Complication Avoidance in Endoscopic Skull Base Surgery

Peleg M. Horowitz, MD, PhD[a,1], Vincent DiNapoli, MD, PhD[a,b,1], Shirley Y. Su, MD[c], Shaan M. Raza, MD[a,c,*]

KEYWORDS

• Endoscopic skull base surgery • Complications • Diabetes insipidus
• Panhypopituitarism • CSF leak • Vascular injury • Cranial nerve injury

KEY POINTS

- Expanded endoscopic resection of pituitary and complex skull base pathology requires an understanding of medical and surgical complications unique to these surgical techniques.
- Common medical complications include anterior and posterior pituitary dysfunction and meningitis; other medical complications such as venous thromboembolism or pneumonia are rare.
- Common surgical complications include vascular injury, cerebrospinal fluid fistula owing to reconstruction failure, cranial nerve injury, and infection/meningitis.

INTRODUCTION

The use of endoscopic endonasal approaches (EEA) to the skull base pathology has evolved significantly over the past 2 decades. Development of the 2-surgeon collaboration between neurosurgery and otolaryngology physicians for the endoscopic resection of pituitary adenomas has led to significant innovation and expansion of this approach to various skull base pathologies. As indications have expanded, surgeons have begun to define the expected incidence of common complications and adopted techniques to avoid or manage these adverse outcomes. With expansion of endoscopic approaches to more challenging tumors (ie, malignancies, intradural pathology), it is imperative that fundamental skull base principles are not short

[a] Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center, 1500 Holcombe Blvd, Houston, TX 77030, USA; [b] Department of Neurosurgery, The Mayfield Clinic, University of Cincinnati, 260 Stetson Street, Suite 2200, Cincinnati, OH 45267, USA; [c] Department of Head and Neck Surgery, The University of Texas M.D. Anderson Cancer Center, 1500 Holcombe Blvd, Houston, TX 77030, USA
[1] Both authors contributed equally to this article.
* Corresponding author. Department of Neurosurgery, 1500 Holcombe Boulevard, Unit 442, Houston, TX 77030.
E-mail address: smraza@mdanderson.org

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changed for the sake of performing an approach. In this article we review the complications and common pitfalls of the EEA for complex cranial base pathology.

ENDOCRINOLOGIC COMPLICATIONS

The most frequent postoperative systemic complications relate directly to the manipulation or disruption of the normal hypothalamic–pituitary axis. Diabetes insipidus (DI), the syndrome of inappropriate antidiuretic hormone release, and panhypopituitarism consequently occur almost exclusively in those populations of patients in which the pituitary gland, stalk, or hypothalamus are involved in either the pathology or the surgical corridor. These can be classified further pathophysiologically as perturbations of anterior gland (secretory function) or posterior gland (osmostat function).

Many studies have suggested equivalent rates of postoperative sodium dysregulation in microscopic and endoscopic pituitary surgery, approximating 11% to 14% in a recent metaanalysis.[1] Risk factors for developing postoperative DI (either transient or permanent) include tumor size, young age, and pathology involving the posterior gland or stalk, that is, Rathke's cleft cyst or craniopharyngioma.[2,3] Pars intermedia tumors and cystic adenomas likely confer an intermediate increase in risk, as does surgical exploration of the posterior gland and intraoperative traction on the pituitary stalk. These perturbations often manifest within the first 48 to 72 hours after surgery. However, delayed postoperative hyponatremia occurs in approximately 15% of cases, peaking between 1 and 2 weeks after surgery, leading to readmission in 6.4% of cases.[4]

Management of postoperative sodium fluctuations starts with vigilant attention to laboratory values, often by checking serum sodium and urine specific gravity every 6 hours for the first 2 to 3 days after surgery, and checking serum sodium on a postoperative clinic visit. The syndrome of inappropriate antidiuretic hormone release is most often mild and managed by fluid restriction and free water deprivation; rare refractory or severe cases may require hypertonic infusion. DI may likewise be mild and managed by drinking free water to thirst, or may require desmopressin by various routes. By carefully educating patients on the signs and symptoms of sodium imbalance and the avoidance of excessive free water on discharge, it may be possible to reduce the incidence of delayed postoperative hyponatremia requiring readmission. Last, the surgeon must be mindful of the possibility of the so-called triple phase response, which encompasses the syndrome of inappropriate antidiuretic hormone release, transient DI, and delayed postoperative hyponatremia.[5]

Hypopituitarism after pituitary surgery likewise has not been shown to differ significantly between microscopic and endoscopic approaches, in approximately 3% to 6% of cases.[1] Adrenal crisis after such surgery can be fatal, although this can be avoided easily with judicious use of preoperative, intraoperative, and postoperative steroid administration. Preoperative low fasting morning cortisol should alert the surgeon that stress dose steroid administration on induction of anesthesia and subsequent taper to a maintenance dose may be indicated. Fasting morning cortisol should likewise be monitored while patients are in house, with cortisol repletion in cases where it is low. Any clinical suspicion for adrenal crisis should prompt swift response to administer steroids because a delay could be life threatening. After cortisol, repletion of thyroid hormone may also be considered, though the longer half-lives of thyroid hormone and other anterior gland hormones make their administration less critical in the immediate postoperative period.