Meningiomas of the orbit: contemporary considerations

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Meningiomas are the most frequently occurring benign intracranial neoplasms. Compared with other intracranial neoplasms they grow slowly, and they are potentially amenable to a complete surgical cure. They cause neurological compromise by direct compression of adjacent neural structures. Orbital meningiomas are interesting because of their location. They can compress the optic nerve, the intraorbital contents, the contents of the superior orbital fissure, the cavernous sinus, and frontal and temporal lobes. Because of its proximity to eloquent neurological structures, this lesion often poses a formidable operative challenge. Recent advances in techniques such as preoperative embolization and new modifications to surgical approaches allow surgeons to achieve their surgery-related goals and ultimately optimum patient outcome. Preoperative embolization may be effective in reducing intraoperative blood loss and in improving intraoperative visualization of the tumor by reducing the amount of blood obscuring the field and allowing unhurried microdissection. Advances in surgical techniques allow the surgeon to gain unfettered exposure of the tumor while minimizing the manipulation of neural structures. Recent advances in technology—namely, frameless computer-assisted image guidance—assist the surgeon in the safe resection of these tumors. Image guidance is particularly useful when resecting the osseous portion of the tumor because the tissue does not shift with respect to the calibration frame. The authors discuss their experience and review the contemporary literature concerning meningiomas of the orbit and the care of patients harboring such lesions.

KEY WORDS • meningioma • orbital tumor • surgical resection

Abbreviations used in this paper: CT = computerized tomography; MR = magnetic resonance.
Meningiomas of the orbit can be thought of as primary and secondary in origin. Primary orbital meningiomas arise from the optic nerve sheath and may extend through the optic canal intracranially, or they may infiltrate the sphenoid wing producing hyperostosis. Nearly two thirds of orbital tumors, however, originate from outside the confines of the orbit. Secondary orbital meningiomas usually arise from the inner and outer aspects of the sphenoid wing but not the middle sphenoid wing. They may infiltrate medially into the orbit, often with intraorbital, intracranial, and intrasosseous portions. They often compress the optic nerve by narrowing the optic canal. They may infiltrate the contents of the supraorbital fissure and cavernous sinus or compress the frontal and temporal lobes. There may also be cases of primary intrasosseous meningiomas in which the tumor arises from within the bone. Tumors originating along the sphenoid wing have a relatively high incidence of bone involvement. It is thought that nearly 30% of orbital meningiomas may involve bone (thus producing so-called hyperostosis), and 12% may originate from within the orbital bones.

The primary purpose of the present discussion is to address comprehensively the unique topic of meningiomas of the orbit. Data from the authors’ experience will be included and the contemporary literature will be reviewed. Additionally, a recent case, complete with radiographic and pathological data and intraoperative video clips will be used to illustrate many of the salient points germane to meningiomas of this region.

**EPIDEMIOLOGY**

Meningiomas comprise approximately 18 to 20% of all intracranial tumors and are the most common benign intracranial neoplasm and the second most common intracranial tumor overall (second to only gliomas). Intracranial meningiomas have been estimated to occur with an incidence of 2.1/100,000 people. Meningiomas account for 3 to 9% of all orbital tumors and are certainly not considered rare. Primary orbital meningiomas represent between 0.4 to 2% of all intracranial meningiomas. Secondary orbital meningiomas are considerably more common than their primary counterparts. Sphenoid ridge meningiomas comprise between 16 to 20% of all meningiomas whereas meningiomas arising from the suprasellar region account for 7 to 30% of intracranial meningiomas.

Orbital meningiomas, as with those located in other regions, have a predilection for afflicting females (73 to 84% occur in females), although this female preponderance does not appear to hold true in children with orbital meningiomas and in meningiomas in general in the pediatric population. Demographic data concerning the age distribution of meningiomas has been the subject of some controversy. Although reported frequently in younger patients, particularly in the early literature, others have found these tumors to be relatively uncommon in patients younger than 20 years of age. Reports of orbital meningiomas in children may have been erroneous in their diagnosis, as gliomas can evoke a surrounding dural reaction that superficially mimics meningioma. Suffice it to say that meningiomas are overall uncommon in children (1.5 to 2.3% of all intracranial tumors); however, the relative incidence of orbital involvement in childhood meningiomas has increased.

