J Neurosurg 116:728–740, 2012

Endonasal management of sellar arachnoid cysts: simple cyst obliteration technique

Technical note

Nancy McLaughlin, M.D., Ph.D.,[1] Alexander Vandergrift, M.D.,[1] Leo F. Ditzel Filho, M.D.,[1] Kiarash Shahlaie, M.D., Ph.D.,[1] Amalia A. Eisenberg, M.S.N., A.R.N.P., C.N.R.N.,[1] Ricardo L. Carrau, M.D.,[2] Pejman Cohan, M.D.,[3] and Daniel F. Kelly, M.D.[1]

[1]Brain Tumor Center, John Wayne Cancer Institute at Saint John's Health Center, Santa Monica, California; [2]Department of Head and Neck Surgery, Ohio State University, Columbus, Ohio; and [3]Department of Medicine, David Geffen School of Medicine, University of California, Los Angeles, California

Object. Symptomatic sellar arachnoid cysts (ACs) have typically been treated via the transsphenoidal route. After sellar cyst wall fenestration, some authors have advocated cyst wall resection and increasing communication between the AC and suprasellar subarachnoid space (SAS). This study is a report of the authors' experience using a simplified approach to reinforce a defective diaphragma sellae or unseen arachnoid diverticulum by deliberately not enlarging the AC-SAS communication and obliterating the cyst cavity with adipose tissue followed by skull base reconstruction.

Methods. A retrospective analysis was conducted of patients who underwent an endonasal transsphenoidal obliteration of symptomatic ACs with a fat graft and skull base repair.

Results. Between July 1998 and September 2010, 8 patients with a sellar AC were identified (6 women and 2 men, mean age 57 years). Clinical presentation included headache, pituitary dysfunction, and visual dysfunction (4 patients each group). Maximal cyst diameter averaged 22 mm (range 15–32 mm). In all cases the sellar communication to the SAS was deliberately not enlarged. The endoscope was used for visualization in 8 of 9 procedures. Postoperatively, headache improved in all 4 patients, vision in all 4 patients, and partial resolution of endocrine dysfunction (hyperprolactinemia and/or recurrent hyponatremia) occurred in 3 (75%) of 4 patients. No new endocrinopathy, CSF leak, meningitis, or neurological deficits occurred. Two patients experienced cyst reaccumulation: 1 symptomatic recurrence was treated with reoperation at 43 months postsurgery, and 1 asymptomatic partial recurrence continued to be monitored at 29 months postsurgery.

Conclusions. Sellar ACs can be effectively treated using endonasal fenestration and obliteration with fat with resultant reversal of presenting symptoms in the majority of patients. This simplified technique of AC cavity obliteration without enlarging communication to the SAS has a low risk of CSF leakage, and in most cases appears to effectively disrupt cyst progression, although longer follow-up is required to monitor for cyst recurrence.
(http://thejns.org/doi/abs/10.3171/2011.12.JNS11399)

Key Words • **arachnoid cyst** • **sella turcica** • **transsphenoidal surgery** • **endonasal surgery** • **endoscope** • **cerebrospinal fluid leak** • **skull base** • **subarachnoid space**

Sellar ACs are relatively rare. Overall, ACs comprise only approximately 1% of intracranial space-occupying masses, and sellar ACs comprise roughly 3% of all intracranial ACs.[24] Excluding our series presented here, only 68 other cases of sellar ACs have been reported in the English and French literature. While other cystic parasellar masses are more common, such as cystic pituitary adenomas, craniopharyngiomas, and Rathke cleft cysts, the rarity of sellar ACs does not preclude the need for surgical intervention when they become symptomatic.

Abbreviations used in this paper: AC = arachnoid cyst; IGF-I = insulin-like growth factor–I; SAS = subarachnoid space; UCLA = University of California, Los Angeles.

Treatment of sellar ACs has typically involved transsphenoidal fenestration of the cyst's anterior wall or, less frequently, through a craniotomy. Some authors have encouraged excision of the cyst's membranes either partially or entirely.[6] Others have advocated that a communication between the cyst cavity and the suprasellar SAS should be largely opened.[6] To our knowledge, no series has reported a homogeneously treated population of sellar ACs using obliteration of the cyst cavity alone. Furthermore, the materials used for cavity obliteration such as fat, muscle, fascia, and biological glue, as well as the reconstruction technique have not been consistently described.[4,6,23] Despite their benign nature, transsphenoidal treatment of sellar ACs has been associated with a relatively high rate of serious complications including visual loss,[32] postop-

erative CSF leak,[1,4,6,17,27,32] meningitis,[23,32] and intracranial abcess.[22]

In this paper we describe the concept and technique of a simplified endonasal approach for symptomatic sellar ACs that aims to eliminate abnormal CSF flow into the AC from the SAS. This goal is accomplished by AC obliteration with fat to reinforce a defective diaphragma sellae or arachnoid diverticulum; the communication between cyst cavity and the SAS is deliberately not enlarged. This technique is contrary to the traditional method employed for disrupting abnormal CSF flow dynamics for most intracranial ACs in which the goal is to enlarge the communication between the AC and SAS. In this report we also emphasize the enhanced visualization of the AC cavity obtained with the endoscope.[4,5]

Methods

Patient Population and Data Collection

All patients in our prospective database between July 1998 and September 2010, who underwent an initial endonasal transsphenoidal surgery for treatment of their symptomatic sellar AC and who had at least a 6-month clinical and imaging follow-up, were considered for the study. Among the 10 cases identified, 2 patients with larger cysts had a deliberate communication created between the sellar AC and the SAS at the time of surgery and were therefore excluded from this technical report. Subsequently, this approach of augmenting the AC-SAS opening was believed to be potentially counterproductive in eliminating the AC-SAS communication and was thereafter abandoned; in all subsequent patients, no communication was made or enlarged between the cyst and the SAS, and instead only cyst obliteration with fat was performed.

Patients' clinical notes, operative notes, imaging studies, and hormonal studies were reviewed. Data on lesion characteristics, detailed intraoperative observations, intra and postoperative complications, and clinical outcomes were collected. All procedures were performed by the senior author (D.F.K.) at UCLA Medical Center or Saint John's Health Center. The institutional review boards of each institution approved this retrospective study of patient data.

Preoperative and Postoperative Evaluation

Endocrine Assessment. Endocrine function was assessed with standard hormonal assays, including levels for plasma adrenocorticotropic hormone and serum cortisol, thyroid-stimulating hormone and thyroxine, luteinizing hormone, follicle-stimulating hormone, testosterone in men and estradiol in women, growth hormone, and IGF-I. Given that patients often underwent their initial and follow-up endocrine assessments at outside laboratories by referring endocrinologists or primary care specialists, a standard reference range is not provided, and instead values were reported as normal or abnormal based on the reference range for a given laboratory test. Additionally, several of the patients had relatively long-standing hormonal deficiencies that were diagnosed and treated well before the diagnosis of their arachnoid cyst, and thus, their original hormonal values were not available. As previously described,[9,10] in the early postoperative period patients were monitored for diabetes insipidus based on urine volume and urine specific gravity. Additionally, for patients without preoperative adrenal insufficiency and not receiving glucocorticoid replacement, serum morning cortisol and adrenocorticotropic hormone levels were monitored. Preoperative or postoperative endocrinopathy includes anterior gland axis deficiencies of hypoadrenalism, hypothyroidism, hypogonadism, and IGF-I deficiency, as well as posterior lobe dysfunction of diabetes insipidus. Two additional forms of endocrinopathy that were documented include stalk compression "hyperprolactinemia"[7,10,20,21] as well as severe hyponatremia, given that 2 patients in the series were diagnosed as a result of hyponatremia.[10,15,29,30]

Visual Function Assessment. Preoperative and postoperative visual function assessment included visual acuity using the handheld eye card and formal visual field testing. Visual function was considered improved if visual acuity assessed by the handheld eye card improved by at least 2 lines and/or if visual field defects, assessed by field confrontation and/or by formal visual field testing reviewed by an ophthalmologist, were resolved or improved.

