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Giant anterior clinoidal meningiomas: surgical technique and outcomes

Clinical article

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Object. Surgery for giant anterior clinoidal meningiomas that invade vital neurovascular structures surrounding the anterior clinoid process is challenging. The authors present their skull base technique for the treatment of giant anterior clinoidal meningiomas, defined here as globular tumors with a maximum diameter of 5 cm or larger, centered around the anterior clinoid process, which is usually hyperostotic.

Methods. Between 2000 and 2010, the authors performed 23 surgeries in 22 patients with giant anterior clinoidal meningiomas. They used a skull base approach with extradural unroofing of the optic canal, extradural clinoidectomy (Dolenc technique), transdural debulking of the tumor, early optic nerve decompression, and early identification and control of key neurovascular structures.

Results. The mean age at surgery was 53.8 years. The mean tumor diameter was 59.2 mm (range 50–85 mm) with cavernous sinus involvement in 59.1% (13 of 22 patients). The tumor involved the prechiasmatic segment of the optic nerve in all patients, invaded the optic canal in 77.3% (17 of 22 patients), and caused visual impairment in 86.4% (19 of 22 patients). Total resection (Simpson Grade I or II) was achieved in 30.4% of surgeries (7 of 23); subtotal and partial resections were each achieved in 34.8% of surgeries (8 of 23). The main factor precluding total removal was cavernous sinus involvement. There were no deaths. The mean Glasgow Outcome Scale score was 4.8 (median 5) at a mean of 56 months of follow-up. Vision improved in 66.7% (12 of 18 patients) with consecutive neuroophthalmological examinations, was stable in 22.2% (4 of 18), and deteriorated in 11.1% (2 of 18). New deficits in cranial nerve III or IV remained after 8.7% of surgeries (2 of 23).

Conclusions. This modified surgical protocol has provided both a good extent of resection and a good neurological and visual outcome in patients with giant anterior clinoidal meningiomas.
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Key Words • **anterior clinoid process** • **Dolenc approach** • **meningioma** • **optic nerve** • **skull base** • **surgical technique**

Although ACP meningiomas were reported in 1938 by Cushing and Eisenhardt,[6] a clear definition of anterior clinoidal meningiomas as a distinct entity is lacking, making analysis of the literature difficult. Meningiomas in the anterior skull base may be referred to as medial sphenoid ridge,[1,14,18,24] optic canal,[2] asymmetrical tuberculum sellae, or cavernous sinus meningiomas, and may be classified as clinoidal meningiomas when they extend to the region of the ACP.[1,4,7,14,18,24]

While an ACP origin may be clearly seen in small clinoidal meningiomas, giant tumors involve relatively large areas of the anterior skull base including the ACP, sphenoid wing, tuberculum sellae, and planum sphenoidale. With such large lesions it is difficult to determine whether the tumor originated from the ACP or extended to the clinoidal area from nearby structures. We defined giant anterior clinoidal meningiomas as globular tumors with a maximum diameter of 5 cm or larger, centered around the ACP, which is usually hyperostotic.

Complete surgical removal of anterior clinoidal meningiomas remains challenging, especially when the tumors become large or giant. These tumors usually compress, displace, or encase vital neurovascular structures in the vicinity of the ACP, such as the optic nerve, the oculomotor nerve, and the ICA and its branches. Peritumoral edema contributes to the difficulty of resecting these deep skull base tumors. An anterior clinoidal meningioma may

Abbreviations used in this paper: AChA = anterior choroidal artery; ACP = anterior clinoid process; CN = cranial nerve; FSR = fractionated stereotactic radiotherapy; GOS = Glasgow Outcome Scale; GTR = gross-total resection; ICA = internal carotid artery; ICH = intracerebral hemorrhage; MCA = middle cerebral artery; PCA = posterior cerebral artery.

also extend into the cavernous sinus. The extent of removal is dependent on the surgeon's ability to safely dissect tumor from these critical structures.

Several patient series describing surgical techniques and outcomes in medial sphenoid ridge and anterior clinoidal meningiomas have been published (Table 1).[1,2,4,7,8,10,13,17,18,20,22–24,27] We present our addition to the literature, which has the following distinguishing features: 1) inclusion of only tumors that satisfied the strict definition of giant anterior clinoidal meningiomas; 2) use of our surgical protocol, initially based on the original Dolenc extradural-intradural technique to the cavernous sinus[9,28] with significant modifications that we have developed for the removal of these difficult tumors; and 3) the large size of the series.

In this article, we describe our surgical technique and nuances in detail. The primary goals of surgery were maximal possible tumor resection with minimal complications and morbidity, and improvement of visual function in patients whose preoperative vision was compromised.

Methods

Patient Population

Between November 2000 and May 2010, 23 surgeries were performed in 22 patients for the removal of giant anterior clinoidal meningiomas. One patient underwent a second operation due to residual tumor regrowth after 42 months. Inclusion criteria for the study were diagnosis of a globular anterior clinoidal meningioma centered around the ACP with a maximum diameter of 5 cm or larger. All patients underwent follow-up on an outpatient basis after surgery.

A prospective database of consecutive patients was maintained. Files and imaging data were analyzed retrospectively to augment information in the database. Data regarding patients' medical history; physical, neurological, and radiological examinations; operative reports; surgical outcome; hospitalization; and long-term follow-up were recorded (Table 2). Tumor presentation on pre- and postoperative as well as follow-up contrast and noncontrast head CT scans, and T1- and T2-weighted MRI studies with and without Gd was recorded. Visual field (Humphrey Field Analyzer II, Zeiss Humphrey Systems, Carl Zeiss Ophthalmic Systems, Inc.) and visual acuity examinations were performed before and after surgery. The Hebrew University-Hadassah Medical Center Institutional Review Board waived the requirement for informed consent for this study.

Surgical Technique

The surgery was performed after induction of general anesthesia, using an operating microscope and neuronavigation (Trion, Medtronic) with microsurgical techniques.