Several groups have endeavored to determine the predisposing factors for orbital meningiomas. Neurofibromatosis appears to be one predisposing factor, particularly in pediatric meningiomas. Exposure to radiation has clearly been associated with meningioma formation (with a latency ranging from 12–27 years), although there have been few cases in which orbital involvement is described. The role of trauma and viral infection in the development of meningiomas is controversial.

**CLINICAL PRESENTATION**

The clinical presentation of orbital meningiomas has not changed since it was described by Cushing and Eisenhardt in 1938. Unilateral visual loss and progressive, usually painless exophthalmos are the most common presentations described in the literature. Visual acuity tends to be involved early and exophthalmos manifests later. The apparent visual loss generally develops gradually, although acute visual loss may occur in 2 to 5% of intracanalicular and 8 to 12% of intracranial meningiomas. Other relatively common clinical features include optic disc changes, diplopia, headaches, and nausea and vomiting. Optic nerve changes may be due to intracranial hypertension, which causes papilledema, or direct compression on the optic nerve, resulting in papilledema or optic atrophy. Additionally, intracranial hypertension may produce papilledema in the contralateral eye. Diplopia may be caused by cranial neuropathies or by direct disruption of the rectus muscles. Headache, as well as nausea and vomiting, are typically associated with intracranial hypertension. Wright emphasized that visual loss was the most common initial symptom, followed by exophthalmos, diplopia, afferent papillary defect, and optic nerve changes with shunt vessels present. So-called optociliary shunt vessels on the disc surface, representative of collateral circulation between the central retinal vein and the choroidal/ciliary venous drainage system, are strongly suggestive of meningiomas involving the anterior visual pathways.

**PATHOLOGICAL CHARACTERISTICS**

The pathological origin of meningiomas is thought to be the arachnoid cap cells. The common sites for the tumor coincide with the presence of arachnoid villi, and they are often tightly bound to areas of arachnoid granulation tissue. Hence, the majority of meningiomas originate in parasagittal, cavernous sinus, tuberculum sellae, sphenoid wing, lamina cribosa, foramen magnum, and torcular regions. Cushing must have appreciated this characteristic distribution, as he described meningiomas by their common locations and clinical syndromes.

Meningiomas of the orbit have similar pathological characteristics to other intracranial meningiomas. They exhibit various histological subtypes. Cushing and Eisenhardt described nine main types and 20 subtypes. In the
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revised World Health Organization classification 15 subtypes are identified (Table 1). Meningothelial meningiomas are solid lobulated masses or sheets of meningothelial cells, in which the cell membrane is not well defined, and this gives an overall appearance of a syncytium; mitotic activity is low. Fibrous meningiomas are composed of bundles of cells that resemble fibroblasts; they typically contain collagen and reticulin, and whorl formation and psammoma bodies may be focally present. Transitional meningiomas are a mixture of the meningothelial and fibrous types. Psammomatous meningiomas contain many psammoma bodies. Bone-related proteins, including osteopontin, are produced by CD68-positive macrophages and may play a role in calcified psammoma body formation. Angiomatous meningiomas contain blood vessels of different shapes and sizes, typically with an endothelial lining. Microcystic meningiomas contain both macroscopic and intracellular microscopic cysts filled with eosinophilic mucin; microcyst formation may be due to tumor cell secretions, tumor cell degeneration, cerebrospinal fluid, penetration into the tumor, or vascular changes. Secretory meningiomas are typically meningothelial or transitional lesions in which epithelial differentiation has produced glandular structures. Clear cell meningiomas contain sheets of polygonal cells with clear cytoplasm. The cytoplasm stains positive for periodic acid–Schiff because of the presence of glycogen. Chordoid meningiomas are composed of eosinophilic, vacuolated cells in trabeculae, resembling chordomas. Lymphoplasmacyte-rich meningiomas are typically meningothelial, fibrous, or transitional with a lymphocytic infiltrate. Metaplastic meningiomas are meningothelial, fibrous, or transitional with metaplastic changes that may be cartilaginous, osseous, xanthomatous, myxoid, or lipomatous.

