



# Endoscopic endonasal resection of symptomatic Rathke cleft cysts: clinical outcomes and prognosis

Zhongzhong Jiang<sup>1</sup> · Mengqiang Yu<sup>1</sup> · Yugang Jiang<sup>1</sup> · Yong Peng<sup>1</sup>

Received: 7 August 2018 / Revised: 1 November 2018 / Accepted: 21 November 2018 / Published online: 10 December 2018  
© Springer-Verlag GmbH Germany, part of Springer Nature 2018

## Abstract

The aim of this study is to investigate the clinical presentation and outcomes associated with endoscopic endonasal resection of Rathke cleft cysts (RCCs). The authors retrospectively studied a series of 13 patients who were diagnosed with RCCs after endoscopic endonasal resection at the Second Xiangya Hospital between June 2016 and December 2017. All 13 patients (8 women) underwent a purely endoscopic endonasal approach (EEA) for fenestration and aspiration of RCCs with excision of the cystic wall. The patient ages varied from 25 to 67 years (mean, 45.1 years), and the follow-up period ranged from 8 to 25 months (mean, 16.6 months). Headache was a presenting symptom in all 13 patients, with 11 (80%) out of the 13 having experienced postoperative improvement of their headaches. Six (46%) of the 13 patients were admitted with pituitary dysfunction, all of them had postoperative improvement. Four (31%) of the 13 patients had temporary postoperative pituitary dysfunction, although there was not any permanent pituitary dysfunction. Six patients had intraoperative complications with CSF leaks, and after the operation, three of them developed temporary diabetes insipidus, one of them had a postoperative infection, and another one had postoperative cerebrospinal fluid leaks, who was treated with absolute bed rest for 7 days. No patient experienced recurrent cysts. EEA is a safe and effective approach for the treatment of symptomatic RCCs. Notably, it is appreciated for protecting and restoring pituitary function; however, the postoperative recurrence rate still lacks a large sample related to the long time follow-up study. Complete aspiration of the cysts' contents with partial excision of the cyst wall is usually sufficient for treatment.

**Keywords** Rathke cleft cysts · Endoscopic endonasal approach · Outcomes · Prognosis

## Introduction

Rathke cleft cysts (RCCs) are the most common incidentally discovered benign sellar lesions, followed by pituitary adenomas. They are thought to arise from the remnants of the embryonic Rathke pouch [10, 25]. Although RCCs are typically asymptomatic, these lesions are often discovered incidentally with head MR imaging [2]. Normally, upon identification of the lesion, it is not large enough to result in pressure effects on adjacent structures such as the optic chiasma, pituitary stalk and/or hypothalamus. However, as it grows, the patient can experience headaches, pituitary dysfunction, or visual disturbances [18]. Surgery is an effective way to decompress and

relieve symptoms. As the neuroendoscopic techniques progress, the position of performing craniotomies for RCC treatment gradually is being replaced by EEA [14, 16, 19–21, 26, 27].

In this study, we report 13 RCC patients who were accepted for EEA fenestration and aspiration of RCC with partial excision of the cyst wall at the Second Xiangya Hospital between June 2016 and December 2017 (Tables 1, 2, and 3). We reviewed each of the cases symptoms, operation methods, effects of the operation, postoperative complications, and recurrence rates.

## Materials and methods

### General information

Thirteen patients were enrolled in the present study. There were 5 males and 8 female patients, aged between 25 and 67, with an

✉ Yong Peng  
foxpy195@csu.edu.cn

<sup>1</sup> Department of Neurosurgery, The Second Xiangya Hospital, Central South University, Changsha, Hunan 410011, China