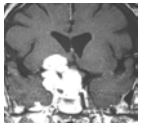
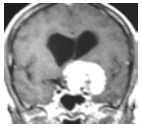
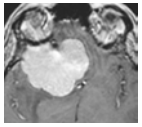
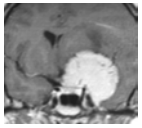
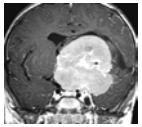
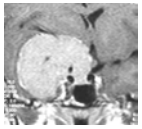
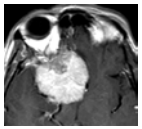
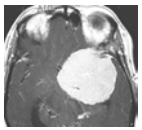
We applied pterional, frontoorbital, and frontoorbi-

TABLE 1: Series of clinoidal and medial sphenoid wing meningiomas reported in the literature*

Authors & Year	Tumor Location	No. of Pts	Mean Tumor Diameter (cm)	GTR (%)†	Improved Vision (%)‡	FU§	Tumor Recurrence/ Regrowth (%)	Mortality (%)
Al-Mefty, 1990	ACP	24	NA	89	10	4.8 yrs	4	8
Risi et al., 1994	ACP	34	NA	59	30	1.9 yrs	21	6
Day, 2000	sphenocavernous	6	>5	66	NA	3 mos	0	0
Goel et al., 2000	ACP	60	NA¶	50	25.4	26 mos	1.67	5
Lee et al., 2001	ACP	15	3.7	87	75	3.1 yrs	0	0
Tomasello et al., 2003	sphenocavernous	13	5.7	77	NA	48.3 mos	15.4	15.4
Abdel-Aziz et al., 2004	sphenocavernous	38	>3	58	NA	96 mos	10.5	0
Nakamura et al., 2006	MSW	108	NA	42.5	40	79 mos	20.3	0
Russell & Benjamin, 2008	MSW	35	4.5	69	73	12.8 yrs	9	0
Behari et al., 2008	MSW	20	6.1	45	15.8	17.6 mos	NA	5
Pamir et al., 2008	ACP	43	3.4**	90.7	84.6	median 39 mos	9.3	0
present study	ACP	22	5.9	30.4	66.7	56 mos	13.6	0

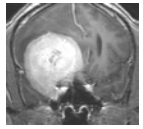
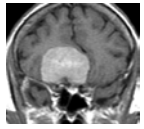
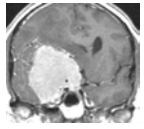
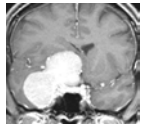
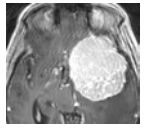
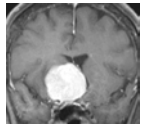
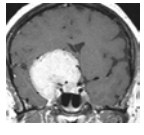
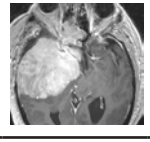
* FU = follow-up; MSW = medial sphenoid wing; NA = not available; Pts = patients.
† Simpson Grade I or II.
‡ Percentage of patients presenting with visual deterioration, who underwent pre- and postoperative visual examinations.
§ Values are the means unless stated otherwise.
¶ A total of 22 giant tumors (> 5 cm) were included as part of a larger series that also included non–giant tumors.
** Mean diameter in 42 patients.

TABLE 2: Demographic and clinical data*

Case No.	Age (yrs), Sex	Tumor Diameter (mm)	Preop Image	CS Involved	Vascular Involvement†	Optic Canal Involved	WHO Grade	Simpson Grade	Location of Residual Tumor	Surgical Complications	Visual Outcome	GOS	Recurrence/ Residual Tumor Regrowth	Adjuvant FSR	FU (mos)
1	73, F	55		no	ICA, ACA, MCA	yes	I	III	supraclinoid ICA, brainstem	partial ptosis	no FU visual exam	5	yes (108 mos)	offered	129
2	52, F	50		no	ICA, ACA, MCA	yes	I	III	CN III	none	improved	5	no	no	129
3	41, F	63		no	ICA, ACA, MCA	yes	I	II	GTR	none	improved	5	no	no	112
4	44, F	61		yes	ICA, ACA, MCA	yes	I	IV	CS, ON, MCA, suprasellar cistern	hemiparesis, dysphasia	deteriorated	4	yes (42 mos), reop	yes	93
5	6, M	63		no	ICA, ACA, MCA	yes	I	II	GTR	none	stable	5	no	no	15
6	49, F	52		yes	ICA, ACA, MCA	yes	I	III	CS	pseudomeningocele‡	stable	5	no	no	74
7	54, F	59		yes	ICA, ACA, MCA	yes	I	III	CS	none	improved	5	no	no	78
8	42, F	58		yes	ICA, ACA, MCA	yes	I	III	CS	pseudomeningocele‡	stable	5	no	no	87

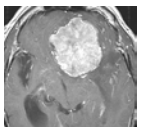
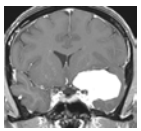
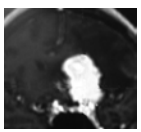
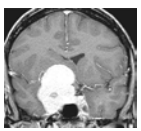
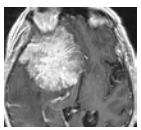
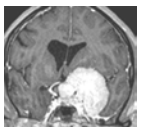
(continued)