Atypical meningiomas deviate to some extent from the aforementioned benign features. They contain increased mitotic activity, increased cellularity, small cells, prominent nuclei, sheet-like growth, and areas of necrosis. Typically they are associated with a greater rate of recurrence. Rhabdoid meningiomas are uncommon. They are aggressive tumors that typically consist of patches or sheets of rhabdoid cells. Rhabdoid cells are round with eccentric nuclei and prominent nucleoli. The cells also contain an eosinophilic cytoplasm with whorls of intermediate filaments. Papillary meningiomas are composed of meningothelial cells in a radiating pattern resembling pseudopseudorosettes. They have a propensity for metastasis and recurrence. Anaplastic meningiomas have a more malignant histological pattern than the atypical meningiomas. They contain higher mitotic activity and greater necrosis. Their incidence is fortunately relatively low, ranging from 0.9 to 10.6% in different series, with an overall mean representation of 2.8% of meningiomas.

Primary orbital meningiomas arising from the optic sheath typically consist of the transitional histological subtype. In addition, psammomatous changes have been noted in orbital meningiomas. Other histological subtypes including fibrous, papillary, and anaplastic meningiomas typically do not involve the orbit primarily. In our evolving understanding of the development of optic sheath meningiomas we have witnessed the replacement of one hypothesis—that they arise primarily extradurally with secondary invasion of the optic dural sheath—with another—that they arise from the dural sheath itself. These lesions then spread along the optic nerve, often enveloping the nerve completely and thereby encompassing the pial blood supply. The optic nerve may be compromised by direct invasion by tumor or by compression leading to atrophy. Furthermore, tumor extension beyond the dura may affect the extracranial muscles.

As previously mentioned, meningiomas with orbital involvement, particularly those originating from the dural wing, commonly demonstrate involvement of the bone. Osseous involvement may consist of hyperostosis associated with an extraosseous meningioma, or the tumor may originate within the bone itself. Hyperostosis associated with intracranial meningiomas is a well-recognized phenomenon. The reported incidence varies from 25% to 49%. Numerous theories have emerged to account for hyperostosis and include vascular disturbances of the bone induced by the tumor, osseous reaction to the adjacent tumor without tumor invasion, production of bone by the tumor itself, stimulation of osteoblasts in adjacent normal bone by tumor-secreted factors, and tumor invasion of the bone.

<table>
<thead>
<tr>
<th>Type of Meningioma</th>
<th>WHO Grade</th>
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<tbody>
<tr>
<td>meningothelial</td>
<td>I</td>
</tr>
<tr>
<td>fibrous (fibroblastic)</td>
<td>I</td>
</tr>
<tr>
<td>transitional (mixed)</td>
<td>I</td>
</tr>
<tr>
<td>psammomatous</td>
<td>I</td>
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<tr>
<td>angiomatous</td>
<td>I</td>
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<tr>
<td>microcystic</td>
<td>I</td>
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<tr>
<td>secretory</td>
<td>I</td>
</tr>
<tr>
<td>lymphoplasmacyte-rich</td>
<td>I</td>
</tr>
<tr>
<td>metaplastic</td>
<td>I</td>
</tr>
<tr>
<td>atypical</td>
<td>II</td>
</tr>
<tr>
<td>clear cell (intracranial)</td>
<td>II</td>
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<tr>
<td>chordoid</td>
<td>II</td>
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<tr>
<td>rhabdoid</td>
<td>III</td>
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<tr>
<td>papillary</td>
<td>III</td>
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<tr>
<td>anaplastic</td>
<td>III</td>
</tr>
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† Defined as WHO = World Health Organization.
‡ Defined as those with a low risk of recurrence and aggressive growth behavior.
§ Defined as those with a greater risk of recurrence and aggressive growth behavior.
to tumor invasion and that complete tumor removal is the desired goal, resection of the hyperostotic bone becomes mandatory.

