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ORIGINAL ARTICLE

Review of empty sella syndrome and its surgical management

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KEYWORDS

Empty sella;
Sellar floor reconstruction;
CSF rhinorrhea

Abstract *Introduction:* Empty sella syndrome (ESS) is a condition in which the sella turcica is partially or completely filled with CSF resulting in a displacement of the normal pituitary gland.
Objective: This study was done to evaluate the clinical features, surgical management and outcome in a consecutive 20 cases with ESS.
Methods: This retrospective study included 20 adult patients suffering from manifestations due to ESS. 12 patients (60%) had symptomatic primary ESS and the rest 8 patients (40%) had manifestations due to secondary ESS. The male to female ratio was 1–3 and their ages ranged from 20 till 56 years with mean age of 41 years. As regards the clinical presentation, manifestations of increase the intracranial pressure were found in 12 patients (60%), visual manifestations in 10 patients (50%), CSF rhinorrhea in 10 cases (50%), and endocrinological dysfunction in 7 patients (35%). All patients had preoperative plain X-ray, CT scan and MRI of the brain. Different surgical procedures were done according to the mode of presentation and the radiological findings. Mean postoperative follow-up period was 30 months, including both clinical and radiological examinations.
Results: After surgery 10 patients (50%) were asymptomatic, 6 patients (30%) improved, and 4 patients (20%) stabilized. (85.3%) of patients with preoperative complaint of headache respond well to surgery, (12 cases out of 14). While 60% of the patients with preoperative visual field defect improved. No patients with preoperative ↓ visual acuity (4 cases) have improved after surgery. Postoperative MRI studies after extradural transsphenoidal packing revealed that the sellar contents appeared satisfactory elevated with upward lifting of suprasellar structures in all cases.

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Conclusion: The surgical outcome of cases with ESS is favorable. Visual disturbances and CSF rhinorrhea are the main indications for surgery. The type of surgery depends on clinical presentation and radiological findings.

1. Introduction

Empty sella syndrome (ESS) is a condition in which the sella turcica is partially or completely filled with CSF resulting in a displacement of the normal pituitary gland.[1,2] Usually the sella is enlarged and the pituitary gland is compressed and reshaped.[3] ESS is divided into primary and secondary types depending on the presence of previous surgery or irradiation to the pituitary gland.[4] Primary empty sella can be due to an inherent weakness of the diaphragma sella and or to an increase in the intracranial pressure which promotes the herniation of arachnoid membrane into the pituitary fossa.[5,6] Although most individuals who have primary ESS are asymptomatic, a few present with clinical symptoms and signs related to the condition.[7,8] It is more common in middle-aged obese females, usually presents with headache, and only occasionally associated with endocrine or visual abnormalities.[9] Surgical therapy is rarely required except for cases presented with CSF rhinorrhea or progressive visual loss.[10]

Aim of the work was to study different diagnostic criteria of primary and secondary ESS and to evaluate the results of its surgical management. Also to compare these results with the results of the other authors reported in literature.

2. Methods

This retrospective study was carried out on 20 adult patients suffering from manifestations due to ESS. Twelve patients had symptomatic primary ESS and the rest 8 patients had manifestations due to secondary ESS. The study was done in Alexandria hospitals over a period of 8 years starting from March 2000 to March 2008. The male to female ratio was 1–3 and their ages ranged from 20 till 56 years with mean age of 41 years.

All the 12 cases with symptomatic primary ESS were middle aged obese females and were complaining of manifestation of increase the intracranial pressure. Also 10 of these 12 patients had CSF rhinorrhea, the other two patients had visual field defect. Also endocrinological dysfunction was found in three cases. All the 8 cases with symptomatic secondary ESS had previous transsphenoidal operations for pituitary adenoma followed by radiotherapy in only one of them. Five patients were males and the other three patients were females. All these patients had visual disturbances in the form of field defect alone or with decrease the visual acuity as occurred in 4 patients. Also associated endocrinological dysfunction was found in 4 patients.

