ScientificScholar®
Knowledge is power
Publisher of Scientific Journals

www.surgicalneurologyint.com

Surgical Neurology International

Editor-in-Chief: Nancy E. Epstein, MD, Clinical Professor of Neurological Surgery, School of Medicine, State U. of NY at Stony Brook.

SNI: General Neurosurgery

Editor
Eric Nussbaum, MD
National Brain Aneurysm and Tumor Center, Twin Cities, MN, USA

SNI
Open Access

Original Article

Rathke's cleft cysts: A single-center case series

Guive Sharifi[1], Arsalan Amin[1], Mahmoud Lotfinia[2], Mohammad Hallajnejad[1], Zahra Davoudi[3], Nader Akbari Dilmaghani[1], Omidvar Rezaei Mirghaed[1]

[1]Skull Base Research Center, Loghman Hakim Hospital, Shahid Beheshti University of Medical Science, Tehran, Iran, [2]Department of Neurosurgery, Medical Center Saarbruecken, Saarland, Germany, [3]Department of Endocrinology, Loghman Hakim Hospital, Shahid Beheshti University of Medical Science, Tehran, Iran.

E-mail: Guive Sharifi - gsharifi@gmail.com; Arsalan Amin - arsalan_amin25@yahoo.com; *Mahmoud Lotfinia - mdla617@yahoo.com; Mohammad Hallajnejad - hallajnejad@gmail.com; Zahra Davoudi - davoudi5842@yahoo.com; Nader Akbari Dilmaghani - nadakbari@sbmu.ac.ir; Omidvar Rezaei Mirghaed - info@drrezaee.ir

***Corresponding author:**
Mahmoud Lotfinia,
Department of Neurosurgery,
Medical Center Saarbruecken,
Saarland, Germany.

mdla617@yahoo.com

Received : 01 November 2021
Accepted : 02 August 2022
Published : 19 August 2022

DOI
10.25259/SNI_1096_2021

Quick Response Code:

ABSTRACT

Background: Rathke's cleft cysts (RCCs) are common benign sellar or suprasellar lesions. The aim of this study is to report our experience on the management of 27 RCC cases.

Methods: We retrospectively analyzed a series of 27 patients with symptomatic RCC who were referred to our department between January 2016 and January 2020. Data regarding patients' demographics, clinical evaluations, laboratory and neuroimaging findings, pathologic records, surgical treatment, and complications were extracted from our electronic database. All patients underwent RCC removal through a direct endoscopic endonasal transsphenoidal (EETS) approach, except for two cases.

Results: Data of 27 patients (3 men and 24 women; mean age: 38 years) with symptomatic RCC were reviewed. The most common presenting symptom was headache, occurring in 20 (74.0%) patients. In 16 (59.2%) cases, the tumor was primarily located in the sella turcica. Nine (33.3%) cases exhibited a secondary suprasellar extension.

Conclusion: Our experience with RCC patients showed that EETS is a safe method of treatment, with minimal recurrence.

Keywords: Neuroendoscopy, Outcome assessment, Pituitary gland, Rathke's cleft cyst

INTRODUCTION

Rathke's cleft cysts (RCCs) are relatively common, benign, nonneoplastic, and intra- and suprasellar lesions originating from the remnant of Rathke's pouch lesions composed typically of a thin cyst wall enclosing a mucous, gelatinous, or caseous liquid core.[4,16,23] The majority of RCCs are small and asymptomatic with an incidence of up to 22% on routine autopsies,[41] a few may grow large enough to cause significant pituitary dysfunction including temporary and permanent diabetes insipidus (DI), visual field deficit, and headache, particularly frontal episodic headache.[1,3,32,46]

RCCs are included in the differential diagnosis with other cystic lesions in such regions, such as craniopharyngiomas, arachnoid cysts, epidermoid cysts, and cystic pituitary adenomas and typically are diagnosed based on the shape, signal intensity, and enhancement characteristics of the lesions on magnetic resonance imaging (MRI).[5] A preoperative delineation between this

several entities is of great importance due to the different treatment approaches.

Several radiological features of RCCs have been described in the literature, but there is a general consensus about the limited sensitivity of each of this imaging findings.[5,7,14,33,34] The aim of the present case series is to add to the current knowledge by sharing our experience on the management of 27 RCC cases.

MATERIALS AND METHODS

Patients

In this study, we retrospectively evaluated records of patients (n = 27) with symptomatic RCC, who were referred to our department between January 2016 and January 2020 and met our inclusion criteria, that is, surgical and histological verification of diagnosis of RCC. We retrieved and reviewed the data regarding preoperative and postoperative clinical manifestations, neurological examination, visual acuity and field, laboratory tests, neuroimaging findings, pathologic records, intervention plans, and complications. The review board and ethics committee of Shahid Beheshti University of Medical Sciences approved the study design. All research was performed in accordance with relevant guidelines and regulations. We contacted the patients and obtained informed consent for the anonymous use of their data in this study. Informed consent was obtained from parent and/or legal guardian of all minor subjects (age under 18).