RADIOLOGICAL FINDINGS

Optic nerve sheath meningiomas may appear normal on plain x-ray films in the early stages of tumor development. Later in tumor development, optic canal enlargement or hyperostosis of the optic canal may be observed. Computerized tomography scanning of specifically optic nerve sheath meningiomas may reveal thickening of the optic nerve or the “tram track sign” in which two strips of lucency are seen around the center of the enlarged optic nerve, signifying that the optic nerve is surrounded by tumor. This feature is in contrast to optic nerve gliomas in which the optic nerve itself is expanded. The presence of calcification is an important feature that is highly suggestive of meningioma. Hyperostosis of the optic canal may also be evident on CT scans. Contrast administration typically reveals homogeneous enhancement of the mass. Magnetic resonance imaging can provide more details about the tumor; specifically it can help differentiate fusiform lesions and loculated from eccentric lesions. Additionally, MR imaging can also detect smaller lesions. Enhancement of a differential diagnosis for orbital meningioma, may also be visualized using MR imaging. Either hyperostosis or erosion of the orbital bone may be seen on radiographs of Sphenoid ridge meningiomas. Computerized tomography examination with and without contrast medium can often reveal the extent of the tumor’s intracranial involvement into the anterior and middle cranial fossae. It can also further delineate the involvement of the bone with hyperostosis or tumor infiltration. Additionally, CT scanning can demonstrate the extent of intraorbital involvement. Magnetic resonance imaging can provide finer detail of the anatomical location of the tumor. A clear dural tail, which would support the diagnosis of meningioma, may also be visualized using MR imaging.

DIFFERENTIAL DIAGNOSIS

An understanding of orbital anatomy facilitates the formulation of a differential diagnosis for orbital meningiomas. Lesions may arise from the optic nerve and its adjacent structures (Table 2). A conglomeration of pathological entities may affect the optic nerve and surrounding orbital contents. Enlargement of the optic nerve may, for example, be due to tumor involvement in addition to a multitude of inflammatory conditions or vascular conditions. Masses arising from the surrounding vicinity may also result from a host of conditions.

NATURAL HISTORY

Rational treatment decisions regarding the treatment of orbital meningiomas are predicated on an appreciation of the natural history of the disease as well as a detailed understanding of the available treatment modalities and their associated benefits, limitations, and risks. It was originally thought, based on anecdotal reports, that meningiomas affecting the orbit did not portend a clinical course of progression and neurological deterioration. As experience has accrued and long-term follow-up data have become available, however, it has become apparent that these tumors almost invariably progress and may grow to overwhelming size with time. As mentioned previously, orbital meningiomas affecting pediatric patients may be associated with a poorer prognosis than in older patients. Analysis of some data suggests that in the pediatric population, these tumors may not only produce visual impairment but also may extend to adjacent areas such as the cavernous sinus, sellar region, the anulus of Zinn, and the pterygomaxillary fossa, particularly with tumor involvement of the medial sphenoid wing. With tumor extension, complete excision without significant neurological compromise becomes exceedingly difficult. Thus it appears that meningiomas with orbital involvement progress, leading to potential neurological compromise. This is particularly so in the younger population in whom these tumors appear to behave more aggressively.

ILLUSTRATIVE CASE

History and Examination. This 35-year-old right-handed woman presented to an ophthalmologist with a history of right eye swelling and exophthalmos. She complained

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of occasional blurry vision and intermittent headaches. On physical examination right-sided exophthalmos was observed, yet otherwise she was neurologically intact. Findings on cranial nerve and sensorimotor examinations were within normal limits.