Headache was the commonest symptom (70% of patients). As regards the clinical presentation, manifestations of increase the intracranial pressure were found in 12 patients (60%), visual manifestations in 10 patients (50%), CSF rhinorrhea in 10 cases (50%), and endocrinological dysfunction in 7 patients (35%) (five women present with oligoamenorrhea with galactorrhea, and two males presents with hypothyroidism) (Table 1).

Before surgery all patients had plain X-ray skull, CT scan and MRI of the brain. CT scan with intrathecal contrast was done only in the 10 cases presented with CSF rhinorrhea. Measurement of intrathecal CSF pressure was done in the 12 cases presented with primary empty sella syndrome. Endocrinological tests especially serum prolactin level was done in all cases, also determination of the visual acuity, field of vision, funds examination were done.

Different surgical procedures were done in all cases according to the mode of presentation and the radiological findings. As regards the 10 cases presented with CSF rhinorrhea, extradural transsphenoidal packing of the sella using fat and fascia with fibrin glue was done on the 6 cases that showed transsellar fistulous tract after intrathecal contrast, the other 4 cases had subfrontal craniotomy with intradural repair of the anterior cranial fossa with fascia. But in the last 4 cases this procedure was ineffective in stopping the CSF leak and another operation was done in the form of application of thecoperitoneal shunt that was effective in stopping the CSF leak.

All the other 10 cases had extradural transsphenoidal packing of the sella turcica using extra peritoneal fat alone or with muscle and fascia followed by reconstruction of the sellar floor using either a fragment of the bone from the anterior sphenoid wall or the bony septum. In cases with previous transsphenoidal surgery (secondary empty sella), the reconstruction of the sellar floor was done using a bone graft from iliac crest or acrylic material.

All patients have been reviewed in the outpatient clinic postoperatively. The last follow-up clinical and MRI examinations were done at a period ranged from 12 to 60 months (mean = 30 months). Clinical presentations, MRI findings, operative findings and outcome of cases are summarized in Tables 2 and 3.

3. Results

Measurement of intrathecal CSF pressure was intermittently high in 7 out of 12 cases but not more than 23 cm in all measurement.

Determination of the visual acuity was found to be counting fingers in two cases, and 6/60 in the other two cases. Field of vision defects was present in the form of constricted field in

Table 1 Clinical features in 20 patients with empty sella syndrome.

Clinical presentation	Cases		Type of ESS
	No.	Percent	
↑ intracranial pressure	12	60	Primary
CSF rhinorrhea	10	50	Primary
Visual field defect	10	50	Primary and secondary
Endocrinological dysfunction	7	35	Primary and secondary
↓ visual acuity	4	20	secondary

Most patients had multiple symptoms.

Table 2 Clinical presentations, radiological findings and outcome of cases with primary ESS.