MRI data acquisition

All MRI studies were performed with a superconducting magnet 1.5-tesla scanner. Before gadolinium injection, T1-weighted SE and T2-weighted turbo SE images, followed by coronal dynamic acquisition (T1-weighted turbo SE), were obtained in the coronal plane using the following protocol: TR/TE, 400/20 ms; 288 • 192 matrix; 2 excitations; 18 • 18 cm field of view; 3 mm in thickness with 0.3-mm intersection gap. Beginning simultaneously with gadolinium injection, coronal and sagittal T1-weighted SE images were obtained 2 min after the injection. The radiologists and surgeons independently reviewed all imaging studies. Unenhanced and enhanced MRI was available for all patients and their reports were retrieved from the databank of the imaging center. MRI features, including location, size, shape, and signal characteristics of the lesion were reviewed. Cyst location and diameter were assessed on the sagittal MRI. We suspected RCC in patients with a midline, homogeneous fluid lesion, with little or no enhancement after GAD injection.

Diagnosis confirmation

The diagnosis was confirmed in each case based on the histological criteria for RCC, that is, dense eosinophilic amorphous mucin containing small strips of simple cuboidal or pseudostratified columnar, ciliated epithelial cyst wall lining. The preoperative and postoperative hormone profile of the patients (prolactin, thyroid function tests, and gonadotropins), as well as the initial clinical manifestations and the effect of surgery on them, were reviewed.

Surgical technique

RCC removal performed through a standard Endoscopic endonasal transsphenoidal (EETS) approach. In this condition and after dural opening, the cyst wall was incised and cyst content was removed by suction if possible. After decompression, the cyst wall was removed as much as it was safe and a gross-total resection was attempted only when it was deemed safe and feasible. Occasionally, the removal involved a direct approach through the anterior-inferior pituitary gland through a low midline vertical glandular incision (i.e., transpituitary approach). At the completion of the cyst removal, the cavity was carefully inspected for residual cyst contents and cyst lining. An angled endoscope was used for assisted visualization in all endoscopic cases. For patients who showed suprasellar extension, we used an extended approach by partial drilling of the pars planum of the sphenoid bone. By this extended transplanum approach, removal of the suprasellar component of the lesion was generally feasible. Cerebrospinal fluid (CSF) leak identified after cyst removal was repaired with fat tissue and collagen sponge. In our experience, there was no need for using fascia lata or other material for preventing CSF leak. For patients without an intraoperative CSF leak, only a collagen sponge was placed.

RESULTS

Twenty-seven patients (three men and 24 women; age range: 9–65 years, mean age: 38 years) with symptomatic RCC were admitted to our department between January 2016 and January 2020. The average follow-up time was 18 months (range: 3–36 months).

Clinical presentation

The most common presenting symptom on hospital admission was headache in 20 (74.0%) patients. Visual field defects occurred in 8 (29.6%) patients. Nine patients had endocrine disorders, including hyperprolactinemia (five patients), menstrual irregularity (five patients), DI (two patients), panhypopituitarism (two patients), galactorrhea (one patient), hypocortisolemia (one patient), and libido changes (one patient). One of the patients was diagnosed incidentally following a diagnostic workup for another condition and after a comprehensive explanation about her situation, she decided to choose surgical treatment. Formal preoperative visual field

perimetry testing demonstrated that 8 (29.6%) of the patients had visual field deficits. A summary of the clinical findings is presented in Table 1.

Imaging

Typical appearance of symptomatic RCC is said to be most often iso- to hyperintense on T1-weighted image (WI) and hyperintense on T2WI, and isointense on CT scans. However, we observed various types of intensity. In six cases, the lesion was isointense on T2, and in one case, the tumor was hypointense on T2-weighted MRI. Enhancement of the cyst wall was not rare and was observed in 7 (25.9%) cases, whereas complete enhancement of the lesion occurred in 4 (14.8%) cases.

In 16 (59.2%) cases, the tumor was primarily located in the sella turcica, whereas in 2 (7.4%) cases, it was primarily suprasellar. Nine (33.3%) cases had a secondary suprasellar extension. One case had a very huge tumor at the time of diagnosis and showed an unusual extension to the parasellar region and the middle fossa [Figure 1]. Table 2 presents the summary of the MRI characteristics of the patients.

Surgical treatment

RCC removal was performed through a standard EETS approach except for two cases [Figure 2]. Both cases showed a huge suprasellar extension, so they were operated by craniotomy through a pterional approach. Precise preoperative attention was drawn to the situation of the pituitary gland nearby the RCC on imaging. In most cases, the cyst was behind the gland.

One patient refused surgical treatment initially but returned after 2 months with exacerbation of his condition. He showed rapid growth of the lesion and presented with acute vision loss and visual field disturbance [Figures 2 and 3].

