraorbital extension through the inferior orbital fissure (black arrows).

H) Frontotemporal craniotomy.

I) Dural infiltration by the angiofibroma.

Postoperative magnetic resonance imaging:

J) Coronal section showing the postoperative defect following resection of the angiofibroma from the nasal cavities, nasopharynx, pterygopalatine fossa, and infratemporal fossa. The left orbit is free of tumor.

K) Residual angiofibroma encasing the left internal carotid artery (red arrows).

L and LL) Axial sections showing persistence of the tumor along the horizontal and paraclival segments of the left internal carotid artery (red arrows).

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Case 2

A 36-year-old man presented with nasal obstruction and mild intermittent epistaxis. He reported left-sided blindness of several years' duration. He had a history of multiple surgeries for juvenile NA, including endonasal resections and two craniofacial approaches performed elsewhere. Ophthalmologic exam confirmed left-sided blindness.

MRI showed a large NA with intracranial, sphenoidal, and left orbital extension. The tumor encased both internal carotid arteries. It was staged as Radkowski III-B (Figure 3A). The extensive intracranial extent of the tumor is observed, encompassing both internal carotid arteries, cavernous sinuses, sellar region, and left orbit at the apex (red arrow).

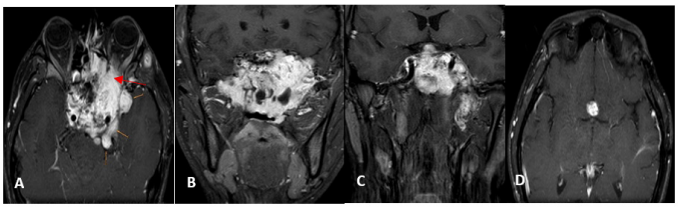

Figure 3A Stage III-B Angiofibroma with a history of multiple surgeries.

Given the extent of the tumor, involvement of vital structures, and probable fibrosis from prior surgeries, a conservative approach was adopted, indicating clinical and imaging monitoring.

During follow-up, the tumor showed no further growth.

Case 3

A 34-year-old man was referred for progressive right-sided visual acuity loss over several months, without sinonasal symptoms. He had undergone surgery for NA during childhood. Ophthalmologic evaluation confirmed decreased visual acuity.

CT and MRI revealed an NA involving the pterygopalatine and infratemporal fossae, right cavernous sinus, and extending intracranially through the middle cranial fossa. It also involved the right orbit and encased the right optic nerve. Radkowski stage III-B (Figure 3B).

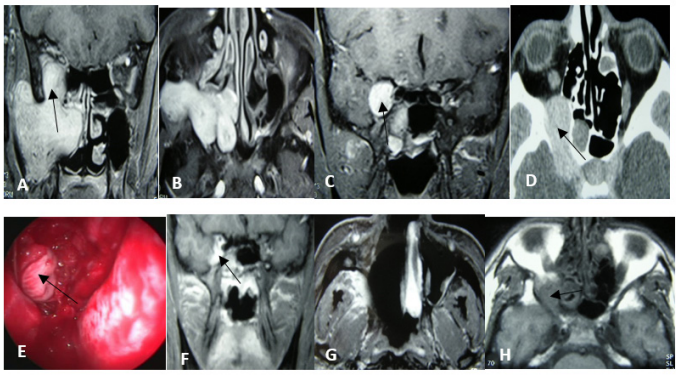

Figure 3B Stage III-B Angiofibroma with a history of multiple surgeries.

Extensive intracranial tumor extension is observed, encasing both internal carotid arteries, the cavernous sinuses, the sellar region, and the left orbit at the apex (red arrow).

The patient underwent endonasal endoscopic resection. A small residual tumor remained at the orbital apex, and radiotherapy was recommended. He was followed for 10 years with no evidence of tumor growth or visual deterioration.

Discussion

Visual loss resulting from optic nerve compression, orbital extension, or chiasmal compression by NA is uncommon. In one series of 18 patients with NA and visual alterations, 8 had preoperative visual acuity decline. All were advanced stages and required external approaches. Total resection was achieved in 12 patients and subtotal in 6. Visual acuity remained stable or improved postoperatively in seven; only one patient developed postoperative blindness after adjuvant radiotherapy.[4]

NA with intracranial extension into the middle cranial fossa may enter the orbit through the superior orbital fissure or cause cavernous sinus compression, leading to unilateral papilledema due to ophthalmic vein obstruction.[5] Asymmetric compression of the optic nerves by the tumor has been reported to produce optic atrophy and contralateral papilledema (Foster Kennedy syndrome).[6]

Bilateral amaurosis has also been described following anterior nasal packing in a patient with NA, likely due to increased tumor pressure on the optic nerves.[7] Orbital involvement corresponds to an advanced stage in most staging systems; in Radkowski, it is stage II-B.

In our cases, orbital invasion was associated with extensive intracranial disease (Radkowski III-B).

Surgery is the treatment of choice for NA. Endoscopic endonasal approaches are widely accepted as first-line therapy in early stages due to excellent visualization, magnification, angled optics, and reduced morbidity compared with external approaches.

In advanced tumors, the role of endonasal surgery is debated. A systematic review including 1,586 cases reported fewer recurrences with endoscopic approaches than with external ones and concluded that endoscopic surgery is preferred for early and moderately advanced stages, while combined endoscopic-assisted or open approaches may be useful for more advanced disease.[8] We consider the endoscopic endonasal approach to be the first option in all stages. In complex cases, it may be complemented with other endoscopic corridors -transoral or transorbital - or, rarely, a craniotomy (as in Case 1).