**Peroperative Treatment.** The patient underwent MR imaging and CT examination. As seen in Fig. 1 *left* and *center*, these studies revealed an enhancing lesion anterior to the tip of the right temporal lobe, as well as widening of the sphenoid ridge with extension of the intraosseous mass into the orbit. Endovascular embolization was performed. As seen in Fig. 1 *right*, external carotid artery angiography revealed a tumor blush, as is frequently seen in meningiomas. The tumor was embolized through selective catheterization of the middle meningeal artery. There was no angiographic evidence of the tumor’s blood supply being derived from the internal carotid artery system.

**Operation.** The patient was taken to the operating room the day after endovascular embolization. A frontotemporal craniotomy was performed. The tumor portion extending into the sphenoid ridge was resected in an extradural fashion. As seen in Video Clip 1, the lateral wall of the orbit was removed to the anterior clinoid process. The soft intraorbital portion of the tumor was then removed.

![Video Clip 1](#) Osseous bone removal. This is an intraoperative video edited to depict several key points of the osseous removal. A variety of clips demonstrates drilling of the sphenoid ridge. The drill is used to thin the tumor-infiltrated lateral wall of the orbit. The punch is then used to remove the thinned wall to the orbit. The final frames demonstrate the absence of the greater and lesser sphenoid ridges, as well as the contents of the supraorbital fissure left intact.

As demonstrated in Video Clip 2, the intradural portion of the tumor was then removed. The procedure was performed with the assistance of computer image guidance, which was found to be particularly useful in this case because much of the tumor was intraosseous. The frameless guidance system is calibrated to the skull with a rigid fixation system. Over the course of the operation the osseous structures do not shift with retraction or decompression; thus, the skull base was believed to be accurate (Fig. 2).

![Video Clip 2](#) Soft tumor removal. This is an intraoperative video demonstrating the removal of the intracranial soft tumor, which was performed in a standard fashion by using bipolar electrocautery and microinstruments.

**Postoperative Course.** The patient awoke from anesthesia without cranial nerve dysfunction. After postoperative swelling had subsided, the exophthalmos resolved. Final pathological examination revealed meningioma with intraosseous spread of tumor cells. Figure 3 shows photomicrographic examples of the intraosseous portion of tumor and the soft tumor.

**MANAGEMENT**

With the advent and characterization of new treatment modalities, there are numerous management options in the treatment of orbital meningiomas. Endovascular embolization can effectively devascularize meningiomas. Embolization is typically used in conjunction with open resection of the tumor. Whole-brain irradiation and stereotactic radiosurgery have been shown to be effective in controlling tumor growth. Radiosurgery can be applied through the gamma knife or through a linear accelerator. Radiosurgery has been used as a primary mode of treatment for intracranial meningiomas. In the skull base it is frequently used as adjuvant therapy, either applied to residual tumor postoperatively or to recurrent tumor. Intraoperatively, the use of computer-assisted image guidance has helped in establishing the tumor borders and localization. A different modification to cranial base approaches has assisted the surgeon in achieving a gross-total resection without compromising neural structures. At
our institution, in cases of orbital meningiomas, we routinely obtain preoperative MR images and perform preoperative embolization. Intraoperative image guidance is frequently used. Aggressive resection is attempted and complete tumor removal is undertaken whenever safely feasible. Radiosurgery is typically reserved for the treatment of patients in whom the tumors recur.

The treatment of optic nerve sheath meningiomas varies from that of secondary orbital meningiomas. Whereas resection is the primary treatment modality for secondary orbital meningiomas, patients with optic nerve sheath meningiomas are often followed clinically, undergoing visual field tests, visual acuity tests, and MR imaging studies. This course of action is typically undertaken after the diagnosis is confirmed by examination of a biopsy sample. Radiation therapy may slow the progression of the disease. As visual loss becomes progressive, or blindness has ensued, surgery may be an option to relieve eye pain or exophthalmos.

