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Transsphenoidal Approach for Pituitary Macroadenoma: Complications

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ABSTRACT

Transsphenoidal surgery (TSS) is a popular method for resecting pituitary tumors, but it has been associated with severe consequences such as perioperative medical issues, endocrine problems, and surgical complications. The pituitary gland, a bilobed endocrine organ, contains eight hormones and is surrounded by the sella turcica, tuberculum sellae, dorsum sellae, and cavernous sinus dura. The gland's structure is complex, with the optic chiasm and sphenoid sinus being significant structures. The sphenoid sinus, which grows with age and has variable septal and cavitary architecture, can make the transsphenoidal approach more difficult to perform in cases of bleeding, vision loss, or cranial nerve palsy. Understanding the anatomy of the sphenoid sinus is crucial for a safe sellar approach and tumor removal. The operative microscope introduced in the 1960s has further improved surgical outcomes with low morbidity and mortality rates.

Pituitary adenomas are extra-arachnoidal tumors that develop outside the boundaries of the cerebrospinal fluid (CSF). They can cause iatrogenic leaks if the arachnoid membrane is ruptured and a cerebrospinal fluid fistula is introduced. Delayed postoperative epistaxis can result from the sphenopalatine artery and the intracavernous internal carotid artery (ICA). Loss of vision can result from physical injuries to the optic nerves or chiasm, which can occur at multiple stages of the operation. Other causes include cerebral vasospasm, traction injury, empty sella syndrome with chiasm prolapse, ophthalmoplegia, and internal injuries to the carotid artery.

Injuries to the sphenoid sinus, including proximity to essential tissues, thin or absent bone, and loss of bone, increase the risk of iatrogenic injury. Packing is the initial therapy, and postoperative angiography should be performed afterward. Embolization is considered in situations where packing is inadequate or when there is development to pseudo-aneurysms or carotid cavernous fistulae.

Chronic Insipidus Diabetes (DI) is a common disturbance in patients following transsphenoidal surgery (TSS), causing water and electrolyte problems. DI can manifest in various clinical patterns, including transitory, permanent, and triphasic phenotypes. Postoperative syndrome of antidiuretic hormone secretion (SIADH) is also a risk factor, with hyponatremia often delayed and symptomatic. Improved or newly developed hypopituitarism is a risk, with the hypothalamic-pituitary-adrenal (HPA) axis being the most susceptible. Transcranial procedures with HPA or other axis deficits may cause damage to normal residual glands, especially in large tumors. Transsphenoidal techniques can mitigate the risk of dissecting an attenuated gland, but understanding pituitary physiology and sellar anatomy is crucial for improving the procedure and encouraging innovation. Transcranial procedures have a low death rate and risk of significant impairment, but understanding the underlying medical, endocrine, and surgical consequences is essential for successful PA excision.

KEYWORDS: transsphenoidal surgery, complications, adenoma

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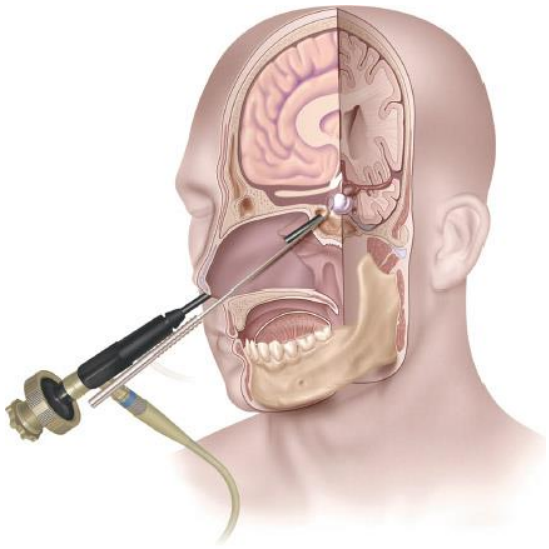
INTRODUCTION