Resection of intraorbital extension can be achieved by combining the endonasal approach with transorbital endoscopic techniques.[9] In two of our cases, intraorbital tumor components were removed endonasally. In one, the inferior fissure was used to dissect and extract the tumor; in the other, the cavernous sinus and orbital apex components were approached through the sphenoid sinus.

A retrospective study of 68 NA patients found recurrence in 38.2%. Recurrence correlated with tumor size (>4 cm), advanced Radkowski stage (III-A/B), preoperative embolization, perioperative transfusion, external approaches, and younger age at presentation. The most frequent sites of recurrence/persistence were the pterygoid process (92.3%), nasopharynx (84.6%), and sphenoid sinus (76.9%). Orbital apex persistence occurred in ~20% of cases.[10]

Residual NA with growth and visual deterioration, when unresectable, may be treated with radiotherapy or radiosurgery.[11,12] In large tumors, resecting the extracranial component may reduce tumor volume, decreasing the field required for radiation therapy and its associated morbidity.

In our series, radiotherapy was indicated in two patients with residual disease: in one to prevent further visual decline due to optic nerve compression at the orbital apex, and in the other due to

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progressive tumor growth invading vital structures, meeting criteria for inoperability.

The third adult patient, with unilateral blindness and multiple prior surgeries, had bilateral carotid involvement. The tumor remained stable, and due to its large volume and involvement of vital structures, observation was chosen (Figure 4).

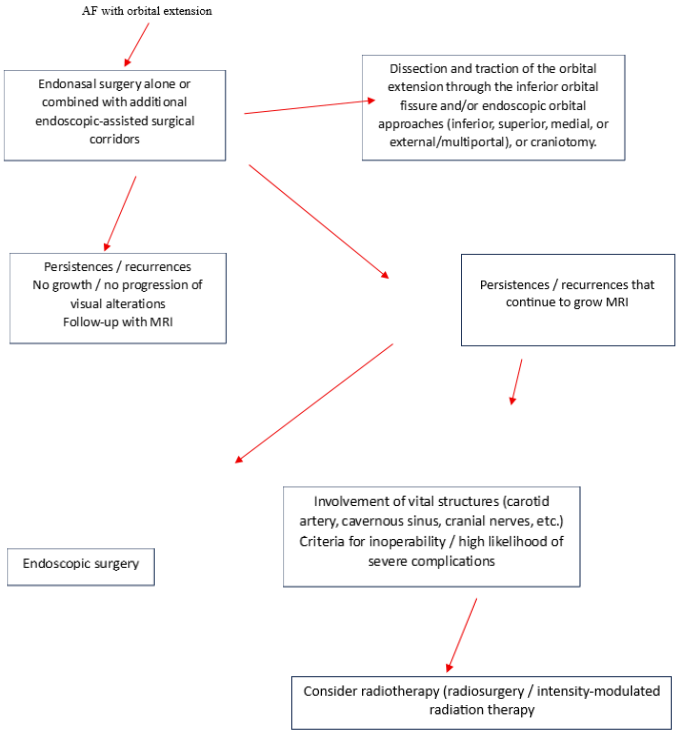

Figure 4 Treatment algorithm for patients with angiofibromas extending into the orbit.

Acknowledgments

None.

Conflict of interest

The authors declare that there are no conflicts of interest.

References

1. Cruz AA, Atique JM, Melo-Filho FV, et al. Orbital involvement in juvenile nasopharyngeal angiofibroma: prevalence and treatment. *Ophthal Plast Reconstr Surg*. 2004;20(4):296–300.
2. Stern RM, Beauchamp GR, Berlin AJ. Ocular findings in juvenile nasopharyngeal angiofibroma. *Ophthal Surg*. 1986;17(9):560–564.
3. Snyderman CH, Pant H, Carrau RL, et al. A new endoscopic staging system for angiofibromas. *Arch Otolaryngol Head Neck Surg*. 2010;136(6):588–594.
4. Xu Y, Lin G, Lin C, et al. Treatment and prognosis of nasopharyngeal angiofibroma involving the eye and optic nerve. *J Laryngol Otol*. 2012;126(11):1108–1113.
5. Patrocínio JA, Patrocínio LG, Martins LP, et al. Vision recovery following nasopharyngeal angiofibroma excision. *Auris Nasus Larynx*. 2002;29(3):309–311.
6. Watnick RL, Trobe JD. Bilateral optic nerve compression as a mechanism for the Foster Kennedy syndrome. *Ophthalmology*. 1989;96(12):1793–1798.
7. Ksahoo A, Preetam C, Kumar R, et al. Bilateral blindness following anterior nasal packing in a case of nasopharyngeal angiofibroma. *J Laryngol Otol*. 2016;130(11):1072–1073.
8. Jurlina M, Pupić-Bakrač J, Pupić-Bakrač A. Endoscopic, endoscopic-assisted and open approaches in the treatment of juvenile angiofibroma: what has been new in the past decade (and 1586 cases)? *Eur Arch Otorhinolaryngol*. 2023;280(5):2081–2089.
9. Vural A, Carobbio ALC, Ferrari M, et al. Transorbital endoscopic approaches to the skull base: a systematic literature review and anatomical description. *Neurosurg Rev*. 2021;44(5):2857–2878.
10. Attya HMA, Hassouna MS, Shawky AA, et al. Recurrent angiofibroma: analysis of risk factors and common sites of recurrence. *Eur Arch Otorhinolaryngol*. 2025;282(7):3611–3618.
11. López F, Triantafyllou A, Snyderman CH, et al. Nasal juvenile angiofibroma: current perspectives with emphasis on management. *Head Neck*. 2017;39(5):1033–1045.
12. Langdon C, Herman P, Verillaud B, et al. Expanded endoscopic endonasal surgery for advanced-stage juvenile angiofibromas: a retrospective multicenter study. *Rhinology*. 2016;54:239–246.