**Table 1** Results of 13 patients with symptomatic Rathke cleft cysts who underwent endoscopic endonasal resection

Patient	Gender	Age (year)	Symptoms	Characteristic on MRI	Lesion size (mm)	Endocrine examination	Complications	Recurrence	Follow-up (month)
1	M	25	H, E	I, N	14	AD	DI	0	12
2	M	27	H	I	22	–	–	0	23
3	M	36	H	I	12	–	–	0	11
4	F	67	H, E	I	23	HG, HT	–	0	9
5	M	47	H	NA(IS)	23	–	–	0	23
6	M	61	H	IS, RE	13	–	CFL	0	13
7	F	30	H, E	I, RE	21	HG, AD	Ii	0	16
8	F	32	H	I, N	13	–	DI	0	25
9	F	51	H, E	I	12	HT	DI	0	25
10	F	46	H, E	I	14	HG, AD	–	0	19
11	F	46	H	IS	12	–	–	0	24
12	F	63	H	IS	18	–	–	0	8
13	F	55	H, E	IS	22	HY	–	0	8

*M* male, *F* female, *H* headache, *E* endocrine dysfunction, *RE* rim enhancement, *I* intrasellar, *IS* intra- and suprasellar, *N* nodules, *NA* not available, *AD* ACTH deficiency, *HG* hypogonadism, *HT* hypothyroidism, *HY* hyperprolactinemia, *DI* diabetes insipidus, *CFL* cerebrospinal fluid leaks, *Ii* Intracranial infection

average of 45.1 years. The duration of the disease took from 5 days to 30 years, with an average of 36.5 months. All patients (100%) had experienced mild and chronic headaches, and six (46%) suffered from pituitary dysfunction. None of the patients complained of visual impairment and diabetes insipidus.

### Image information

All patients underwent MRI of the pituitary region. The maximal dimensions of the cysts (as measured in the

plane) ranged from 12 to 23 mm (mean, 17 mm), and the location of the cysts is different: in 8 patients, the cyst was purely intrasellar and the remaining 5 patients had both intra- and suprasellar components. The signal intensity varied widely (hypo-, iso-, or hyperintense). Two patients also had nodules in the cysts with low signal intensity on T2-weighted and high signal intensity on T1-weighted images. Moreover, 2 of 13 patients were found to have peripheral rim enhancement after MRI tests.

**Table 2** Endocrine laboratory results of 13 patients with symptomatic Rathke cleft cysts who underwent endoscopic endonasal resection

Patient no	Preoperative	Postoperative			
		1d	7d	30d	90d
1	ACTH-	N	N	N	N
2	N	N	N	N	N
3	N	N	N	N	N
4	E2-; T3-; T4-;	E2-; LH-; FSH-; T3-; T4-	E2-; LH-; T4-; FSH-	E2-; T4-	E2-
5	N	N	N	N	N
6	N	N	N	N	N
7	LH-; ACTH-	LH-;	N	N	N
8	N	N	N	N	N
9	T3-; T4-;	T3-; T4-; TSH-	T4-	N	N
10	E2-; ACTH-	LH-; E2-; T4-; ACTH-	E2-; T4-; ACTH-	E2-; ACTH-	E2-
11	N	N	N	N	N
12	N	N	N	N	N
13	PRL+	N	N	N	N

*ACTH* adrenocorticotropin, *E2* estradiol, *FSH* follicle stimulating hormone, *LH* luteinizing hormone, *TSH* thyroid stimulating hormone, *T3* triiodothyronine, *T4* thyroxine, *PRL* prolactin

**Table 3** Patient characteristics and literature review of endoscopic endonasal resection of symptomatic Rathke cleft cysts

