International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

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ARTICLE TITLE	A COMPREHENSIVE REVIEW OF RATHKE'S CLEFT CYSTS: DIAGNOSTIC CHALLENGES AND MANAGEMENT TRENDS
DOI	https://doi.org/10.31435/ijitss.3(47).2025.3616
RECEIVED	11 July 2025
ACCEPTED	31 August 2025
PUBLISHED	17 September 2025
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A COMPREHENSIVE REVIEW OF RATHKE'S CLEFT CYSTS: DIAGNOSTIC CHALLENGES AND MANAGEMENT TRENDS

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ABSTRACT

Introduction and purpose: Rathke's cleft cysts (RCCs) are benign, epithelium-lined lesions located in the sellar or suprasellar region, originating from remnants of Rathke's pouch. While often asymptomatic and discovered incidentally, RCCs may lead to significant clinical symptoms due to compression of adjacent structures, including the pituitary gland and optic chiasm. The aim of this review is to provide a comprehensive overview of RCCs and emphasize the importance of accurate diagnosis and individualized management, especially in symptomatic cases.
Material and methods: The review is based on scientific literature published between 2005 and 2025, retrieved from PubMed, Scopus and Google Scholar using the following keywords: "Rathke's cleft cyst,", "Rathke's cyst diagnosis," "Rathke's cyst treatment," "pituitary cyst surgery," and "Rathke's cyst recurrence".
Results: Analysis of the literature indicates that although most RCCs remain asymptomatic, a significant subset causes symptoms such as headaches, visual disturbances, and endocrine dysfunction. MRI remains the most effective diagnostic tool, while treatment typically involves transsphenoidal surgical drainage. Recurrence rates vary depending on the surgical technique and inflammatory changes within the cyst wall. Emerging approaches, such as steroid-eluting stents, show promise in reducing recurrence, though further research is needed.
Conclusions: RCCs, though generally benign, may result in substantial morbidity in symptomatic patients. Accurate diagnosis, appropriate imaging, and tailored treatment are essential for optimal outcomes. Long-term follow-up is recommended due to the potential for recurrence, and novel interventions offer promising avenues for future management strategies.

KEYWORDS

Policy Development, School Management, Indiscipline, Leadership, Challenges and Strategies

CITATION

Martyna Musiorska, Julia Mazurek, Joanna Kośka, Gabriela Łocik, Katarzyna Moliszewska, Paweł Kukiełka, Julia Załęcka, Alicja Nowik, Kacper Dywan, Michał Błaszkiewicz. (2025) A Comprehensive Review of Rathke's Cleft Cysts: Diagnostic Challenges and Management Trends. *International Journal of Innovative Technologies in Social Science*. 3(47). doi: 10.31435/ijitss.3(47).2025.3616

Introduction

Rathke's cleft cysts (RCCs) are benign, epithelium-lined, non-neoplastic lesions that arise within the sellar or suprasellar region of the brain. Although frequently discovered incidentally during imaging or postmortem examinations, a subset of RCCs can cause clinically significant symptoms due to compression of adjacent neuroendocrine structures. Despite being relatively common findings, particularly in autopsy series, the underlying mechanisms of their development, growth, and symptomatic transformation remain poorly understood. Recent advances in neuroimaging, molecular biology, and endocrinology have deepened our understanding of their embryologic origin, clinical course, and therapeutic management, yet many questions persist.

This paper provides a comprehensive overview of the current knowledge regarding the pathogenesis, epidemiology, clinical presentation, diagnostic approach, and management of RCCs. Special attention is given to the factors influencing recurrence following surgical intervention, as well as emerging strategies aimed at reducing the risk of postoperative complications and long-term disease relapse(Kanter et al., 2006; Zhong et al., 2012).

Methodology

A focused literature review was conducted to synthesize current knowledge on Rathke's cleft cysts (RCCs), including their pathogenesis, clinical presentation, diagnostic imaging, treatment options, and recurrence. Sources were identified using databases such as PubMed, Scopus and Google Scholar with search terms including:"Rathke's cleft cyst, ""RCC diagnosis, ""sellar cyst treatment, ""pituitary cyst surgery, " and"Rathke's cyst recurrence." Only articles published in English from 2005 to 2025 were considered. Additional literature was selected through manual review of reference lists from relevant publications. Each study was assessed for clinical relevance based on title, abstract, and full-text analysis. Language refinement and formatting were assisted using AI-supported grammar correction tools.

Results

Pathogenesis of Rathke's Cleft Cyst

The pathogenesis of RCCs remains incompletely understood. Various hypotheses have been proposed to explain their development, suggesting that RCCs may originate from neuroepithelial or endodermal tissue, or result from metaplastic transformation of anterior pituitary cells(Kanter et al., 2006) Experimental studies also indicate that mutations or defects in the *Isl1* transcription factor gene may contribute to RCC formation.