Imaging. All patients underwent pre- and postoperative pituitary MR imaging with and without Gd, including early postoperative MR imaging on postoperative Day 1 or 2 and then subsequent MR images at 3- to 6-month intervals. A presumptive diagnosis of sellar AC was made when the sellar MR imaging showed CSF signal on all sequences and there was absence of abnormal enhancement in the cyst wall region.

Surgical Techniques

A direct endonasal transsphenoidal approach to the sellar region was performed in all cases of this series using either an endoscopically assisted microsurgical technique or more recently a fully endoscopic approach.[9,18] The various techniques to approach the sellar region have been previously described by the senior authors and are discussed in the literature.[9,18] A brief summary of the surgical procedure as it pertains specifically to treatment of the sellar AC follows (Fig. 1).

After a wide sphenoidotomy and sellar bone opening, the dura is incised in a U-shaped fashion and is flapped upward. A relatively small dural opening is performed that is large enough to work through and pass a 4-mm rigid endoscope but is not so large as to complicate skull base closure. Specifically, the dural opening should not extend to the inferior sellar pole or the lateral sellar edges so that a dural margin will remain circumferentially to help hold the eventual fat graft in position. Care must also be taken to selectively open the dura only, while not penetrating the pituitary gland that may be located anteroinferiorly, or the AC itself. If the pituitary is pushed anteriorly, a vertical gland incision may be needed to enter the cyst (Case 2). The AC membrane is then opened sharply with a microblade (in all cases, copious amounts of clear

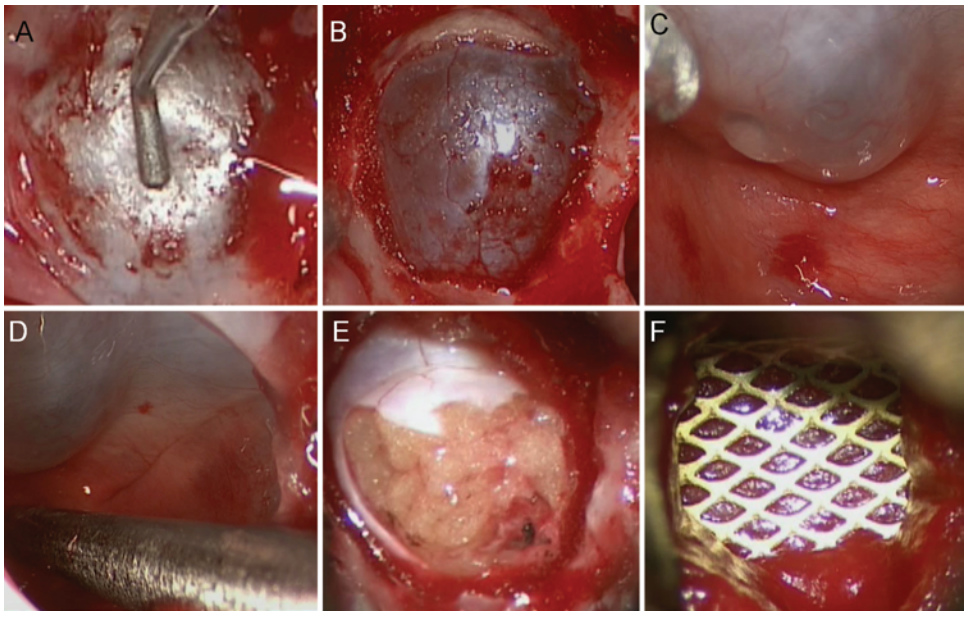

Fig. 1. Intraoperative steps of the management of an intrasellar AC (Case 8). **A:** Exposure of the thinned-out sellar floor. **B:** View of the cystic intrasellar lesion through the dura using the endoscope. **C and D:** View of the cyst's cavity using the endoscope. **E:** Filling of the cavity with adipose tissue. **F:** Skull base reconstruction using micromesh.

CSF immediately poured forth). A small window is cut into the anterior AC membrane and the specimen is sent to pathology. An inspection of the AC cavity is performed initially using the microscope and more recently with the endoscope to visualize the cyst walls, making sure the lesion is not a cystic tumor, and looking for potential diaphragmatic defects or arachnoid diverticula. After cyst drainage the superior portion of the cyst typically herniates downward into the enlarged sella and must be manipulated carefully to avoid enlarging the communication into the SAS. The insertion of the infundibulum into the diaphragma should be carefully inspected, as defects have often been noted in this location and although small, they may be a site for CSF entry into the sella.[6] Widening or dissection through the diaphragmatic defect to establish a larger communication to the suprasellar SAS is specifically avoided. Additionally, the cyst wall is not dissected off of the pituitary gland (which is typically thinned and attenuated) given the risk of worsening pituitary dysfunction.

Next, the AC cavity is obliterated with a fat graft. If a definitive diaphragmatic defect is visualized, the fat graft should extend up to and partially through this defect as shown in the postoperative MR imaging in the case illustration (below). The fat graft should fill the cavity sufficiently while not causing excessive optic apparatus compression. After cyst obliteration, the dura should remain pulsatile but no or minimal CSF should be noted weeping through the now-filled cyst cavity. A Valsalva maneuver is performed to further assess the adequacy of the fat graft and degree of CSF leakage and whether the fat will remain within the sella. If there is substantial egress of CSF at the time of the Valsalva maneuver, additional fat should be placed within the sella but with care not to overpack the sella.

Next, a layer of collagen sponge (Helistat, Integra Life Sciences) is placed over the sellar dura and adjacent sphenoid bone. A semirigid buttress using titanium micromesh (Leibinger) or other buttress material is then placed in the intrasellar extradural space to hold the repair firmly in position. Care is taken to cut the buttress just wide enough to extend 1–2 mm lateral to the bony sellar defect. Another Valsalva maneuver is performed to be certain the mesh is well-wedged into position. Additional fat is placed in the posterior sphenoid to cover the mesh, followed by a second larger piece of collagen sponge to cover the outer fat graft, followed by tissue glue (either Tisseal or DuraSeal).[8] Earlier in the senior author's experience, a lumbar drain was used for CSF diversion of high-grade (Grade 3) CSF intraoperative leaks, but in the last 8 procedures no lumbar drain was used and instead acetazolamide was used for 48 hours. Although we have not used a pedicled vascularized flap, this is an alternative skull base repair that may also be effective in these patients.[11,19]