	Present study	Xie et al [26].	Madhok et al [14].	Mendelson et al [16].	Frank et al [7].
<b>General information</b>					
Male	5 (38%)	11 (48%)	–	6 (55%)	8 (36%)
Female	8 (62%)	12 (52%)	–	5 (45%)	14 (64%)
Total	13	23	32	11	22
Mean age (year)	45.1	43.3	34	43	37
<b>Manifestations</b>					
Headache	13 (100%)	15 (65%)	26 (81%)	9 (82%)	3 (14%)
Pituitary dysfunction	6 (46%)	6 (26%)	6 (19%)	5 (45%)	14 (64%)
Visual impairment	0 (0%)	9 (39%)	0 (0%)	6 (55%)	5 (23%)
<b>Characteristic on MRI</b>					
Intrasellar	8 (67%)	7 (30%)	20 (63%)	–	14 (64%)
Intra-/supra-sellar	4 (33%)	15 (65%)	10 (31%)	–	8 (36%)
Suprasellar	0 (0%)	1 (5%)	2 (6%)	–	0 (0%)
Nodules	2	2	0	0	0
Peripheral rim enhancement	2	11	0	6	0
Mean lesion size, mm	17	–	10	15	20
<b>Post-operative complications</b>					
CSF leak	1 (8%)	3 (13%)	0 (0%)	0 (0%)	8 (36%)
DI transient	3 (23%)	1 (4%)	0 (0%)	1 (9%)	1 (5%)
New pituitary	0 (0%)	0 (0%)	3 (9%)	0 (0%)	0 (0%)
Dysfunction	1 (8%)	2 (8%)	0 (0%)	1 (9%)	0 (0%)
<b>Infection</b>					
Recovery rate of pituitary function	100%	50%	33%	100%	29%
Recurrence	0 (0%)	2 (9%)	2 (6%)	2 (18%)	1 (5%)
Mean follow-up(range), mo	16.6	3–36	19	24	33

CSF cerebrospinal fluid, DI diabetes insipidus

### Endocrine examination information

Six patients suffered from pituitary dysfunction before operation. Among them, three patients experienced ACTH deficiency, three patients had hypogonadism, two patients had hypothyroidism, and one patient had hyperprolactinemia. In addition, three of the six patients had multiple pituitary hormones deficiency, they presented with hypogonadism combined with hypothyroidism, hypogonadism combined with ACTH deficiency, and hypogonadism combined with ACTH deficiency, respectively.

### Surgical method

A standard binary approach was used to access the sphenoid sinus. The operation was carried out through the nostril. In most cases, the right nostril was selected; in other cases, the nostril was selected according to the location of the lesion. The bilateral middle turbinates were lateralized and the natural sphenoid ostium on the left was opened to create wide bilateral sphenoidotomies. A small portion of the posterior nasal septum was then resected. With a power drill and rongeur, the

sphenoid sinus interval and sphenoid sinus mucosa was removed to reveal the base of the sella, the bilateral internal carotid artery, and the inferior saddle crypt. After the floor of the sella was carefully removed, the dura was opened according to the position of the pituitary gland, as revealed by pre-operative imaging. The cyst contents (pale, yellowish-white or yellowish-brown gelatin) suddenly overflowed and were cleaned. Meanwhile, the cyst wall was examined pathologically. If cerebrospinal fluid (CSF) leakage occurred during the operation, repair of the sella was performed by plugging it with abdominal fat. A small amount of biomedical fibrin sealant and artificial dural membrane was used to support the floor of the sella.

## Results

### Postoperative outcome

All patients were reported to survive in this study. Six patients had intraoperative complications with CSF leaks and the sella

were repaired instantly. After the operation, three of them developed temporary diabetes insipidus and the urine volume returned to normal after ingesting half a tablet or 1 tablet of vasopressin for 3–5 days. In addition, one of the six patients had postoperative cerebrospinal fluid leaks and improved by absolutely lying in bed for 7 days. Another one of the six patients had a postoperative infection and was cured after antibiotic treatment.

Preoperative pituitary dysfunction were not aggravated as seen from the endocrine examination 7 days or 1 months or 3 months after surgery, and four of the six (67%) patients had normal endocrine laboratory results 3 months after surgery, two of the six presented with hypogonadism. In addition, hormone replacement therapy was performed (scheme for oral hydrocortisone 20 mg at 8 PM, 10 mg at 4 PM, oral levothyroxine 25 µg/Qd) after operation without gonadal hormones.

The results of pathological examination were all RCCs, which revealed that the contents were unstructured substance of homogenous red dye or pink dye. The cystic walls were a single layer of ciliated cuboidal or columnar epithelium.