Pathological analysis

The cyst walls and removed contents from all 27 patients were sent for analysis. Of the latter, 23 (85.1%) were identified as cuboidal or columnar epithelium. Four (14.8%) patients showed squamous metaplasia on histologic examination, among them two showed enhanced signal, while two were hypointense on T2-weighted MRI.

Surgical complications outcome

There was no major complication following surgery, except one patient who presented with acute visual deterioration 3 days after surgery. This event was due to chiasmal herniation into the sella, which resolved completely after chiasmopexy surgery. Transient DI happened in three cases which prolonged the duration of hospitalization to control hypernatremia; however, no permanent DI occurred following the surgery. Although we performed an extended endoscopic transplanum approach in ten cases to remove the suprasellar component of the lesion, we experienced no CSF leak following surgery.

Headaches resolved in 19 out of 20 patients who had it (95% recovery). Only one patient continued to have an intermittent low-intensity headache and was later treated medically. Seven (87.5%) out of eight patients with preoperative visual loss experienced an improvement in their vision following surgery. Six patients had complete improvement and one case had partial improvement. The preoperative visual acuity defect in one patient remained unchanged. Of those patients presenting with endocrinopathy, 55% had improvement of the anterior pituitary axis; hyperprolactinemia resolved in all patients; normalization of menstruation occurred in 3 (out of 5) women with oligomenorrhea. In the two patients presenting with DI, two with panhypopituitarism, and one with hypocortisolemia, persistent postoperative hormone replacement was commenced. Table 1 presents a summary of the surgical outcomes of the patients.

Table 1: Summary of clinical findings and surgical outcomes of our patients.

Findings*	Preoperative (*n*=27)	Postoperative improvement	Postoperative new deficit
Headache	20 (74.0)	19 (95.0)	0
Visual deficit	8 (29.6)	6 (75.0)	1
Visual field	8 (29.6)	8 (100)	1
Visual acuity	2 (7.4)	1 (50.0)	1
Endocrinopathy	9 (33.3)	5 (55.5)	3
Menstrual irregularity	5 (18.5)	3 (60.0)	0
Galactorrhea	1 (3.7)	1 (100)	0
Diabetes insipidus	2 (7.4)	0 (0)	3 (transient)
Libido change	1 (3.7)	1 (100)	0
Hyperprolactinemia	5 (18.5)	5 (100)	0
Panhypopituitarism	2 (7.4)	0 (0)	0
Hypocortisolemia	1 (3.7)	0 (0)	0

Number of patients (%)

Table 2: Summary of personal and MRI characteristics.

Characteristic	Value (*n*=27)
Age, year	38 ( )
Female sex, *n* (%)	24 (88.8)
MRI findings, *n* (%)	
Location	
Intrasellar	16 (59.2)
Intra and suprasellar	9 (33.3)
Suprasellar	2 (7.4)
Signal on T1WI*	
Isointense	17 (62.9)
Hypointense	4 (14.8)
Hyperintense	6 (22.2)
Signal on T2WI	
Isointense	6 (22.2)
Hypointense	1 (3.7)
Hyperintense	20 (74.0)
Enhancement	11 (40.7)
Rim enhancement	7 (25.9)
Complete enhancement	4 (14.8)
Intra cystic nodule	6 (22.2)

*WI: Weighted image

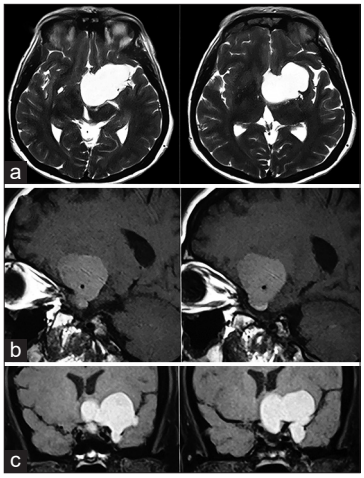

Figure 1: Axial (a), sagittal (b), and coronal (c) MRI images of a 52-year-old woman who presented with headache and bitemporal hemianopia. Images show a huge sellar and suprasellar lesion with lateral extension to the middle fossa, which showed no enhancement after Gadolinium injection (b).

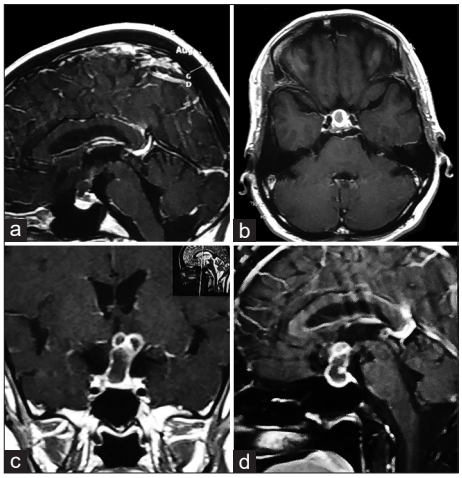

Figure 2: Sagittal (a) and axial (b) MRI images of a 42-year-old woman who presented with headache and showed a sellar lesion with ring enhancement. After 2 months, this patient refrained from surgery and returned with acute vision loss and severe headache. The new MRI (c and d) showed the lesion's rapid progression, which caused a compressive effect on the optic structures. The patient was operated by extended EETS and almost all symptoms resolved after surgery.

