MEDS Clinical Medicine (2026)
Clausius Scientific Press, Canada

DOI: 10.23977/medsc.2026.070119
ISSN 2616-1907 Vol. 7 Num. 1

Advances in the Analysis and Prevention of Diabetes Insipidus Following Pituitary Adenoma Resection

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Keywords: Pituitary adenoma; Diabetes insipidus; Pathological mechanism; Risk factors; Prevention and treatment strategies

Abstract: Pituitary adenoma is a common tumor of the central nervous system, for which transsphenoidal surgery is the mainstay of treatment. However, postoperative diabetes insipidus (DI) is a frequent complication, with an overall incidence ranging from 2% to 54%, of which approximately 4.6% may progress to persistent DI. This article aims to provide a systematic review of the epidemiology, pathophysiology, risk factors, and management strategies for DI following pituitary adenoma surgery. The core pathogenic mechanism involves surgical injury to the hypothalamic-pituitary axis, leading to insufficient synthesis or secretion of arginine vasopressin (AVP) or renal resistance to its action. The occurrence of DI results from the interplay of multiple factors, with primary risks pertaining to patient-, tumor-, and surgery-related aspects. Current prevention emphasizes meticulous intraoperative techniques as the cornerstone, including protection of the pituitary stalk, controlled use of electrocautery, and gentle manipulation. Treatment requires a tiered approach: mild cases can often be managed conservatively with fluid replacement and monitoring, while moderate to severe cases necessitate the standard use of desmopressin. Existing research is predominantly retrospective with heterogeneous diagnostic criteria. Future studies should focus on conducting multicenter prospective research to develop risk prediction models and to optimize individualized prevention and long-term management strategies for high-risk patients.

1. Introduction

Pituitary adenomas are common benign tumors of the central nervous system, with an estimated annual clinical incidence of approximately 3.23 to 5.1 per 100,000 individuals. Autopsy studies suggest a significantly higher prevalence [1, 2]. Advancements in microsurgical and endoscopic techniques have established transsphenoidal surgery as the primary treatment for symptomatic pituitary adenomas, particularly non-functioning adenomas[1, 3]. Despite the trend towards

minimally invasive approaches, postoperative complications remain challenging to eliminate completely due to the complex anatomy of the sellar region and its proximity to the hypothalamic-pituitary axis, a central regulator of water-electrolyte balance. Among these complications, diabetes insipidus (DI) is frequent, with reported incidence rates varying widely across studies from 2% to 54% [4, 5]. This complication not only significantly prolongs hospital stays and increases healthcare costs but, more critically, can progress to permanent DI in a subset of patients, necessitating long-term pharmacologic replacement therapy and substantially impairing quality of life[6].

The pathogenesis of postoperative DI is clearly linked to surgical injury to the hypothalamus, pituitary stalk, or posterior pituitary gland, resulting in deficient synthesis, secretion, or action of arginine vasopressin[7-9]. However, the risk factors involved are multifaceted and complex, encompassing patient age, tumor characteristics (such as size, consistency, and invasiveness), and surgical details (e.g., diaphragma sellae opening, cerebrospinal fluid leak), with the role of many factors still debated[10-13]. Regarding management strategies, while a prevention philosophy centered on meticulous intraoperative technique and a treatment paradigm based on medications like desmopressin have been established, significant challenges persist. These include achieving individualized prevention based on precise preoperative assessment and optimizing the long-term management of patients with permanent DI [14-16]. Therefore, systematically reviewing the epidemiological features of DI after pituitary surgery, analyzing the multifactorial pathophysiology in depth, and summarizing current advances in prevention and treatment are of great importance for guiding clinical practice and improving patient outcomes. This article aims to provide a comprehensive review focusing on these key aspects, intending to offer a theoretical basis and reference for the standardized management of this complication.

2. Overview of the Epidemiology, Pathological Mechanism and Clinical Treatment of Pituitary Adenoma

Pituitary adenomas (PA) are relatively common tumors of the central nervous system. Their annual clinical incidence is approximately 3.23 to 5.1 per 100,000 individuals[2, 17], while autopsy studies reveal a significantly higher prevalence of up to 16.7%[18]. A slight female predominance is noted, and these tumors are rare in children. According to the 2022 World Health Organization classification of pituitary tumors [19], this category includes tumors originating from the adenohypophysis (anterior lobe), neurohypophysis (posterior lobe), and the hypothalamus. Specifically, anterior lobe tumors are primarily classified into: (1) pituitary neuroendocrine tumors (PitNETs), which are well-differentiated adenohypophyseal neoplasms corresponding to the former entity of pituitary adenoma; (2) pituitary blastoma; and (3) two distinct subtypes of craniopharyngioma. PitNETs can be further subclassified into three groups based on cell-lineage-specific transcription factors: PIT1, TPIT, and SF1[19, 20]. Clinically, they are often categorized by size as macroadenomas (≥10 mm) or microadenomas (<10 mm). The distribution of various types is as follows: non-functioning adenomas constitute 57%, while functioning types account for 43% (e.g., growth hormone [GH]-secreting, prolactin [PRL]-secreting, adrenocorticotropic hormone [ACTH]-secreting).