No	Age in year	Sex	Clinical presentation					Radiological and MRI findings	Operative procedures	Outcome and follow up period
			↑ ICP	CSF rhinorrhea	Visual field defect	↓ Visual acuity	Endocrine dysfunction			
1	25	F	+	+				MRI and CT with intrathecal contrast → the site of fistula can not be found	Subfrontal craniotomy with intradural repair of the anterior cranial fossa with fascia → failed thecoperitoneal shunt → successful	Asymptomatic 42 months
2	31	F	+	+			+	MRI and CT with intrathecal contrast → transsellar fistulous tract	Transsphenoidal packing of the sella using fat and fascia with fibrin glue + floor reconstruction	Asymptomatic 42 months
3	29	F	+	+				MRI and CT with intrathecal contrast → the site of fistula can not be found	Transsphenoidal packing of the sella using fat and fascia with fibrin glue + floor reconstruction	Asymptomatic 40 months
4	34	F	+	+				MRI and CT with intrathecal contrast → transsellar fistulous tract	Subfrontal craniotomy with intradural repair of the anterior cranial fossa with fascia → failed thecoperitoneal shunt → successful	Asymptomatic 34 months
5	44	F	+	+				MRI and CT with intrathecal contrast → transsellar fistulous tract	Transsphenoidal packing of the sella using fat and muscle + reconstruction of the sellar floor	Asymptomatic 60 months
6	41	F	+	+				MRI and CT with intrathecal contrast → transsellar fistulous tract	Transsphenoidal packing of the sella using fat + reconstruction of the sellar floor	Asymptomatic 40 months
7	36	F	+	+			+	MRI and CT with intrathecal contrast → the site of fistula	subfrontal craniotomy with intradural repair of the anterior cranial fossa with fascia → failed thecoperitoneal shunt → successful	Asymptomatic 20 months
8	38	F	+	+				MRI and CT with intrathecal contrast → transsellar fistulous tract	Transsphenoidal packing of the sella using fat and muscle + reconstruction of the sellar floor	Asymptomatic 48 months
9	48	F	+	+				MRI and CT with intrathecal contrast → transsellar fistulous tract	transsphenoidal packing of the sella using fat + reconstruction of the sellar floor	Asymptomatic 14 months
10	45	F	+	+				MRI and CT with intrathecal contrast → the site of fistula through cribriform plat	Subfrontal craniotomy with intradural repair of the anterior cranial fossa with fascia → failed thecoperitoneal shunt → successful	Asymptomatic 18 months
11	47	F	+		+		+	MRI → intrasellar CSF collection with posterior displacement flattening of pituitary gland	Transsphenoidal packing of the sella using fat and fascia + reconstruction of the sellar floor	Improved 24 months
12	41	F	+		+			MRI → intrasellar CSF collection with posterior displacement flattening of pituitary gland	Transsphenoidal packing of the sella using fat + reconstruction of the sellar floor	Improved 14 months

Table 3 Clinical presentations, radiological findings and outcome of cases with secondary ESS.

No	Age in year	Sex	Clinical presentation				Radiological and MRI findings	Operative procedures	Outcome and follow up period
			↑ ICP	CSF rhinorrhea	Visual field defect	↓ Visual acuity	Endocrine dysfunction			
1	51	M			+	+		MRI → intrasellar CSF collection with posterior displacement flattening of pituitary gland eccentric position of the stalk	Transsphenoidal packing of the sella using fat + reconstruction of the sellar floor	Stabilized 50 month
2	31	M			+		+	MRI → intrasellar CSF collection with posterior displacement flattening of pituitary gland eccentric position of the stalk	Transsphenoidal packing of the sella using fat + reconstruction of the sellar floor	Improved 12 months
3	51	M			+	+		MRI → intrasellar CSF collection with posterior displacement flattening of pituitary gland eccentric position of the stalk	Transsphenoidal packing of the sella using fat + reconstruction of the sellar floor	Stabilized 20 months
4	44	F			+		+	MRI → intrasellar CSF collection with posterior displacement flattening of pituitary gland eccentric position of the stalk	Transsphenoidal packing of the sella using fat + reconstruction of the sellar floor	Improved 20 months
5	36	F			+	+	+	MRI → intrasellar CSF collection with posterior displacement flattening of pituitary gland eccentric position of the stalk	Transsphenoidal packing of the sella using fat + reconstruction of the sellar floor	Stabilized 39 months
6	38	M			+	+		MRI → intrasellar CSF collection with posterior displacement flattening of pituitary gland	Transsphenoidal packing of the sella using fat + reconstruction of the sellar floor	Stabilized 31 months
7	57	M			+		+	MRI → intrasellar CSF collection with posterior displacement flattening of pituitary gland, eccentric position of the stalk	Transsphenoidal packing of the sella using fat + reconstruction of the sellar floor	Improved 14 months
8	50	F			+			MRI → intrasellar CSF collection with posterior displacement flattening of pituitary gland	Transsphenoidal packing of the sella using fat + reconstruction of the sellar floor + radiotherapy	Improved 18 months

6 cases, binasal hemianopia in 2 cases and multiple scotomas in two cases.

Endocrinological tests showed hyperprolactinemia in five women with DI in two of them, and hypothyroidism in the third case.

Plain X-ray showed enlarged sella turcica in 18 cases. CT scan with intrathecal contrast showed transsellar fistulous tract in 6 cases, CSF leak through cribriform plate in two cases and failed to identify the exact site of leak in two cases.

