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Original Article

The clinical course of Rathke's cleft cysts in pediatric patients: impact on growth and pubertal development

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Highlights

- The long-term course for symptomatic Rathke cleft cysts in children remains elusive.
- Growth impairment and precocious puberty were common and were managed medically.
- Follow-ups are warranted because of the potential for imaging and endocrine changes.

Abstract. Rathke's cleft cysts (RCCs) are non-neoplastic epithelial lesions in the sellar or suprasellar regions. RCCs are usually asymptomatic; however, some patients experience headaches, visual disturbances, and endocrine disorders. The best treatment for associated endocrinopathy remains elusive. We aimed to investigate the clinical course, magnetic resonance imaging findings, and response to therapy in 10 pediatric patients with RCCs and endocrinopathy. Growth impairment and precocious puberty were observed to be prevalent. One patient with suprasellar extension of RCC underwent surgery, while the others were treated medically. Of the nine patients, seven patients showed stable cyst size, while two patients displayed reduction in cyst size. Hormone replacement and gonadotropin suppression therapy were found to be effective. Imaging and endocrine follow-ups are warranted because of the potential for changes in the cyst size and hormonal changes.

Key words: Rathke's cleft cysts, GH deficiency, central precocious puberty, pituitary imaging

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Introduction

Rathke's cleft cysts (RCCs) are non-neoplastic epithelial lesions of the sellar or suprasellar regions. RCCs arise from Rathke's pouch remnants, which are embryonic structures located in the pars intermedia region (1). RCCs are relatively common in adults and are found in 3–33% of autopsy cases, and most RCCs are asymptomatic; however, they are occasionally accompanied by headaches, visual deficits, and endocrine dysfunction. These symptoms may be induced by compression of the pituitary and adjacent structures, while some studies suggest that inflammation is implicated in endocrine dysfunction (2, 3). Surgical management is usually selected to treat headaches and visual disturbances, whereas it is reportedly less efficacious in treating endocrinopathy and may even cause further complications (4).

RCCs are considered less frequent in the pediatric population than in the adult population. One study reported that asymptomatic pituitary cysts were detected on magnetic resonance imaging (MRI) in 1.2% of 341 patients aged < 15 yr (5). A recent study showed that RCC was detected on MRI in 13.5% of pediatric patients with endocrine-related diseases (6). Little is known about the clinical course and ideal treatment for pediatric patients with symptomatic RCCs (7). In the present study, we investigated the clinical course, MRI findings, and treatment response of pediatric patients with RCCs with endocrinopathy.

Methods

Patients

This study included patients with RCC aged <15 yr from April 2000 to March 2017 at three institutions. All but one RCC was diagnosed radiographically, differentiated from other cystic lesions by the location (between the midline of the anterior and posterior pituitary gland), and lack of calcification (8). The demographic, auxologic, and clinical data, family history, and follow-up duration were recorded.

Clinical Assessment

The height, height velocity SD, and body mass index (BMI) SD scores were calculated using the body index calculators of Japanese children and adolescents (9, 10). Sexual maturation was classified according to the Tanner stage (11, 12). The IGF-1, TSH, free T4, ACTH, cortisol, PRL, LH, FSH, and sex hormone levels were assessed. The IGF-1 SD score was also calculated (9, 13).

Short stature associated with GH deficiency (GHD) was defined as height ≤ –2.0 SD, or height velocity ≤ –1.5 SD over two years, and peak GH level < 6.0 ng/mL on stimulation test. The adult GH deficiency was reevaluated using a peak GH level < 9.0 ng/mL on GHRP-2 test or < 1.8 ng/mL on other stimulation tests. Idiopathic short stature (ISS) was defined as height ≤ –2.0 SD and peak GH levels > 6.0 ng/mL on any GH stimulation. Small for gestational age (SGA) was defined as sex- and parity-specific birth weight below the 10th percentile for gestational age based on the Japanese population reference. Central precocious puberty (CPP) was defined as: the onset of breast development by 7.5 yr of age or menarche by 10 yr of age in girls: testicular volume ≥ 4 mL by 9 yr of age in boys, together with height ≥ 2.0 SD or height velocity ≥ 1.5 SD over two years and basal LH levels > 0.3 mIU/mL, or GnRH stimulated peak LH levels > 5.0 mIU/mL (14). Central hypothyroidism (CH) was defined as low free T4 levels with normal or low TSH levels. Central adrenocortical insufficiency (CAI) was initially screened for morning serum cortisol levels < 10 μg/dL with normal or low ACTH levels and confirmed by peak cortisol levels < 20 μg/dL on an insulin-induced hypoglycemia test. Hyperprolactinemia (HPL) was defined as > 20 ng/mL. Central diabetes insipidus (CDI) was defined as urine volume > 3,000 mL per body surface area per day and responded to desmopressin. The bone age was assessed using the Tanner-Whitehouse 2, radius-ulna-short bone method standardized for Japanese children (15). The RCC MRI features (anterior-posterior diameter, height, localization, signal intensity compared with the anterior lobe, and size change) were assessed on sagittal T1- and T2-weighted images (WI). The patients with GHD or CPP had a follow-up bone age, height SD score, and BMI SD score.