Clinical manifestations typically follow three patterns: firstly, syndromes related to hormone excess or deficiency, such as hyperprolactinemia, acromegaly, and Cushing's disease from hypersecretion, or hypogonadism from partial/complete hypopituitarism; secondly, neurological symptoms due to mass effect, including headaches and visual disturbances; and thirdly, incidental discovery on imaging performed for unrelated conditions.

Current standard management encompasses surgical resection, medical therapy, and radiotherapy, with the choice of strategy highly dependent on tumor type, size, secretory activity, and clinical presentation. For functioning pituitary adenomas, medical therapy plays a central role. This is based

on the expression of hypothalamic neuropeptide receptors that regulate hormone secretion and tumor growth in most secretory adenomas, allowing for targeted receptor ligand therapy [1, 21]. For instance, most prolactinomas and GH-secreting adenomas, as well as some ACTH-secreting and TSH-secreting tumors, respond well to drugs, achieving long-term biochemical control and tumor growth inhibition. Conversely, for symptomatic non-functioning pituitary adenomas (NFPAs), surgical resection is the primary treatment modality [2, 3]. Postoperative complications for patients with PAs are varied and can include surgical site hemorrhage, cerebrospinal fluid rhinorrhea, intracranial infection, diabetes insipidus, and pituitary insufficiency[18]. Among these, diabetes insipidus, resulting from injury to the hypothalamic-pituitary axis, warrants particular attention.

3. Definition and Pathophysiological Basis of Diabetes Insipidus

Diabetes insipidus is a disease characterized by the excretion of a large amount of hypotonic urine, leading to symptoms such as polyuria, polydipsia, low urine specific gravity and electrolyte disturbance in patients, and even inducing epilepsy[22, 23]. Its overall prevalence is approximately 1 case per 25,000 population. The incidence is similar in males and females, with no significant gender difference. The age of onset is closely related to the etiology type: hereditary diabetes insipidus mostly onsets in infants or adolescents, while acquired diabetes insipidus mostly occurs in adulthood[24].

The currently commonly used diagnostic criteria are as follows: 24-hour urine output > 4000 ml or urine output > 200 ml/h for 2 consecutive hours; urine specific gravity < 1.005 or urine osmotic pressure < 300 mOsm/kg; no kidney or adrenal gland-related diseases; no use of exogenous antidiuretic hormone; exclusion of polyuria caused by diuretics or hyperglycemia[24]. For a long time, the water deprivation-vasopressin test has been regarded as the classic reference method for identifying diabetes insipidus, but it still has certain limitations in distinguishing partial diabetes insipidus from primary polydipsia. The newly developed plasma copeptin detection technology provides a new and effective approach for the accurate diagnosis and differentiation of diabetes insipidus[25].

According to the etiology, diabetes insipidus (DI) can be divided into acquired and hereditary types, among which acquired diabetes insipidus is far more common than the hereditary type, accounting for about 90% of all DI cases[26]. Acquired diabetes insipidus mainly includes central and nephrogenic types. The causes of central diabetes insipidus mainly include damage from trauma, tumors, inflammation, etc., while the causes of nephrogenic diabetes insipidus are often factors such as drugs and metabolic disorders[24]. Among them, acquired central DI is most common after neurosurgery, especially after pituitary region tumor resection, with an incidence of approximately 20%; such postoperative DI is often transient, but its recovery depends on the degree of neuronal damage to the hypothalamic-neurohypophyseal axis, and severe cases can develop into permanent diabetes insipidus[27]. Diaphragma sellae opening and cerebrospinal fluid leakage are two key risk factors for postoperative diabetes insipidus. The former compresses and stretches the hypothalamic-pituitary axis mainly through the disturbance of cerebrospinal fluid circulation, while the latter further damages neurological function due to a sudden drop in intracranial pressure and subsequent packing compression and inflammatory response. Both interfere with the normal secretion of antidiuretic hormone, thereby increasing the risk of postoperative diabetes insipidus[28]. Other acquired causes include primary brain tumors (e.g., pituitary adenomas rarely cause it directly but may lead to it through local compression), hypophysitis, infiltrative diseases (e.g., Langerhans cell histiocytosis, sarcoidosis), exposure to drugs or toxins, osmotic receptor dysfunction (lipogenic DI), and infectious diseases (e.g., meningitis, encephalitis or tuberculosis). Notably, approximately 25-50% of adult-onset central DI cases have an unknown etiology at the initial diagnosis and are classified as idiopathic