Follow-up

In a mean follow-up of 18 months, we observed no recurrence of RCC in our patients. Almost 95% of the cases had complete relief after surgery, and in one patient, the chronic persistent headache became an intermittent headache with the lower intensity in comparison with the preoperative condition. His headache responded to medication successfully.

DISCUSSION

RCC is a rare diagnosis in neurosurgery that can occur in every age group and it becomes symptomatic when it is large enough to compress the adjacent structures or rupture.[28] It may present with various clinical presentations such as headache, transient or permanent adenohypophysis dysfunction, diabetes insipidus, and visual impairment.[32,46] These lesions show variable manifestation and different schema of intensities on T1WI, T2WI, and various pattern of enhancement on MRI images which cause preoperative diagnosis difficult.[5,38,42,44] Although the natural history of RCC is not well defined; yet, it has been observed that surgical intervention results in the resolution of symptoms with minimal complications and risk of recurrence.[28] Our experience with RCC patients indicated that it can be managed successfully by transsphenoidal surgery with minimal complications in the studied population.

Endocrine dysfunction has been described in 17–81% of patients with RCC, and hyperprolactinemia was the most common abnormality.[22] As noted in other published series, we found a high rate of pituitary dysfunction in our patients. Our data revealed 33.3% of patients presented with pituitary dysfunction, and hyperprolactinemia, as the top disorder, occurred in nearly 55.5% of them. The menstrual irregularity was a common finding in women. All patients with hyperprolactinemia showed improvement in their postoperative prolactin level to normal or near-normal references. DI is also a relatively common finding

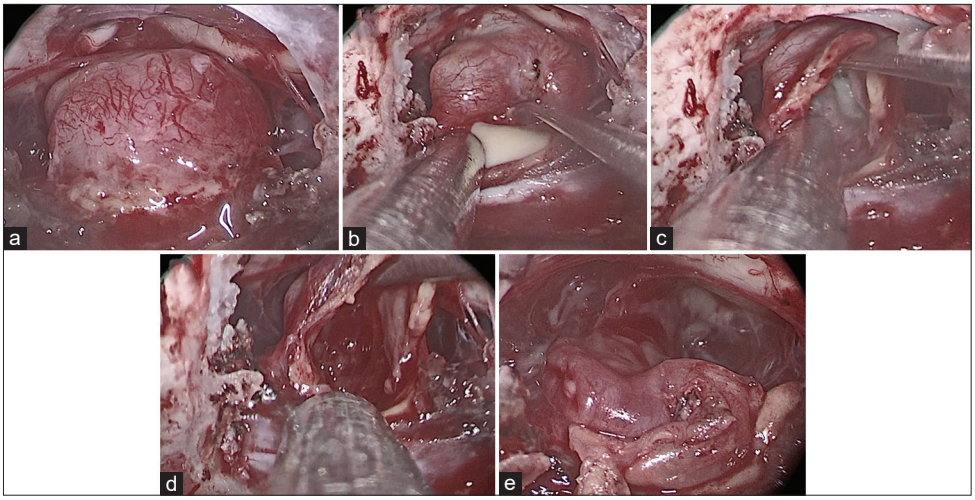

Figure 3: (a) Endoscopic transsphenoidal view of a Rathke's cleft cyst patient after drilling sellae and opening the dura. The pituitary gland surrounded the cyst circumferentially. Pituitary stalk and optic chiasma can be seen above the surgical field. (b) Trans-pituitary approach to Rathke's cleft cyst. Colloid-like material drained after pituitary incision. (c) Cyst wall adhesion to the pituitary gland can be seen. (d) Removing cyst material and cyst wall as far as feasible. (e) Saving pituitary gland and stalk at the end of the operation.

at the initial presentation in patients with RCC, as compared to pituitary adenomas. The reported rates of DI for RCC patients range from 2.3% to as high as 37% of patients.[15,22] In our series, 22.2% of patients with endocrinopathy presented with diabetes insipidus. None of these patients showed improvement postoperatively, similar to panhypopituitarism and hypocortisolemia. Wait *et al*. reported female sex as the sole independent predictor of recovery of the pituitary axis function.[43] The growth hormone most likely recovers first, followed by the thyroid-stimulating hormone, testosterone, and cortisol. Due to the variability in recovery and the relatively small numbers of those recovering, it is impossible to predict a patient's chance of recovery in the function of a specific axis.[43]

Surgical intervention is the mainstay for the management of symptomatic RCC. The transsphenoidal approach has become the standard route for intrasellar lesion and EETS has proved its efficacy to standard approaches as reported in the previous studies.[2,9,10,18] Some authors proposed a transcranial approach for lesions with supra-sellar extension,[17,36] but we used extended EETS for patients with suprasellar extension. By partial the pars planum of the sphenoid bone and coagulating the anterior intercavernous sinus, we achieved a great view to the suprasellar part of the lesion and successfully removed it in all patients who were operated with this approach. No surgically-related complications, like CSF leak, happened in our series. We used transcranial approach for only two patients: one of them had pure suprasellar lesion and the other one had a gross lateral extension to the middle fossa, which is an exotic behavior for RCC.