The most widely accepted theory posits that RCCs originate from the Rathke's pouch (RP) a crucial embryonic structure involved in pituitary gland development. RP gives rise to the anterior lobe, pars intermedia, and pars tuberalis of the pituitary. RCCs are believed to develop when this pouch fails to close properly during early fetal life, leading to its abnormal persistence(Oyesiku & Post, 2011).

RCCs are benign, non-neoplastic lesions and represent the most common incidental finding in the sellar and suprasellar regions. They are typically unilocular, lined with a single layer of epithelial cells, and filled with mucoid material(Dadej et al., 2021; Raghunath et al., 2010).

Epidemiology

Symptomatic RCCs are relatively uncommon, which makes it difficult to determine their precise prevalence. In an analysis of 1, 000 unselected pituitary glands from routine postmortem examinations, subclinical RCCs were found in 113 cases (11.3%)(Larkin et al., 2014). Another study indicates that, in routine autopsies, they are identified in 13–33% of cases (Trifanescu et al., 2012).

RCCs have been documented in individuals of all ages, but they are more frequently observed in adults, with the highest incidence occurring between the ages of 30 and 50 years, and average ages ranging from 34 to 44 years. Other sources report a peak frequency between the 4th and 6th decades of life (Larkin et al., 2014; Trifanescu et al., 2012). Cases in children are rare(Larkin et al., 2014).

Published reports indicate a clear female predominance in the occurrence of RCCs, with a reported female-to-male ratio as high as 3:1, although some sources cite a ratio of 2:1. This may be partly attributed to earlier detection in women, possibly due to menstrual irregularities prompting medical attention (Kanter et al., 2006; Trifanescu et al., 2012).

According to another source, female sex hormones may have a direct impact on the development of RCC. Estrogen receptors have been identified at both the mRNA and protein levels in the anterior pituitary, which originates from RP, his suggests that estrogen may contribute to the growth and pathogenesis of RCC. Research has shown that 69% of women developed these cysts before menopause, while 31% did so after menopause, indicating a potential role of female sex hormones in their formation (Hacioglu et al., 2025).

Clinical Presentation

Although RCCs are generally benign, they have the potential to cause a range of burdensome clinical manifestations. Small cysts are typically asymptomatic and are often discovered incidentally. However, when they grow large enough to exert pressure on adjacent structures—such as the pituitary gland, pituitary stalk, optic chiasm, or hypothalamus—they may lead to chronic symptoms, primarily including headaches, visual disturbances, and endocrine dysfunction (Kanter et al., 2006; Trifanescu et al., 2012).

A retrospective analysis of medical records from 102 patients diagnosed with RCC revealed that headaches were the most prevalent symptom, reported in 42% of cases. Another review reported headache prevalence ranging from 44% to 81% among symptomatic patients(Han et al., 2014). The second most common symptom was vertigo or dizziness (20%), followed by visual impairment (15%)(Dadej et al., 2021).

Among these, headache is perhaps the most challenging to assess. While endocrine dysfunction and visual disturbances are well-established complications of sellar lesions—objectively evaluated via hormone panels or visual field tests—headaches are subjective, often intermittent, and common even among healthy individuals. They are generally not correlated with cyst size, location, or the presence of pituitary dysfunction. Instead, they are more commonly associated with hyperintense or isointense signals on T1-weighted MRI, mucous content within the cyst, or chronic inflammation of the cyst wall (Altuwaijri et al., 2018).

Several authors have also suggested that headaches may result from cyst wall infarction, hemorrhage, or the leakage of inflammatory contents (Altuwaijri et al., 2018; Han et al., 2014). As inflammation can potentially damage the pituitary gland, episodic frontal headaches have been proposed as a clinical marker of intermittent inflammatory activity (Trifanescu et al., 2012). These headaches typically occur in the frontal region but may radiate to the frontotemporal or occipital areas, or even affect the entire head. The pain may

manifest as a dull ache or throbbing sensation, often persisting chronically, though in some patients it may be sudden in onset, episodic, or progressively worsening. For some individuals, headache may be the sole symptom; for others, it coexists with additional complaints (Hacioglu et al., 2025).

Visual disturbances are present in approximately one-third of patients with RCCs, most commonly presenting as reduced visual acuity, quadrantanopia, or hemianopia. In rarer cases, cranial nerve dysfunction and diplopia may occur (Kanter et al., 2006). One source describes a case of chiasmal syndrome caused by an RCC; unfortunately, despite prompt surgical intervention, the patient's visual function could not be restored (Lešták et al., 2025).