Results

Patient Demographics and Clinical Presentation

As shown in Tables 1 and 2, the patient population consisted of 6 women and 2 men with a mean age of 57 years (range 43–81 years). Clinical presentation included constitutional symptoms (headache, malaise, low energy, weight gain), visual dysfunction, and episodic severe hyponatremia (Table 1). The most common presenting constitutional symptom was fatigue and low energy occurring in 6 patients (75%) followed by headache in 4 patients (50%) and decreased libido in 2. Of 4 patients with visual dysfunction, all 4 had visual field defects and 2 had decreased visual acuity. Endocrinopathy was document-

TABLE 1: Patient demographics, preoperative presentation, and postoperative outcome*

Case No.	Age (yrs), Sex	Preop Presentation			Postop Outcome		
		Constitutional	Visual Function	Pituitary Function	Constitutional	Vision Function	Pituitary Function
1	45, M	HA	superior bitemporal quadranopsia, VA: 20/25 bilat	normal	improved HA	resolution of VF defect, VF full, VA same	normal
2	52, F	HA, fatigue, decreased libido	VF full, VAR: 20/50, VAL: 20/25	normal	improved HA & energy	VF full, VA same (after 1st and 2nd operation)	normal (after 1st and 2nd operation)
3	54, F	fatigue	bitemporal hemianopsia, VAR: 20/30, VAL: 20/200	hypogonadism, hyperprolactinemia (35 ng/ml, range 2–18 ng/ml)	improved energy	improved VF, VAR: 20/40, improved VAL: 20/50	normalized prolactin (11 ng/ml, range 5–27 ng/ml)
4	67, M	fatigue, decreased libido	VF full, VAR: 20/20, VAL: 20/50	hypoadrenalism, hypothyroidism, hypogonadism	improved energy	VF full, VA same	same
5	81, F	HA, fatigue, recurrent severe hyponatremia over 5 yrs (low 118 mmol/L)	VF full, VAR: 20/100, VAL: 20/40	hypoadrenalism, hypogonadism, hyperprolactinemia (36.8 ng/ml, range 5–26 ng/ml), recurrent hyponatremia	improved HA	VF full, VA same	normalized prolactin postop but at 29 months postop, recurrent mild hyperprolactinemia (31.5 ng/ml; normal 5–20 ng/ml) and partial cyst reaccumulation noted on new MRI; no further hyponatremia since surgery
6	47, F	none	bitemporal hemianopsia, VA: 20/25 bilat	normal	none	improved VF, VA same	normal
7	70, F	fatigue, recurrent severe hyponatremia over 3 years (low 114 mmol/L)	bitemporal hemianopsia, VAR: 20/30, VAL: 20/40	hypoadrenalism, hypothyroidism, hypogonadism, recurrent hyponatremia	improved energy	resolution of VF defect, VF full, VAR: 20/30, improved VAL: 20/20	same; no further hyponatremia for 21 mos since surgery
8	43, F	HA, fatigue	VF full, VAR: 20/25, VAL: 20/25	normal	improved HA & energy	VF full (same), VA same	normal

* HA = headache; VA = visual acuity; VAL = visual acuity of the left eye; VAR = visual acuity of the right eye; VF = visual fields.

TABLE 2: Preoperative imaging, intraoperative findings, and postoperative follow-up*

	Preop MRI		Intraop		Postop FU	
Case No.	PG Max Diameter (mm)	PG Location	Endoscopically Assisted Technique	Findings	Last Postop MRI (mos/reaccumulation)	Last Clinical FU (mos)
1	32	superior, lateral	yes	no communication noted	47/no	53
2	18	superior, anterior, bilat	yes	vertical gland incision, defect in diaphragma sella	42/yes	53 in all (11 after repeat surgery)
	15	superior, anterior, bilat	fully endoscopic	defect in the diaphragma sella, residual fat graft noted	4/no	
3	20	superior, anterior, bilat	yes	no communication noted	6/no	40
4	19	superior, anterior, lateral	yes	low horizontal gland incision, no communication noted	22/no	39
5	27	inferior, lateral	yes	no communication noted	29/partial	29
6	26	posterior, bilat	yes	no communication noted	14/no	16
7	31	superior, posterior, bilat	yes	no communication noted	18/no	18
8	17	superior, lateral	yes	no communication noted	10/no	10

* FU = follow-up; PG = pituitary gland.

ed in 4 patients (50%), including 4 with hypogonadism, 3 with hypothyroidism and/or adrenal insufficiency, and 2 with hyperprolactinemia. Two of these patients (Cases 5 and 7, Table 1) both with multiple axes deficiencies also had recurrent episodes of symptomatic severe hyponatremia over several years with serum sodium levels less than 120 mmol/L.

On preoperative MR imaging, the cystic lesion was presumptively believed to be a sellar AC in all cases. In all 8 patients, MR imaging showed a bowing upward of the diaphragma sellae and thinning of the pituitary gland, confirming the presence of mass effect within the expanded sella. The average maximal cyst diameter was 22 mm (range 15–32 mm) and all cysts had some degree of suprasellar extension. Cyst contents were consistent with CSF on all imaging sequences and no suspicious enhancement was noted in any case to suggest possible tumor (Fig. 2). As detailed in Table 2, the pituitary gland was most commonly displaced superiorly (in 6 patients), laterally (in 4 patients), or bilaterally (in 4 patients).

Endonasal Treatment

The pituitary gland was found to be displaced anteriorly in 3 cases requiring a vertical incision in the gland to enter the arachnoid cyst in 1 case (Case 2) and a low horizontal incision to provide better access to the cyst in another (Case 4). A clear direct communication between the SAS and the cyst was visualized in only 2 procedures (Case 2 at both initial and repeat surgery). An endoscopically assisted technique was performed in 8 procedures (88.9%; Table 2). Visualization of the cyst's cavity and the suprasellar anatomy was clearly better using the endoscope with 0° and angled lenses in these 8 procedures.

After cyst decompression, simple obliteration of the sellar space with an autologous adipose tissue graft and sellar floor reconstruction was performed in all cases as described in *Methods*. In this series, a high-flow (Grade 3) CSF leak was noted intraoperatively in Cases 1 and 2 and was reconstructed accordingly.[8]

A cyst membrane was sent in 3 cases and was confirmatory of arachnoid tissue in 2 cases and in the other specimen it was too small for a definitive diagnosis.

Surgical, Endocrine, Visual, and Imaging Outcome

All 8 patients underwent an initially successful endonasal cyst fenestration and obliteration. Early postoperative Day 1 imaging using either a CT scan or MR imaging demonstrated cyst obliteration in all patients (Fig. 2). There were no postoperative CSF leaks, meningitis, new neurological deficits, or vascular injuries. One patient developed postoperative sinusitis that resolved with oral antibiotics (Case 8; see illustrative case below).

The mean clinical follow-up was 32 months (range 10–53 months). Of the 4 patients that presented with headache, all improved following surgery. All 4 patients with preoperative visual field disturbances experienced resolution or marked improvement in visual fields, and 2 of these patients (Cases 3 and 7; Tables 1 and 2) also had improved visual acuity; no patients developed new visual deficits. Regarding endocrine dysfunction, none of the 3 patients with multiple preoperative anterior pituitary axes deficiencies experienced improved pituitary function, but there was no worsening of pituitary function. Of 2 patients with stalk compression hyperprolactinemia (Cases 3 and 5), both had prolactin normalization but as detailed below, one has had recurrent mild hyperprolactinemia. The 2 patients with episodic severe symptomatic hyponatremia have had no further episodes of hyponatremia documented for 29 months (Case 5) and 21 months (Case 7) since surgery. Thus, 3 of 4 patients experienced partial improvement in endocrine dysfunction.