ENDOVASCULAR PREOPERATIVE EMBOLIZATION

Embolization involves selective catheterization of the blood vessels supplying the tumor. With the catheter in place, embolic material is positioned within the lumen, generating clot and thrombus. There has been some debate as to whether embolization of meningiomas is a beneficial intervention. In 1973 Manelfe, et al. first described the microcatheter technique of meningioma embolization. Since that time many authors have demonstrated the potential benefits of embolization—namely, decreased intraoperative blood loss, decreased complication rates, and potentially reduced operative times. In many of these studies the authors examined intracranial meningiomas in their entirety. It is clear that there are different surgery-related considerations for meningiomas in different locations. Convexity meningiomas, in which the blood supply is derived mainly from the external carotid artery system, are less challenging in terms of endovascular embolization, yet on the other hand, intraoperative cauterization of the feeding vessels before tumor resection is also an effective technique for diminishing intraoperative blood loss. Embolization is perhaps more helpful for selective cases such as when the surgeon must treat large-sized meningiomas and those of the skull base. In these cases the blood supply is derived from both the internal and external carotid systems and is not as easily sur-
Orbital meningiomas

gically accessible. An orbital meningioma is a good example of a tumor that may derive its blood supply from both the internal and external carotid systems. In cases in which the tumor’s blood supply is derived from both systems, and only the external system is embolized, the benefit achieved remains controversial. Subjectively, we have found that in the management of skull base osseous tumors preoperative embolization reduces the rate of intraoperative blood loss, allowing the surgeon to visualize the tumor more effectively and work in an unhurried fashion.

SURGICAL APPROACHES

The surgical approach must be tailored to the individual patient and his/her clinical status and pathological anatomy. Primarily intracranial lesions should be approached intracranially, and anteriorly located orbital lesions should be approached through the orbit.87

Transorbital access may be achieved using a variety of approaches in which a lower-lid, subciliary, superior lid crease, or an eyebrow incision is made. These provide different angles of approach to the anterior orbit. Many advances have been made over the years because of improved technology and accrued surgical experience. The transorbital approach is typically used for lesions located solely in the anterior orbit. An anteromedial microorbotomy can be performed to treat lesions located in the anteromedial orbit. A small operative field limits this approach. A lateral orbitotomy permits retraction of the globe laterally to expose the medial tumor, and this can expand the operative field. For lesions located in the superior temporal or inferior compartment of the orbit a lateral orbitotomy can be performed. The extent of resection from the lateral rim of the orbit bone and the sphenoid ridge depends on the location of the tumor (superiorly compared with inferiorly), as well as the depth of the tumor. Currently Newman and Jane87 advocate the use of a lateral orbitotomy in which a Burke incision is made. Transcranially the orbit can be reached via a pt erional, frontotemporal, supraorbital ridge, or subfrontal approach. A frontotemporal route can be used to approach primary orbital tumors that extended intracranially. This approach provides good exposure of the intraorbital contents, including the optic canal. This is achieved by excising the orbital roof and the lateral orbital wall. The optic nerve can be identified intradurally to facilitate the resection of the orbital roof. The frontotemporal approach also provides good exposure of the anterior and middle cranial fossae. A pterional approach can be used to reach sphenoid wing meningiomas that invade the orbit; this approach permits radical resection of the greater and lesser wings of the sphenoid bone, as well as exposure of the lateral orbit, optic canal, and the supraorbital fissure, foramen rotundum, and foramen ovale. Fraizer90 first advocated the removal of the orbital rim to obtain additional exposure of orbital contents. Jane and colleagues17,54 modified this approach by removing the supraorbital bone flap in one piece. Colohan, et al.30 introduced the concept of using the frontal sinus to access the anterior cranial fossa thereby providing better cosmesis.30 Jho86 has advocated a minimally invasive approach: though an eyebrow incision, one can gain access to the posterior orbit and anter-