DI[26]. Hereditary diabetes insipidus is relatively rare, and it should be highly suspected if the onset is in the early stage of life with a clear family history. Among them, central familial DI is mainly autosomal dominant inheritance, mostly caused by AVP gene mutations[29].

The occurrence of diabetes insipidus is closely related to the dysfunction of the hypothalamic-pituitary axis. The normal water and electrolyte regulation function of the hypothalamic-neurohypophyseal system depends on magnocellular neurons in the supraoptic nucleus and paraventricular nucleus of the hypothalamus. The axons of these neurons form the hypothalamic-neurohypophyseal tract, descending to the neurohypophysis, and release the synthesized arginine vasopressin (AVP) into the blood through the pituitary portal system. Arginine vasopressin (AVP), also known as antidiuretic hormone (ADH), is the core hormone maintaining body water homeostasis. After entering the blood, it mainly acts on the renal collecting ducts, binding to vasopressin V2 receptors on the basolateral membrane, thereby activating and up-regulating aquaporin 2 (AQP-2) on the luminal membrane. The normal operation of this signaling pathway can promote water reabsorption, thus precisely regulating urine concentration and dilution according to plasma osmotic pressure[7-9]. The physiological mechanism diagram is shown in Figure 1. The dysfunction of the above water homeostasis regulation mechanism leads to impaired urine concentration function and the occurrence of diabetes insipidus (DI). According to different pathological links, DI is mainly divided into two types: central diabetes insipidus, caused by insufficient synthesis or secretion of AVP due to damage to the hypothalamic-neurohypophyseal system; and nephrogenic diabetes insipidus, resulting from the lack of renal response to normal or high levels of AVP, mostly caused by V2 receptor or AQP-2 dysfunction. In the field of neurosurgery, direct damage to the hypothalamic-neurohypophyseal system by trauma, tumors or surgery is a common cause of central diabetes insipidus[18, 24].