The mean length of imaging follow-up for these 8 patients was 21 months (range 6–47 months). To date, 2 patients (Cases 2 and 5) have developed cyst reaccumulation at 3 and 29 months after surgery, respectively. In Case 2, this 52-year-old woman was noted to have a relatively large SAS-AC defect at the time of her original surgery. She experienced initial resolution of her headaches, but these recurred and progressively increased in frequency and intensity over the next 3 years. Consequently, at 43

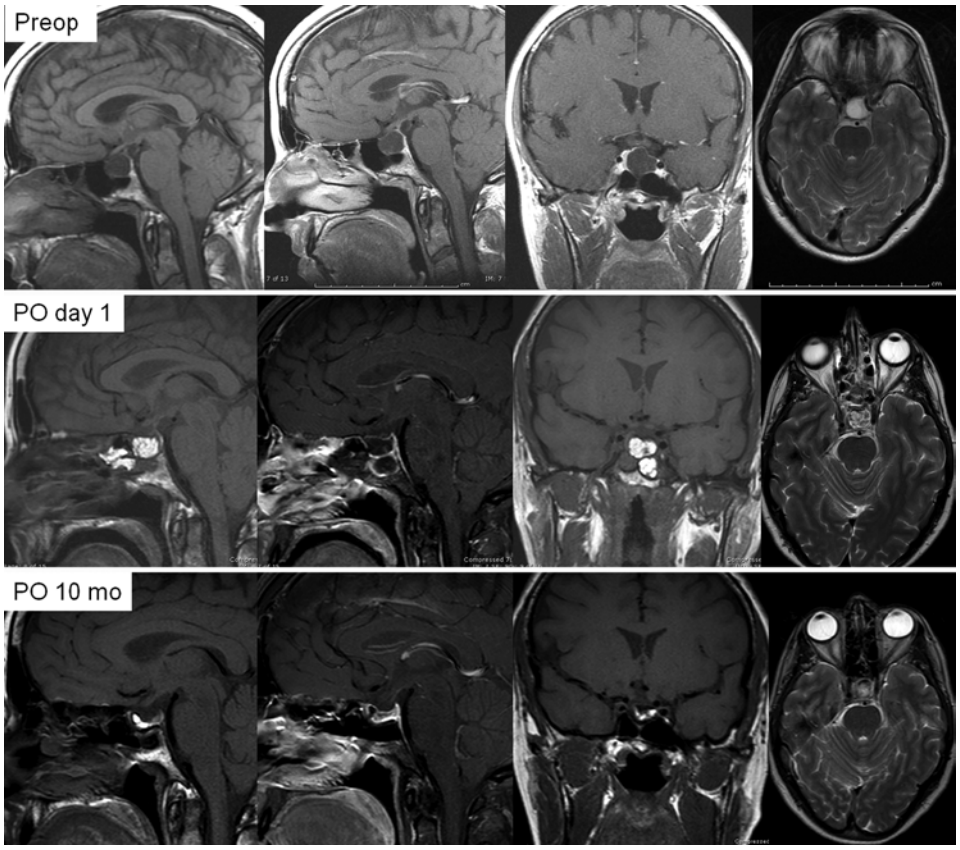

Fig. 2. Case 8. Preoperative MR imaging (*upper row;* sagittal, sagittal, coronal, and axial orientations from left to right) showing a cystic sellar lesion with suprasellar extension measuring 14 × 16 × 17 mm following CSF signal intensity on all sequences. The gland was thinned and pushed toward the right, as was the pituitary stalk. Postoperative (PO) Day 1 MR imaging *(center row)* showing cyst drainage and obliteration with fat, and skull base reconstruction. At 10 months after surgery *(lower row)*, MR imaging shows continued collapse of the AC with minimal residual fat graft and thicker pituitary gland.

months after her original surgery, she underwent repeat endoscopic cyst obliteration. At reoperation, the great majority of the fat was noted to have been reabsorbed and the large communication between the sella and SAS persisted, thus a new larger fat graft was placed. Currently at 11 months after her second surgery, her headaches have improved and her last MR imaging (4 months postoperatively) showed fat filling the enlarged sella with no reaccumulation of the AC. In Case 5, at 12 months postsurgery, this 81-year-old woman experienced resolution of her preoperative headaches, no further bouts of severe hyponatremia, a normal prolactin level, and complete disappearance of her arachnoid cyst on MR imaging. However, at 29 months after surgery, her endocrine studies revealed recurrent mild hyperprolactinemia to 31.5 ng/ml, and MR imaging showed partial cyst reacculumation. Because she remains asymptomatic, conservative management with endocrine and imaging follow-up is planned.

Illustrative Case

This 43-year-old healthy woman (Case 8) had a several-month history of fatigue and a 1-month history of severe daily vertex headaches. Her sellar MR imaging showed a cystic sellar lesion with suprasellar extension measuring 14 × 16 × 17 mm following CSF signal intensity on all sequences; the pituitary gland and stalk were pushed toward the right (Fig. 2). Her preoperative endocrine workup was significant for a low-normal IGF-I level of 62 ng/ml (normal range 58–318 ng/ml); a formal stimulation test for growth hormone deficiency was not performed. She had normal visual acuity and full visual fields. Given her symptoms and the relatively large size of her AC with severe gland distortion, an endonasal cyst fenestration and obliteration with abdominal fat was performed (Fig. 1). Her postoperative Day 1 MR imaging showed obliteration of the cyst cavity with fat, and skull base reconstruction (Fig. 2). Endoscopic examination of the nasal cavity resulted in a diagnosis of acute sinusitis, which resolved with antibiotic treatment. Her latest hormonal studies 10 months after surgery were all normal including an improved IGF-I level to 155 ng/ml, and her 10-month postoperative MR imaging showed no AC reaccumulation, minimal residual fat graft, and a fuller thickened pituitary gland (Fig. 2). Her headaches completely resolved and her energy has improved.

Discussion

Pathophysiology of Intrasellar ACs

The pathophysiology of intrasellar ACs remains con-

troversial. Currently, two hypotheses may explain the formation of these benign lesions. The first states that intrasellar ACs result from a defective diaphragma sellae through which the basal arachnoid membrane herniates. According to this mechanism intrasellar ACs do not represent true intraarachnoid cysts but more an arachnoid diverticulum. This diverticulum may remain patent, responsible for communication between the SAS and the cyst's cavity, qualified accordingly as communicating.[1,6,14] The aperture through which the diverticulum herniates may also close, creating a noncommunicating cyst. This closure may result from either a dynamic reconfiguration of the cyst's superior wall and displacement of normal structures, or as a result of an arachnoiditic phenomenon following a meningitis, hemorrhagic, or inflammatory event.[1,6,14]