The majority of pituitary tumor resections are performed using the transsphenoidal technique; nevertheless, current pituitary surgery is founded on a lengthy history of utilizing a variety of methods to the sella turcica because of its historical significance. Beginning with Caton and Paul's first

attempted subtemporal approach in 1893, pioneering surgeons championed several ingenious approaches to the pituitary fossa, comprising both transcranial and transfacial routes and including the transsphenoidal approach utilized by Hermann Schloffer in 1907 and subsequently popularized by Hirsch and Cushing in the 1910-20s.1 Although transsphenoidal surgery (TSS) grew out of favor for a period of several decades, it has gained widespread use since Hardy introduced the operative microscope in the 1960s, and since then, a large number of studies have demonstrated excellent surgical outcomes with low rates of morbidity and mortality. Even though TSS has been around for a very long time, it continues to be a possible cause of a number of severe consequences that are linked with it. These complications can range from perioperative medical concerns to endocrine problems to surgical problems. It is possible that the complications that arise during TSS might be partially explained by the fact that there are several potential for procedural mistake in a limited anatomic area that possesses a distinct physiology and structural diversity. With the purpose of highlighting surgical and perioperative difficulties associated with TSS, as well as specific characteristics of sellar architecture and physiology that contribute to the dangers that are intrinsic to TSS, this review helps to emphasize these aspects.



Anatomy and physiology

Pituitary Gland (Glo)

In the pituitary gland, which is a bilobed endocrine organ, there are eight different hormones that are secreted. Two of these hormones come from the posterior lobe, while the other six come from the anterior lobe. Not only does the posterior lobe, which is an extension of the central nervous system and is composed of specialized glial cells, lack a blood–brain barrier, but it also functions as a conduit for vasopressin and oxytocin that are generated from the hypothalamus. On the other hand, the anterior lobe, which is generated from the ectoderm, is composed of pituitary cells that are actively secreting and wraps around the caudal pituitary stalk. A slender collection of magnocellular hypothalamic axons is known as the pituitary stalk. This stalk connects the pituitary gland to the hypothalamus at a superior position through the pituitary gland's posterior lobe.

Complication	***Description***
CSF Leak	Leakage of cerebrospinal fluid, leading to risk of meningitis.
Infection	Postoperative infection, including meningitis and sinusitis.
Hemorrhage	Bleeding during or after surgery, which can lead to hematoma or increased intracranial pressure.
Diabetes Insipidus	Due to damage to the pituitary or hypothalamus, resulting in excessive urination and thirst.
Hormonal Imbalance	Hypopituitarism due to damage to pituitary gland, leading to deficiencies in one or more hormones.
Vision Changes	Damage to optic chiasm or nerves, potentially causing visual field defects or loss of vision.
Nasal Complications	Persistent nasal discharge, crusting, or nasal septum perforation.
Cranial Nerve Injury	Potential injury to cranial nerves, leading to symptoms like double vision or facial numbness.
Vascular Injury	Injury to major blood vessels, which can cause significant bleeding or stroke.
Recurrence	Incomplete tumor removal, leading to regrowth and recurrence of symptoms.

Regional Cavernous Sinuses and the Sellar Region

Not only is the structure of the pituitary gland itself complicated, but the bone anatomy in which it is located is also rather complicated. The pituitary gland is completely encircled by sphenoid bone, with the exception of the aperture of the extremely thin and frequently rectangular diaphragma sellae that lies on top of it. The gland is located in the sella turcica, and it is surrounded by the tuberculum sellae and the sellar floor in the anterior direction, the dorsum sellae in the posterior direction, and the cavernous sinus dura in the lateral direction. The internal carotid artery and the abducens nerve are both located inside the lateral walls of the cavernous sinuses. Additionally, the oculomotor nerve, the trochlear nerve, and the ophthalmic and maxillary divisions of the trigeminal nerve are all located within these sinuses.

An Area of Suprasellarity

As is the case in the majority of the population, the optic chiasm can either be "prefixed" above the tuberculum sellae or "postfixed" above the dorsum sellae. The optic chiasm is formed by the confluence of optic tracts posterolaterally and gives rise to the optic nerves anterolaterally. It is also possible for the optic chiasm to sit directly above the diaphragma

sellae. The oculomotor nerve, which travels in a direction parallel to the optic nerve as it moves toward the superior orbital fissure, and the olfactory tracts, which cross the optic nerves inferiorly and bifurcate superior to the anterior clinoid processes, are two of the other cranial nerves that are located in this region.