MRI is the ideal modality for preoperative assessment of RCCs and for distinguishing RCCs from other cystic sellar lesions. RCCs can be diagnosed on MRI based on shape, signal intensity, enhancement features, and an intracystic nodule. The diverse appearance of RCCs on MRI makes the neuroimaging diagnosis of an RCC difficult.[19] The low intensity on T1WI occurs if protein concentration is low, whereas the high intensity is associated with a high protein concentration.[39,41] Therefore, the most common MRI signal patterns of RCC can be one of the following combinations: hypointensity on T1WI and hyperintensity on T2WI; isointensity on T1WI and hyperintensity on T2-WI; or hyperintensity on both T1WI and T2WI.[37,40] In the majority of cases, administration of Gadolinium reveals little or no enhancement of the cyst wall or contents on MRI images,[3,5] while a thin enhancing rim has been regarded to be the result of the inflammation or squamous metaplasia of the cyst wall, or to a rim of displaced hypophysis.[7,25] Chotai *et al*. propose that the hypointensity of the cyst content on T2WI image has an independent predicting value in terms of recurrence.[13] Wajima *et al*. classified RCCs based on their signal intensity on T2WI in two types. Type 1 was considered for lesions with hypointensity on T1WI and hyperintensity on T2WI and type 2 included other types of appearance on imaging.[44] They proposed that type-1 lesions have a more aggressive course and rapid growth and type 2 lesions are more indolent.[44] An intracystic nodule could also be present in over 40–75% of RCCs with high signal intensity on T1WI and low signal intensity on T2WI.[8] In our study similar to other studies iso- to hypointensity on T1WI, and hyperintensity on T2WI was the most common pattern on MRI (76%). Hypointensity on T2WI was perceived in 28% of the cases, but we observed no relationship between this parameter and an increase in the recurrence rate. Rim or complete enhancement was seen in 40% of patient and 22% showed a nodular mass in their cyst content, so lack of typical imaging characteristics regarding signal intensity and pattern of enhancement was observed in

our series. As far as location and size are concerned, most of our cases (59%) were primarily located in the sella turcica, and (7.4%) of 27 being primarily suprasellar. Nine (33%) of 27 intrasellar cysts had a secondary suprasellar extension. One case was very huge at diagnosis time and showed an unusual extension to the parasellar region and middle fossa. Up to the best of our knowledge, this type of RCCs extension was not reported in the literature up to now [Figure 1].

Many other neoplastic and nonneoplastic suprasellar lesions can clinically and radiologically mimic the RCC, including pituitary adenomas, craniopharyngiomas, germ cell tumors, suprasellar meningiomas, midline gliomas, hypothalamic hamartomas, Langerhans cell histiocytosis, tuberculoma, and sarcoidosis.[6,12,30] In the preoperative assessment of patients with cystic sellar lesions, it is critical to differentiate between RCC and other pathologies, most importantly cystic pituitary adenomas, craniopharyngiomas, and optochiasmatic-hypothalamic gliomas. The presence of a fluid-fluid level, a hypointense rim on T2WI, septation, and an off-midline location were more common with pituitary adenomas, whereas the presence of an intracystic nodule was more common with RCC.[35] On the other hand, craniopharyngioma is generally irregular, and large and is located in the suprasellar region. Homogenous and low-density cysts without enhancement are common on CT scans, with isodensity, high-density, and calcification also reported.[7,42] Optochiasmatic-hypothalamic gliomas are usually large and may contain areas of cystic degeneration. Contrast enhancement is seen in half of these lesions, which usually has a homogenous pattern. These tumors are mostly seen as hypo- to isointense on T1WI and hyperintense on T2WI.[27]