The second hypothesis proposed by Meyer et al.[23] states that the intrasellar cyst develops in the same fashion as other intracranial ACs—between the arachnoid layers. This diverticulum would either originate above the diaphragm and expand through its aperture or develop from a subdiaphragmatic arachnoid layering.[23] More recently, the diaphragma sellae and the pituitary stalk's anatomy have been studied in great detail.[3,31] Although it was believed that there is no arachnoid tissue below the diaphragma sellae, Campero and colleagues[3] have elegantly shown that the basal arachnoid membrane covering the diaphragma sellae extends not only superiorly along the pituitary stalk but also inferiorly, along a variable distance. These findings support the hypothesis of Meyer et al.[23] Given the presence of arachnoid along the pituitary stalk extending down to the pituitary gland, well beyond the diaphragma sellae, an intrasellar AC may potentially arise from one of these arachnoid sleeves. Cerebrospinal fluid may penetrate via a ball-valve mechanism and favor expansion similarly as for other intracranial ACs. Therefore, some intrasellar ACs may actually be true intraarachnoid cysts, accounting for some of the cases in which no communication between the cyst and the suprasellar SAS is noted during surgery.[23]

Surgical Management of Intrasellar ACs

Although some rare sellar ACs have been successfully treated by stereotactic intracavitary irradiation, symptomatic ACs are most frequently addressed surgically.[6,26] Early on, some cases were addressed through a transcranial approach.[2,17,24,25] However, the majority of more recent symptomatic ACs have been fenestrated via the transsphenoidal route (Table 3). Although fenestration of the anterior cyst membrane is an essential initial entry point using the transsphenoidal approach, the subsequent intrasellar management of these ACs has been inconsistent among case reports and even within series. In most reports, some degree of augmenting communication between the AC and SAS has been performed and removal of some of the cyst lining is also often performed.[6] Although we took this approach in our early experience with sellar ACs, for all cases in this series we deliberately did not create or augment communication between the cyst and the SAS, nor have we excised the cyst membrane given potential damage to the pituitary gland and infundibulum. Instead, by simply filling the enlarged sella with a generous fat graft and leaving all arachnoid membranes and diaphragma sellae intact, we augmented the natural but deficient barrier between the SAS and sellar space. Acutely, it appears the fat graft prevents the sella from refilling with CSF, and over time, it likely induces scar formation to the diaphragma and parasellar arachnoid, recreating the natural partition between the sella and SAS. This minimalist technique aims to recreate the anatomical and physiological state in which the diaphragma sellae acts more as a true barrier to arachnoid descent and CSF entry into the sella turcica. As can be seen in the case examples presented, there appears to be varying degrees of fat reabsorption over time (Figs. 3 and 4) but complete cyst reaccumulation was observed in only 1 patient (Case 2) and partial reaccumulation was noted in a second patient (Case 5). Although simple cyst obliteration with adipose tissue appears to eliminate the cyst and has a high rate of symptomatic improvement, given the relatively short follow-up in some patients, it remains unproven whether this technique will provide a lasting solution.

Another advantage of this simple technique is that the degree of communication between the sella and SAS is minimized since the diaphragma and the suprasellar arachnoid membranes are not further opened or removed. Leaving these barriers in place likely reduces the risk of a postoperative CSF leak because a high-flow leak into the sella is minimized. In this series, we found no postoperative CSF leaks using this technique. In contrast, totaling prior reports, 11 (16.2%) of 68 cases have experienced postoperative CSF leaks.[1,4,6,13,17,26,27,32] Saeki et al.[27] even stipulated that a lumbar drain should be placed in every case of a sellar AC regardless of the efficiency of the skull base repair due to this high leak rate. In our experience, using sellar obliteration with fat and a semirigid sellar floor buttress, CSF diversion with a lumbar drain is infrequently needed. In our series, only 2 cases (1 and 2) had high-flow intraoperative CSF leaks and only Case 1 was treated with CSF lumbar diversion.

While this simple technique appears to work well for this subset of smaller ACs up to 3 cm in size, it is quite possible that for larger cysts with a significant suprasellar extension, sellar packing alone would not efficiently occlude the AC-SAS communication and reaccumulation would be likely. These larger, more extensive suprasellar ACs may be best managed by a traditional approach of transcranial fenestration into the suprasellar SAS.

Until recently, most case reports or series of sellar ACs have used the purely microscopic transsphenoidal approach.[1,5,6,12,14,16,17,22–28,32–34] Dietemann and colleagues[5] first described the use of the endoscope to explore the cystic cavity, which lead to the finding of a communication between the cyst and the suprasellar SAS in one of their cases. More recently, Cavallo and colleagues[4] in a multicenter study recognized the role of the endoscope in the transsphenoidal management of sellar cystic lesions. For sellar ACs with a large sella, they observed that the angled scopes allowed a much wider and more panoramic visualization of the suprasellar cistern and the entire cavity. In our series, we noted a communication between the

TABLE 3: Studies of surgical management of intrasellar ACs*

Authors & Year	Case No.	Mean Age (yrs), Sex	Surgical Approach	Intraop Observation	Cyst Cavity Inspection	Sella Packing	Skull Base Repair	Complications	Recurrence
Benedetti et al., 1977	1	35, M	subfrontal	clear CSF, grayish membrane	NS	NS	NS	none	none at 12 mos
	2	65, M	subfrontal	clear CSF, grayish membrane	NS	NS	NS	none	none at 6 mos
Leo et al., 1979	3	49, F	microscopic TS	NS	NS	NS	NS	pituitary abscess 3 wks after	death from septic shock
Harter et al., 1980	4	60, F	microscopic TS	clear CSF	inspection of the cyst roof showed no abnormally large aperture	NS	NS	none	none at 8 mos
Spaziante et al., 1981	5	24, M	microscopic TS	clear CSF	NS	NS	NS	blindness a few hrs postop; second operation documented prolapsed of optic chiasm into sella – 2nd operation for packing	no FU mentioned
	6	69, M	microscopic TS	clear CSF	NS	yes, no mention on material used	NS	none	no FU mentioned
	7	42, F	microscopic TS	clear CSF	at 2nd operation, communication between sella and SAS	yes, no mention on material used	NS	CSF leak Day 5, meningitis, 2nd surgery needed (repacking)	none at 3 mos
Baskin & Wilson, 1984	8–15	58, M	microscopic TS	clear CSF	in almost all, pinhole communication of the cyst with supradiaphragmatic SAS	adipose tissue	nasal cartilage	CSF leak, closed with LD; CSF leak and meningitis, 2nd surgery needed (repacking and LP shunt)	no FU mentioned
Meyer et al., 1987	16–28	42.4 (6 M, 2 F)	microscopic TS	clear CSF	in 1 case pinhole communication of the cyst with supradiaphragmatic SAS	muscle or adipose tissue	nasoseptal bone or cartilage	meningitis (resulting in death)	1 death, 7 others no FU mentioned
Hasegawa et al., 1991	29	46 (5 M, 8 F)	microscopic TS	clear CSF	descent of roof with no leakage of CSF noted	muscle, fascia, and adipose tissue	cartilage, chemical glue	CSF leak postop Day 4 & meningitis postop Day 6 (treated w/ LD)	symptomatic recurrence post Day 46: 2nd operation needed; no recurrence as of 16 mos after 2nd operation
Hornig & Zervas, 1992	30	53, M	microscopic TS			adipose tissue		none	no, 6 yrs
Iida et al., 1996	31	57, M	microscopic TS	clear CSF	NS	NS	NS	no mention	no FU mentioned

(continued)