Nose of the Sphenoid

Additionally, the sphenoid sinus, which is a component of the body of the sphenoid bone, is located anteriorly and inferiorly to the sella. The sinus grows with age and has highly variable septal and cavitary architecture, factors that reduce the reliability of anatomic landmarks and reinforce the importance of thorough preoperative imaging. Also variable are the characteristics of the sphenoid ostia that drain the sphenoid sinus into the nasal cavity, specifically the spheno-ethmoidal recesses situated superiorly and posteriorly to the superior nasal turbinates. To ensure a safe sellar approach and tumor removal, it is essential to have a comprehensive awareness of the diversity that exists inside the gland, in addition to the physical features of the gland itself.

Having a grasp of the anatomy of an individual's sphenoid sinus is equally as significant as having an understanding of their anatomy lateral to the walls of their sphenoid sinus. This region is home to a number of significant structures, including as the optic canal, the maxillary division of the trigeminal nerve, and the internal carotid artery. These formations are located in close proximity to the exterior surface of the sinuses, and in many instances, they protrude or erode through the areas of the sinus wall that include bone dehiscence. It is reasonable to assume that the protrusion of these tissues, in addition to any bone dehiscence into the sinus, can make the transsphenoidal approach more difficult to perform in the event of bleeding, vision loss, or cranial nerve palsy.

The leakage of cerebrospinal fluid

Pituitary adenomas are classified as extra-arachnoidal tumors, which means that they develop outside of the boundaries of the cerebrospinal fluid (CSF). Even in cases of large invasive illness, tumors do not often break through the arachnoid membrane and expand into the subarachnoid region. Consequently, this indicates that the process is typically of an iatrogenic origin if the arachnoid membrane is ruptured and a cerebrospinal fluid fistula is introduced. It is possible for this to take place at a number of different phases of a certain procedure, at a number of distinct places. It is most usual for CSF fistulae to develop in the diaphragma sellae, which is also the most common site. In the case of resections of bigger pituitary adenomas, this is especially relevant because the massive bulk of the tumor can induce redundancy in the arachnoid, which, once the tumor is gone, may descend or herniate into the expanded sellar cavity. CSF fistula can also develop as a result of operating too far superiorly, which can cause cribriform plate injury in the ethmoidal region. This type of injury may also be associated with outfracturing of the middle turbinates. Not only could this result in a leak of CSF, but it also has the potential to cause direct damage to the olfactory epithelium of the superior nasal cavity, which can result in either temporary or permanent anosmia. In the event that a CSF leak occurs, the repercussions can be severe regardless of the origin of the leak. It is possible that fluid loss might be severe enough to produce a fall in cerebrospinal fluid pressure below atmospheric pressure, which would facilitate the advancement of pneumocephalus, tension pneumocephalus, or perhaps meningitis if the primary repair is not performed or if therapy is insufficient.

The Epistaxis

The sphenopalatine artery and the intracavernous internal carotid artery (ICA) are two of the most significant causes of delayed postoperative epistaxis. Other notable sources include the sphenopalatine artery. When the sphenoid sinus is opened anteriorly and inferiorly, there is a possibility that the sphenopalatine artery will be injured. It is of the utmost importance to protect the sphenopalatine artery and the associated vascular pedicle to the nasal septum in the event that a pedicled nasal-septal flap is required for the repair of a high-grade CSF leak.

Loss of Vision;

Loss of vision can be the consequence of a physical injury to the optic nerves or chiasm, which can be caused by either direct trauma or excessive traction respectively. It is possible for this to happen at multiple stages of the operation, both by advancing too far into the sphenoid body and by deviating the approach to its superior lateral wall. As was mentioned earlier, the bony structures that separate the sinus from adjacent structures in this location, such as the optic canal, are frequently thin or absent, which makes any deviation in approach potentially dangerous. Ischemic damage can occur as a result of mechanical deformation of the optic canal and subsequent compression of the optic nerve, regardless of the origin of the distortion. The nerve is particularly vulnerable at the proximal entrance of the optic canal, which is where the overlaying dura forms the falciform process. Vision loss associated with TSS arises more frequently as a consequence of overpacking of the sella during the process of rebuilding the skull base. The reversal of edema and monocular blindness can be accomplished with rapid decompression and the injection of high-dose steroids, provided that timely action is taken by the patient.