Preoperative MRI showed flattened pituitary gland against sellar floor and stretched pituitary stalk in all cases (Fig. 1), while postoperative MRI studies after transsphenoidal extradural packing revealed that the sellar contents appeared satisfactory elevated both on sagittal and coronal planes with

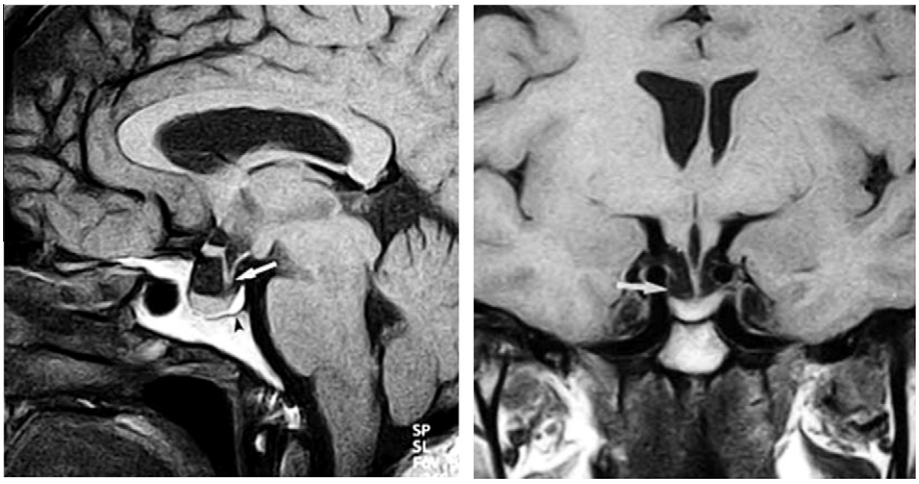

Figure 1 Preoperative sagittal and coronal T1-weighted MRI in patient with primary ESS showing the pituitary gland is flattened against the sellar floor, and the stretched pituitary stalk.