TABLE 2: Demographic and clinical data* ***(continued)***

Case No.	Age (yrs), Sex	Tumor Diameter (mm)	Preop Image	CS Involved	Vascular Involvement†	Optic Canal Involved	WHO Grade	Simpson Grade	Location of Residual Tumor	Surgical Complications	Visual Outcome	GOS	Recurrence/ Residual Tumor Regrowth	Adjuvant FSR	FU (mos)
9	48, M	62		yes	ICA, ACA, MCA	yes	II	IV	CS, ON	pseudomeningocele‡	improved	5	no	yes	70
10	62, F	50		no	ICA	yes	I	II	GTR	pseudomeningocele‡	deteriorated	5	no	no	54
11	55, M	66		yes	ICA, ACA, MCA	yes	I	IV	CS, CN III, posterior fossa	none	improved	5	no	yes	51
12	61, M	56		yes	ICA, ACA, MCA	yes	I	IV	CS, CN III, posterior fossa	ptosis‡, CN IV palsy pseudomeningocele‡	improved	5	no	yes	51
13	75, F	65		yes	ICA, ACA, MCA	yes	I	IV	CS, CN III, MCA	pseudomeningocele‡	improved	5	yes (27 mos)	offered	48
14	55, F	52		no	ICA, ACA	yes	I	II	GTR	none	improved	5	no	no	46
15	51, F	52		yes	ICA, ACA, MCA	yes	I	III	CS	ptosis‡	improved	5	no	no	43
16	56, M	85		yes	ICA, MCA	yes	II	III	CS	intraop pneumothorax, ICH, coma‡, hemiparesis	no preop exam, no FU visual exam	3	no	no	31

(continued)

TABLE 2: Demographic and clinical data* *(continued)*

Case No.	Age (yrs), Sex	Tumor Diameter (mm)	Preop Image	CS Involved	Vascular Involvement†	Optic Canal Involved	WHO Grade	Simpson Grade	Location of Residual Tumor	Surgical Complications	Visual Outcome	GOS	Recurrence/ Residual Tumor Regrowth	Adjuvant FSR	FU (mos)
17	75, M	62		no	ICA (displaced)	no	II	II	GTR	none	no preop exam	5	no	no	27
18	54, F	50		no	ICA, MCA	no	I	II	GTR	none	improved	5	no	no	24
19	68, F	55		no	ICA (displaced)	no	I	II	GTR	none	improved	5	no	no	24
20	42, F	50		yes	ICA, ACA, MCA, AChA	no	I	IV	CS, CN III, posterior fossa, PCoA, AChA	stroke, hemiparesis, hydrocephalus, CN III palsy, traumatic PCA aneurysm§	stable	4	no	yes	16
21	63, F	76		yes	ICA, ACA, MCA	yes	I	IV	CS, ON, CN III, posterior fossa	ptosis‡, pseudomeningocele‡	no preop exam, no FU visual exam	5	no	no	15
22	58, F	61		yes	ICA, ACA, MCA	no	I	III	CS	PE, ptosis‡, pseudomeningocele‡	improved	5	no	no	16

* ACA = anterior cerebral artery; CS = cavernous sinus; exam = examination; ON = optic nerve; PCoA = posterior communicating artery; PE = pulmonary embolism.

† Vascular structures were encased in all cases except those in which the structures are noted as displaced.

‡ Transient.

§ A traumatic PCA aneurysm was diagnosed and coiled 8 months after surgery.

tozygomatic craniotomies using techniques that are well described,[3,16,29] and we proceeded with the extradural approach. With the aid of a microdrill, the superior orbital fissure was exposed, and the meningoorbital band was transected, facilitating access to the ACP. For these steps, extradural cerebral retraction was needed. In most cases we made several tiny cuts at the basal frontal dura to release CSF. When the brain was edematous and tight, we used a transdural tumor-debulking maneuver, which provided more room to continue the extradural approach. For this maneuver, we debulked the tumor with the aid of a Cavitron ultrasonic surgical aspirator (CUSA, ValleyLab) under neuronavigation for safe transdural entry into adjacent tumor, avoiding injury to neurovascular structures.

Then, using a diamond bur with copious irrigation, the superior wall of the optic canal was unroofed extradurally (Fig. 1), the dural sleeve of the optic nerve was exposed, and complete clinoidectomy was accomplished.[20] At this point the optic nerve medially, the clinoidal segment of the ICA lateral and inferior to the optic nerve, and the oculomotor nerve lateral to the ICA came into view (Fig. 2 upper). In cases in which the tumor reached the lateral wall of the cavernous sinus and/or invaded the cavernous sinus, we peeled the outer layer of the lateral wall of the cavernous sinus extradurally, from anterior to posterior, exposing the inner membranous layer.

Opening of the Dura Mater

If the dura mater had been opened at the frontal base for transdural tumor debulking, we continued the incision medially through the falciform ligament and optic sheath. If there was no need for transdural debulking, we opened the dura in the lateral part of the optic canal and continued the incision medially to the optic nerve through the falciform ligament, and then laterally along the distal carotid ring (Fig. 2 lower). We also added a perpendicular incision corresponding to the basal projection of the medial sylvian fissure. This dural opening was sufficient for detaching the tumor from the skull base, accessing and opening the basal cisterns, and releasing additional CSF, which immediately relieved brain and dural tension. As the tumor was removed, we extended the dural incision from inside out.

Tumor Resection

We continued to work intradurally and localized the following structures: 1) the prechiasmatic part of the optic nerve, by going backward from its exposed intracanalicular segment; 2) the supraclinoid segment of the ICA, by following its clinoidal segment distally; and 3) the oculomotor nerve lateral to the ICA. After exposing these important neurovascular structures, we began to debulk the adjacent basal part of the tumor using the usual techniques. After the deep basal and central parts of the tumor had been removed, we opened the dura mater of the convexity and dissected the peripheral parts of the tumor from the frontal and temporal lobes. The distal sylvian fissure was split, and the distal MCA branches and M_2 segments were located. Then we used a bidirectional dissection technique to remove the rest of the tumor.

