Management of Orbital Involvement in Sinonasal and Ventral Skull Base Malignancies

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KEYWORDS

• Sinonasal malignancy • Orbit • Orbital invasion • Orbital exenteration
• Orbital clearance • Orbital sacrifice • Orbit preservation • Endoscopic resection

KEY POINTS

- Multimodality therapy with surgery and radiation therapy is usually necessary to manage the orbit infiltrated with sinonasal or ventral skull base malignancy.
- Surgical resection with negative margins is the cornerstone of management.
- In carefully selected situations, orbital preservation does not adversely affect survival.
- Imaging and frozen section histopathology are critical in assessing candidacy for orbital preservation.
- Appropriate reconstruction of surgical defects is essential to minimize complications and optimize functional and aesthetic outcomes.

Video content accompanies this article at http://www.oto.theclinics.com.

INTRODUCTION: ORBITAL INVOLVEMENT IN SINONASAL AND VENTRAL SKULL BASE MALIGNANCIES

Malignancies of the sinonasal cavity and skull base involve the orbit in in 50% to 80% of cases.[1–4] The incidence of orbital involvement depends on primary tumor site and histopathology, being reported in 62% to 82% of ethmoid tumors and 46% of nasal cavity tumors.[2,5] Orbital invasion bodes poorer prognosis for overall and

Disclosure Statement: No relevant disclosures or Conflicts of Interest.
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Otolaryngol Clin N Am 50 (2017) 347–364
http://dx.doi.org/10.1016/j.otc.2016.12.010
oto.theclinics.com

Table 1
Staging of orbital invasion

Primary tumor (T)	
Maxillary sinus, nasal cavity and ethmoid sinus	
T3	Tumor invades floor or medial wall of the orbit
T4a	Moderately advanced local disease: tumor invades the anterior orbital contents
T4b	Very advanced local disease: tumor invades orbital apex

From Edge SB, Byrd DR, Compton CC, et al, editors. AJCC cancer staging manual. 7th edition. New York: Springer; 2010.

disease-free survival[2,5,6] and is associated with poorer outcome from salvage surgery.[7] Involvement of the orbit therefore upgrades local tumor stage to at least T3, with invasion of the orbital apex and beyond (T4b) having the gravest prognosis (Table 1).[8]

DIAGNOSIS

The proximity of the orbit to the sinonasal and ventral skull base facilitates tumor infiltration into the eye through many pathways (Table 2). Although diplopia, epiphora, chemosis, visual changes, and proptosis may be present in approximately 50% of cases,[3,5] the absence of these findings does not rule out tumor invasion. Symptoms can result from orbital compression, nasolacrimal duct obstruction, and true invasion. The periorbita is a robust barrier against invasion. However, once the tumor invades through the periorbita, there are no further barriers to diffuse orbital infiltration. Computerized tomography (CT) of the paranasal sinuses is useful in studying loss of orbital bone and enlargement of fissures and foramina. MRI is superior for delineating orbital soft tissue

Table 2
Pathways for orbital invasion by sinonasal and ventral skull base tumors

Route of Extension	Orbital Involvement
Direct invasion through bone	• Lamina papyracea, orbital floor and orbital roof → orbital periosteum, extraconal fat, extraocular muscles, intraconal fat, globe, orbital apex • Nasal bone, frontal process of maxilla (nasal tumor) → skin of medial canthal area, eyelids • Lateral sphenoidal wall → optic canal, orbital apex, cavernous sinus, cranial fossa
Direct extension through preformed pathways	• Sphenopalatine foramen → pterygopalatine fossa → inferior oblique fissure → orbit • Inferior orbital fissure • Superior orbital fissure • Anterior and posterior ethmoidal foramina
Perineural extension	• Infraorbital nerve • Supraorbital and supratrochlear nerves
Subperiosteal or intraperiosteal extension	• Orbital apex, cavernous sinus, cranial fossa
Nasolacrimal duct	• Lacrimal sac, medial and inferior orbit, upper and lower eyelids
Blood borne	• Metastatic tumors (eg, renal cell carcinoma)