Among endocrine dysfunctions, elevated prolactin levels are the most frequently observed, followed by pituitary insufficiency (Zada, 2011). The type and severity of endocrinological symptoms may vary depending on the patient's age and sex. In men, hypogonadism may result in fatigue and decreased libido. Premenopausal women often report menstrual irregularities and galactorrhea, while postmenopausal women more commonly present with panhypopituitarism, accompanied by fatigue and cognitive impairment (Han et al., 2014).

Diabetes insipidus (DI) is identified as an initial symptom in approximately 7% to 20% of RCC cases, though other reports estimate this range to be between 2.3% and 37% (Han et al., 2014; Zada, 2011).

Less common complications include aseptic meningitis, sphenoid sinusitis, and precocious puberty, though such cases are rare in the literature (Dadej et al., 2021).

Diagnostics

The development of CT and MRI has led to the more frequent detection of symptomatic RCCs, although they are often misdiagnosed as other intrasellar lesions due to similarities in clinical and radiological characteristics (Zhong et al., 2012). RCCs are typically identified on MRI as ovoid lesions located within the sella, occasionally extending into the suprasellar cistern. They are well-demarcated from surrounding structures and usually show no significant mass effect or invasion. On T2-weighted MRI sequences, RCCs generally appear hyperintense. On T1-weighted images, their signal intensity may vary: hyperintensity often indicates mucinous cyst fluid with high protein content and inflammation, while hypointensity may reflect clear fluid with low protein content. One study suggests that RCCs with high or isointense signals on T1-weighted images are more commonly associated with headaches and pituitary dysfunction than those with low signal intensity, emphasizing the diagnostic value of MRI in assessing inflammation (Hasebe et al., 2025).

Intracystic nodules are seen in over 75% of cases. They typically appear hyperintense on T1-weighted images and hypointense on T2-weighted ones. Their presence is considered highly suggestive of RCC and may even support diagnosis independently of clinical symptoms (Hacioglu et al., 2025). These nodules often appear intraoperatively as yellow, waxy solid masses and are histologically confirmed to contain mucin clumps. While most float freely within the cyst and shift with patient movement, some may adhere to the cyst wall (Hacioglu et al., 2025).

The main differential diagnoses for RCC include pituitary adenoma, pituitary cyst, and intrasellar craniopharyngioma. RCCs are characteristically located in the midline, without pituitary stalk deviation, and lie between the anterior and posterior lobes of the pituitary on sagittal MRI scans (Han et al., 2014; Raghunath et al., 2010).

A significant diagnostic challenge is distinguishing between RCC and cystic craniopharyngioma. Contrast-enhanced 3D-FLAIR MRI has been noted as particularly useful due to its ability to show differences in cyst wall enhancement(Azuma et al., 2020). One study suggests that when there is uncertainty between RCC and papillary craniopharyngioma on imaging, a molecular BRAF test may offer diagnostic value. Nearly all papillary craniopharyngiomas exhibit a clonal BRAF V600E mutation, whereas RCCs do not. Although BRAF testing is not routinely performed for RCCs and depends on institutional resources, it represents a promising avenue for future research (Candy et al., 2025).

Treatment

Small, usually slow-growing and asymptomatic RCCs typically do not require surgical intervention and can be safely monitored over extended periods (Altuwaijri et al., 2018; Trifanescu et al., 2012). On average, asymptomatic RCCs grow at a rate of 5.3% during follow-up (Hacioglu et al., 2025).

In contrast, symptomatic RCCs often require surgical resection, usually performed using a microscopic or endoscopic endonasal transsphenoidal approach. Most cases can be effectively managed via transnasal exposure of the sella, with minimal perioperative morbidity (Altuwaijri et al., 2018; Kasperbauer et al., 2002).

Even symptomatic RCCs should not undergo immediate surgery, as spontaneous regression or resolution may occur, as documented in certain case reports(Lee & Park, 2019). Spontaneous regression of RCCs has been observed in 3.2% to 38% of patients during follow-up, with some experiencing resolution of headaches without the need for surgical intervention. In certain instances, significant cyst shrinkage has been noted following glucocorticoid therapy, indicating that suppression of inflammation may play a role in reducing cyst size (Hacioglu et al., 2025).

The most commonly used surgical methods include fenestration and drainage via a transnasal or sublabial transsphenoidal approach, which are typically effective in relieving pressure-related symptoms (Trifanescu et al., 2012). Surgical drainage is highly effective, with over 90% of RCCs resolving completely. More than 80% of patients have also experienced significant relief from headaches and improvements in visual function (Zada, 2011)

In 2005, Aho et al. published the largest study to date on adult RCC treatment, involving 118 patients. They reported a 97% initial gross-total resection rate and an 18% recurrence rate over a five-year period (Aho et al., 2005).