Traumatic injuries to the orbit itself are another condition that can result in vision loss. Furthermore, the bivalve speculum, which was traditionally preferred for the transsphenoidal approach, has the potential to fracture not only the sphenoid body but also the orbital wall. This is despite the fact that it is not frequently utilized in the present era of endoscopic endonasal techniques. This might have caused a hemorrhage, either intrasellar or suprasellar, which could then lead to orbital compartment syndrome and subsequent ischemia. It is essential to do prompt hematoma evacuation in the event of such occurrences.

Other notable causes of postoperative visual loss include cerebral vasospasm and traction injury, particularly in revision surgeries for previous craniotomies. The superior hypophyseal artery is an important vessel to identify and preserve during extended endonasal approaches for craniopharyngiomas and entities of the suprasellar region. Additionally, traction injury is a cause of postoperative visual loss. Finally, empty sella syndrome with chiasm prolapse has been recorded as a cause of progressive postoperative vision deterioration. Documentation of this condition has been found.

A condition known as ophthalmoplegia

Oculomotor nerve palsy has also been documented after surgical manipulation in the cavernous sinus or excessive packing of the sella. This condition frequently manifests itself with a down-and-out gaze, ptosis, and mydriasis. The abducens nerve is the most frequently injured of all the cranial nerves that occur in the cavernous sinus. It travels medial to the ICA and lateral to V1.

Injuries to the Carotid Artery Internally

Despite the fact that cadaveric studies have demonstrated that the intracavernous ICA is typically between 1 and 3 millimeters and can be as far away as 7 millimeters from the pituitary gland, it is possible for it to occasionally protrude through the wall of the cavernous sinus and abut the gland itself. The close proximity of the gland to the ICA, in conjunction with the possibility of inadequate bony protection, emphasizes the significance of maintaining a midline approach, even when adenomas extend laterally into the cavernous sinuses. Additional measures for lowering the risk of iatrogenic ICA injury linked with TSS include neuronavigation, routine use of a micro-Doppler flow probe, and avoidance using sharp equipment in the cavernous sinus region. These strategies are in addition to having a comprehensive grasp of the anatomy of the sellar region.

Similar dangers can be seen within the sphenoid sinus, including proximity to essential tissues, bone that is either thin or absent, and loss of bone. There have been many cadaveric and computed tomography (CT)-based investigations that have showed ICA protrusion in 26% to 41% of instances and dehiscence in 4% to 8% of cases. These are characteristics that enhance the risk of iatrogenic injury. The most feared consequence of such injuries is the possibility of bleeding, which can be serious. Packing is the initial therapy that should be administered in these situations, and postoperative angiography should be performed afterward. There is a risk of cerebral infarcts and occlusion or stenosis if the patient is overpacked. Embolization is something that should be considered in situations when packing is inadequate or when there is development to pseudo-aneurysms or carotid cavernous fistulae.

Chronic Insipidus Diabetes

It is largely agreed upon that the spectrum of water and electrolyte problems that are found following TSS can be attributed to excessively forceful stimulation of the pituitary stalk. Diabetes insipidus (DI) is one of the most common of these disturbances, occurring transiently in approximately 4% to 18% of resections. Traction to the stalk or direct posterior lobe injury can damage the magnocellular hypothalamic axons within and lead to subsequent deficits in antidiuretic hormone (ADH) production and secretion. The persistence and degree of DI is often associated with the level of injury to the infundibulum and posterior pituitary gland, with higher level stalk injuries posing a greater risk for permanent DI.