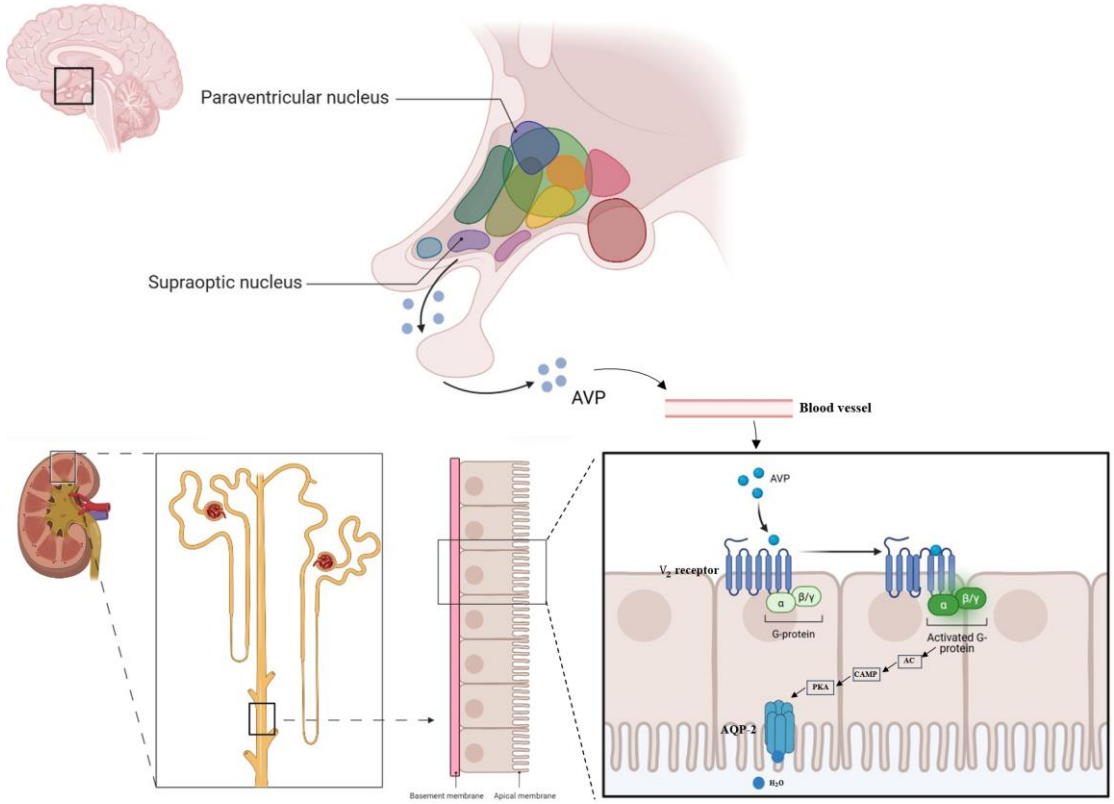

Figure 1. Normal Physiology and Pathogenesis of Diabetes Insipidus

Under physiological conditions, arginine vasopressin (AVP) synthesized in the hypothalamic supraoptic and paraventricular nuclei is released into the circulation from the neurohypophysis. It then activates the V2 receptor on renal collecting ducts, upregulating aquaporin-2 (AQP-2) expression to promote water reabsorption and urine concentration. In central diabetes insipidus (CDI), injury to the hypothalamic-neurohypophyseal system leads to deficient AVP synthesis/secretion. In nephrogenic diabetes insipidus (NDI), dysfunction of the V2 receptor or AQP-2 causes renal unresponsiveness to AVP. Both disorders ultimately result in impaired urine concentrating ability, manifesting as polyuria and hypotonic urine.

Abbreviations: AVP, arginine vasopressin; AQP-2, aquaporin-2; CDI, central diabetes insipidus; NDI, nephrogenic diabetes insipidus.

4. Incidence and Risk Factors of Diabetes Insipidus after Pituitary Adenoma Resection

With the advancement of minimally invasive neurosurgical techniques, endoscopic and microscopic transsphenoidal surgery has become the mainstream surgical method for the treatment of pituitary tumors[30]. However, due to the complex anatomical structure of the pituitary region, surgery is prone to damage the key structures of the hypothalamic-pituitary axis, leading to complications such as postoperative diabetes insipidus. Central and transient diabetes insipidus is the most common type after pituitary tumor surgery, with a widely reported overall incidence ranging from 2% to 54%[4, 5]. Studies have shown that the incidence of postoperative diabetes insipidus is 14.7%, among which permanent diabetes insipidus accounts for 4.6%[6].

The incidence of postoperative diabetes insipidus varies among different types of pituitary adenomas. Among different pathological types, the incidences of postoperative diabetes insipidus in ACTH adenomas, TSH adenomas and FSH/LH adenomas are 14.7%, 16.7% and 15.8% respectively. In addition, the difference in incidence between microadenomas and macroadenomas is mostly not statistically significant[6].

Postoperative diabetes insipidus after pituitary tumor surgery is the result of the combined action of multiple factors, involving patient-related, tumor-related and surgical-related factors. Adequate preoperative assessment of risk factors and delicate intraoperative operation are of great significance for the prevention of diabetes insipidus.

4.1 Patient-related Factors

Regarding age, current research findings are not yet unanimous; however, multiple lines of evidence support that younger age (e.g., <50 years) is a risk factor for postoperative diabetes insipidus (DI), including its progression to the permanent form[11-13]. The underlying mechanisms may involve a more active hypothalamic-pituitary axis and greater sensitivity to surgical injury in younger patients, coupled with a clinical tendency toward more aggressive resection strategies. Of course, some smaller-scale studies have reached different conclusions[31]. Concerning sex, most studies suggest no significant association with the occurrence of postoperative DI. Although isolated studies have indicated a higher risk in either females or males[32], these discrepancies are likely attributable to differences in study populations, diagnostic criteria, surgical eras, and techniques, and a consensus has not been established.

4.2 Tumor-related Factors

Tumor characteristics are key determinants influencing the occurrence of DI after pituitary adenoma surgery. Tumor size, volume, and invasiveness are core risk factors, primarily increasing the risk of injury to the hypothalamic-pituitary axis and surgical difficulty through direct mechanical

compression. Tumor consistency is also significant; firm tumors often adhere tightly to surrounding tissues, increasing the risk of traction injury to structures like the pituitary stalk during dissection[10, 13]. After tumor resection relieves the mass effect, the pituitary gland and adjacent structures undergo a process of repositioning and settling. This process can be divided into acute and chronic phases, with traction during the acute phase considered a critical event leading to pituitary stalk injury and subsequent DI[33]. Consequently, for patients with a preoperatively assessed high-riding pituitary gland, judicious Gelfoam packing within the tumor cavity to buffer traction forces during the acute repositioning phase may serve as a potential strategy to reduce postoperative DI risk.

