Orbital apex syndrome
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Purpose of review
Visual loss from optic neuropathy and ophthalmoplegia involving multiple cranial nerves are the hallmarks of an orbital apex syndrome. Historically, the terms superior orbital fissure, orbital apex, and cavernous sinus have been used to define the anatomic locations of a disease process. However, the diagnostic evaluation and management is similar for each of these entities. The authors reviewed the literature on the diagnosis and evaluation of disorders involving the orbital apex.

Recent findings
High-resolution MRI is the preferred modality for evaluating most lesions involving the orbital apex. CT is a useful tool in the setting of trauma, to evaluate bone involvement, or when MRI is contraindicated. Although laboratory studies may be useful adjuncts in the diagnostic evaluation of lesions involving the orbital apex, surgical biopsy is often required for definitive diagnosis.

Summary
Orbital apex syndromes may result from a variety of inflammatory, infectious, neoplastic, iatrogenic/traumatic, and vascular conditions. A detailed history with review of systems is important in narrowing the differential diagnosis. Management is directed at the underlying cause and may be guided by surgical biopsy. Corticosteroids may be useful if an inflammatory etiology is suspected, but should be used with caution.

Keywords
orbital apex, superior orbital fissure, cavernous sinus, Tolosa–Hunt syndrome, magnetic resonance imaging


Introduction
An orbital apex syndrome (OAS) has been described previously as a syndrome involving damage to the oculomotor nerve (III), trochlear nerve (IV), abducens nerve (VI), and ophthalmic branch of the trigeminal nerve (V1) in association with optic nerve dysfunction. The cavernous sinus syndrome (CSS) may include the features of an OAS with added involvement of the maxillary branch of the trigeminal nerve (V2) and oculosympathetic fibers (Fig. 1) [1]. Cavernous sinus lesions are also more commonly bilateral.

The term superior orbital fissure syndrome (SOFS) or Rochon–Duvigneaud syndrome is often applied to lesions located immediately anterior to the orbital apex, including the structures exiting the annulus of Zinn and often those external to the annulus (Fig. 2) [2]. In this clinical setting, multiple cranial nerve palsies may be seen in the absence of optic nerve pathology.

The superior orbital fissure, orbital apex, and cavernous sinuses are all contiguous, and although these terms define the precise anatomic locations of a disease process, the etiologies of these syndromes are similar. In some instances, patients who have features of a SOFS may subsequently develop orbital apex and cavernous sinus pathology. Thus, we have chosen to discuss these entities together under the heading of OAS. We present a diagnostic algorithm to evaluate an OAS and review recent literature on the diagnosis and treatment of these disorders.

Symptoms and signs
Visual loss and ophthalmoplegia are often the initial manifestations of an OAS. Thus, the ophthalmologist may be the first physician consulted by a patient with an OAS.

Periorbital or facial pain may reflect involvement of the ophthalmic (V1) or maxillary (V2) branch of the trigeminal nerve. Periorbital pain is one of the diagnostic criteria for Tolosa–Hunt syndrome (THS), an idiopathic inflammatory syndrome of the orbital apex. However, the absence of pain does not exclude a process within the orbital apex. It is important to test the periorbital skin and the corneal reflexes to detect asymmetry in sensation.

Infectious, inflammatory, and neoplastic conditions may be associated with proptosis. Vascular causes of a CSS,
such as a carotid–cavernous fistula, classically are associated with pulsatile proptosis.

To assess for optic nerve dysfunction, measurement of best-corrected visual acuity, examination of the pupils for the presence of an afferent pupillary defect, and color vision testing should be included. Visual field testing with kinetic or static perimetry may reveal subtle visual field deficits when visual acuity is normal. We routinely use Humphrey automated perimetry to assess for visual field defects.

Optic atrophy typically develops over weeks to months in patients with an OAS. Thus, optic atrophy may or may not be present in a patient with an OAS, and its absence should not preclude further evaluation if other clinical findings suggest that an optic neuropathy is present.

Diplopia may be the presenting symptom in SOFS, OAS, or CSS. The pattern of an ocular deviation is especially important in the evaluation of a single ocular motor nerve palsy (e.g., an esotropia greater in gaze ipsilateral to a sixth nerve palsy or hypertropia increasing in contralateral gaze and on ipsilateral head tilt in a fourth nerve palsy). However, because multiple cranial nerves may be involved, a distinct pattern may be difficult to detect.