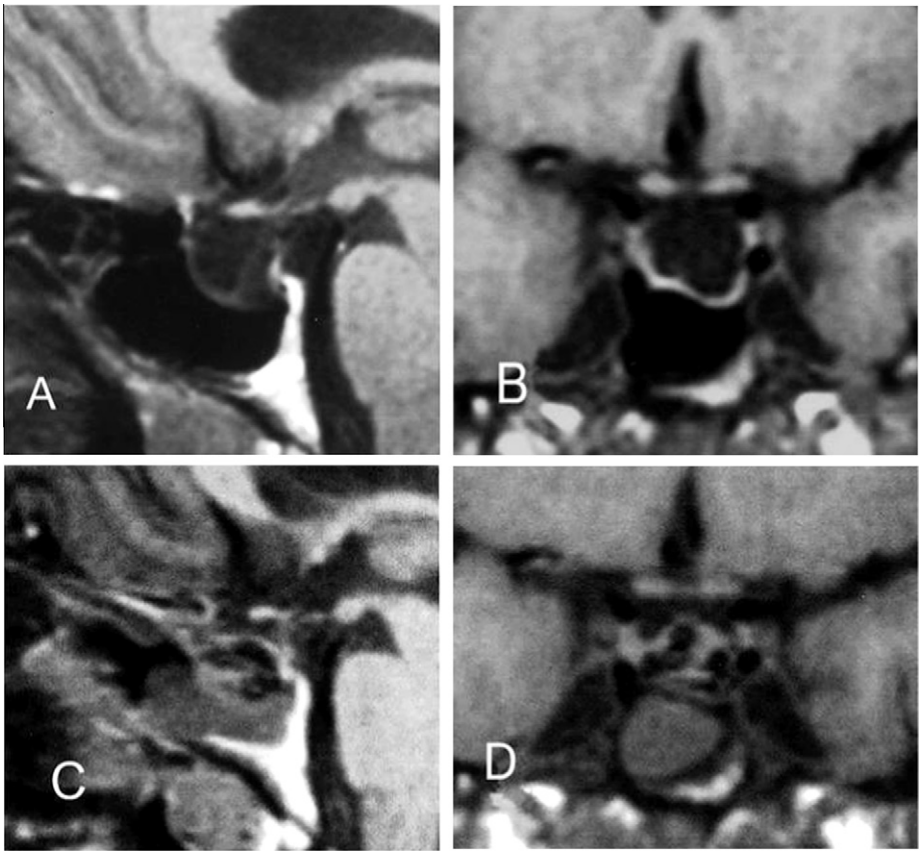

Figure 2 A & B: Preoperative sagittal and coronal T1-weighted MRI in patient with primary ESS showing downward traction of chiasm and the pituitary gland is flattened against the sellar floor, and the stretched pituitary stalk. C & D: Postoperative sagittal and coronal MRI of the same patient 6 months after extradural transsphenoidal packing of the sella with muscle & fat showing that the chiasm and pituitary stalk are in normal position.

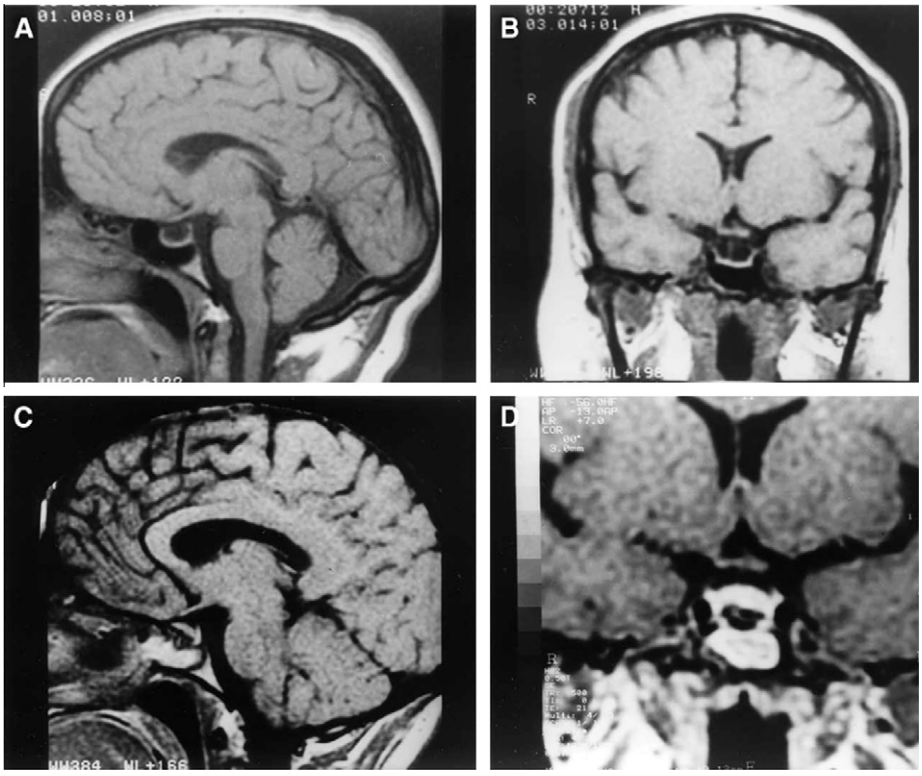

Figure 3 A & B: Preoperative sagittal and coronal T1-weighted MRI in patient with primary ESS showing downward traction of chiasm and the pituitary gland is flattened against the sellar floor, and the stretched pituitary stalk. C & D: Postoperative sagittal and coronal MRI of the same patient 12 months after extradural transsphenoidal packing of the sella with fat showing that the chiasm and pituitary stalk are in normal position.

upward lifting of suprasellar structures in all the 16 cases (Figs. 2–4).

Patients with preoperative complaint of headache respond well to surgery, with complete resolution in 85.3% of cases (12 cases out of 14). On contrast only 60% of the patients with preoperative visual field defect improved (6 cases out of 10). No patients with preoperative ↓ visual acuity (4 cases) have improved after surgery.