There is no general consensus regarding the extent of resection and debate still exists between radical cyst wall excision and partial cyst excision with cyst drainage. Xie *et al*. suggested that fenestration and aspiration of the cysts with partial excision of the cyst wall are usually sufficient.[45] Zhong *et al*. achieved the same conclusion in their series.[47] Fan *et al*. concluded that gross-total resection does not appear to reduce the recurrence rate but increases the risk of postoperative complications.[17] Likewise, in a series of 78 surgical interventions for RCC, Higgins *et al*. showed 13% of patients with RCC required repeat surgery in the future and attempted that gross-total resection did not appear to decrease the overall rate of recurrence.[21] Furthermore, more invasive resections in this study were associated with a higher rate of complications such as hyperprolactinemia, DI, and CSF leaks. On the contrary, Koutourousiou *et al*. emphasized their experience of aggressive surgical approach with a good surgical outcome and a low recurrence rate in their retrospective analysis[26] an Kim *et al*. recognized that the extent of resection is an central predictor for relapse.[25] Supporters of this conclusion suggest total resection of the cyst wall as more as it can be performed safely[29] or, at least, making cyst fenestration as wide as possible.[25] In a meta-analysis, Mendelson *et al*. attempted to address this challenging question.[31] They reported that less aggressive technique is associated with less postoperative dysfunction of the pituitary gland; however, the recurrence rate in relation to extent of resection has not been described. Nevertheless, they concluded that the microsurgical approach has resulted in slightly higher recurrence and endocrine dysfunction rates, as compared to the endoscopic approach. In our series, we achieved gross-total resection in 64% of our patients, and fortunately, we saw no rise in the rate of complications, considering pituitary dysfunction and visual problems. Just in one case, using an aggressive approach and generous exposure to achieve gross-total resection resulted in chiasmal prolapse and visual deterioration. This complication was treated instantly by chiasmopexy, and visual problems resolved after the intervention. In the cases with cyst wall adhesion to stalk or pituitary gland, or especially when the pituitary gland bounds the cyst wall, we preferred to fenestrate the cyst wall. This was done even with the transpituitary approach if necessary, including aspiration of the cyst content and partial removal of the cyst wall. We saw no increase in the recurrence rate with this approach on the primary course of follow-up. However, a longer follow-up will provide more information to validate or decline our argument. In general, we saw no postoperative exacerbation in the pituitary dysfunction, except for three cases who suffered from transient DI. However, these cases were successfully treated by medication.

Recent case-series have reported an improvement in visual symptoms in 68–98% of the patients; however, total recovery and improvement were not differentiated.[3,15] In our series, 29.6% of the patients had visual problems and 87% of the patients with visual disturbances showed various degrees of improvement postoperatively. Furthermore, 75% showed complete improvement of symptoms, which all of them had a visual field defect on preoperative perimetry. In the two patients with concomitant visual acuity diminution, one case showed partial improvement and the defect remained permanent in the other one.

RCC may remain stable throughout the patient's life. Conservative management and MRI follow-up are suitable for silent RCC or smaller cysts with mild symptoms, particularly in the elderly or younger patients without fertility.[1] There are also some reports of spontaneous involution of RCC (even with a large size), which supports conservative management in asymptomatic patients.[24] However, there is a shred of evidence that RCCs can rapidly get symptomatic as a result of cyst wall rupture or intracystic hemorrhage, such as pituitary adenoma apoplexy.[11] That happened in one of our patients who refused surgical treatment at first and came back with severe headache, sudden visual impairment and doubling

of tumor size, which underwent surgical operation urgently. Fortunately, his neurologic deficit recovered after surgery. Thus, in the case of conservative management, precise follow-up and patient education should be emphasized.

The recurrence rate in the literature varies from 0% to 40%.[15,20] Overall, among the three largest surgical series, the studies by Aho *et al*.[1] and Benveniste *et al*.[3] have reported rates of 18% recurrence at 5 years, and 16% recurrence at 2 years, respectively. Mendelson *et al*., in their meta-analyses of 28 articles reporting recurrence rates, totaling 1151 patients, identified the weighted estimate of recurrence to be 12.5%.[31] Interestingly, in our series, recurrence had no relationship with the contributing factors which were mentioned above. Moreover, out of the four cases with squamous metaplasia, none has had recurrences yet in our series.

Study limitations

Our sample size and retrospective nature of the study are the major limiting factors of our work. Furthermore, this was a single-center experience and as we are one of the few centers for skull base surgery in Iran, it is probable that more complicated patients present to our center, and this may influence our findings. We also think that a longer follow-up period could help to achieve more clear-cut results.

CONCLUSION

Our experience with RCC patients showed that EETS is a safe and effective method in the treatment of patients with minimal recurrence.

Ethics approval

Ethics committee of Shahid Beheshti University of Medical Sciences.

Availability of data and material

Data are available.

Declaration of patient consent

Patients' consent not required as patients' identities were not disclosed or compromised.