Several authors have reported a correlation between the initial number of cranial nerves involved and the likelihood of having a CSS. In their retrospective study of 68 patients with cranial neuropathies, CSS was found in 7 of 34 patients (18%) who presented with a cranial mononeuropathy and in seven of nine patients (78%) who presented with involvement of four cranial nerves. The oculomotor and abducens nerves were the most frequently involved cranial nerves, followed by the trochlear nerve [3••].

Causes
Orbital apex syndromes may be caused by inflammatory, infectious, neoplastic, iatrogenic/traumatic, or vascular processes:

Inflammatory

1. Sarcoidosis
2. Systemic lupus erythematosus
3. Churg–Strauss syndrome
4. Wegener granulomatosis
5. THS
6. Giant cell arteritis
7. Orbital inflammatory pseudotumor
8. Thyroid orbitopathy

Infectious

1. Fungi: *Aspergillus, Mucormycosis*
3. Spirochetes: *Treponema pallidum*
4. Viruses: Herpes zoster
Neoplastic
1. Head and neck tumors: nasopharyngeal carcinoma, adenoid cystic carcinoma, squamous cell carcinoma
2. Neural tumors: neurofibroma, meningioma, ciliary neurinoma, schwannoma
3. Metastatic lesions: lung, breast, renal cell, malignant melanoma
5. Perineural invasion of cutaneous malignancy

Iatrogenic/traumatic
A. Iatrogenic
1. Sinonasal surgery
2. Orbital/facial surgery
B. Traumatic
1. Penetrating injury
2. Nonpenetrating injury
3. Orbital apex fracture
4. Retained foreign body

Vascular
1. Carotid cavernous aneurysm
2. Carotid cavernous fistula
3. Cavernous sinus thrombosis
4. Sickle cell anemia

Other
A. Mucocele

The incidence of each cause differs depending on the source of the report. In a retrospective review of 151 lesions causing a CSS, tumors were the most frequent cause (45 patients, 30%). When surgical causes were included with trauma, an iatrogenic/traumatic etiology (53 patients, 35%) was the most common cause. Self-limited inflammation was the third most frequent cause (34 patients, 23%), whereas vascular causes, infections, and other causes constituted the remaining 12% of CSS [4].

A retrospective review of 130 published cases of SOFS identified an inflammatory etiology in 45 of 63 patients (71%) in whom a neuroradiologic evaluation was pursued. Neoplastic causes and hematoma were each found in 5 of 63 patients (8%). The causes of SOFS were unidentified in 8 of 63 patients (13%) [5].

Inflammatory
Inflammatory disease within the orbital apex may present as painful ophthalmoplegia with or without associated optic neuropathy (Fig. 3). Typically, the onset of symptoms is abrupt with progression over days to weeks. Associated conditions include Wegener granulomatosis [6], sarcoidosis [7], systemic lupus erythematosus [8], and Churg–Strauss syndrome [9]. Giant cell arteritis may also rarely mimic an OAS and present with periorbital pain and ophthalmoplegia [10].

Both the systemic form of Wegener granulomatosis, with pulmonary and renal involvement, and its limited form may involve the cavernous sinus. Seizures and CSS were reported in a patient with antineutrophil cytoplasmic antibody-associated vasculitis in the limited form of Wegener granulomatosis. In this patient, enhancing lesions of the cavernous sinus and thickened pachymeninges were observed [6].

Although patients with neurosarcoidosis often have other systemic manifestations, CSS has been reported as the only manifestation of this inflammatory condition [7]. In this patient, laboratory testing revealed an elevated erythrocyte sedimentation rate and a normal serum angiotensin-converting enzyme, chest radiograph, and gallium scan. Definitive diagnosis was made by cavernous sinus biopsy, which revealed noncaseating granulomas with multinucleated giant cells.

Churg–Strauss syndrome may also cause a CSS, with one recent report in the literature [9]. This patient, with a history of asthma, developed severe headaches, progressive left-sided ophthalmoplegia, and visual loss. Laboratory studies revealed eosinophilia, an elevated erythrocyte sedimentation rate, and an elevated perinuclear antineutrophil cytoplasmic antibody level. MRI showed enhancement involving the left superior orbital fissure, cavernous sinus, and dura of the temporal base.