### Follow-up outcomes

The average follow-up time was 16.6 months (range 8–25 months), preoperative headaches were relieved in 11 out of 13 patients. A continued mild headache was seen in two patients, but recurrence was not found through the latest MRI after 3 months of surgery.

### Discussion

RCCs are non-neoplastic lesions in the sellar region that contain residual material of the embryonic Rathke pouch. There is a mucinous, gelatinous, or caseous cystic fluid within the cyst, and the wall is formed by a thin epithelial membrane [10, 13]. Most patients are asymptomatic; 11.3% of RCCs are found by chance during routine autopsy. However, the detection rate is higher after MRI is applied to the head [23].

Symptomatic RCCs typically present in all age groups, with a peak frequency between the fourth and sixth decades. It is associated with a female preponderance: female/male ratio up to 2 [15]. Our study is concordant with others in gender (F:M = 8:5) and in age (M = 45.1 years). Of course, these epidemiological characteristics require large sample studies owing to selection bias and a small number of cases. When the RCC volume is small, it never produces symptoms. As it grows, the patient may experience headaches, pituitary dysfunction, or visual disturbances [25, 26]. Symptomatic RCCs may be associated with headaches (14%–83%), visual impairments (0%–55%), and/or pituitary dysfunction (18.8%–78%) [4, 5, 11, 14, 16, 19, 20, 26, 28]. In our study, the most common symptom was headache (100%), followed

by pituitary dysfunction (46%). RCCs may also develop atypical symptoms including hypothalamic dysfunction, cystic abscess, and sphenoidal sinusitis.

Headaches are the most common clinical manifestation of RCCs. Most patients have chronic and intermittent headaches located at the forehead or unilateral or bilateral temporal distension. Headaches are caused by cystic compression or aseptic inflammation originated from cystic formation. Few reports state that acute headaches are caused by RCCs internal bleeding, which is similar to pituitary adenoma apoplexy [3]. If RCC internal bleeding occurs, early surgery should be performed to stop bleeding and remove blood clots to relieve pressure. In this study, 11 (80%) of the 13 patients experienced postoperative improvement of their headaches. Whereas, the remaining two patients still reported a mild headache. Six (46%) of the 13 patients presented with pituitary dysfunction; all of them had postoperative improvement. All patients in this study performed the vision and visual field tests as well as underwent fundus photography before the operation. No patients experienced visual impairment or temporal hemianopia. It may be that the cyst volume was too small to press the optic chiasm (with an average of 1.7 cm on the preoperative MRI), or that the compression time was too shorter to show visual impairment or temporal hemianopia.

For asymptomatic patients with RCC, clinical observation (conduct a review of magnetic resonance imaging every 6 months to 1 year) was satisfactory. If the cyst size increased during conservative treatment, surgical intervention was indicated [8, 12]. In Silas a. Culver's study, 75 patients received conservative treatment, and the follow-up time was 1–126 months (median 24 months), and only 21 cases (28%) increased cyst size [6]. Because of the variable signal intensities shown by the MRI imaging, it was sometimes difficult to distinguish RCCs from other saddle lesions. However, Mandy J. Binning et al. reported that except for the marginal enhancement of cystic lesions, signal intensity pouch nodules on T1- and T2- weighted images were noticed, especially on the T2- weighted images [2]. When nodules are always visible, it can aid the preoperative diagnosis of RCCs and help doctors to select conservative treatment. However, the RCC diagnostic gold standard is histopathological examination.

The surgical indications of this condition include headaches, vision and visual field changes, enlarged cysts, pituitary endocrine diseases, and diagnosis uncertainty [11]. In recent years, the EEA has become the preferred surgical approach [14, 16, 19–21, 26, 27]. However, for complicated, large, and complete saddle RCCs, or in patients with contraindicated transsphenoidal surgery, a transcranial approach is still required [27]. It has also been reported that the transsphenoidal expansion approach and the use of angular endoscopy can resect lesions in the saddle [12, 16]. Compared with surgical intervention, endoscopy provides good lighting, possesses the advantages of a large angle and close observation, and clearly displays the internal structure of the sella to distinguish the

normal pituitary and the cystic wall. In addition, it results in reduced trauma, less complications, and quick postoperative recovery [7, 19].