Ethical consideration

This study was approved by the institutional review board of each participating institution. Informed consent was obtained from the parents or guardians as well as from the patient when applicable. All procedures performed in the studies involving human participants were in accordance with the 1964 Helsinki Declaration and its subsequent amendments.

Results

A total of 12 patients with RCC were identified; 2 patients were excluded because of insufficient endocrinological data. Subsequently, we enrolled 10 patients (four boys) with a mean age at diagnosis of 7.6 yr (range, 1–14 yr) and the mean follow-up period was 72.7 mo (range, 35–200 mo; growth charts are shown in the supplementary material). The birth weight and gestational age data were recorded in seven patients, and two patients were SGA (Cases 3 and 10). Growth impairment (short stature or growth retardation) was the most common manifestation, followed by precocious puberty, headache, visual disturbance, and polyuria. Four patients were initially diagnosed with GHD and CPP, followed by CDI, CAI, and CH. As for patients with CPP, their mother's mean age at menarche was 12.3 yr (range, 11–14 yr). One patient (Case 5) showed linear growth acceleration for two years (height velocity score +

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4.00 SD) and breast development (Tanner stage III). The prolactin levels were measured in six patients, and one patient had HPL (Case 5). The clinical characteristics are summarized in **Table 1**.

The median diameter of the cysts was 5.5 mm (range, 2–24 mm), and most cysts existed between the anterior and posterior lobes; however, the largest cyst developed a suprasellar extension. The MRI signal intensities were as follows: four cysts were observed to be hypointense on T1WI and hyperintense on T2WI, four cysts were observed to be isointense on T1WI and hypointense on T2WI, and two cysts were hyperintense on T1WI and isointense on T2WI. The representative images of the patterns are shown in **Fig. 1A–F**.

A total of nine patients were managed conservatively; seven patients showed no detectable change in the cyst size during the follow-up period. In the two remaining patients, one patient showed a decrease in the cyst size, and another (Case 10) exhibited near-complete cyst regression after eight months of follow-up with gradual height improvement (from –2.62 to –1.15 SD); the GH provocation test showed a normal response at 3-yr-old. Four patients with initial GHD received recombinant GH (rGH) treatment, and the height SD scores were promoted. The bone age to chronological age (BA/CA) ratio increased following GH treatment in three patients. One of these patients (Case 1) was subsequently treated with adult GH deficiency. Three patients with simple CPP received a GnRH analog (within a range of 30–100 μg/kg/dose); all the patients showed suppression of pubertal progression and a decreased height SD score and BA/CA ratio. Other endocrine disorders were managed with hormone replacement therapy. The patient who had a suprasellar extension (Case 5) underwent fenestration and partial cyst wall resection. Headaches, visual deficits, and HPL resolved temporarily; however, the rest of the endocrine dysfunction persisted. Subsequently, the patient had recurrent cyst enlargement, and the cyst was re-fenestrated. Later, she developed growth retardation and hypogonadotropic hypogonadism secondarily. She underwent step-by-step rGH, estrogen, and progesterone replacement therapy. The treatment outcomes are summarized in **Table 2**.