Furthermore, pituitary apoplexy, as a special circumstance, can significantly elevate the risk of DI by directly damaging the posterior pituitary and disrupting the synthesis and release pathway of antidiuretic hormone[34, 35]. It is noteworthy that with the continuous advancement and widespread adoption of minimally invasive techniques like endoscopy, the actual impact of some previously reported risk factors (e.g., intraoperative cerebrospinal fluid leak) on postoperative DI may have changed[36, 37].

4.3 Surgical-related Factors

The occurrence of DI after pituitary adenoma surgery is closely associated with the surgical approach, intraoperative maneuvers, and extent of resection. Regarding the surgical approach, the transnasal transsphenoidal route (endoscopic and microscopic) is the mainstream technique, but its impact on DI incidence remains debated. Some studies report a higher incidence in endoscopic groups[38], while others suggest endoscopic techniques may reduce incidence in elderly patients[39], with surgeon experience and technical proficiency being key moderating factors. Intraoperative complications such as diaphragma sellae opening and cerebrospinal fluid (CSF) leak are significant risk factors. The former alters CSF dynamics and traction on hypothalamic-pituitary structures, while the latter exacerbates neural injury through abrupt intracranial pressure changes and subsequent packing-related compression, increasing DI risk by 3.6-fold and the risk of permanent DI by over 20-fold[13, 28]. Concerning the extent of resection, gross-total or near-total resection may increase the risk of injury to the pituitary stalk and surrounding tissues due to the expanded operative field, thereby elevating DI incidence[40]. However, other studies have found no significant association[38], which may be related to heterogeneity in tumor characteristics, protective strategies, and diagnostic criteria.

4.4 Other Factors

Beyond tumor characteristics and surgical maneuvers, the occurrence of postoperative DI is also associated with various other factors. Operative duration is an independent risk factor; surgery lasting more than three hours significantly increases the risk of mechanical injury to the pituitary stalk and posterior lobe, thereby raising DI incidence[11]. Preoperative visual impairment often indicates suprasellar tumor extension and optic chiasm compression, making intraoperative involvement of the hypothalamic-pituitary stalk structures more likely and increasing postoperative DI risk[41]. Imaging assessments show that the degree of third ventricular and hypothalamic distortion correlates positively with postoperative DI occurrence, and postoperative hemorrhage is also strongly associated with its development[42]. Additionally, certain pathologies are linked to DI, such as Rathke's cleft cysts, found in 3.7% to 22% of normal pituitaries[43, 44]. Resection of these cysts is associated with an increased overall and permanent incidence of DI, primarily because cyst rupture induces a local inflammatory response that can directly damage the neurohypophysis, leading to a high and often persistent rate of DI[44-47].

5. Prevention and Treatment Strategies and Research Progress of Postoperative Diabetes Insipidus

The prevention and management of postoperative diabetes insipidus (DI) adhere to the principle of "prevention first, early intervention," with the core objective of minimizing iatrogenic injury to the hypothalamic-pituitary neuroendocrine axis[14, 15]. The root cause of this complication lies in intraoperative damage to the pituitary stalk, posterior pituitary, and their vascular supply. Therefore, meticulous preoperative planning and precise intraoperative technique are paramount. The foremost principle is gentle manipulation, avoiding excessive traction and compression on the pituitary stalk and hypothalamic region. Specific intraoperative measures to mitigate risk include: 1) Identification and active preservation of the pituitary stalk; 2) Use of low-power electrocautery coupled with continuous irrigation for cooling to reduce thermal conduction injury; 3) Prioritizing compression for hemostasis over electrocautery when feasible; and 4) For soft or cystic tumors, employing slow suction or staged drainage to prevent rapid collapse of the tumor wall and subsequent traction injury[14, 15]. These techniques aim to preserve the anatomical integrity of the hypothalamic-pituitary axis. Studies indicate that the incidence of surgical complications is inversely correlated with the experience level of the medical center. As institutional and surgeon case volumes increase, the incidence of complications like postoperative DI decreases significantly[48]. This underscores that systematic training, standardized surgical protocols, and mentorship from senior surgeons are crucial external safeguards for enhancing overall prevention and enabling continuous quality improvement.