All patients in this group underwent the neuroendoscopic transnasal sphenoidal approach, which is now the main means of surgery. Some advocate for the complete removal of the cyst wall; however, this easily produces cerebrospinal fluid nasal leakage and iatrogenic decline of pituitary function [1]. Moreover, studies have shown there is no significant differences in the postoperative recurrence rate between the two strategies for cyst wall resection [4]. In our study, postoperative headache symptoms were 85% relieved. Pituitary dysfunction was 100% relieved, but the recovery time was from 7 days to 3 months after surgery, and hormone replacement therapy was required in the early stage. In the previous studies, postoperative headache and vision loss were both improved, but the recovery of pituitary function was 28.6–56% for varying degrees of pituitary injury before or during surgery, and different operation method (partial resection or radical resection), except for lesion size [5, 7, 9, 14, 16, 19, 24, 26]. In our study, 67% (4/6) had recovery of pituitary function, the other two patients who did not fully restore their pituitary function showed hypogonadism, which may be related to no giving gonadal hormones irregularly after surgery. Besides, recovery of pituitary function after operation was more common in patients with a single pituitary hormone deficiency [20]. In our study, three of the four (75%) cases of complete recovery of pituitary function are the single pituitary hormone deficiency. In addition, it has been proposed that anterior hypopituitarism is observed in cysts with high and iso-intensity on T1-weighted MR images, so the signal difference on MR may be related to the recovery rate of pituitary function [17].

Postoperative complications after transsphenoidal surgery were mainly cerebrospinal fluid leaks (0–36%), infection (0–9%), diabetes insipidus transient (0–9%), and new pituitary dysfunction (0–33%) [5, 7, 14, 16, 26]. In this group, there was 1 case (7%) of cerebrospinal fluid nasal leakage, 1 case of infection (7%), 3 cases of diabetes insipidus transient (23%), and 1 case of new pituitary dysfunction (7%), which was roughly consistent with other studies. There were six cases of intraoperative cerebrospinal fluid leakage, autologous fat stuffing in the saddle, with artificial dura mater, gelatin sponge, and fibrin glue to seal the bottom of the saddle. After this, there was only one case of postoperative cerebrospinal fluid leakage, so the repair success rate was 83%, and the patient improved by absolutely lying in bed for 7 days. Although secondary intracranial infection occurred on another one patient, she recovered after antibiotics treatment.

Recurrence was mainly detected by head MRI examination after the occurrence of the original symptoms or new symptoms, but it was also found accident during the review of head MRI examination. Zachary S. Mendelson et al. reported that the overall relapse rate was 12.5% [15]. Recurrence time

varied; the average recurrence time was 14±6 months [4]. This group's postoperative follow-up time was from 8 to 25 months, with an average of 16.6 months. In other similar surgical methods, the postoperative follow-up time was 1–60 months, and the recurrence rate was 5.7–18%. Silky Chotai et al. reported that the pathologic examination results of squamous metaplasia and intensity on T2-weighted magnetic resonance imaging (MRI) were independent predictor of recurrence of RCCs, and the extent and type of surgical resection was not associated with recurrence [4]. At the same time, some studies have shown possible cystic wall enhancement by using MRI and that RCC infection may lead to its recurrence [8, 12, 22]. In this group, there was only one case of equal intensity on T2-weighted MRI. Therefore, the low recurrence rate was low because of the absence of independent predictors of recurrence; however, it also may be due to the short follow-up or the lack of samples. Patients with a high probability of recurrence should be very closely followed up.

## Conclusion

EEA is a safe and effective approach for the treatment of symptomatic RCCs, and it is appreciated for protecting and restoring pituitary function. However, the postoperative recurrence rate remains unclear because of the lack of a large sample of follow-up cases over a long period of time. Complete aspiration of the cysts' contents with partial excision of the cyst wall is usually sufficient for treatment.