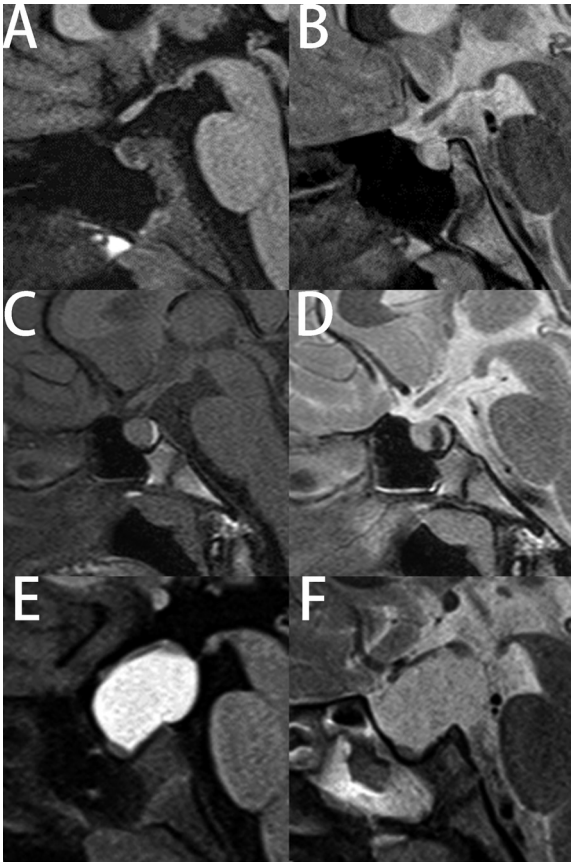

Fig. 1. Representative images of the three magnetic resonance imaging signal intensity patterns observed in pediatric patients with Rathke's cleft cysts: T1 weighted image (WI) hypointensity (A) and T2WI hyperintensity (B) (Case 1); T1WI isointensity (C) and T2WI hypointensity (D) (Case 3); and T1WI hyperintensity (E) and T2WI isointensity (F) (Case 5).

Table 1. Clinical characteristics of pediatric patients with Rathke's cleft cysts

Case	Sex/age (yr)	Presenting symptoms	Endocrine dysfunction	MRI findings			Surgery	Follow-up period (mo)
				T1/T2 signal intensity	Location	Size (diameter × height, mm)		
1	M/3	Short stature	CAI, CH, CDI, GHD	Hypo/Hyper	Sella	10 × 6	–	200
2	M/1	Short stature	GHD	Hypo/Hyper	Sella	2 × 2	–	77
3	F/11	Short stature	GHD	Iso/Hypo	Sella	6 × 2	–	40
4	M/14	Growth retardation	GHD	Iso/Hypo	Sella	7 × 6	–	49
5	F/8	Headaches, visual disturbance, breast development	CAI, CH, CPP, CDI, HH, HPL, GHD	Hyper/Iso	Sella-Supra	24 × 15	+	79
6	F/9	Premature menarche	CPP	Hypo/Hyper	Sella	3 × 2	–	57
7	F/8	Premature menarche	CPP	Iso/Hypo	Sella	3 × 2	–	35
8	F/8	Premature menarche	CPP	Iso/Hypo	Sella	4 × 1	–	39
9	M/8	Polyuria, polydipsia, enuresis	CAI, CDI	Hyper/Iso	Sella	6 × 6	–	108
10	F/1	Short stature	ISS	Hypo/Hyper	Sella	4 × 3	–	43

CAI, central adrenocortical insufficiency; CDI, central diabetes insipidus; CH, central hypothyroidism; CPP, central precocious puberty; F, female; GHD, GH deficiency; HH, hypogonadotropic hypogonadism, HPL, hyperprolactinemia; ISS, idiopathic short stature; M, male; MRI, magnetic resonance imaging.

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Table 2. Treatment outcomes of patients with growth hormone deficiency or central precocious puberty