The therapeutic approach for established postoperative DI focuses on correcting polyuria to restore normal urine output and preventing electrolyte disturbances, with active correction required if imbalances are already present. Following continuous urine output monitoring confirming DI, management is tiered: For conscious patients with 24-hour urine output < 4000 mL, providing adequate oral fluid intake alongside electrolyte monitoring to maintain balance is often sufficient. No pharmacological intervention is typically needed, and the condition frequently resolves spontaneously within 3-5 days. For higher urine output (>4000 mL/24h) or prolonged duration, pharmacological intervention with oral desmopressin acetate tablets (e.g., DDAVP) is initiated to control urine volume. Desmopressin acts by increasing water reabsorption in the renal collecting ducts and distal tubules. At appropriate doses, it provides effective and safe treatment for all patients with central DI[16]. A common starting dose is 0.05 mg orally every 8 hours, titrated based on urine output response, with a maximum dose typically of 0.4 mg every 8 hours. If desmopressin tablets are ineffective, intramuscular pituitrin (5 units every 8 hours) can be used as an alternative. However, its use should be short-term due to the risk of antidiuretic excess. Strict monitoring of 24-hour urine output is mandatory to avoid oliguria or even renal failure. For patients who develop permanent DI, long-term desmopressin replacement therapy is indicated. The goal is to identify and maintain the minimal effective dose that sustains a normal quality of life[49].

6. Summary and Prospect

Diabetes insipidus (DI) is a common complication following pituitary adenoma surgery, with its pathogenesis involving injury at multiple levels of the hypothalamic-pituitary axis. This systematic review indicates that the overall incidence of this complication ranges from 2% to 54%, with permanent DI accounting for approximately 4.6% of cases[4-6]. The risk factors for its development encompass patient characteristics (e.g., age), tumor features (size, consistency, invasiveness, and apoplexy), and surgical details (diaphragma sellae opening, cerebrospinal fluid leak), among others[10, 13, 34, 35]. The pathophysiological core lies in surgical trauma leading to dysfunction in the synthesis, secretion, or action of antidiuretic hormone, thereby disrupting water and electrolyte

homeostasis. Current prevention and management strategies emphasize meticulous intraoperative technique and comprehensive perioperative care. Key technical measures, such as protecting the pituitary stalk and posterior pituitary vasculature, controlling electrocautery intensity, and standardizing tumor resection methods, are of significant importance for preventing this complication[14, 15]. Regarding treatment, mild cases can often be managed with hydration and electrolyte monitoring, while moderate to severe cases require the standardized use of medications like desmopressin, with vigilance for the risk of electrolyte imbalances[16].

This study has certain limitations. First, the existing literature primarily consists of single-center retrospective studies, and the level of evidence needs improvement. Second, diagnostic criteria and severity grading for DI are not yet standardized across studies, affecting the comparability of data. Furthermore, risk prediction models for postoperative DI specific to different pathological subtypes of adenomas remain underdeveloped, lacking a systematic review of the likelihood of DI occurrence stratified by pathology.

Future research could explore the following directions: First, establishing multicenter, prospective registries to integrate radiomics, surgical video data, and postoperative follow-up information for constructing accurate prediction models. Second, conducting research on individualized prevention and treatment strategies for patients with different risk stratifications, particularly optimizing perioperative management protocols for high-risk groups (e.g., invasive macroadenomas, Rathke's cleft cysts). Third, strengthening long-term follow-up studies on postoperative DI to clarify the risk factors for its progression to permanence and its impact on patients' quality of life. Through multidisciplinary collaboration and evidence-based research, it is anticipated that the prevention and management of postoperative DI in pituitary adenoma surgery can be further enhanced.

References

[1] Melmed, S., Pituitary Medicine From Discovery to Patient-Focused Outcomes. J Clin Endocrinol Metab, 2016. 101(3): p. 769-777.

[2] Molitch, M.E., Diagnosis and Treatment of Pituitary Adenomas: A Review. JAMA, 2017. 317(5): p. 516-524.

[3] Lucas, J.W., et al., Congress of Neurological Surgeons Systematic Review and Evidence-Based Guideline on Primary Management of Patients With Nonfunctioning Pituitary Adenomas. Neurosurgery, 2016. 79(4): p. E533-5.

[4] Fountas, A., et al., Central diabetes insipidus (vasopressin deficiency) after surgery for pituitary tumours: a systematic review and meta-analysis. Eur J Endocrinol, 2024. 191(1): p. S1-S13.