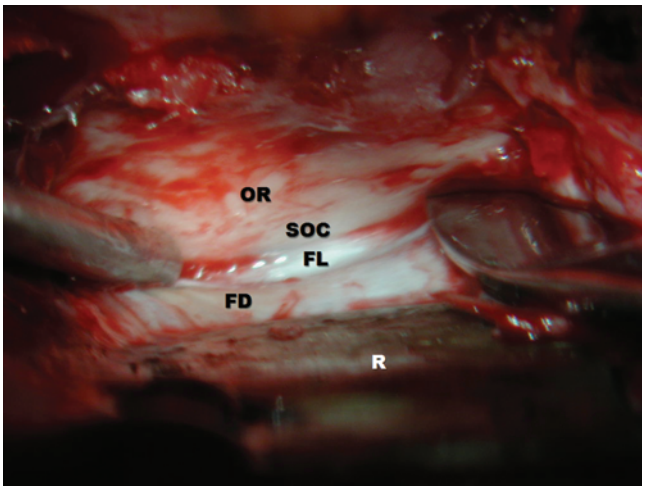


Fig. 1. Extradural exposure of the optic canal. After a left-sided craniotomy, the frontal dura (FD) is elevated, exposing the orbital roof (OR) and falciform ligament (FL), which covers the optic nerve. R = retractor blade; SOC = superior wall of the optic canal.

Whenever possible, we performed a GTR, including removal of the dura mater involved by the tumor. We left residual tumor in cases in which it had invaded the cavernous sinus or when it was very adherent to the optic nerve, oculomotor nerve, and/or the ICA and its branches.

Adjuvant Treatment

Fractionated stereotactic radiotherapy was offered in some cases of large residual tumors or residual growth, especially in younger patients (Table 2).

Results

Patient Population

The mean age at surgery was 53.8 years (range 6–75 years). There were 6 male and 16 female patients. The mean tumor diameter was 59.2 mm (range 50–85 mm) (Table 2); 13 meningiomas were located on the right side and 9 were on the left. The most common presenting symptoms were visual impairment (86.4%), cognitive and memory deficit (50.0%), headache (45.5%), limb weakness (27.3%), and seizures (22.7%) (Table 3). All patients underwent clinical follow-up at a mean of 56 months after surgery (range 15–129 months).

Radiological Presentation and Intraoperative Findings

The most prominent radiological findings were clinoid hyperostosis (90.9%), midline shift (90.9%), and cerebral edema (86.4%). The tumor invaded the optic canal in 17 patients (77.3%) and was adherent to the lateral wall of the cavernous sinus in 18 (81.8%). The superior orbital fissure and the cavernous sinus were involved by the tumor in 13 patients (59.1%) (Table 4). The prechiasmatic region of the optic nerve was involved by the tumor in all patients; in 13 (59.1%) it was compressed by the tumor and in 9 (40.9%) it was encased. In the vast majority of patients, the ICA and its main branches were encased by tumor (Table 2).

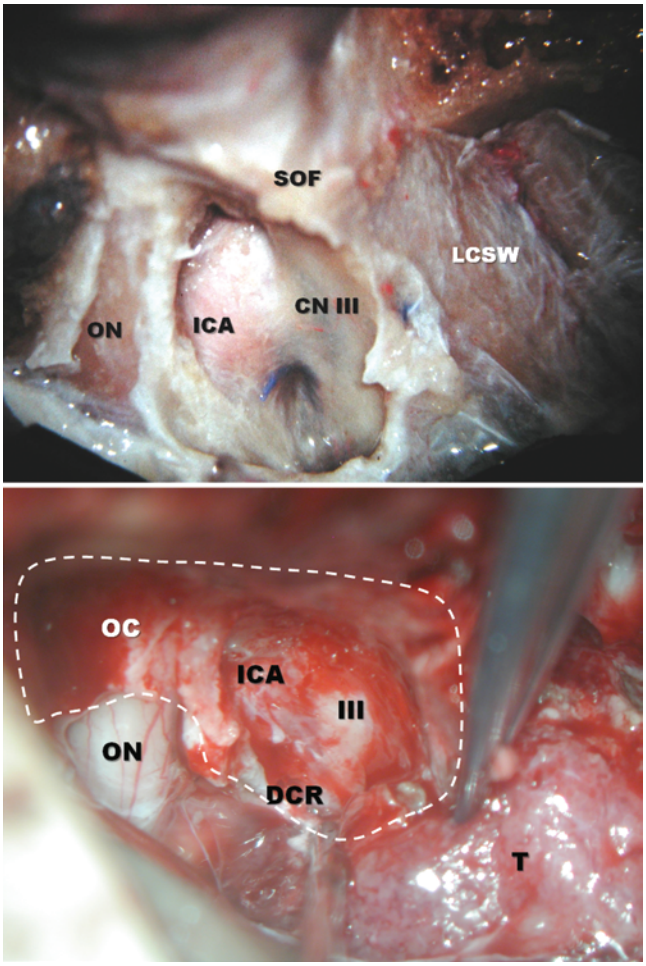


Fig. 2. Exposure after extradural anterior clinoidectomy. **Upper:** Cadaver dissection of the right side demonstrating the surgical anatomy after extradural unroofing of the optic canal and extradural clinoidectomy. The optic nerve (ON) has been exposed. The clinoidal segment of the ICA is inferior and lateral to the optic nerve; the oculomotor nerve is seen lateral to the ICA en route from the lateral wall of the cavernous sinus (LCSW) into the superior orbital fissure (SOF). **Lower:** Intraoperative photograph of the right side demonstrating the preserved extradural neurovascular anatomy, which is not violated by the intradural tumor. This cornerstone of well-preserved anatomy serves as the starting point of a "roadmap" for safer tumor resection. DCR = distal carotid ring; OC = optic canal; T = tumor; III = oculomotor nerve.

Surgical Results

A frontoorbital craniotomy was the most frequent approach. Typically, craniotomy was followed by extradural clinoidectomy, which was complete in 16 patients (72.7%) and partial in 3 (13.6%) (Figs. 3 and 4).