## References

1. Aho CJ, Liu C, Zelman V, Couldwell WT, Weiss MH (2005) Surgical outcomes in 118 patients with Rathke cleft cysts. *J Neurosurg* 102:189–193. <https://doi.org/10.3171/jns.2005.102.2.0189>
2. Binning MJ, Gottfried ON, Osborn AG, Couldwell WT (2005) Rathke cleft cyst intracystic nodule: a characteristic magnetic resonance imaging finding. *J Neurosurg* 103:837–840. <https://doi.org/10.3171/jns.2005.103.5.0837>
3. Chaiban JT, Abdelmannan D, Cohen M, Selman WR, Arafah BM (2011) Rathke cleft cyst apoplexy: a newly characterized distinct clinical entity. *J Neurosurg* 114:318–324. <https://doi.org/10.3171/2010.5.JNS091905>
4. Chotai S, Liu Y, Pan J, Qi S (2015) Characteristics of Rathke's cleft cyst based on cyst location with a primary focus on recurrence after resection. *J Neurosurg* 122:1380–1389. <https://doi.org/10.3171/2014.12.JNS14596>
5. Cohan P, Foulad A, Esposito F, Martin NA, Kelly DF (2004) Symptomatic Rathke's cleft cysts: a report of 24 cases. *J Endocrinol Investig* 27:943–948. <https://doi.org/10.1007/Bf03347537>
6. Culver SA, Grober Y, Ornan DA, Patrie JT, Oldfield EH, Jane JA Jr, Thomer MO (2015) A case for conservative management: characterizing the natural history of radiographically diagnosed Rathke