Financial support and sponsorship

Publication of this article was made possible by the James I. and Carolyn R. Ausman Educational Foundation.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Aho CJ, Liu C, Zelman V, Couldwell WT, Weiss MH. Surgical outcomes in 118 patients with Rathke cleft cysts. J Neurosurg 2005;102:189-93.
2. Alfieri A, Schettino R, Tarfani A, Bonzi O, Rossi GA, Monolo L. Endoscopic endonasal removal of an intra-suprasellar Rathke's cleft cyst: Case report and surgical considerations. Minim Invasive Neurosurg 2002;45:47-51.
3. Benveniste RJ, King WA, Walsh J, Lee JS, Naidich TP, Post KD. Surgery for Rathke cleft cysts: Technical considerations and outcomes. J Neurosurg 2004;101:577-84.
4. Billeci D, Marton E, Tripodi M, Orvieto E, Longatti P. Symptomatic Rathke's cleft cysts: A radiological, surgical and pathological review. Pituitary 2004;7:131-7.
5. Binning MJ, Gottfried ON, Osborn AG, Couldwell WT. Rathke cleft cyst intracystic nodule: A characteristic magnetic resonance imaging finding. J Neurosurg 2005;103:837-40.
6. Bommakanti K, Panigrahi M, Yarlagadda R, Sundaram C, Uppin MS, Purohit AK. Optic chiasmatic-hypothalamic gliomas: Is tissue diagnosis essential? Neurol India 2010;58:833-40.
7. Brassier G, Morandi X, Tayiar E, Riffaud L, Chabert E, Heresbach N, *et al*. Rathke's cleft cysts: Surgical-MRI correlation in 16 symptomatic cases. J Neuroradiol 1999;26:162-71.
8. Byun WM, Kim OL, Kim D. MR imaging findings of Rathke's cleft cysts: Significance of intracystic nodules. AJNR Am J Neuroradiol 2000;21:485-8.
9. Cappabianca P, Cavallo LM, Colao A, de Divitiis E. Surgical complications associated with the endoscopic endonasal transsphenoidal approach for pituitary adenomas. J Neurosurg 2002;97:293-8.
10. Cavallo LM, Prevedello D, Esposito F, Laws ER Jr., Dusick JR, Messina A, *et al*. The role of the endoscope in the transsphenoidal management of cystic lesions of the sellar region. Neurosurg Rev 2008;31:55-64; discussion 64.
11. Chaiban JT, Abdelmannan D, Cohen M, Selman WR, Arafah BM. Rathke cleft cyst apoplexy: A newly characterized distinct clinical entity. J Neurosurg 2011;114:318-24.
12. Chibbaro S, Signorelli F, Milani D, Cebula H, Scibilia A, Bozzi MT, *et al*. Primary endoscopic endonasal management of giant pituitary adenomas: Outcome and pitfalls from a large prospective multicenter experience. Cancers (Basel) 2021;13:3603.
13. Chotai S, Liu Y, Pan J, Qi S. Characteristics of Rathke's cleft cyst based on cyst location with a primary focus on recurrence after resection. J Neurosurg 2015;122:1380-9.
14. Christophe C, Flamant-Durand J, Hanquinet S, Heinrichs C, Raftopoulos C, Sariban E, *et al*. MRI in seven cases of Rathke's cleft cyst in infants and children. Pediatr Radiol 1993;23:79-82.
15. El-Mahdy W, Powell M. Transsphenoidal management of 28 symptomatic Rathke's cleft cysts, with special reference to visual and hormonal recovery. Neurosurgery 1998;42:7-16; discussion 16-7.
16. Famini P, Maya MM, Melmed S. Pituitary magnetic resonance imaging for sellar and parasellar masses: Ten-year experience in 2598 patients. J Clin Endocrinol Metab 2011;96:1633-41.
17. Fan J, Peng Y, Qi S, Zhang XA, Qiu B, Pan J. Individualized