Case	Endocrine dysfunction	Age at start of treatment (yr)	Pretreatment				Post-treatment				Treatment period (mo)
			BA/CA ratio	Height	BMI	IGF-1	BA/CA ratio	Height	BMI	IGF-1	
1	GHD	5	0.60	–2.75	–1.50	–2.49	1.00	–1.68	–1.20	–3.33	154
2	GHD	2	N/A[a]	–2.41	–1.56	–2.24	0.68	–0.56	–1.36	+0.95	69
3	GHD	11	0.87	–2.77	–1.16	–2.33	0.90	–1.06	–1.14	+0.97	38
4	GHD	14	0.89	–1.73	–0.91	–0.68	1.00	–0.05	–0.91	–1.22	19
5	CPP, GHD	8	1.08	–0.78	+1.72	–1.23	1.00	–2.75	+2.13	–4.98	76
6	CPP	9	1.20	+0.50	+1.89	+1.64	1.07	–0.81	+2.18	–1.25	30
7	CPP	9	1.37	+2.36	+0.30	+1.22	1.16	+0.10	–0.83	–1.15	30
8	CPP	8	1.22	–0.73	+0.62	+0.65	0.94	–1.19	+0.68	–0.18	37

Data are shown as standard deviation scores unless otherwise specified. [a] Not evaluated due to bone age < 3.2 yr. BA/CA ratio, ratio of bone age to chronological age; BMI, body mass index; IGF-1, insulin-like growth factor 1; GHD, GH deficiency; CPP, central precocious puberty.

Discussion

The present study reported the clinical characteristics, MRI findings, and treatment response of pediatric patients with RCC.

Growth impairment was the most prevalent symptom at presentation, whereas headaches and visual disturbances were less frequent. Previous studies reported that more than half of pediatric patients with RCCs had headaches; however, Müller *et al*. reported that growth retardation was the most frequent (57%) and headaches were less frequent (23%) (1, 16, 17). In patients with childhood-onset craniopharyngioma, which is considered part of the spectrum of cystic epithelial lesions of the sellar region like RCCs, headaches were present in approximately 50% of cases. Regarding the pituitary function, GH (75%), gonadotropins (40%), ACTH (25%), and TSH (25%) abnormalities were observed (18). Endocrine dysfunction is also assumed to be affected by inflammation, which is implicated in the pathogenesis of craniopharyngioma (1, 16). Furthermore, lymphocytic hypophysitis induces GHD (76%), gonadal dysfunction (32%), hypothyroidism (30%), and adrenal insufficiency (21%) in pediatric patients (19). Although somatotrophs are the most abundant cell population in the adenohypophysis, these findings suggest that inflammation might attenuate GH secretion. HPL was found in one patient with the most massive cyst. It is a primary endocrine dysfunction caused by compression of the pituitary stalk and subsequent dopamine transport impairment (4).

CPP was prevalent in girls in this study, similar to other reports (6, 16). Hypothalamic-pituitary organic lesions, infectious or granulomatous conditions, and brain insults can cause CPP (20). It seems that the paradoxical situation in which other adenohypophyseal hormone-secreting cell damage may be associated with excessive gonadotropin secretion. RCCs have been occasionally found in patients with CPP. It has not been unclear whether the findings are incidental or not; however, inflammation might affect the inhibitory pathways of GnRH (21). Notably, one patient (Case 5) developed hypogonadotropic hypogonadism without HPL after receiving GnRH treatment. Higher doses of radiation may be more likely to cause GnRH deficiency, whereas lower doses may lead to CPP. Chronic damage due to inflammation could lead to CPP, followed by hypogonadotropic hypogonadism.

The signal intensity of RCCs on T1WI and T2WI is variable, and high mucosal content is likely to show as hyperintense on T1WI and hypointense T2WI (22). The association between T1WI signal intensity and endocrine dysfunction has been debated. Hypopituitarism was associated with T1WI iso-or hyper-intensity in prior studies, but not with cyst size (3, 23). The high mucosal content is thought to induce an inflammatory response that affects the surrounding tissue. Our results showed that six of ten patients (including two of three complex hypopituitarism patients) showed iso-or higher T1 signal intensity. Meanwhile, another study reported that the T1WI hypointensity/T2WI hyperintensity signal pattern was significantly more frequent in patients with endocrine disorders than in those without (7). Our results imply an association between T1WI iso- or hyper-signal intensity and endocrine dysfunction, but are not definitive. A further accumulation of cases is needed to clarify the relationship between endocrinopathy and signal intensity. Some reports indicate that the majority of RCCs are stable in size and that some RCCs may experience change; however, a retrospective RCC study reported that the predicted mean cystic growth rate was not significantly different from zero (24–26). Moreover, the spontaneous regression of RCCs is often accompanied by recovery from hypopituitarism (27). Likewise, one of our patients experienced regression of the cyst and height gain. Although most catch-up growth in those with SGA generally occurs by three years of age, it may also be contributed to by involution. Although RCCs typically remain stable in size, radiology and endocrine follow-up are warranted.