Total resection (Simpson Grade I or II) was achieved in 7 surgeries (30.4%); subtotal (Simpson Grade III) and partial (Simpson Grade IV) resections were achieved in 8 surgeries (34.8%) each.[26] The main factor precluding total removal was cavernous sinus involvement (Fig. 5). The cavernous sinus was not involved by the tumor in any patient who underwent total resection. Six patients with only minimal residual tumor in the cavernous sinus were considered to have undergone Simpson Grade III resections. In some cases, residual tumor adherent to the oculomotor nerve, the optic nerve, or blood vessels remained. At postoperative imaging, 7 patients (31.8%) had no evidence of residual tumor, and residual tumor was seen in 15 patients (68.2%) (Table 2).

TABLE 3: Presenting symptoms and signs

Signs & Symptoms	No. of Patients (%)
visual impairment	19 (86.4)
cognitive & memory deficit	11 (50.0)
headaches	10 (45.5)
limb weakness	6 (27.3)
seizures	5 (22.7)
drowsiness	3 (13.6)
confusion	2 (9.1)
vomiting	2 (9.1)
proptosis	1 (4.5)
behavioral changes	1 (4.5)
dysphasia	1 (4.5)
dizziness	1 (4.5)

World Health Organization Grade I tumors were diagnosed in 19 patients (86.4%), and WHO Grade II tumors were diagnosed in 3 (13.6%) (Table 2).[21] The most frequent pathology subtypes were meningothelial and transitional meningioma.

At a mean follow-up of 56 months after surgery, GOS scores[12] ranged from 3 to 5 (median 5, mean 4.8) (Table 2).

Morbidity and Mortality

There were no deaths during postoperative or long-term follow-up. The most common postoperative compli-

TABLE 4: Imaging findings

Finding	No. of Patients (%)
clinoid hyperostosis	20 (90.9)
cerebral displacement & herniation	20 (90.9)
calcifications	12 (54.5)
diffuse edema	11 (50.0)
local edema	8 (36.4)
hydrocephalus	6 (27.3)
pneumosinus dilatans	3 (13.6)
tumor extension	
sphenoid ridge	20 (90.9)
lat wall of CS	18 (81.8)
roof of CS	18 (81.8)
tuberculum sella	17 (77.3)
optic canal	17 (77.3)
superior orbital fissure	13 (59.1)
CS	13 (59.1)
planum sphenoidale	5 (22.7)
posterior fossa	5 (22.7)
middle fossa floor	4 (18.2)

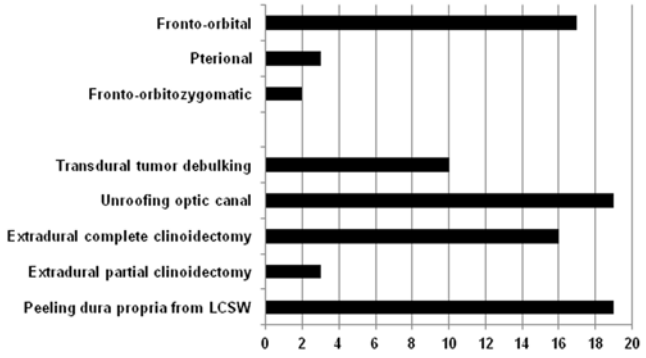


Fig. 3. Graph showing the craniotomies and extradural skull base procedures performed in 22 patients.

cation was temporary pseudomeningocele after 8 surgeries (34.8%) (Fig. 6). This was resolved with compressive dressing and wound collection tap or lumbar drainage. Postoperative CN deficit, the second most common complication, developed after 6 surgeries (26.1%). Four patients developed transient ptosis, including one who also had a permanent trochlear nerve deficit. Partial ptosis remained in another patient at the 129-month follow-up. A postoperative complete oculomotor palsy in 1 patient was improved at the 16-month follow-up. At long-term follow-up, 1 new partial CN III and 1 new CN IV deficit remained (Table 2 and Fig. 7).

New hemiparesis developed after 3 surgeries (13.0%) (Fig. 6). One patient (Case 4), who underwent reoperation 42 months after her first surgery due to residual growth, had an infarction at the distribution of the perforating branches of the MCA during the second surgery, leading to hemiparesis and dysphasia. At the 51-month follow-up, there was significant improvement in the patient's dysphasia, but her hemiparesis remained. Another patient (Case 16) had multifocal diffuse remote ICHs due to increased venous pressure and venous bleeding due to intraoperative pneumothorax and salvage positive end-expiratory pressure ventilation for correction of severe hypoxia. Postoperatively, the patient remained intubated and was comatose with significant worsening of his preexisting hemiparesis. At the 31-month follow-up, he was awake, spoke slowly, and followed commands. He remained hemiparetic and needed assistance to walk with a walker. One patient (Case 20) developed hemiparesis due to postoperative infarction at the posterior limb of the internal capsule, resulting from damage to the AChA, which was encased by tumor and bled during dissection. The patient's hemiparesis improved significantly at the 16-month follow-up. A traumatic PCA aneurysm was diagnosed and successfully treated with coils 8 months after surgery. Two patients (9.1%) developed hydrocephalus postoperatively, and ventriculoperitoneal shunts were inserted.

Eleven patients (50%) had cognitive and memory deficits at presentation; 6 (54.5%) of these patients were deficit free at follow-up. One patient (4.5%) had a persistent new short-term memory deficit after surgery at the 31-month follow-up. Ten patients presented with headaches (45.5%), which resolved completely after surgery. Four of 6 patients presenting with hemiparesis had complete resolution after surgery.

Five patients presented with seizures; 2 are seizure-free after surgery at the 24- and 27-month follow-up points. All preoperative drowsiness, confusion, vomiting, proptosis, behavioral change, dysphasia, and dizziness resolved completely after surgery.

Visual Outcome

The only patient with normal vision before surgery remained stable postoperatively and at 54-month follow-up examination. Preoperative and postoperative neuroophthalmological findings were available for comparison in 18 patients (Table 2). Vision improved after surgery in 12 (66.7%) of these 18 patients. In 8 patients both eyes improved; in 4 patients the ipsilateral eye improved and the contralateral eye was stable. In 4 other patients (22.2%) vision was stable in both eyes. Vision deteriorated in 2 patients (11.1%) (Fig. 6), including one patient who experienced ipsilateral deterioration and contralateral improvement and another who experienced ipsilateral deterioration with stable vision in the contralateral eye. No patient experienced bilateral visual deterioration after surgery. The mean visual follow-up examination was 33.1 months.