Outcome of cases: At the last follow-up examination period after surgery, 10 patients (50%) were asymptomatic, 6 patients (30%) improved, and 4 patients (20%) stabilized. No patient in the improved group developed new deficits or had progression of an existing preoperative deficit. The differences between the age of the patients among the various outcome groups were not significant ($p > 0.05$). The differences in the main clinical presentation before surgery among the various outcome groups were statistically significant ($p < 0.05$) (Table 4).

Postoperative morbidity and mortality: There was no post operative mortality, and surgery did not provoke any permanent neurological aggravation of any of the cases. Three patients had severe postoperative headache, lasting from three to seven days after surgery. One patient had temporary postoperative decrease of his visual acuity lasting for two days then improved on cortisone therapy.

4. Discussion

The term ESS describes a distinct radiological and anatomical entity in which the subarachnoid space extends significantly through an incompetent diaphragma sella into the sella turcica.[11] The diaphragma sella almost completely covers the pituitary body with only a small central opening for the passage of the infundibulum. Even though the term empty sella is the one most commonly used to refer to this clinical condition, it is well known that the term is incorrect, because in these cases the sella is not empty, but rather completely filled by the pituitary gland, with its stalk, the arachnoid, the CSF and occasionally, the optic system and the third ventricle. That is why some authors prefer to use the term intrasellar arachnoidocele because it expresses in a simple and clear way the findings in this entity.[5]

The condition is more common and benign in adults, with a female predominance, but its occurrence in children has been reported.[12–14] It has been found in association with several conditions, such as obesity, hypertension, and migraine.[15] A number of hypotheses have been offered to explain the cause of primary ESS such as pituitary infarction, pituitary apoplexy, and rupture of an intrasellar cyst.[16] Although one or more of these conditions may play role in the development of ESS, a reasonable explanation is that the condition arises in a patient who has either a transient or constant elevation in intracranial pressure and who has incompetent diaphragma sella that allows the subarachnoid space to be forced into the sella by the hydrostatic pressure and pulsatile movement of CSF.[17] Recently, shrinkage of the pituitary gland by antipituitary antibodies was advocated as another possible cause of primary ESS.[18] Secondary ESS is generally associated with a previous surgery, radiotherapy, or medical treatment for tumors of the sellar region.[1,2,4]