TABLE 3: Studies of surgical management of intrasellar ACs* *(continued)*

Authors & Year	Case No.	Mean Age (yrs), Sex	Surgical Approach	Intraop Observation	Cyst Cavity Inspection	Sella Packing	Skull Base Repair	Complications	Recurrence
Nomura et al., 1996 (1 case previously reported by Hasegawa et al.)	32	44, M	microscopic TS	NS	NS	NS at first OR	NS	CSF leak (time not specified) and meningitis, 2nd operation needed (packing and LP shunt)	no FU mentioned
Dietemann et al., 1997	33	57, M	microscopic TS endoscopic exploration	clear CSF	communication of the cyst with SAS	adipose tissue and biological glue	NS	none	no FU mentioned
	34	45, F	microscopic TS	clear CSF	NS	NS	NS	none	no FU mentioned
Saeki et al., 1999	35	50, F	microscopic TS	clear CSF	upper aspect of the cyst wall not visible, no leak discovered even w/ Valsalva	none	fascia lata, adipose tissue, fibrin glue	CSF leak postop Day 7; LD and 2nd surgery, not all arachnoid membrane could be viewed; although no site of CSF leak was identified, there was a leak from the suprasellar direction; packing at 2nd operation	no FU mentioned
Miyamoto et al., 1999	36	62, M	microscopic TS	clear CSF	roof inspected and no leak viewed	muscle, fibrin glue	bony septum	none	no FU mentioned
	37	67, F	frontotemporal craniotomy	clear CSF	NS	NS	NS	NS	no FU mentioned
Shin et al., 1999	38–42	40, M	microscopic TS	clear CSF	NS	NS	NS	NS	mean FU of 33 mos, 1 case recurrence at 99 mos
Weil, 2001	43	53 (2 M, 3 F)	microscopic TS	clear CSF, transparent membrane	continuous egress of CSF during operation despite negative cisternogram	NS	fat, nasal bone, tissue adhesive	NS	none at 6 mos
Murakami et al., 2003	44	74, F	microscopic TS	clear CSF	cyst wall collapsed and clear fluid spurted out in rhythmic fashion, no perforation seen	adipose tissue	bony septum	none	symptomatic recurrence at 52 mos
Yasuda et al., 2005	45	48, M	microscopic TS	clear CSF	no CSF leak was discovered	adipose tissue	cartilage and fibrin glue	none	none at 6 mos

(continued)

TABLE 3: Studies of surgical management of intrasellar ACs* *(continued)*

Authors & Year	Case No.	Mean Age (yrs), Sex	Surgical Approach	Intraop Observation	Cyst Cavity Inspection	Sella Packing	Skull Base Repair	Complications	Recurrence
Dubuisson et al., 2007	46–54	67, M	microscopic TS	clear CSF (3), mildly xanthochromic (5), mildly hemorrhagic (1)	communication was discovered in 3 and leakage of CSF around the stalk was observed in 2 others; a broad fenestration toward the SAS was created in 4/5 of these patients	adipose tissue in 4/9	nasoseptal bone, adipose tissue, biological glue	CSF leak postop Day 6, LD & needed 2nd operation (repacking); CSF leak, operation 3 years postop	FU from 2 mos to 27 yrs (mean 11 mos), 1 death (unrelated cause)
Cavallo et al., 2008 (series collected from 3 institutions)	55–65	48.6 (4 M, 6 F)	microscopic endoscopically assisted TS approach or endonasal endoscopic approach	clear CSF	NS	adipose tissue and/or collagen sponge	material not specified	CSF leak (time not specified), LD and 2nd operation; CSF leak w/ meningitis (time not specified), LD and 2nd operation	FU varying from 10 to 94 mos, 1 case recurrence at 16 mos
present study	66–73	57 (6 F, 2 M)	microscopic TS, microscopic endoscopically assisted TS approach or endonasal endoscopic approach	clear CSF	see details in Table 2	apidose tissue	graded skull-base reconstruction	none	mean FU 32 mos, 1 case of symptomatic recurrence at 43 mos, 1 case of asymptomatic partial recurrence

* LD = lumbar drain; LP = lumboperitoneal; NS = not specified; TS = transsphenoidal surgery.

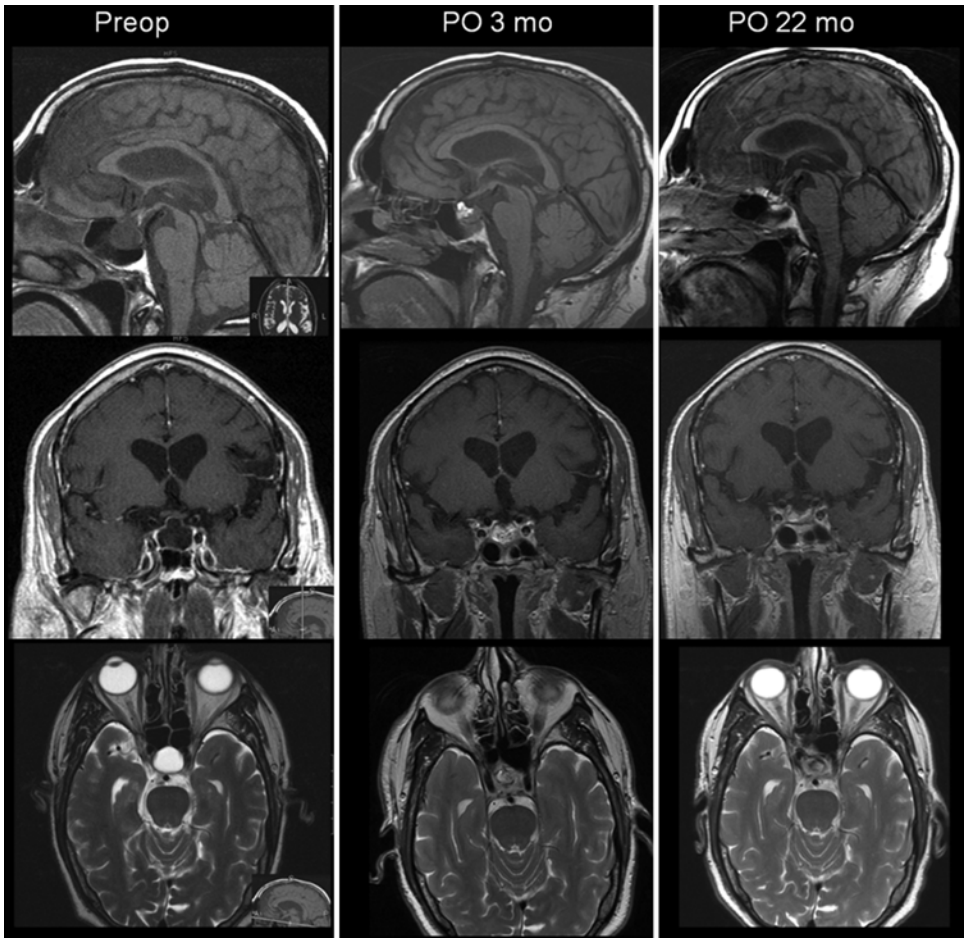

Fig. 3. Case 4. Sagittal *(upper row)*, coronal *(center row)*, and axial *(lower row)* MR images showing rapid fat resorption. Preoperative imaging *(left column)* shows an intrasellar AC with suprasellar extension. Postoperative imaging at 3 *(center column)* and 22 months *(right column)* shows progressive fat tissue resorption but no recurrence of the cystic cavity.

suprasellar SAS and the AC in Case 2 at initial and repeat surgery with the help of the endoscope. For the remaining cases in whom no obvious communications were observed, this was much more confidently determined using the endoscope. This visualization technique allows a better view of the superior arachnoid membrane and the flattened pituitary gland and insertion site of the infundibulum. It provides a better exploration of the cyst's cavity, inspecting all the walls of the cyst to verify that there is no evidence of cystic tumor.