In the context of pituitary surgery, DI can manifest itself in a variety of clinical patterns. These patterns include a transitory phenotype, a permanent phenotype, and a traditional triphasic phenotype, which is observed in situations of severe pituitary injury. This triphasic phenotype is characterized by a temporary DI, which is caused by early decreases in ADH. This results in a clinical appearance of hyponatremia or normonatremia, depending on the severity of the necrotic posterior lobe, which releases stored ADH several days later. While there is limited research that quantifies decreases in ADH secretion following pituitary damage, it is understood that reduced traction and careful handling of the gland decreases the risk for endocrine complications. Finally, any SIADH-like presentation is replaced by a permanent DI around postoperative day 7 to 10 for any SIADH-like presentation. In a recent study, the hormonal changes that are associated with incremental pituitary damage were mathematically modeled. The study predicted that there could be a transient diastolic dysfunction (DI) after 20% posterior pituitary damage and a permanent DI after 20% to 40%. The characteristics and size of the tumor are also important, as macroadenomas can provide the stalk with a fibrous layer that helps prevent traction injury.

Inappropriate secretion of antidiuretic hormone

Manipulation of the posterior lobe, stalk, and hypothalamus are all examples of processes that are assumed to be responsible for the development of postoperative syndrome of antidiuretic hormone secretion (SIADH). These mechanisms are also thought to be responsible for the development of DI. The onset of hyponatremia is often delayed, occurring sometime around the seventh postoperative day. Hyponatremia can be asymptomatic or present clinically with symptoms such as fatigue, headache, nausea, or vomiting when blood sodium levels drop below 130 mEq/L. This is dependent on the degree of the ADH deviation. In addition, any indication for more comprehensive gland investigation would raise the risk of postoperative water and electrolyte disturbances. This is because, as was mentioned earlier, a transitory DI that occurred before to the operation is a substantial risk factor for the release of stored ADH, which can then lead to hyponatremia and euvolemia.

Improved or newly developed hypopituitarism

Following the somatotropic axis (growth hormone), the gonadal axis (follicle stimulating hormone and luteinizing hormone), the thyroid axis (thyroid stimulating hormone), and the adrenal axis (adrenocorticotrophic hormone) are

thought to be the most vulnerable. Preoperative hypopituitarism is thought to be caused by a growing adenoma. This hypopituitarism is thought to involve the hypothalamic-pituitary axes in a predictable sequence. An interruption of these axes can occur as a consequence of a number of different causes, which can range from the displacement of the stalk and gland to the compression and obliteration of the axes. The resultant restriction in hormone transport can therefore be either reversible or permanent in nature, depending on the size of the adenoma and the development history of the tumor. It is therefore possible that selective adenomectomy and gland or stalk decompression would not only maintain the pituitary function that is already there, but would frequently augment it under ideal circumstances.

In spite of the fact that TSS has the ability to normalize endocrine function, it also carries with it the danger of a worsening of preoperative hypopituitarism or the development of new hypopituitarism. Although few studies differentiate among the axes involved in new or worsened hypopituitarism, a recent retrospective study suggested the hypothalamic-pituitary-adrenal (HPA) axis may be the most susceptible, with affected patients lacking postoperative ACTH and cortisol stress responses. For these surgeries complicated by HPA or other axis deficits, damage to normal residual gland may occur whenever the anterior pituitary gland is manipulated, with the risk especially high in large tumors that distort, diminish, and lift the gland superiorly towards the diaphragma sellae. The greater prevalence of new hypopituitarism following transcranial procedures, which essentially include a superior sellar approach, may be partially explained by this unique relocation of residual gland, which may serve as a possible explanation. Logically, the risk of dissecting an attenuated, albeit normal, gland is mitigated by a transsphenoidal approach that accesses the tumor from below. Additionally, careful identification of any residual gland while working in the sella is also beneficial. Normal gland can be distinguished by its orange color and structural integrity that is resistant to curettage and suction. It is important to correlate the glandular architecture that was investigated on preoperative T1-weighted imaging with the visual anatomy.

CONCLUSION

When done by qualified surgeons, the transsphenoidal technique continues to be the primary method of therapy for the majority of PAs. This surgery has a low death rate and an extremely low risk of significant impairment. In spite of this, it is essential to recognize the wide range of unfavorable medical, endocrine, and surgical consequences that can make PA excision more difficult, whether it be in terms of reaching the gland or addressing the disease that it causes. In order to do this, it is necessary to have a full understanding of pituitary physiology as well as typical variation in sellar anatomy. These are elements that will not only increase the effectiveness of the procedure, but will also encourage innovation in order to enhance patient safety.

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