The THS refers to a condition of unknown cause involving painful ophthalmoplegia resulting from granuloma-
tous inflammation within the cavernous sinus or orbital apex [11,12]. In 2004, the International Headache Society defined the diagnostic criteria of THS to include episodes of unilateral orbital pain persisting for weeks if untreated; associated paralysis of one or more of the third, fourth, or sixth cranial nerves; and/or demonstration of a granuloma by MRI or biopsy [13••]. Cranial nerve paresis typically coincides with the onset of pain or follows it within a period of as long as 2 weeks, and pain or paresis resolves within 72 hours after adequate corticosteroid therapy. THS is diagnosed according to International Headache Society criteria only after exclusion of other causative lesions [13••]. The pain is often described as a “gnawing” or “boring” sensation and may precede or occur with ophthalmoplegia. Oculosympathetic nerve fibers adjacent to the internal carotid artery may also be involved in THS. THS may have a relapsing and remitting course; however, residual neurologic deficits may persist after remission [2,14]. Some authors have suggested that other causative conditions should be excluded using blood and cerebrospinal fluid examination and possible biopsy if positive findings are seen on MRI or CT. They recommend that follow-up examinations must then be performed for at least 2 years before the diagnosis of THS is made [15]. Neuroimaging may not be specific for THS, because meningioma, lymphoma, and sarcoidosis may have identical signal intensities on T1- and T2-weighted MRI [16].

Infectious Diseases

Infectious diseases involving the central nervous system, paranasal sinuses (Fig. 4), and periorbital structures may lead to an OAS. These include fungal organisms such as *Mucormycosis* [17,18] and *Aspergillosis* [19,20], bacteria [21–23,24•,25–29], and syphilis [30]. Early identification of an infectious cause is paramount, because failure to recognize these conditions may be fatal.

*Mucormycosis* and *Aspergillosis* should be suspected in individuals with predisposing conditions including diabetes mellitus, alcoholism, hematologic malignancies, and immunosuppression [17]. A fungal cause should be considered in any patient requiring immunomodulatory, antineoplastic, or long-term corticosteroid therapy. However, *Aspergillosis* and *Mucormycosis* have been reported in immunocompetent individuals as well [20,31]. Although fungal infections of the orbit and paranasal sinuses may present with pain, local tissue invasion and necrosis, and typical radiographic findings, they may also occur without pain and in an insidious fashion, making the diagnosis more difficult. Otolaryngologic consultation may be helpful to identify and to sample areas of tissue necrosis if the cause of an orbital apex syndrome is unclear [18].

Tuberculosis has also been reported to cause both unilateral and bilateral CSS [21]. CSS resulting from tuberculosis has rarely been reported in the literature, but may occur in the absence or presence of other central nervous system findings [22]. A cavernous sinus tuberculoma may also occur in the absence of pulmonary findings [23].

Bacterial infection may result in cavernous sinus thrombosis, most commonly from contiguous spread of infections from the paranasal sinuses. Organisms most commonly implicated include *Staphylococcus aureus*, *Streptococcus pneumoniae*, other streptococci, Gram-

**Figure 3.** Axial T1-weighted MRI (left) showing contrast enhancement within the orbital apex (larger arrow) and cavernous sinus (smaller arrow) in a 77-year-old woman who developed a left sixth nerve palsy and optic neuropathy. Total ophthalmoplegia developed, prompting a craniotomy and cavernous sinus biopsy, which revealed nongranulomatous inflammatory changes. Despite corticosteroids and the addition of other immunomodulatory agents, the patient lost vision to no light perception, and repeat neuroimaging revealed progression of the inflammatory process (right).

**Figure 4.** Axial CT (left) showing opacification within the ethmoid (larger arrow) and sphenoid (smaller arrow) sinuses. Coronal CT (right) confirms opacification within the ethmoid sinus in a 43-year-old man who developed fevers, visual loss, and a sixth nerve palsy. Endoscopy revealed mucosal inflammation within the paranasal sinuses and pus within the orbital apex. His symptoms improved after surgical drainage and treatment with intravenous antibiotics.
negative bacilli, and anaerobes [24–26]. Bilateral cavernous sinus involvement has also been associated with central nervous system *Actinomyces israelii* [27,28]. A mixed infection with *S. aureus* and *Pseudomonas aeruginosa* has also been observed to cause an OAS with cavernous sinus thrombosis [29].

**Neoplastic**

The possibility of a neoplasm should be considered in the differential diagnosis of an OAS, especially in any patient with a known history of cancer. Primary ocular or orbital tumors, neoplasms of the paranasal sinuses, or central nervous system tumors may invade the orbital apex. Metastatic disease may also involve the cavernous sinus. Tumors that most commonly cause a CSS include nasopharyngeal cancer, lymphoma, pituitary adenoma, meningioma, and metastatic disease [4].