Simple drainage is associated with a low rate of endocrine complications, it carries a higher risk of recurrence. To minimize recurrence, more aggressive cyst wall resection is recommended, although it increases the risk of pituitary dysfunction. In cases where the cyst is entirely suprasellar, a transcranial approach is advised. One notable advantage of craniotomy over the transsphenoidal route is the absence of pituitary incision, which helps preserve pituitary function (Dolovac et al., 2025; Hacioglu et al., 2025).

The least understood form of RCC treatment is radiotherapy or radiosurgery, and these methods remain limited in current treatment protocols. There are two types of radiotherapy: endocavitary irradiation and stereotactic radiosurgery. In rare cases, cytostatic agents or radioactive substances may be injected directly into the cyst (Kanter et al., 2006).

Surgical Procedure Complications

The most commonly reported complications of surgical procedures include visual disturbances, affecting 35%–50% of patients undergoing surgical intervention. These may involve both visual field deficits and decreased visual acuity (Zada, 2011).

A significant postoperative concern following RCC resection is iatrogenic pituitary injury, leading to hypopituitarism, most notably DI.

Less frequently reported complications include postoperative cerebrospinal fluid rhinorrhea, occurring in fewer than 3% of patients after transsphenoidal surgery, as well as postoperative meningitis (Kanter et al., 2006).

Recurrences and Approaches to Their Prevention

The likelihood of recurrence varies and is influenced by several factors, including the chosen surgical method, operative technique, duration of follow-up, and imaging assessments. Since recurrences may occur years after the initial surgery, regular imaging follow-up is recommended for at least ten years. Reported recurrence rates in the literature range from 0% to over 30% (Kanter et al., 2006; Kasperbauer et al., 2002).

While some studies suggest that cyst size may influence the risk of recurrence, others have found no such correlation (Ference et al., 2019; Kanter et al., 2006).

Inflammation has also been implicated in recurrence, as it can stimulate cyst secretion and squamous metaplasia. MRI enhancement, which correlates with these inflammatory changes, has been associated with a higher risk of recurrence. These findings may help predict recurrence risk prior to surgical intervention (Ference et al., 2019; Zhong et al., 2012).

One novel therapeutic approach for managing recurrent RCC involves the implantation of a bioabsorbable, steroid-eluting stent following marsupialization. This technique aims to prevent scar tissue from obstructing the drainage pathway, thereby reducing the likelihood of further recurrence. However, more research is needed to determine the effectiveness of this method, particularly in patients with multiple recurrences (Ference et al., 2019).

Discussion

This review highlights current evidence on the epidemiology, pathogenesis, diagnosis, and management of Rathke's cleft cysts (RCCs). Although most RCCs are benign and asymptomatic, a subset becomes clinically significant through mass effect or endocrine dysfunction. The embryologic origin from Rathke's pouch is well established, but factors driving growth, symptom onset, and recurrence remain unclear. Hormonal influences—particularly estrogen—and inflammatory changes within the cyst wall have been proposed as contributors. Diagnosis relies primarily on MRI, with features such as midline location and intracystic nodules aiding differentiation from other sellar lesions. However, overlap with craniopharyngiomas and cystic adenomas can complicate diagnosis, and advanced techniques such as contrast-enhanced 3D-FLAIR or BRAF mutation testing may improve specificity. Management depends on symptoms. Asymptomatic lesions can be safely monitored due to slow growth and occasional spontaneous regression. Symptomatic cases are typically treated via endonasal transsphenoidal surgery, with simple drainage offering low morbidity but higher recurrence, and more aggressive wall resection reducing recurrence at the expense of greater pituitary risk. Novel options, such as steroid-eluting stents, are under investigation. Recurrence rates vary widely (0–30%) and can occur many years postoperatively, necessitating long-term imaging follow-up. MRI evidence of cyst wall enhancement may predict recurrence risk by reflecting inflammation. Overall, RCCs require individualized management balancing symptom relief, recurrence prevention, and preservation of pituitary function. Further research into their molecular biology and inflammatory pathways will be key to refining treatment strategies.

Conclusions

RCCs are relatively common but often asymptomatic lesions that can, in certain cases, cause significant clinical symptoms such as headaches, visual disturbances, and endocrine dysfunction. Despite advances in imaging and surgical techniques, the precise mechanisms behind their formation and progression remain unclear. Regular follow-up through imaging is essential, as recurrences can occur years after initial surgery. Current treatment strategies, including surgical resection and emerging techniques like the use of steroid-eluting stents, aim to alleviate symptoms and reduce recurrence rates. However, further research is needed to better understand the pathophysiology of RCCs and to refine therapeutic approaches. Given their benign nature, most RCCs can be managed conservatively, but appropriate intervention is crucial in symptomatic cases to improve patient outcomes and prevent complications.

Conflicts of Interest: No conflicts of interest to declare.

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