RADIATION THERAPY

Radiation therapy is a treatment option for meningiomas as a primary treatment modality or as adjuvant therapy. Wara, et al.127 found in their 1975 series that adjuvant postoperative radiotherapy decreased recurrence rates from 74 to 29%. In several series and case reports authors have reported an improvement in visual acuity in patients in whom orbital meningiomas were treated primarily with radiotherapy.63,109 One issue concerning irradiation as a primary therapy is whether a tissue biopsy sample should be obtained for diagnosis. Some authors have found that inflammatory diseases can sometimes be misdiagnosed as optic nerve sheath meningiomas.35 Others have suggested that, at a minimum, a fine needle or open biopsy sampling should be contemplated.39,77

Radiosurgery has become increasingly popular for the treatment of meningiomas at many sites. In their series of 99 patients, Kondziolka, et al.,62 found that 63% of the tumors were reduced in size, 32% remained unchanged, and 5% enlarged. Resection was performed in 7% of patients in whom there was continued neurological dysfunction or continued tumor growth. Four of the tumors were located at the sphenoid ridge. These patients were followed for up to 10 years. In reviewing 62 patients with petroclival meningiomas, Subach, et al.,116 found that tumor volumes decreased in 23%, remained unchanged in 68%, and increased in 8%; the median follow-up period was 37 months. Hakim, et al.,96 examined 155 meningiomas treated with linear accelerator–based radiosurgery and demonstrated that 89% had good tumor growth control at 5 years posttreatment. Progression of disease was demonstrated in 15.7%. Radiosurgery is not without complications, however. Kalaparakal, et al.,33 reported on 42 patients treated with radiotherapy, of whom 11 developed cerebral edema, necessitating steroid therapy, and one patient developed radiation-induced necrosis. In the series published by Subach, et al.,116 5 patients sustained cranial nerve palsies, and one developed hydrocephalus. Although some success in controlling growth of meningiomas at multiple sites in the calvarium, has been achieved with radiosurgery it remains to be seen if its application to orbital meningiomas is efficacious and safe. The morbidity associated with orbital meningiomas is caused primarily by compression of neural structures. It has yet to be determined if radiosurgery can sufficiently reduce tumor volume to relieve symptoms associated with orbital meningiomas.

PROGNOSIS

Meningiomas are thought to be a slow-growing but progressive tumors. The majority of recurrences are thought to be due to residual tumor in the operative bed, which happen because of fear of causing severe functional deficits, were complete resection attempted. Meningiomas of all locations have a recurrence rates ranging from 10 to
Orbital meningiomas can invade the cavernous sinus, the dura of the sella turcica, the lateral part of the sphenoid body, the anulus of Zinn, the pterygomaxillary fossa, and the lateropharyngeal space. In their report Bonnal, et al., have argued against exploration of these areas out of desire for a complete resection to preserve neurological function postoperatively. In their series of invading meningiomas of the sphenoid ridge, residual tumor was left in 18 of 21 cases for the aforementioned reasons. Recurrence rates of sphenoid ridge meningioma are much higher than those located in other regions, ranging from 25 to 50%.[2-4] Maroon and colleagues[5] examined recur- rent sphenoorbital meningiomas and found that they tended to be large infiltrating tumors that had a propensity for invading vital neurological structures. They advocated performing an extensive resection that includes the entire greater and lesser wings of the sphenoid to the superior orbital fissure, the anterior clinoid process, and the orbital roof, as well as decompression of the foramen rotundum, foramen ovale, and the optic canal. They do not recommend exploration of the cavernous sinus or the superior orbital fissure.

CONCLUSIONS
The location of orbital meningiomas poses a particular challenge to the surgeon. As such, residual tumor may be left after resection, resulting in higher recurrence rates than for meningiomas located in other regions. Fortunately, meningiomas are typically slow-growing benign tumors that can be followed clinically and radiographically for recurrences. Should they recur, treatment options remain. Radiation therapy has been shown to be effective in controlling local tumor growth. Additional resection remains an option. A complete resection is still the most desired result, as it may be curative. Current advances in peri- and operative management—specifically preoperative endovascular embolization, intraoperative computer-assisted image guidance, and refined surgical approaches—assist the surgeon in achieving optimum outcomes.

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