In this study, rGH therapy elevated the height SD scores and no cyst growth was observed during the

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treatment period. Similarly, GnRH analog therapy was also effective. A previous study reported that the treatment results for CPP and GHD with and without RCCs were comparable; however, the follow-up period was one year (7). The patient who underwent surgery experienced relief from headaches and visual disturbance; however, most endocrine dysfunctions remained. The majority of the headaches and visual problems were ameliorated postoperatively; however, anterior pituitary function was resolved by less than 30% (4, 28). Extensive resection of the cyst wall may increase the risk of cerebrospinal fluid leakage and hypopituitarism, including permanent CDI (28, 29). Moreover, recurrence of RCCs occurs in 0–48% of cases (1). Hormonal treatment is reasonable for relatively small RCCs, given the potential for spontaneous regression, irreversible pituitary damage, and recurrent risk.

This study has several limitations. First, it is controversial whether the presenting symptoms were attributable to cysts or incidentalomas because some cyst sizes were relatively small. Although patients with suspected endocrinopathy are more likely to undergo MRI and RCCs could be the most common incidentaloma, a recent study showed that nearly one in 10 patients with endocrine-related disease had RCC, whose size was similar to our results (median 5.3 mm, range 1–13.4 mm) (6, 8). Second, pars intermedia cysts, which can also form along the posterior margin of the Rathke's pouch, are typically observed to be less than 3 mm with overlapping features (30). We recognized non-neoplastic lesions at the midline of the anterior and posterior regions as RCCs in this study because RCCs arise from the pars intermedia (31). Third, the study design was retrospective in nature.

In conclusion, growth impairment and precocious puberty were the major symptoms in pediatric patients with relatively small RCCs and would be better served by hormonal therapy. Imaging and endocrine follow-ups are warranted because of the potential for changes in the cyst size and hormonal changes.

Conflict of interests: The authors declare that they have no competing interests.

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References

1. Trifanescu R, Ansorge O, Wass JAH, Grossman AB, Karavitaki N. Rathke's cleft cysts. Clin Endocrinol (Oxf) 2012;76: 151–60. [Medline] [CrossRef]
2. 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. [Medline] [CrossRef]
3. Nishioka H, Haraoka J, Izawa H, Ikeda Y. Magnetic resonance imaging, clinical manifestations, and management of Rathke's cleft cyst. Clin Endocrinol (Oxf) 2006;64: 184–8. [Medline] [CrossRef]
4. 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. [Medline] [CrossRef]
5. Takanashi J, Tada H, Barkovich AJ, Saeki N, Kohno Y. Pituitary cysts in childhood evaluated by MR imaging. AJNR Am J Neuroradiol 2005;26: 2144–7. [Medline]
6. Güneş A, Özbal Güneş S. The neuroimaging features of Rathke's cleft cysts in children with endocrine-related diseases. Diagn Interv Radiol 2020;26: 61–7. [Medline] [CrossRef]
7. Oh YJ, Park HK, Yang S, Song JH, Hwang IT. Clinical and radiological findings of incidental Rathke's cleft cysts in children and adolescents. Ann Pediatr Endocrinol Metab 2014;19: 20–6. [Medline] [CrossRef]
8. Shareef M, Nasrallah MP, AlArab N, Atweh LA, Zadeh C, Hourani R. Pituitary incidentalomas in paediatric population: Incidence and characteristics. Clin Endocrinol (Oxf) 2021;94: 269–76. [Medline]
9. Japanese children and adolescents body index calculator. Japanese Soc Pediatr Endocrinol Japanese Assoc Hum Auxol [cited 2020 Jun 1]; Available from: http://jspe.umin.jp/taikakushisu_v3.3.xlsx.
10. Isojima T, Kato N, Ito Y, Kanzaki S, Murata M. Growth standard charts for Japanese children with mean and standard deviation (SD) values based on the year 2000 national survey. Clin Pediatr Endocrinol 2016;25: 71–6. [Medline] [CrossRef]
11. Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. Arch Dis Child 1970;45: 13–23. [Medline] [CrossRef]
12. Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child 1969;44: 291–303. [Medline] [CrossRef]
13. Isojima T, Shimatsu A, Yokoya S, Chihara K, Tanaka T, Hizuka N, *et al*. Standardized centile curves and reference intervals of serum insulin-like growth factor-I (IGF-I) levels in a normal Japanese population using the LMS method. Endocr J 2012;59: 771–80. [Medline] [CrossRef]
14. Harrington J, Palmert MR, Hamilton J. Use of local data to enhance uptake of published recommendations: an example from the diagnostic evaluation of precocious puberty. Arch Dis Child 2014;99: 15–20. [Medline] [CrossRef]
15. The Japanese Association for Human Auxology and The Japanese Society for Pediatric Endocrinology. Atlas of Japanese Standard of TW2 Skeletal Maturity. Medical Review Co., Ltd., 2018.
16. Zada G, Ditty B, McNatt SA, McComb JG, Krieger MD. Surgical treatment of rathke cleft cysts in children. Neurosurgery

