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Vertical Diplopia and Ptosis from Removal of the Orbital Roof in Pterional Craniotomy

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Purpose: To describe a newly recognized clinical syndrome consisting of ptosis, diplopia, vertical gaze limitation, and abduction weakness that can occur after orbital roof removal during orbito-zygomatic-pterional craniotomy.

Design: Case series.

Participants: Eight study patients (7 women), 44 to 80 years of age, with neuro-ophthalmic symptoms after pterional craniotomy.

Methods: Case description of 8 study patients.

Main Outcome Measures: Presence of ptosis, diplopia, and gaze limitation.

Results: Eight patients had neuro-ophthalmic findings after pterional craniotomy for meningioma removal or aneurysm clipping. The cardinal features were ptosis, limited elevation, and hypotropia. Three patients also had limitation of downgaze and 2 patients had limitation of abduction. Imaging showed loss of the fat layers that normally envelop the superior rectus and levator palpebrae superioris. The muscles appeared attached to the defect in the orbital roof. Ptosis and diplopia developed in 2 patients despite Medpor titanium mesh implants. Deficits in all patients showed spontaneous improvement. In 2 patients, a levator advancement was required to repair ptosis. In 3 patients, an inferior rectus recession using an adjustable suture was performed to treat vertical diplopia. Follow-up a mean of 6.5 years later revealed that all patients had a slight residual upgaze deficit, but alignment was orthotropic in primary gaze.

Conclusions: After pterional craniotomy, ptosis, diplopia, and vertical gaze limitation can result from tethering of the superior rectus–levator palpebrae superioris complex to the surgical defect in the orbital roof. Lateral rectus function sometimes is compromised by muscle attachment to the lateral orbital osteotomy. This syndrome occurs in approximately 1% of patients after removal of the orbital roof and can be treated, if necessary, by prism glasses or surgery. *Ophthalmology 2015;122:631-638 © 2015 by the American Academy of Ophthalmology.*

The development of the fronto-temporal-sphenoidal approach has greatly facilitated neurosurgical access to lesions around the base of the skull.[1–5] It is also known as the pterional approach because it involves the conjunction of the frontal, parietal, sphenoid, and temporal bones. In a refinement of the pterional approach, an orbitozygomatic craniotomy was added to allow a more tangential approach to lesions in the anterior cranial fossa, such as aneurysms and meningiomas.[6–9] After surgery, a permanent defect often remains in the orbital roof, bringing orbital contents into direct contact with meningeal tissue at the base of the frontal lobe.

Over the past 15 years, we have encountered 8 patients with a constellation of neurovisual findings that constitute a new clinical syndrome caused by orbito-zygomatic-pterional craniotomy. The patients had various combinations of vertical diplopia, limited upgaze, restricted downgaze, and ptosis after neurosurgical exploration of the anterior cranial fossa. Some patients also had a horizontal component to their gaze misalignment with limited abduction. This article describes the neuro-ophthalmic complications of pterional craniotomy involving the orbit, with emphasis on the likely mechanism, prevention, and treatment.

Methods

This study was approved by the University of California, San Francisco, Institutional Review Board and adhered to the tenets of the Declaration of Helsinki. The electronic patient records of a single neuro-ophthalmologist (J.C.H.) from 1999 through 2013 were searched to identify patients who were referred after pterional craniotomy for either ptosis, diplopia, or limitation of eye movements. Patients with a malignancy, benign tumor invading the orbit, or cranial nerve palsy were excluded. There were 8 patients who met the search criteria (Table 1). In each case, a portion of the orbital roof had been removed to gain better access to the anterior cranial fossa during the pterional approach. The operative report, computed tomography (CT) or magnetic resonance imaging (MRI), intracranial pathologic features, neurovisual examination, ocular deviation, and degree of ptosis were reviewed. The subsequent course of treatment was recorded. Patients were contacted and invited to return to the clinic to document their long-term outcome.

The operative technique varied from case to case, depending on the lesion being resected. The patient was placed supine on the operating room table with the head stabilized in a Mayfield head holder and turned 45° to the side. A pterional skin incision was made with a scalpel and clips were applied to the skin edges for hemostasis. The scalp was elevated anteriorly and the superficial temporal fascia was dissected as a free flap from the deep

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Table 1. Summary of Patients with Neuro-ophthalmic Complications Resulting from Orbital Roof Removal During Pterional Craniotomy