Four patients had ipsilateral blindness at their preoperative ophthalmological examination. Vision in 1 of these patients had improved to normal 9 months after surgery; 3 patients showed no improvement at long-term follow-up.

Three patients were unable to undergo neuroophthalmological examination before surgery due to compromised neurological status. In all 3 patients visual function was preserved on a level enabling normal daily activities.

Fractionated Stereotactic Radiotherapy

Residual tumors have remained stable in 14 of 17 cases (82.4%) that were treated with subtotal or partial resection (Table 2).

Four younger patients (Cases 9, 11, 12, and 20) who underwent partial resection (Simpson Grade IV) received adjuvant FSR after surgery. One patient (Case 4) received FSR after her second surgery, which was performed due to residual growth. Residual tumors in these 5 patients have remained stable. In addition, 2 older patients were offered FSR when residual regrowth was seen after 108 and 27 months (Cases 1 and 13, respectively).

Discussion

The traditional approach for the resection of anterior clinoidal meningiomas and medial sphenoid wing meningiomas has been the pterional intradural transsylvian approach, which begins with splitting the sylvian fissure, releasing CSF, and debulking the tumor, and then proceeds with peripheral tumor dissection from neurovascular structures.[8,24,29] In this series we present our experience using the original Dolenc skull base approach to the cavernous sinus[9,28] via frontoorbital, frontoorbitozygomatic, or pterional craniotomy, with a combination of extradural

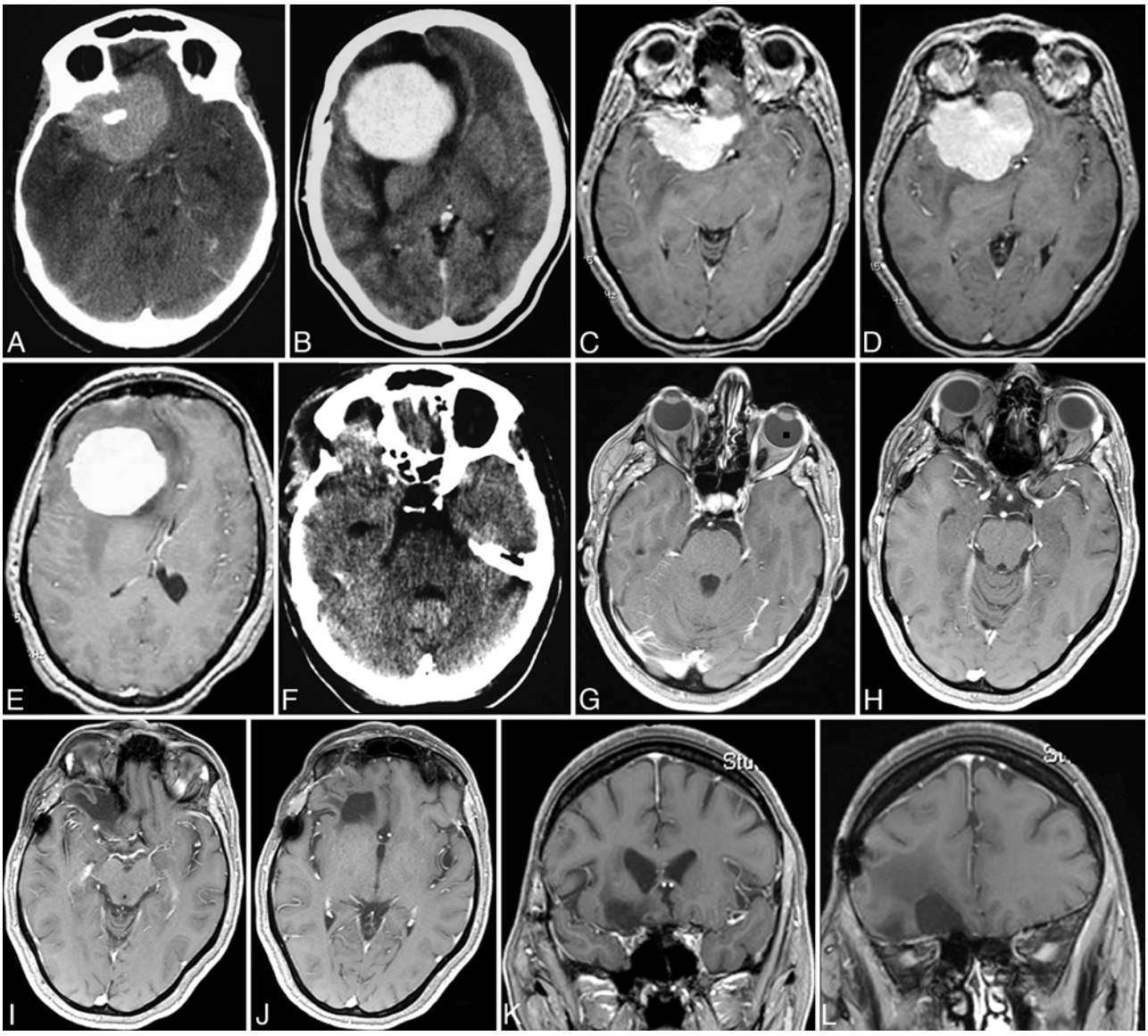


Fig. 4. Case 3. Images obtained in a 41-year-old woman with a giant clinoidal meningioma. The patient had presented with headaches, cognitive deficit, and visual complaints. **A–E:** Computed tomography scans (A and B) and MRI studies (C–E) demonstrating a giant (63-mm) right clinoidal meningioma. **F:** Postoperative CT scan showing that GTR was achieved after extradural unroofing of the optic canal and extradural clinoidectomy. **G–L:** Follow-up MRI studies obtained 8.3 years after surgery, showing no residual tumor. The patient is doing well (GOS Score 5), and her vision has improved.

and intradural techniques to remove these lesions (Figs. 3 and 4).[5,7,13,14,19]

The surgical challenges associated with these giant tumors stem from their size and difficult location, as well as the risks inherent in finding, dissecting, and preserving the critical neurovascular structures that they inevitably involve or encase, for example, the cavernous sinus, CNs, ICA, MCA, and AChA. These challenges are increased by tensed brain, secondary edema, and tumor mass effect.