[5] Qari, F.A., E.A. AbuDaood, and T.A. Nasser, Diabetes insipidus following neurosurgery at a university hospital in Western Saudi Arabia. Saudi Med J, 2016. 37(2): p. 156-60.

[6] Burke, W.T., et al., Diabetes Insipidus After Endoscopic Transsphenoidal Surgery. Neurosurgery, 2020. 87(5): p. 949-955.

[7] Nielsen, S., et al., Aquaporins in the kidney: from molecules to medicine. Physiol Rev, 2002. 82(1): p. 205-244.

[8] Xin Peiyuan, et al., The Role and Mechanism of Arginine Vasopressin in the Central Nervous System. Journal of Neuroanatomy, 2017. 33(02): p. 225-228.

[9] Robertson, G.L., Antidiuretic hormone. Normal and disordered function. Endocrinol Metab Clin North Am, 2001. 30(3): p. 671-694, vii.

[10] Micko, A.S., et al., Invasion of the cavernous sinus space in pituitary adenomas: endoscopic verification and its correlation with an MRI-based classification. J Neurosurg, 2015. 122(4): p. 803-811.

[11] Zhang Zhongyuan, Analysis of Complications and Related Risk Factors of Transsphenoidal Endoscopic Pituitary Adenoma Resection. 2017, Zhejiang University.

[12] Ajlan, A.M., et al., Diabetes Insipidus following Endoscopic Transsphenoidal Surgery for Pituitary Adenoma. J Neurol Surg B Skull Base, 2018. 79(2): p. 117-122.

[13] Araujo-Castro, M., et al., Is it possible to predict the development of diabetes insipidus after pituitary surgery? Study of 241 endoscopic transsphenoidal pituitary surgeries. J Endocrinol Invest, 2021. 44(7): p. 1457-1464.

[14] Wang Y H, Qi S T. Relationship between diabetes insipidus and pituitary stalk injury[J]. Chinese Journal of Minimally Invasive Neurosurgery, 2005, 10(1): 35-38.

[15] Berker, M., et al., Complications of endoscopic surgery of the pituitary adenomas: analysis of 570 patients and review of the literature. Pituitary, 2012. 15(3): p. 288-300.