2009;64: 1132–7, author reply 1037–8. [Medline] [CrossRef]
17. Müller HL, Gebhardt U, Faldum A, Warmuth-Metz M, Pietsch T, Pohl F, *et al*. Kraniopharyngeom 2000 Study Committee. Xanthogranuloma, Rathke's cyst, and childhood craniopharyngioma: results of prospective multinational studies of children and adolescents with rare sellar malformations. J Clin Endocrinol Metab 2012;97: 3935–43. [Medline] [CrossRef]
18. Müller HL, Merchant TE, Puget S, Martinez-Barbera JP. New outlook on the diagnosis, treatment and follow-up of childhood-onset craniopharyngioma. Nat Rev Endocrinol 2017;13: 299–312. [Medline] [CrossRef]
19. Kalra AA, Riel-Romero RMS, Gonzalez-Toledo E. Lymphocytic hypophysitis in children: a novel presentation and literature review. J Child Neurol 2011;26: 87–94. [Medline] [CrossRef]
20. Latronico AC, Brito VN, Carel JC. Causes, diagnosis, and treatment of central precocious puberty. Lancet Diabetes Endocrinol 2016;4: 265–74. [Medline] [CrossRef]
21. Monzavi R, Kelly DF, Geffner ME. Rathke's cleft cyst in two girls with precocious puberty. J Pediatr Endocrinol Metab 2004;17: 781–5. [Medline] [CrossRef]
22. Bonneville F, Cattin F, Marsot-Dupuch K, Dormont D, Bonneville JF, Chiras J. T1 signal hyperintensity in the sellar region: spectrum of findings. Radiographics 2006;26: 93–113. [Medline] [CrossRef]
23. 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. [Medline] [CrossRef]
24. Jung JE, Jin J, Jung MK, Kwon A, Chae HW, Kim DH, *et al*. Clinical manifestations of Rathke's cleft cysts and their natural progression during 2 years in children and adolescents. Ann Pediatr Endocrinol Metab 2017;22: 164–9. [Medline] [CrossRef]
25. Barkhoudarian G, Palejwala SK, Ansari S, Eisenberg AA, Huang X, Griffiths CF, *et al*. Rathke's cleft cysts: a 6-year experience of surgery vs. observation with comparative volumetric analysis. Pituitary 2019;22: 362–71. [Medline] [CrossRef]
26. Culver SA, Grober Y, Ornan DA, Patrie JT, Oldfield EH, Jane JA Jr, *et al*. A case for conservative management: characterizing the natural history of radiographically diagnosed Rathke cleft cysts. J Clin Endocrinol Metab 2015;100: 3943–8. [Medline] [CrossRef]
27. 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. [Medline] [CrossRef]
28. 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. [Medline] [CrossRef]
29. Laws ER, Kanter AS. Rathke cleft cysts. J Neurosurg 2004;101: 571–2, discussion 572. [Medline] [CrossRef]
30. Delman BN. Imaging of pediatric pituitary abnormalities. Endocrinol Metab Clin North Am 2009;38: 673–98. [Medline] [CrossRef]
31. McKinney AM. Atlas of Normal Imaging Variations of the Brain, Skull, and Craniocervical Vasculature. Cham, Springer International Publishing, 2017.

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