- cleft cysts. *J Clin Endocrinol Metab* 100:3943–3948. <https://doi.org/10.1210/jc.2015-2604>
7. Frank G, Sciarretta V, Mazzatenta D, Farneti G, Modugno GC, Pasquini E (2005) Transsphenoidal endoscopic approach in the treatment of Rathke's cleft cyst. *Neurosurgery* 56:124–129. <https://doi.org/10.1227/01.neu.0000144824.80046.1f>
  8. Han SJ, Rolston JD, Jahangiri A, Aghi MK (2014) Rathke's cleft cysts: review of natural history and surgical outcomes. *J Neuro-Oncol* 117:197–203. <https://doi.org/10.1007/s11060-013-1272-6>
  9. Ito M, Matsuda K, Kuge A, Sato S, Kayama T, Sonoda Y (2018) Treatment of Rathke's cleft cyst: technical note for preservation of pituitary function. *No Shinkei Geka* 46:313–317. <https://doi.org/10.11477/mf.1436203722>
  10. Jahangiri A, Molinaro AM, Tarapore PE, Blevins L Jr, Auguste KI, Gupta N, Kunwar S, Aghi MK (2011) Rathke cleft cysts in pediatric patients: presentation, surgical management, and postoperative outcomes. *Neurosurg Focus* 31:E3. <https://doi.org/10.3171/2011.5.FOCUS1178>
  11. Kim E (2012) Symptomatic Rathke cleft cyst: clinical features and surgical outcomes. *World Neurosurg* 78:527–534. <https://doi.org/10.1016/j.wneu.2011.12.091>
  12. Kim JE, Kim JH, Kim OL, Paek SH, Kim DG, Chi JG, Jung HW (2004) Surgical treatment of symptomatic Rathke cleft cysts: clinical features and results with special attention to recurrence. *J Neurosurg* 100:33–40. <https://doi.org/10.3171/jns.2004.100.1.0033>
  13. Laws ER, Kanter AS (2004) Rathke cleft cysts. *J Neurosurg* 101:571–572; discussion 572. <https://doi.org/10.3171/jns.2004.101.4.0571>
  14. Madhok R, Prevedello DM, Gardner P, Carrau RL, Snyderman CH, Kassam AB (2010) Endoscopic endonasal resection of Rathke cleft cysts: clinical outcomes and surgical nuances. *J Neurosurg* 112:1333–1339. <https://doi.org/10.3171/2009.10.JNS09348>
  15. Mendelson ZS, Husain Q, Elmoursi S, Svider PF, Eloy JA, Liu JK (2014) Rathke's cleft cyst recurrence after transsphenoidal surgery: a meta-analysis of 1151 cases. *J Clin Neurosci* 21:378–385. <https://doi.org/10.1016/j.jocn.2013.07.008>
  16. Mendelson ZS, Husain Q, Kanumuri VV, Eloy JA, Liu JK (2015) Endoscopic transsphenoidal surgery of Rathke's cleft cyst. *J Clin Neurosci* 22:149–154. <https://doi.org/10.1016/j.jocn.2014.08.002>
  17. Nishioka H, Haraoka J, Izawa H, Ikeda Y (2006) Magnetic resonance imaging, clinical manifestations, and management of Rathke's cleft cyst. *Clin Endocrinol* 64:184–188. <https://doi.org/10.1111/j.1365-2265.2006.02446.x>
  18. Raper DM, Besser M (2009) Clinical features, management and recurrence of symptomatic Rathke's cleft cyst. *J Clin Neurosci* 16:385–389. <https://doi.org/10.1016/j.jocn.2008.04.023>
  19. Ratha V, Patil S, Karmarkar VS, Shah NJ, Deopujari CE (2017) Surgical management of Rathke cleft cysts. *World Neurosurg* 107:276–284. <https://doi.org/10.1016/j.wneu.2017.07.164>
  20. Sala E, Moore JM, Amarin A, Carosi G, Martinez H Jr, Harsh GR, Arosio M, Mantovani G, Katznelson L (2018) Natural history of Rathke's cleft cysts: a retrospective analysis of a two centres experience. *Clin Endocrinol* 89:178–186. <https://doi.org/10.1111/cen.13744>
  21. Solari D, Cavallo LM, Somma T, Chiamonte C, Esposito F, Del Basso De Caro M, Cappabianca P (2015) Endoscopic endonasal approach in the management of Rathke's cleft cysts. *PLoS One* 10:e0139609. <https://doi.org/10.1371/journal.pone.0139609>
  22. Tate MC, Jahangiri A, Blevins L, Kunwar S, Aghi MK (2010) Infected Rathke cleft cysts: distinguishing factors and factors predicting recurrence. *Neurosurgery* 67:762–769; discussion 769. <https://doi.org/10.1227/01.NEU.0000377017.53294.B5>
  23. Teramoto A, Hirakawa K, Sanno N, Osamura Y (1994) Incidental pituitary lesions in 1,000 unselected autopsy specimens. *Radiology* 193:161–164. <https://doi.org/10.1148/radiology.193.1.8090885>
  24. Trifanescu R, Stavriniades V, Plaha P, Cudlip S, Byrne JV, Ansorge O, Wass JA, Karavitaki N (2011) Outcome in surgically treated Rathke's cleft cysts: long-term monitoring needed. *Eur J Endocrinol* 165:33–37. <https://doi.org/10.1530/EJE-11-0142>
  25. Trifanescu R, Ansorge O, Wass JA, Grossman AB, Karavitaki N (2012) Rathke's cleft cysts. *Clin Endocrinol* 76:151–160. <https://doi.org/10.1111/j.1365-2265.2011.04235.x>
  26. Xie T, Hu F, Yu Y, Gu Y, Wang X, Zhang X (2011) Endoscopic endonasal resection of symptomatic Rathke cleft cysts. *J Clin Neurosci* 18:760–762. <https://doi.org/10.1016/j.jocn.2010.10.014>
  27. Zada G (2011) Rathke cleft cysts: a review of clinical and surgical management. *Neurosurg Focus* 31:E1. <https://doi.org/10.3171/2011.5.FOCUS1183>
  28. Zhong W, You C, Jiang S, Huang S, Chen H, Liu J, Zhou P, Liu Y, Cai B (2012) Symptomatic Rathke cleft cyst. *J Clin Neurosci* 19:501–508. <https://doi.org/10.1016/j.jocn.2011.07.022>