[16] Oiso, Y., et al., Clinical review: Treatment of neurohypophyseal diabetes insipidus. J Clin Endocrinol Metab, 2013. 98(10): p. 3958-3967.
[17] Daly, A.F. and A. Beckers, The Epidemiology of Pituitary Adenomas. Endocrinol Metab Clin North Am, 2020. 49(3): p. 347-355.
[18] Schreckinger, M., N. Szerlip, and S. Mittal, Diabetes insipidus following resection of pituitary tumors. Clin Neurol Neurosurg, 2013. 115(2): p. 121-6.
[19] Asa, S.L., et al., Overview of the 2022 WHO Classification of Pituitary Tumors. Endocr Pathol, 2022. 33(1): p. 6-26.
[20] Tsukamoto, T. and Y. Miki, Imaging of pituitary tumors: an update with the 5th WHO Classifications-part 1. Pituitary neuroendocrine tumor (PitNET)/pituitary adenoma. Jpn J Radiol, 2023. 41(8): p. 789-806.
[21] Ho, K.K., The year in pituitary 2014. J Clin Endocrinol Metab, 2014. 99(12): p. 4449-4454.
[22] Coleman, D.M., et al., Intraoperative Diagnosis and Management of Arginine Vasopressin Disorder During Pituitary Tumor Resection via Transsphenoidal Endoscopic Navigation. Cureus, 2025. 17(4): p. e82096.
[23] De Marco, R., et al., Exploring factors behind Arginine-Vasopressine deficiency in endoscopic endonasal surgery for PitNET: a single-center analysis of 349 patients. Neurosurg Rev, 2025. 48(1): p. 449.
[24] Christ-Crain, M., et al., Diabetes insipidus. Nat Rev Dis Primers, 2019. 5(1): p. 54.
[25] Li P. Clinical analysis of 389 cases of diabetes insipidus[D]. Zhengzhou: Zhengzhou University, 2018.
[26] Christ-Crain, M., B. Winzeler, and J. Refardt, Diagnosis and management of diabetes insipidus for the internist: an update. J Intern Med, 2021. 290(1): p. 73-87.
[27] Winzeler, B., et al., Postoperative Copeptin Concentration Predicts Diabetes Insipidus After Pituitary Surgery. J Clin Endocrinol Metab, 2015. 100(6): p. 2275-2282.
[28] Cai S H. Analysis of related factors of diabetes insipidus after endoscopic transsphenoidal resection of pituitary neuroendocrine tumors[D]. Nanchang: Nanchang University, 2024.
[29] Patti, G., et al., Familial neurohypophyseal diabetes insipidus in 13 kindreds and 2 novel mutations in the vasopressin gene. Eur J Endocrinol, 2019. 181(3): p. 233-244.
[30] Mu Y M. Advances in the diagnosis and treatment of pituitary tumors[J]. Medical Journal of Chinese People's Liberation Army, 2017. 42(07): p. 576-582.
[31] Zhang G M. Analysis of curative effect and related factors of postoperative complications of pituitary tumor resection under neuroendoscopy[D]. Nanjing: Nanjing Medical University, 2023.
[32] Yasuda, M.E., et al., Risk Factors Related to Transient Diabetes Insipidus Development Following Transsphenoidal Pituitary Adenoma Resection: A Multicentric Study. World Neurosurg, 2023. 175: p. e636-e643.
[33] Lin, K., et al., Change in cephalocaudal tumor cavity diameter after transsphenoidal surgery is a predictor of diabetes insipidus in pituitary adenoma. Eur J Med Res, 2022. 27(1): p. 72.
[34] Zhao Y B. Analysis of influencing factors and prevention strategies of diabetes insipidus after transsphenoidal surgery for pituitary adenoma[D]. Yinchuan: Ningxia Medical University, 2018.
[35] Xu H C, et al. Clinical characteristics and therapeutic efficacy of patients with pituitary apoplexy: a single-center retrospective study[J]. Journal of Chongqing Medical University, 2023. 48(09): p. 1084-1088.
[36] Mortini, P., et al., Pituitary Surgery. Presse Med, 2021. 50(4): p. 104079.
[37] Kinoshita, Y., et al., Predictive factors of postoperative diabetes insipidus in 333 patients undergoing transsphenoidal surgery for non-functioning pituitary adenoma. Pituitary, 2022. 25(1): p. 100-107.
[38] Li L, et al. Analysis of related factors of diabetes insipidus after pituitary tumor surgery[J]. Journal of Qiqihar Medical University, 2019. 40(23): p. 2932-2935.
[39] Ding Q, Shi G X, Yang K H. Effects of different minimally invasive transsphenoidal approaches on efficacy, perioperative indicators and postoperative complications in elderly patients with pituitary tumors[J]. Chinese Journal of Gerontology, 2023. 43(14): p. 3381-3384.
[40] Yu Y H, et al. Analysis of microsurgical treatment via transsphenoidal approach for 1194 cases of pituitary adenomas[J]. Chinese Journal of Neurosurgical Disease Research, 2018. 17(04): p. 366-368.
[41] Dong X, Yang R, Yan HM, et al. Research progress on risk factors of diabetes insipidus after pituitary tumor surgery[J]. China Medical Herald, 2025, 22(29): 157-161.
[42] Wang, S., et al., Clinical Predictors of Diabetes Insipidus after Transcranial Surgery for Pituitary Adenoma. World Neurosurg, 2017. 101: p. 1-10.
[43] Osborn, A.G. and M.T. Preece, Intracranial cysts: radiologic-pathologic correlation and imaging approach. Radiology, 2006. 239(3): p. 650-64.
[44] Han, S.J., et al., Rathke's cleft cysts: review of natural history and surgical outcomes. J Neurooncol, 2014. 117(2): p. 197-203.
[45] Yuyama, R., et al., [Secondary pan-hypophysitis caused by rupture of Rathke's cleft cyst: case report]. No Shinkei Geka, 2002. 30(1): p. 95-99.
[46] Aho, C.J., et al., Surgical outcomes in 118 patients with Rathke cleft cysts. J Neurosurg, 2005. 102(2): p. 189-193.

[47] Nishikawa, T., et al., Hypophysitis caused by Rathke's cleft cyst. Case report. Neurol Med Chir (Tokyo), 2007. 47(3): p. 136-139.
[48] Barker, F.G., 2nd, A. Klibanski, and B. Swearingen, Transsphenoidal surgery for pituitary tumors in the United States, 1996-2000: mortality, morbidity, and the effects of hospital and surgeon volume. J Clin Endocrinol Metab, 2003. 88(10): p. 4709-4719.
[49] Lv C X. Analysis of influencing factors and discussion on prevention and treatment strategies of diabetes insipidus after transsphenoidal pituitary adenoma resection[D]. Dalian: Dalian Medical University, 2020.