Patient No.	Age (yrs)	Gender	Neurosurgical Lesion	Motility Deficit	Primary Gaze Deviation	Ptosis	Follow-up/Intervention
1	55	M	Anterior communicating artery aneurysm	Right elevation & abduction	30 PD right hypotropia & 12 PD right esotropia	No	Left inferior rectus recession
2	69	F	Right sphenoid wing meningioma	Right elevation & depression	Orthotropia	Yes	Levator repair
3	58	F	Right sphenoid wing meningioma	Right elevation	20 PD right hypotropia	Yes	Prism glasses
4	44	F	Right supraclinoid internal carotid artery aneurysm	Right elevation & depression	Right hypotropia	Yes	No light perception right eye, no intervention
5	53	F	Anterior communicating artery aneurysm	Right elevation	10 PD right hypotropia	Yes	Right inferior rectus recession & levator repair
6	55	F	Anterior communicating artery aneurysm	Left elevation	Orthotropia	Yes	No treatment needed
7	54	F	Left middle cranial fossa meningioma	Left elevation & abduction	25 PD left hypotropia & 25 PD left esotropia	Yes	Left inferior & medial rectus recession
8	80	F	Left sphenoid wing meningioma	Left elevation & depression	40 PD left hypertropia	Yes	Monitoring

F = female; M = male; PD = prism diopters.

temporalis fascia to protect the facial nerve. After the zygoma and the superior and lateral orbital rims were exposed, the periorbita was dissected away from the inner surface of the orbital wall. The temporalis muscle was reflected inferiorly. A pterional craniotomy was performed with a Midas Rex drill saw (Medtronic, Minneapolis, MN) equipped with a footplate attachment. After the bone flap was removed, the dura was opened and tacked up circumferentially to the bone edge.

At this point, a series of osteotomy cuts with a reciprocating saw were made to release the superior and lateral orbital wall as a single unit. These consisted of a linear, parasagittal cut in the medial orbit, a perpendicular cut in the posterior orbit extending mediolaterally, and a cut in the lateral orbital wall from the inferior orbital fissure to the pterion (Fig 1). Additional bone was removed from the orbital roof as necessary using a Lempert rongeur. The intraorbital contents were retracted from the orbital roof with a malleable retractor to guard them from injury during the saw cuts. Moist Telfa pads or cottonoid sponges were placed over the orbital tissues for protection. The dura was opened in a curvilinear fashion over the orbital roof to provide access to the tumor or aneurysm located underneath the frontal lobe.

After lesion resection, the dura was closed with running 4-0 Dexon suture. The pterional bone flap and orbital bone fragment were replaced with hydroxyapatite cement, plates, and screws. In some patients a Medpor titanium mesh (Stryker, Kalamazoo, MI) was used to close the orbital roof defect. The temporalis muscle and its fascia were reapproximated with interrupted 3-0 Dexon sutures and the galea was closed with running 2-0 Dexon sutures. A subgaleal drain was placed and brought out through a separate skin incision.

Results

Patient 1

Patient 1 is a 55-year-old man who underwent clipping of a ruptured anterior communicating artery aneurysm. The patient reported vertical diplopia immediately after the operation. One year later, there was nearly complete absence of elevation (Fig 2). In primary position, there were 30 prism diopters (PD) of right hypotropia and 12 PD of right esotropia. In upgaze, there were 45 PD of right hypotropia. In downgaze he could fuse. Horizontal gaze was full, except for slight limitation of abduction in the right eye. Surprisingly, there was no appreciable ptosis.

A 3-dimensional CT reconstruction showed a 2-cm defect in the roof of the orbit (Fig 3). An oblique CT image revealed that the superior rectus muscle followed an abnormal trajectory to the orbital apex. It was tented upward in mid course, where it appeared adherent to soft tissue filling the bony defect. A coronal T1-weighted MRI scan showed disruption of the superior rectus and levator palpebrae superioris muscles (Fig 4A). The normal fat plane between the 2 muscles could not be identified. In addition, the layer of fat that usually surrounds the 2 muscles was absent. A contrast-enhanced image confirmed that the superior rectus—levator palpebrae superioris complex was damaged (Fig 4B). In addition, the lateral rectus appeared displaced toward the temporalis muscle.

Prism glasses were prescribed but were unsatisfactory for restoration of fusion in primary gaze. An 8-mm recession of the inferior rectus muscle was performed 16 months after the pterional craniotomy using an adjustable suture technique. Forced ductions under anesthesia just before the muscle surgery showed restriction of elevation. After surgery, the patient was able to fuse in primary gaze.

Patient 2

Patient 2 is a 69-year-old woman with a history of vision loss in the right eye resulting from a sphenoid wing meningioma. The lesion was resected via a right fronto-temporal-sphenoidal craniotomy. The orbital roof was removed up to the superior orbital fissure using rongeurs. Difficulty was encountered because of the abnormal thickness of the sphenoid bone. A Midas Rex drill was used, requiring bipolar cautery, bone wax, and Surgicel (Ethicon, Somerville, NJ) to achieve hemostasis. Although the orbital contents were protected with cottonoid sponges, the levator palpebrae superioris and superior rectus muscles were damaged.

Immediately after surgery, the patient had complete right ptosis and little vertical eye movement. Ten weeks later, the ptosis was still complete. There was 80% elevation and 30% depression of the globe. Six months later, the right palpebral fissure measured 5 mm. The eyelid just cleared the pupil and the patient reported no diplopia in primary gaze. However, there was still limitation of

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