The concept of an extradural approach to skull base tumors is not new. After the pioneering work of Dolenc, the technique he introduced as an approach to the cavernous sinus evolved in the hands of other surgeons for removal of medial sphenoid wing/clinoidal meningiomas. What is unusual and original in our series is the application of extradural techniques for the removal of giant clinoidal meningiomas along with the mass effect and reactive brain edema that they produce.

Strictly speaking, there are 2 main challenges in the safe removal of giant tumors: 1) how to safely locate the important arteries and the optic apparatus inside these giant tumors, and 2) how to avoid damage to tensed brain during approach, dissection, and tumor removal.

We presumed that the best way to avoid damaging the ICA and optic nerve was to locate and dissect them in areas in which the anatomy remains relatively normal, with minimal distortion from the tumor. Extradural clinoidectomy solves this problem.

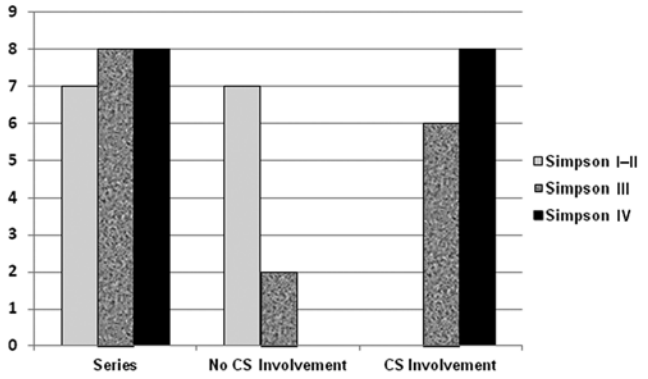


Fig. 5. Bar graph showing the extent of resection and cavernous sinus involvement in 23 surgeries for removal of giant clinoidal meningiomas. CS = cavernous sinus.

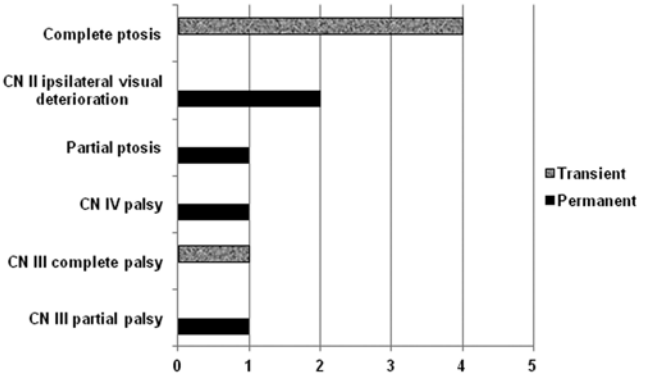


Fig. 7. Bar graph showing the number of new CN deficits after 23 surgeries for removal of giant clinoidal meningiomas.

Management of tight brain and the technique for opening the dura mater—the best brain protector—are of crucial importance here. We use the following maneuvers. As soon as the craniotomy is complete and extradural basal subfrontal retractors have been inserted, we make several tiny incisions in the frontobasal dura to allow for gradual CSF drainage. If this is not sufficient, we enter through the dura beneath the adjacent meningioma, under neuronavigation, and debulk the tumor transdurally using the Cavitron ultrasonic aspirator. The brain, contained inside the dura mater, pushes more and more of the tumor toward the surgeon, facilitating debulking and releasing brain pressure. In addition, the fact that we open the basal dura first, starting with the dural sleeve of the optic nerve and the falciform ligament, provides early optic nerve decompression and eases CSF drainage from the basal arachnoid cisterns, allowing for excellent brain relaxation. Tumor removal continues "from the inside out" with the addition of the usual transsylvian technique. This technique has the additional benefits of early tumor devascularization and detachment from the skull base.

We attribute to this technique our excellent visual outcomes, the lack of injury to major vessels during surgery, and good postoperative neurological function in this series in comparison with other studies (Table 1). The extradural skull base approach that was used in our patients is similar to the technique reported by Lee et al.[13,14]

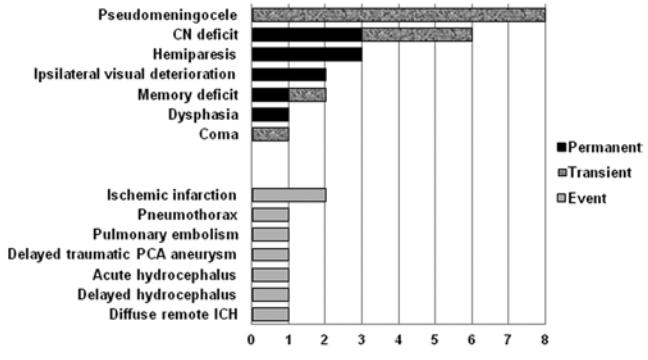


Fig. 6. Bar graph showing the number of surgical complications after 23 surgeries for removal of giant clinoidal meningiomas.

with some modifications. They reported on 15 patients with somewhat smaller anterior clinoidal meningiomas (mean diameter 3.7 cm), including 8 patients presenting with preoperative visual deficits. After surgery, vision improved in 75%.[13] This good result could be related to their extradural approach and early optic nerve decompression.