surgical strategies for Rathke cleft cyst based on cyst location. J Neurosurg 2013;119:1437-46.
18. Frank G, Sciarretta V, Mazzatenta D, Farneti G, Modugno GC, Pasquini E. Transsphenoidal endoscopic approach in the treatment of Rathke's cleft cyst. Neurosurgery 2005;56:124-8; discussion 129.
19. Gaddikeri S, Vattoth S, Riley KO, DeHoff GW, Smith CB, Combs JT, *et al.* Rathke cleft cyst. MRI criteria for presumptive diagnosis. Neurosciences (Riyadh) 2013;18:258-63.
20. Hama S, Arita K, Nishisaka T, Fukuhara T, Tominaga A, Sugiyama K, *et al*. Changes in the epithelium of Rathke cleft cyst associated with inflammation. J Neurosurg 2002;96:209-16.
21. Higgins DM, Van Gompel JJ, Nippoldt TB, Meyer FB. Symptomatic rathke cleft cysts: Extent of resection and surgical complications. Neurosurg Focus 2011;31:E2.
22. Isono M, Kamida T, Kobayashi H, Shimomura T, Matsuyama J. Clinical features of symptomatic Rathke's cleft cyst. Clin Neurol Neurosurg 2001;103:96-100.
23. Kanter AS, Sansur CA, Jane JA Jr., Laws ER Jr. Rathke's cleft cysts. Front Horm Res 2006;34:127-57.
24. Kim CW, Hwang K, Joo JD, Kim YH, Han JH, Kim CY. Spontaneous involution of rathke's cleft cysts without visual symptoms. Brain Tumor Res Treat 2016;4:58-62.
25. Kim JE, Kim JH, Kim OL, Paek SH, Kim DG, Chi JG, *et al*. Surgical treatment of symptomatic Rathke cleft cysts: Clinical features and results with special attention to recurrence. J Neurosurg 2004;100:33-40.
26. Koutourousiou M, Grotenhuis A, Kontogeorgos G, Seretis A. Treatment of Rathke's cleft cysts: Experience at a single centre. J Clin Neurosci 2009;16:900-3.
27. Kumar J, Kumar A, Sharma R, Vashisht S. Magnetic resonance imaging of sellar and suprasellar pathology: A pictorial review. Curr Probl Diagn Radiol 2007;36:227-36.
28. Larkin S, Karavitaki N, Ansorge O. Rathke's cleft cyst. Handb Clin Neurol 2014;124:255-69.
29. Laws ER, Kanter AS. Rathke cleft cysts. J Neurosurg 2004;101:571-2; discussion 572.
30. Lemm D, de Oliveira FH, Bernays RL, Kockro RA, Kollias S, Fischer I, *et al*. Rare suprasellar glioblastoma: Report of two cases and review of the literature. Brain Tumor Pathol 2012;29:216-20.
31. Mendelson ZS, Husain Q, Elmoursi S, Svider PF, Eloy JA, Liu JK. Rathke's cleft cyst recurrence after transsphenoidal surgery: A meta-analysis of 1151 cases. J Clin Neurosci 2014;21:378-85.
32. Nishioka H, Haraoka J, Izawa H, Ikeda Y. Headaches associated with Rathke's cleft cyst. Headache 2006;46:1580-6.
33. Niwa J, Tanabe S, Ibayashi Y, Hashi K. Clinicopathological findings in symptomatic Rathke's cleft cyst: Correlation between enhancement effects on MRI and histopathology of the cyst wall. Neurol Surg 1996;24:125-33.
34. Park M, Lee SK, Choi J, Kim SH, Kim S, Shin NY, *et al*. Differentiation between cystic pituitary adenomas and Rathke cleft cysts: A diagnostic model using MRI. Am J Neuroradiol 2015;36:1866-73.
35. Park M, Lee SK, Choi J, Kim SH, Kim SH, Shin NY, *et al*. Differentiation between cystic pituitary adenomas and rathke cleft cysts: A diagnostic model using MRI. AJNR Am J Neuroradiol 2015;36:1866-73.
36. Peng Y, Fan J, Li Y, Qiu M, Qi S. The supraorbital keyhole approach to the suprasellar and supra-intrasellar rathke cleft cysts under pure endoscopic visualization. World Neurosurg 2016;92:120-5.
37. Rao VJ, James RA, Mitra D. Imaging characteristics of common suprasellar lesions with emphasis on MRI findings. Clin Radiol 2008;63:939-47.
38. Ratha V, Patil S, Karmarkar VS, Shah NJ, Deopujari CE. Surgical management of rathke cleft cysts. World Neurosurg 2017;107:276-84.
39. Ross DA, Norman D, Wilson CB. Radiologic characteristics and results of surgical management of Rathke's cysts in 43 patients. Neurosurgery 1992;30:173-8; discussion 178-9.
40. Saeki N, Sunami K, Sugaya Y, Yamaura A. MRI findings and clinical manifestations in Rathke's cleft cyst. Acta Neurochir (Wien) 1999;141:1055-61.
41. Teramoto A, Hirakawa K, Sanno N, Osamura Y. Incidental pituitary lesions in 1,000 unselected autopsy specimens. Radiology 1994;193:161-4.
42. Tominaga JY, Higano S, Takahashi S. Characteristics of Rathke's cleft cyst in MR imaging. Magn Reson Med Sci 2005;2:1-8.
43. Wait SD, Garrett MP, Little AS, Killory BD, White WL. Endocrinopathy, vision, headache, and recurrence after transsphenoidal surgery for Rathke cleft cysts. Neurosurgery 2010;67:837-43; discussion 843.
44. Wajima D, Yonezawa T, Masui K, Aketa S. Relationship between clinical features and T2-weighted magnetic resonance images in symptomatic rathke cleft cysts. World Neurosurg 2016;88:421-7.
45. Xie T, Hu F, Yu Y, Gu Y, Wang X, Zhang X. Endoscopic endonasal resection of symptomatic Rathke cleft cysts. J Clin Neurosci 2011;18:760-2.
46. Zada G, Lin N, Ojerholm E, Ramkissoon S, Laws ER. Craniopharyngioma and other cystic epithelial lesions of the sellar region: A review of clinical, imaging, and histopathological relationships. Neurosurg Focus 2010;28:E4.
47. Zhong W, You C, Jiang S, Huang S, Chen H, Liu J, *et al*. Symptomatic rathke cleft cyst. J Clin Neurosci 2012;19:501-8.

How to cite this article: Sharifi G, Amin A, Lotfinia M, Hallajnejad M, Davoudi Z, Dilmaghani NA, *et al*. Rathke's cleft cysts: A single-center case series. Surg Neurol Int 2022;13:368.