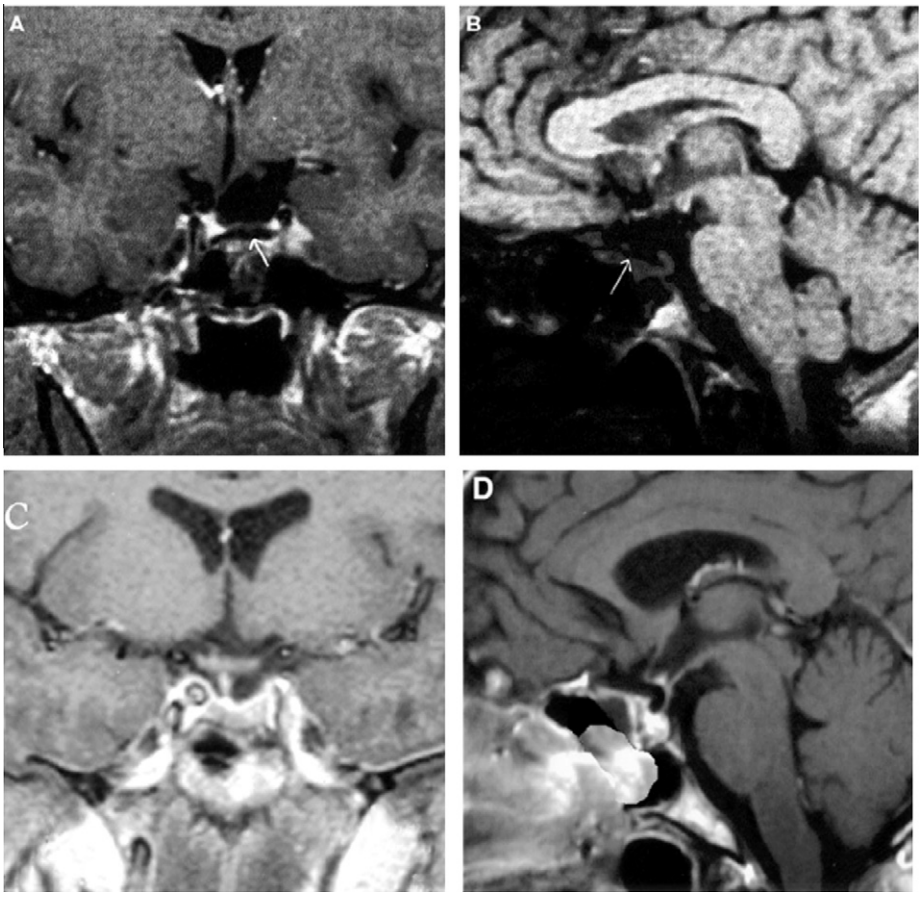

Figure 4 A & B: Preoperative sagittal and coronal T1-weighted MRI in patient with secondary ESS showing downward traction of chiasm, and the stretched pituitary stalk. C & D: Postoperative sagittal and coronal MRI of the same patient 8 months after extradural transsphenoidal packing of the sella with fat showing that the chiasm and pituitary stalk are in normal position.

Table 4 Outcome in 20 cases with EES versus main clinical presentation and age of patients.

Outcome	Cases		Main clinical presentation	Average age in years
	No.	(%)		
Asymptomatic	10	50	CSF rhinorrhea	37,5
Improved	6	30	Visual field defect	45
Stabilized	4	20	visual field defect and ↓visual acuity	44
Aggravated	0	0		0

In a review study by Bjerre[19] an empty sella (ES) of normal size was considered as a normal variant, whereas an enlarged ES is associated with pituitary disease or other clinical disorders. Enlarged sella was found in 90% of the cases in this study. Many theories explain enlargement of the sella. CSF pulsations against the parasellar structures can cause remodeling of the bony sellar floor. The bony erosion, especially if augmented by increased intracranial pressure, can cause communication of the intrasellar subarachnoid space with the sphenoid sinus.[20] CSF rhinorrhea may be also attributed to benign intracranial hypertension, which is frequently associated with ESS.[1,2,20] CSF pressure elevation has been recorded in 65% of patients with CSF rhinorrhea.[19] The site of the leak is usually into the sphenoid sinus but may be through the cribriform plate and can be distinguished after the injection of the intrathecal contrast.[20] In this study, the 10 cases presented with CSF rhinorrhea the intrathecal CSF pressure was intermittently high in 7 of them (70%). Also the site of the CSF leak was through the sellar floor in 6 cases, through cribriform plate in two cases and cannot be identified in the two cases.

Bjerre[19] discounted that theory of being the cause of chiasmatic cistern herniation into the sella because as many patients with an enlarged empty sella have normal intracranial pressure. In addition, patients with hydrocephalus rarely have an enlarged ES. The theory receiving his support is that an enlarged ES reflects a stage in the spontaneous course of some pituitary adenomas. The bony enlargement is a result of the growing adenoma, whereas spontaneous necrosis may allow

herniation of the subarachnoid space after absorption of the necrotic material. This theory explains the enlargement of the sella, the presence of pituitary hypersecretions, chiasmal lesions, spontaneous CSF rhinorrhea, and possibly even pseudotumor cerebri. Necrosis of a pituitary adenoma also explains the lack of further sellar enlargement once an enlarged ES has been diagnosed. The onset of pituitary necrosis with hemorrhage can have no symptoms or variable symptoms such as classic pituitary apoplexy or a minor attack.