Lymphomatous infiltration of the cavernous sinus has been reported both in pediatric [32–33] and adult patients [34,35]. Lymphoma may be found in the paranasal sinuses or within the cavernous sinus alone. Neural tumors of the orbital apex are most commonly benign and include meningiomas (Fig. 5), schwannomas, neurofibromas, and, rarely, ciliary neuromas [36–37]. These benign lesions typically result in slowly developing orbital symptoms in the absence of pain.

**Vascular**

Vascular causes of CSS include cavernous carotid aneurysms, carotid–cavernous fistulas, and cavernous sinus thrombosis. Cavernous carotid aneurysms may cause a unilateral or bilateral cavernous sinus syndrome via compression of adjacent cranial nerves [4,53]. A carotid–cavernous fistula may cause pulsatile proptosis, severe conjunctival injection, and glaucoma from elevated episcleral venous pressure. A history of head trauma is often elicited; however, carotid–cavernous fistulas may also occur spontaneously and have been observed in patients with longstanding hypertension or connective tissue disorders such as Ehlers–Danlos syndrome [54,55]. Septic thrombosis of the cavernous sinus may occur in the setting of systemic or, more commonly, periorbital infections [56,57], but aseptic cavernous sinus thromboses have also been noted [58]. Cavernous sinus thrombosis may be visualized on contrast-enhanced high-resolution CT or with MRI. Typical findings include filling defects Local invasion of the orbital apex from adjacent head and neck tumors has also been frequently reported in the literature. Although patients with tumors of the head, neck, and paranasal sinuses most frequently have other findings, OAS has been reported as the initial sign in a patient with maxillary sinus carcinoma [38]. Adenoid cystic carcinoma [39], mucoepidermoid carcinoma [40], and poorly differentiated squamous cell carcinoma [41] have also presented with primary involvement of the orbital apex.

Metastatic disease to the cavernous sinus has been reported from the breast [42], lung [43], kidney [44], and from malignant melanoma [45]. Extramedullary hematopoiesis in a patient with polycythemia rubra vera caused an OAS from compressive neuropathy. Bone marrow biopsy revealed myelofibrosis in this patient [46].

**Iatrogenic/traumatic**

Iatrogenic causes of an OAS have been reported following sinonasal and periorbital surgical procedures. An OAS has been reported after ethmoidal artery ligation for recurrent epistaxis [47], intranasal ethmoidectomy for nasal polyposis [48], and septorhinoplasty [49]. Optic neuropathy during sinus surgery may occur from direct or indirect injury to the optic nerve or its blood supply [50].

OAS has been observed after both penetrating and blunt orbital trauma [51]. Penetrating trauma leading to OAS is rare in the absence of a bony fracture; however, an OAS has been reported secondary to a retained intraorbital foreign body without bone involvement. Although delayed onset of neurologic symptoms is rare after an acute traumatic injury, symptoms of a SOFS were delayed for 72 hours in a patient with a retained foreign body within the orbit and cavernous sinus [52].

**Figure 5.** Axial T1-weighted MRI showing an enhancing lesion within the left cavernous sinus and orbital apex (larger arrow) and involvement of the meninges around the left temporal lobe (smaller arrow) in a 50-year-old woman with a left third nerve palsy and optic neuropathy

Neurosurgical resection confirmed that the lesion was a meningioma.
within the cavernous sinus and expansion of tributary veins and venous sinuses [59].

**Evaluation and management**

Neuroimaging should be performed in a patient with findings consistent with an OAS (Fig. 6). High-resolution (1.5-T magnet or greater) MRI is the preferred imaging modality in the evaluation of most patients with an OAS. To evaluate the orbital apex and cavernous sinus, we perform MRI of the brain and orbits with contrast and fat suppression sequences. The 3-T MRI has been found to be superior to the standard 1- to 1.5-T MRI magnet to define parasellar anatomy and to diagnose tumor invasion of the cavernous sinus [60••].

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**Figure 6. Evaluation and management of a patient with an orbital apex syndrome**

Detailed H&P with emphasis on ophthalmic and neurologic findings – Pain, Cranial nerve palsies, Afferent pupillary defect

Multiple cranial nerve palsies (III, IV, V, VI) +/- pupil involvement +/- V2 involved

III, IV, V, VI involved

Optic nerve involved

V2 involved

Superior orbital fissure syndrome

Orbital apex syndrome

Cavernous sinus syndrome

Neuroimaging – MRI or CT brain and orbits w/ and w/o contrast

Yes

? Hx of trauma or surgery

No

Findings suggestive of vascular cause

Yes

Consider angiography

No

Meningeal, systemic Sx/Si of infection?