Improvement in Preoperative Symptoms

Among the 4 other largest cases series concerning intrasellar ACs, visual symptoms have improved in 67% to 100%, although most series reported 100% improvement.[4,6,23,28] Spaziante et al.[32] reported a case of acute blindness following surgery that was believed to be due to prolapse of the optic chiasm into the sellar cavity. Although the patient was brought back to the operating room to refill the sella, his vision never improved.[32] Headache improvement occurs in 33% to 50% of cases.[4,6,28] Regarding pituitary function, hyperprolactinemia resolved postoperatively in 100% of cases.[4,6,23] Patients with anterior pituitary dysfunction improved between 80% and 100%,[6,28] although those with preoperative complete pituitary failure did not improve in the series by Dubuisson et al.[6] New pituitary dysfunction has been reported in 11% to 15% of patients.[6,23] In our small series, both headache and visual function improved in 4 of 4 patients. Regarding endocrine outcome, none of the 3 patients with 3 or more axes deficiencies experienced resolution of an axis deficiency, but stalk compression "hyperprolactinemia" resolved in 2 patients and recurrent symptomatic hyponatremia has not recurred in 2 patients. Thus, overall there was partial improvement in pituitary dysfunction in 3 of 4 patients but this was transient in Case 5; there was no new endocrine dysfunction. Overall, this small clinical series and prior reports of sellar ACs indicate that surgical treatment results in significant improvement of preoperative symptoms including headaches, visual function, and to some degree, endocrine abnormalities. Also, our results appear to indicate that even though the fat graft obliterates the potential space of the enlarged sella, it does not create such excessive mass effect that it results in worsened gland function or new visual loss.

Conclusions

Symptomatic intrasellar ACs are uncommon sellar

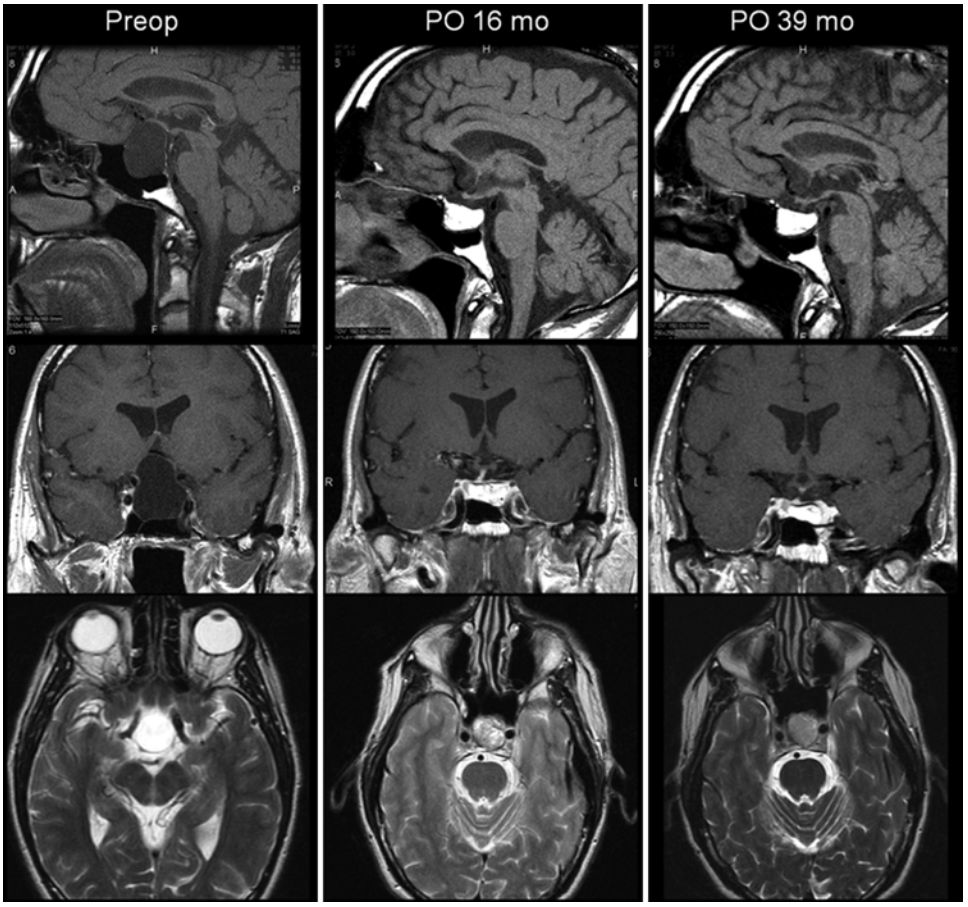

Fig. 4. Case 1. Sagittal *(upper row)*, coronal *(center row)*, and axial *(lower row)* MR images showing minimal fat resorption over time. Preoperative imaging *(left column)* documents an intrasellar AC with suprasellar extension. Postoperative imaging at 16 *(center column)* and 39 months *(right column)* shows minimal fat resorption and no cyst recurrence.

lesions that can be effectively treated by simple obliteration of the sellar space with an abdominal fat graft and sellar floor reconstruction. By deliberately not augmenting or enlarging the communication into the SAS, this technique aims to recreate the natural barrier between the sella and the suprasellar space. In this small series with a relatively short follow-up duration, this approach appears to achieve a high success rate in terms of reversing headaches, visual loss, and endocrinopathy, and produces a low complication rate. Endoscopy is recommended for all such cases given the enhanced panoramic visualization of the sellar and suprasellar space.

Disclosure

Dr. Kelly has a royalty agreement with Mizuho-America, Inc.

Author contributions to the study and manuscript preparation include the following. Conception and design: Kelly, McLaughlin, Vandergrift. Acquisition of data: McLaughlin, Vandergrift, Ditzel Filho, Shahlaie, Eisenberg. Analysis and interpretation of data: Kelly, McLaughlin, Ditzel Filho, Cohan. Drafting the article: McLaughlin, Vandergrift. 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: Kelly. Study supervision: Kelly, McLaughlin, Carrau.