As noted earlier, direct comparison with outcomes after resection of clinoidal meningiomas that were reported in earlier series is difficult due to the lack of a clear definition for anterior clinoidal meningioma and variable tumor size. The difference between medial sphenoid wing and anterior clinoidal meningiomas is not only semantic; there are clinical implications. Involvement of the optic canal is much more frequent in anterior clinoidal meningiomas than in medial sphenoid wing meningiomas.

Among 20 patients with preoperative visual deficits due to giant medial sphenoid wing meningiomas, Behari et al.[4] attained visual improvement in 3 patients and stable visual function in 11; 5 patients experienced deterioration of vision in the ipsilateral eye at a mean of 17.6 months of follow-up. The majority of patients in the series of Behari et al. had stable vision after surgery, while in our experience most patients' vision improved. Although tumors in the series of Behari et al. were slightly larger (6.12 vs 5.92 cm in our series), the main difference is more likely in technique. The team of Behari et al. performed early extradural unroofing of the optic canal and optic nerve decompression in only 15.8% of their patients, whereas we used this technique in 86.4%.

In a series of 35 patients with medial sphenoid wing meningiomas (mean diameter 4.5 cm), Russell and Benjamin[24] reported visual improvement in 73%, stable vision in 20%, and deterioration in 7% of patients who had visual loss before surgery. Pamir et al.[20] also reported improvements in visual function in the majority of 43 patients with anterior clinoidal meningiomas with a mean tumor diameter of 3.35 cm, including 16 patients with tumors larger than 4 cm in diameter. Among 26 patients who had preoperative visual deficits, 84.6% improved and 15.4% remained stable. The Russell and Pamir teams used an intradural approach, and tumors in these 2 series were smaller on average than those presented here (Table 1).

We think that our technique also has advantages in the prevention of death, severe neurological deficit, and

vascular damage, although 1 patient in this series did develop a PCA aneurysm that was probably caused by the surgical dissection. In our series there were no deaths, in comparison with 5%–15.4% mortality in some recent series (Table 1). This emphasizes the fact that resection of medial sphenoid wing and anterior clinoidal meningiomas remains challenging, especially in cases of giant tumors. Tomasello et al.,[27] whose patients had a mean tumor diameter of 5.7 cm and of whom 77% underwent GTR, reported the highest mortality rate in patients treated via the conventional pterional intradural transsylvian approach. Behari et al.[4] and Goel et al.[10] both reported 5% mortality.

In our series 3 patients (13.6%) had postoperative hemiparesis. In 1 patient (Case 16), the hemiparesis was caused by a remote ICH due to intraoperative pneumothorax. In 2 other patients the deficit was most likely due to manipulation of perforating vessels encased by the tumor. Goel et al.[10] reported postoperative hemiplegia in 6.6% of patients. Behari et al.[4] reported 10% temporary and 5% permanent hemiparesis, and Tomasello et al.[27] reported 7.6% hemiparesis. The encasement of small perforating vessels in clinoidal/medial sphenoid wing meningiomas is a serious problem. Injury to small perforating arteries during tumor resection is a known cause of neurological deterioration, even when the large parent vessels are well preserved.

Regarding the choice of craniotomy, we prefer to use frontoorbital craniotomies for high-riding, deeply located tumors to obtain a better angle of attack. The disadvantage of this approach is a higher rate of transient postoperative ptosis (Fig. 7). There was no pupil involvement or extraocular movement deficit in 4 patients with transient isolated postoperative ptosis, or in 1 patient with permanent partial ptosis in our series. This suggests direct damage to the levator palpebrae muscle by the retractor blade as the cause of ptosis in these patients.

The most common postoperative complication in our series was subgaleal CSF collection (pseudomeningocele) at the site of surgery in 36.3% of patients (Fig. 6). Pseudomeningocele, which was resolved within 5 weeks with conservative management in all patients, is the price of a skull base approach with aggressive resection and extensive removal of dura involved by the tumor at the level of the skull base, precluding a watertight closure. We apply a collagen substitute, reinforced by fibrin glue, in lieu of dura mater. Using a frontotemporal approach, Behari et al.[4] reported pseudomeningocele at the site of surgery in 20% of patients. Nakamura et al.[18] reported pseudomeningocele in 6.5% of patients using pterional and frontolateral craniotomies.

Cavernous sinus involvement was the main impediment to complete resection.[25] In 13 patients, we left residual tumor in the cavernous sinus. We agree with other authors' recommendations regarding the use of adjuvant FSR in these patients. There is increasing evidence that radiosurgery provides good control of residual tumor in the cavernous sinus.[11,15] Fractionated stereotactic radiosurgery was performed in 5 patients with cavernous sinus residual tumors, and it was offered to 2 additional patients.

In the Russell series,[24] the Karnofsky Performance Scale score improved in 32.4% patients and worsened in 11.8% at the 3-month follow-up. Behari et al.[4] reported a good functional outcome in 65% of patients and a fair outcome in 30%. Tomasello et al.[27] noted good outcomes in 76.9% of patients and fair outcomes in 7.7%. Goel et al.[10] found that 90% of patients are leading independent and active lives at long-term follow-up. We had a very good overall outcome, with GOS scores of 5, 4, and 3 in 19 patients (86.3%), 2 patients (9%), and 1 patient (4.5%), respectively.

Conclusions

Giant anterior clinoidal meningiomas are challenging tumors. We prefer a skull base extradural approach to the tumor, including extradural unroofing of the optic canal, extradural clinoidectomy, transdural debulking of the tumor when needed, early optic nerve decompression, and early identification and control of key vascular structures, followed by removal of the remaining tumor. This technique has provided a good extent of resection, as well as a good visual and clinical outcome.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Spektor, Umansky, Shoshan. Acquisition of data: Spektor, Attia, Paldor. Analysis and interpretation of data: Spektor, Attia, Dotan. Drafting the article: Spektor, Attia, Paldor. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Spektor. Study supervision: Spektor.

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