Yes

Blood Cs, LP, CSF Cs, IV Abx, Consider Bx

No

GCA Sx?

No

ESR, CBC w/ differential, ANA, ACE, PPD, CXR, RPR, MHA-TP, HIV C-ANCA, P-ANCA, LP

Yes

Hx of autoimmune or inflammatory conditions?

No

Hx of cancer or cancer risk factors?

Yes

Corticosteroids and TABx

Tx failure

Consider corticosteroids vs. biopsy

No

Consider LP and Bx

Tx failure

Iatrogenic vs. traumatic

Consider IV steroids and surgical intervention

Yes

Treat underlying condition

No

Repeat neuroimaging - Consider other immunomodulatory agents or radiation if inflammatory condition suspected vs. additional biopsy

Pos

Neg
CT also plays an important role in the setting of trauma or in patients who are suspected of having magnetic foreign bodies, surgical clips, or who have other contraindications to MRI. CT is superior to MRI for the depiction of bony anatomy and is especially helpful if an orbital apex fracture is suspected clinically [61].

If a vascular lesion of the cavernous sinus is suspected, magnetic resonance angiography (MRA) or CT angiography may be helpful. If the index of suspicion remains high despite negative neuroimaging studies, cerebral angiography may be necessary.

Laboratory testing for inflammation and infection (eg, erythrocyte sedimentation rate, complete blood count with differential, rapid plasma reagin, microhemagglutination assay for antibody to Treponema pallidum, angiotensin-converting enzyme, perinuclear antineutrophil cytoplasmic antibody, cytoplasmic antineutrophil cytoplasmic antibody, antinuclear antibody, purified protein derivative, chest radiography, HIV, lumbar puncture) should also be considered if the clinical findings are suggestive of these processes.

When a specific cause cannot be determined, the primary management options in a patient with an OAS include observation, an empiric trial of corticosteroids, and surgical biopsy. In the absence of systemic signs of infection and when inflammatory disease is likely, we offer corticosteroids and a period of close observation. If visual loss or ophthalmoplegia progresses, repeat neuroimaging and a biopsy are then pursued. Neurosurgical, otoaryngologic, and radiologic consultation may be warranted if a cavernous sinus or paranasal sinus lesion is accessible for surgical biopsy [62••]. Internal medicine or neurologic consultation may also be sought when clinical circumstances suggest a systemic or neurologic process.

Management of an OAS is aimed at the underlying cause. The distinction between inflammation, infection, and neoplasia is often difficult, and all may respond initially to corticosteroids. Judicious use of corticosteroids is recommended, because the presence of an occult infection, especially fungal disease, may result in severe morbidity or mortality. Methotrexate [63] and azathioprine [64] have provided clinical benefit in a limited number of patients with THS. Radiotherapy also has reportedly alleviated symptoms of THS in one patient refractory to immunosuppressive therapy and in another patient who became steroid dependent [65]. Following traumatic or iatrogenic operative injury, corticosteroids may be instituted while surgical intervention is considered.

Conclusion

OASs represent a heterogeneous group of disorders that result from a number of etiologies. A systemic disease (ie, infection, neoplasm, autoimmune condition) may precede the OAS, and a thorough history with review of systems may offer diagnostic clues; however, an OAS may also be isolated and herald a systemic process. Neuroimaging, preferably with MRI, can confirm the clinical findings of an OAS and identify sites that may be sampled if the diagnosis remains unclear.

Treatment is directed at the underlying cause and may require neurosurgical, otoaryngologic, neurologic, or medical consultation. Corticosteroids may be helpful if an inflammatory cause for an OAS is suspected, but should be used judiciously, particularly if an infectious etiology is being considered.

References and recommended reading

Papers of particular interest, published within the annual period of review, are highlighted as:

- Of special interest
- Of outstanding interest

hours when treated with corticosteroids; and exclusion of other causes of painful ophthalmoplegia.