References

1. Baskin DS, Wilson CB: Transsphenoidal treatment of nonneoplastic intrasellar cysts. A report of 38 cases. **J Neurosurg 60:**8–13, 1984
2. Benedetti A, Carbonin C, Colombo F: Possible aetiopathogenetic correlation between primary empty sella and arachnoid cyst. **Acta Neurochir (Wien) 38:**269–278, 1977
3. Campero A, Tróccoli G, Martins C, Fernandez-Miranda JC, Yasuda A, Rhoton AL Jr: Microsurgical approaches to the medial temporal region: an anatomical study. **Neurosurgery 59 (4 Suppl 2):**ONS279–ONS308, 2006
4. Cavallo LM, Prevedello D, Esposito F, Laws ER Jr, Dusick JR, Messina A, et al: The role of the endoscope in the transsphenoidal management of cystic lesions of the sellar region. **Neurosurg Rev 31:**55–64, 2008
5. Dietemann JL, Guessoum M, Schultz A, Zöllner G, Sanoussi S, Maitrot D, et al: [Intrasellar arachnoid cysts: computed tomography and MRI. Apropos of 2 cases.] **J Neuroradiol 24:** 168–173, 1997 (Fr)
6. Dubuisson AS, Stevenaert A, Martin DH, Flandroy PP: Intrasellar arachnoid cysts. **Neurosurgery 61:**505–513, 2007
7. Dusick JR, Fatemi N, Mattozo C, McArthur D, Cohan P, Wang C, et al: Pituitary function after endonasal surgery for nonadenomatous parasellar tumors: Rathke's cleft cysts, craniopharyngiomas, and meningiomas. **Surg Neurol 70:**482–491, 2008
8. Esposito F, Dusick JR, Fatemi N, Kelly DF: Graded repair of cranial base defects and cerebrospinal fluid leaks in trans-

sphenoidal surgery. **Neurosurgery 60 (4 Suppl 2):**295–304, 2007
9. Fatemi N, Dusick JR, de Paiva Neto MA, Kelly DF: The endonasal microscopic approach for pituitary adenomas and other parasellar tumors: a 10-year experience. **Neurosurgery 63 (4 Suppl 2):**244–256, 2008
10. Fatemi N, Dusick JR, Mattozo C, McArthur DL, Cohan P, Boscardin J, et al: Pituitary hormonal loss and recovery after transsphenoidal adenoma removal. **Neurosurgery 63:**709–719, 2008
11. Hadad G, Bassagasteguy L, Carrau RL, Mataza JC, Kassam A, Snyderman CH, et al: A novel reconstructive technique after endoscopic expanded endonasal approaches: vascular pedicle nasoseptal flap. **Laryngoscope 116:**1882–1886, 2006
12. Harter LP, Silverberg GD, Brant-Zawadzki M: Intrasellar arachnoid cyst: case report. **Neurosurgery 7:**387–390, 1980
13. Hasegawa M, Yamashima T, Yamashita J, Kuroda E: Symptomatic intrasellar arachnoid cyst: case report. **Surg Neurol 35:**355–359, 1991
14. Hornig GW, Zervas NT: Slit defect of the diaphragma sellae with valve effect: observation of a "slit valve". **Neurosurgery 30:**265–267, 1992
15. Hsu YJ, Chau T, Yang SS, Tsai WS, Lin SH: Rathke's cleft cyst presenting with hyponatremia and transient central diabetes insipidus. **Acta Neurol Scand 107:**382–385, 2003
16. Iida S, Fujii H, Tanaka Y, Hayashi S, Nagareda T, Moriwaki K: An intrasellar cystic mass and hypopituitarism. **Postgrad Med J 72:**441–442, 1996
17. Iqbal J, Kanaan I, Al Homsi M: Non-neoplastic cystic lesions of the sellar region presentation, diagnosis and management of eight cases and review of the literature. **Acta Neurochir (Wien) 141:**389–398, 1999
18. Kassam A, Snyderman CH, Mintz A, Gardner P, Carrau RL: Expanded endonasal approach: the rostrocaudal axis. Part I. Crista galli to the sella turcica. **Neurosurg Focus 19(1):**E3, 2005
19. Kassam AB, Thomas A, Carrau RL, Snyderman CH, Vescan A, Prevedello D, et al: Endoscopic reconstruction of the cranial base using a pedicled nasoseptal flap. **Neurosurgery 63 (1 Suppl 1):**ONS44–ONS53, 2008
20. Kruse A, Astrup J, Gyldensted C, Cold GE: Hyperprolactinaemia in patients with pituitary adenomas. The pituitary stalk compression syndrome. **Br J Neurosurg 9:**453–457, 1995
21. Lees PD, Pickard JD: Hyperprolactinemia, intrasellar pituitary tissue pressure, and the pituitary stalk compression syndrome. **J Neurosurg 67:**192–196, 1987
22. Leo JS, Pinto RS, Hulvat GF, Epstein F, Kricheff II: Computed tomography of arachnoid cysts. **Radiology 130:**675–680, 1979
23. Meyer FB, Carpenter SM, Laws ER Jr: Intrasellar arachnoid cysts. **Surg Neurol 28:**105–110, 1987
24. Miyamoto T, Ebisudani D, Kitamura K, Ohshima T, Horiguchi H, Nagahiro S: Surgical management of symptomatic intrasellar arachnoid cysts—two case reports. **Neurol Med Chir (Tokyo) 39:**941–945, 1999
25. Murakami M, Okumura H, Kakita K: Recurrent intrasellar arachnoid cyst. **Neurol Med Chir (Tokyo) 43:**312–315, 2003
26. Nomura M, Tachibana O, Hasegawa M, Kohda Y, Nakada M, Yamashima T, et al: Contrast-enhanced MRI of intrasellar arachnoid cysts: relationship between the pituitary gland and cyst. **Neuroradiology 38:**566–568, 1996
27. Saeki N, Tokunaga H, Hoshi S, Sunada S, Sunami K, Uchino F, et al: Delayed postoperative CSF rhinorrhea of intrasellar arachnoid cyst. **Acta Neurochir (Wien) 141:**165–169, 1999
28. Shin JL, Asa SL, Woodhouse LJ, Smyth HS, Ezzat S: Cystic lesions of the pituitary: clinicopathological features distinguishing craniopharyngioma, Rathke's cleft cyst, and arachnoid cyst. **J Clin Endocrinol Metab 84:**3972–3982, 1999
29. Sivakumar W, Cole CD, Couldwell WT: Rathke cleft cyst presenting with hyponatremia: an unusual presentation. **Neurosurg Focus 31(1):**E4, 2011
30. Son JH, Fujimaki T, Tsuchiya Y, Ishii T, Takagi K, Nakagomi T: Pituitary cyst presenting with hyponatremia and increased secretion of brain natriuretic peptide. Case report. **J Neurosurg 103:**1092–1094, 2005
31. Song-tao Q, Xi-an Z, Hao L, Jun F, Jun P, Yun-tao L: The arachnoid sleeve enveloping the pituitary stalk: anatomical and histologic study. **Neurosurgery 66:**585–589, 2010
32. Spaziante R, de Divitiis E, Stella L, Cappabianca P, Donzelli R: Benign intrasellar cysts. **Surg Neurol 15:**274–282, 1981
33. Weil RJ: Rapidly progressive visual loss caused by a sellar arachnoid cyst: reversal with transsphenoidal microsurgery. **South Med J 94:**1118–1121, 2001
34. Yasuda K, Saitoh Y, Okita K, Morris S, Moriwaki M, Miyagawa J, et al: Giant intrasellar arachnoid cyst manifesting as adrenal insufficiency due to hypothalamic dysfunction—case report. **Neurol Med Chir (Tokyo) 45:**164–167, 2005

Manuscript submitted March 7, 2011.
Accepted December 12, 2011.
Please include this information when citing this paper: published online January 27, 2012; DOI: 10.3171/2011.12.JNS11399.
Address correspondence to: Daniel F. Kelly, M.D., Brain Tumor Center, John Wayne Cancer Institute at Saint John's Health Center, 2200 Santa Monica Boulevard, Santa Monica, California 90404. email: kellyd@jwci.org.