The pathogenesis of the visual changes is attributed to herniation of the suprasellar cisterns into the sellar space. This causes downward displacement of the optic nerves, optic chiasm and exposes the optic structures to a more intense CSF pulsation, which results in a force directed toward the sellar floor that does not exist under normal conditions.[21,22]

The MRI is most effective tool for diagnosis of the empty sella that appeared large and filled with CSF extending downward from the suprasellar cistern. The pituitary gland is flattened along the floor of the sella turcica, usually in the posteroinferior portion. The pituitary stalk can be seen to traverse this CSF space from the median eminence of the hypothalamus down to the flattened pituitary gland. This is an important feature to ascertain, because it excludes the possibility that the sella turcica is occupied by a space occupying cyst.[1,2,23] Cysts and other space occupying lesions deviate the stalk away from its normal course.[2,3]

Considering the high risk of CSF rhinorrhea and infection, the technique of intradural packaging was replaced by the extradural technique that had been widely accepted and put into use as the current treatment modality. The extradural packing of the sella was performed with minimal trauma, and avoid the risk of overpacking, because the intradural and suprasellar structures were not directly manipulated as they were protected by the dural and arachnoidal planes, also their upward displacement was self limited by the insertion of the dura mater of the sellar floor on the medial wall of the cavernous sinus.[24]

Several materials have been suggested for filling the sellar space and reconstruction the sellar floor. They include bioabsorbable materials, muscle, fat, dural substitutes, cartilage, bone fragments, ceramic substances, titanium plates, and others.[24,25] As recorded by many authors in this study the fat was preferable to muscle because it results in less necrosis or scar retraction over time, so loss of initial volume was not excessive. This allowed for a more proportionate amount of initial packing material.[3,24]

In cases with secondary (ESS) adequate bone for reconstruction of the sellar floor may not be available, in such cases a bone graft from iliac crest, titanium plates, ceramic substances or acrylic material may be used.[24–29]

Transcranial operations were done formerly in secondary cases for the release of adhesions and elevating the herniated optic chiasm (chiasmaspexy). Outcome has generally been disappointing in these operations, as the visual failure commonly worsened.[30,31] Mortara and Norell[32] suggested opening of the lamina terminalis to direct CSF pulsation away from the optic chiasm. Recently, filling of the sellar cavity by the way of treatment. The procedure consists of inserting inside the sella an amount of fat sufficient to push the optic structure into their normal suprasellar position.[3,4,24] Extradural transsphenoidal chiasmapexy was indicated when the optic chiasm herniates inside the sella and the herniation causes progressive visual abnormalities. Other techniques of chiasmapexy that involves insertion of an extradural inflatable balloon or silastic coil into the sellar space were proved successful. These techniques allow the sellar space to be monitored by intraoperative fluoroscopic means after the balloon is inflated with contrast material.[30–34] These techniques were criticized by some authors[32,35,36] who noted that basal dura was continuous with the medial wall of the cavernous sinus, with no clear dissection plan between them; therefore dura could not be elevated su½ciently for the placement of the balloon into the sellar cavity and bleeding from the cavernous sinuses could occur.

In this study, the indication for surgery was based on the presence of neurological deficit (visual field or acuity) or CSF rhinorrhea, prophylactic surgery was not done to any one of the cases. In this study, extradural transsphenoidal packing of the sellas with fat alone or with fat, muscle and fascia were done in 16 out of 20 cases with no permanent postoperative neurological complications. Postoperative improvement of headache occurred in 85.3% of cases (12 cases out of 14), and of the visual field defect in 60% of the patients (6 cases out of 10). No patients with preoperative ↓ visual acuity (4 cases) have improved after surgery. CSF rhinorrhea was not resolved by the first operation in 4 out of 10 cases. In this series good results were obtained in 16 cases (80%) after a mean follow up period of 30 months. Nearly the same results were marked by Gallardo et al.[37] who reported the results of treatment in 76 successive cases with ESS (73 primary and 3 secondary). Transsphenoidal packing of the sellas was done in 56 of their cases with 7.1% postoperative complications. They reported postoperative improvement of headache in 71% and of visual disturbances in 46% of their cases. Also CSF rhinorrhea was not resolved by the first operation in 6 out of 13 cases. In 20 patients without surgery, headache improved in 64.4% of patients.

5. Conclusion

Surgical indications for correction of symptomatic ESS remain controversy and rare. Visual disturbances and CSF rhinorrhea are the main indications for surgery. When surgery is indicated, the type of surgery depends on clinical presentation and radiological findings. The surgical outcome of cases with ESS is favorable, as most patients report improvement or stabilization of their symptoms.

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