30 This is a case report and literature review of Burkitt lymphoma presenting with bilateral cavernous sinus lesions. A 9-year-old boy presented with left-sided toothache, headache, and, subsequently, vertical diplopia. Examination revealed a third nerve palsy, and cranial MRI showed bilateral cavernous sinus lesions. Blood smear and bone marrow biopsy revealed Burkitt lymphoma, which was treated with chemotherapy.


35 The authors discuss their treatment of five orbital peripheral nerve tumors. Two cases, both schwannomas, involved the orbital apex and superior orbital fissure. Their surgical management is outlined.


37 This is a case report of a 75-year-old woman who presented with a 1-month history of decreased vision, diplopia, retroorbital pain, and exophthalmos. CT and MR revealed a 12 × 16-mm mass involving the orbital apex. The mass was surgically removed and pathology revealed a benign ciliary neurinoma. The patient’s pain resolved and visual acuity was partially restored.


46 This is a case report of a 21-year-old fair-skinned man with a 10-day history of progressive bifrontal headache and diplopia. Partial third, fourth, and sixth nerve palsies were observed initially, and the patient’s pupil reaction was blunted. CT of the brain and orbits was normal. Symptoms progressed to complete ptosis and ophthalmoplegia with a fixed, dilated pupil. Routine blood tests and MRI of the brain and orbits with gadolinium contrast were normal at this time. Excisional biopsy of two skin nevi showed dysplastic nevi but no malignant features. Diagnosis of malig- nant melanoma was made with a core biopsy of a 4.1-cm axillary lymph node. Despite whole-brain radiotherapy, the patient died 4 months after initial presenta- tion after developing multiple metastases.


49 This is a case report of a 34-year-old man with severe, recurrent epistaxis who underwent external anterior and posterior ethmoidal artery ligation on the right side. Severe visual loss from optic neuropathy and complete ophthalmoplegia devel- oped after surgery. CT revealed surgical clips within the right orbital apex. Emer- gent removal of the surgical clips and medial wall decompression were performed. Despite prompt recognition and treatment, severe visual loss and ophthalmoplegia persisted.


56 This is a case report of an 89-year-old woman with a history of hypertension and Paget disease who developed right-sided headache. Two years earlier she had fallen, resulting in a fracture of her arm; weeks later, she developed pain and swell- ing in the right eye followed by numbness of the right forehead and blurred vision. Neurologic examination revealed right-sided ptosis, esotropia, complete ophthal- moplegia, and decreased sensation in the V1 and V2 dermatomes. MRI and MR angiography showed bilateral cavernous carotid aneurysms with thrombi. At- tempted endovascular occlusion of the aneurysm on the right side was unsuccess- ful.


This is a retrospective review of 101 cases of direct dural carotid–cavernous and orbital arteriovenous fistulas. The diagnostic triad of arterIALIZED loops, exophthalmos, and glaucoma are discussed. Diagnostic procedures such as ultrasound, color Doppler of the orbit and carotid systems, and MRI and MR angiography are reviewed. Management strategies including conservative therapy, balloon embolization, and direct or indirect surgery are discussed. Of the 10 orbital arteriovenous shunts with signs of dural fistulas, findings spontaneously resolved in eight patients, one patient required direct surgery (which was successful), and one patient’s nonprogressive orbital findings persisted.


This is a study to determine the value of high-field MRI for diagnosis and surgery of sellar lesions. High-field MR images were obtained with 3-T MRI, with emphasis on sellar and parasellar structures in 21 patients. Three-tesla MR images were compared with standard 1- to 1.5-T MR images already obtained with intraoperative findings with attention to the medial border of the cavernous sinus to assess for possible invasion of a sellar tumor. Three-tesla MRI was superior to standard MRI for predicting tumor invasion through the medial cavernous sinus border. Better delineation of the lateral sinus compartment was also observed with 3-T MRI. Identification of the cavernous sinus segments of the third, fourth, fifth (V1 and V2), and sixth cranial nerves was also improved with 3-T MRI. Three-tesla MRI was found to be superior to standard MRI for delineation of parasellar anatomy and tumor infiltration of the cavernous sinus, and may be valuable for intraoperative navigation.


This is a retrospective review of the treatment and clinical outcomes of 22 orbital inflammations and infections, with a subgroup involving the orbital apex. The surgical approach in each of these cases was determined by the anatomic location of the disease process within the orbit. A transantral approach was used in one patient with a mucocele involving the orbital apex and maxillary sinus. A pterional extradural approach was useful in two patients with lesions of the orbital apex. A pterional intradural operation was performed in five patients with inflammation of the optic canal extending into the intracranial space.

