



Review Article

Rathke cleft cyst apoplexy: Hormonal and clinical presentation

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Received : 18 April 2021

Accepted : 01 July 2021

Published : 06 October 2021

DOI

10.25259/SNI_382_2021

Quick Response Code:



ABSTRACT

Background: Rathke cleft cyst (RCC) apoplexy is an uncommon type of lesion that is challenging to diagnose without histopathological samples. Very few articles have been published describing the details of RCC apoplexy. We studied a good number of published articles to analyze its demographics, clinical and hormonal presentations, and outcomes.

Methods: A literature review of English language publications about RCC apoplexy or pituitary apoplexy was conducted using Medline and EMBASE search engines. Thirty articles with histological diagnosis of RCC apoplexy were identified, the earliest of which was published in 1990 and the latest in 2019. We combined the findings of these articles with our own case report and then compared the demographics, clinical and hormonal presentations, and outcomes between RCC apoplexy and pituitary adenoma apoplexy.

Results: Our data included 29 patients with RCC, with a mean age of 36.87 years (8–72) and a predominance of female patients (68%). The hemorrhagic type was most common, reported in 86%. Headache was the most common presenting symptom, being reported in 93% followed by hypogonadism (73%) and hormonal deficits (52%). All but three patients improved neurologically (90%); however, 45% of patients required long-term hormonal replacement, mostly thyroid hormone. No cases of worsening neurological or hormonal status were reported.

Conclusion: RCC apoplexy presents with less severe neurological and hormonal abnormalities than pituitary adenoma apoplexy; it also has a better prognosis in endocrine functional recovery. We recommend applying current management guidelines of pituitary adenoma apoplexy to RCC apoplexy.

Keywords: Rathke cyst, Apoplexy, Surgery, Pituitary

INTRODUCTION

Rathke cleft cyst (RCC) is an epithelium-lined benign congenital cystic lesion found in the sellar or suprasellar area. RCC originates from the craniopharyngeal duct (Rathke's pouch).^[21,23] Such lesions are more common among females, with a male-to-female ratio of 1:1.5.^[11,20,21] Frequently, RCC is asymptomatic and found in 20% (out of 1000 samples) of postmortem examinations; however, it is prevalent in 2–9% of those who underwent transsphenoidal surgery due to symptomatic sellar mass.^[15,20,22] If symptomatic, they usually present in adulthood between the 2nd and 6th decades.^[11,15,22] RCC apoplexy is a rare event with scarce case reports published in the

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literature. We present a case of RCC apoplexy in a 23-year-old man; additionally, we reviewed the literature to study the common demographic profile, clinical and hormonal presentation, and outcome.

MATERIALS AND METHODS

A literature review in Medline, EMBASE, Web of Science, and Scopus was used to identify existing case reports. The search included the following terms: “Rathke’s cleft cyst” AND “Apoplexy” OR “Pituitary apoplexy” OR “Hemorrhagic apoplexy” OR “Nonhemorrhagic apoplexy.” We included all articles with a histological confirmation of RCC apoplexy in any age group, and we excluded non-English articles (two articles). The earliest article found was published in 1990.

The items of interest in each article were patient demographics, clinical presentation, presenting hormonal profile, hemorrhagic versus nonhemorrhagic RCC apoplexy, clinical outcome, and hormonal outcome. Improvement was recorded based on clinical signs and symptoms after management. The database was last searched in February 2020. The article published by Chaiban *et al.*^[8] did not have individual patient information; rather, patients’ data were combined into frequency, average, and percentage. Kleinschmidt-DeMasters *et al.*^[10] were excluded from the analysis due to unknown hormonal status at presentation and in the final outcome. The decreased level of consciousness was defined as a Glasgow Coma Scale $\leq 14/15$. Headache, visual acuity or field deficits, and diplopia were measured subjectively based on history and physical examination. We added one patient treated at our medical center to the overall data [Figures 1-6].

RESULTS

We identified 29 patients in articles published between 1990 and 2019 [Table 1]. The average age was 36.87 (8–72), with 20 female patients (68%). Of the 29 patients, 25 had hemorrhagic RCC apoplexy (86%) [Table 2]. The most common clinical presentation was headache in 27/29 patients (93%), followed by visual deficits in 9/29 (31%), diplopia in 3/29 (10%), and altered mental status in 3/29 (10%). Of the 15/29 patients (52%) who presented with hormonal deficits, 11 had hypogonadism (73%), followed by hypocortisolism in 9 patients (60%), hypothyroidism in 7 patients (47%), high prolactin in 6 patients (40%), and low sodium in 2 patients (13%) [Figure 7].

Of the 29 patients in this series, 15 improved without hormonal replacement therapy (52%), 11 improved with continued hormonal replacement therapy (38%), 1 was stable without hormonal replacement therapy (3%), and 2 were stable with continued hormonal replacement therapy (7%)

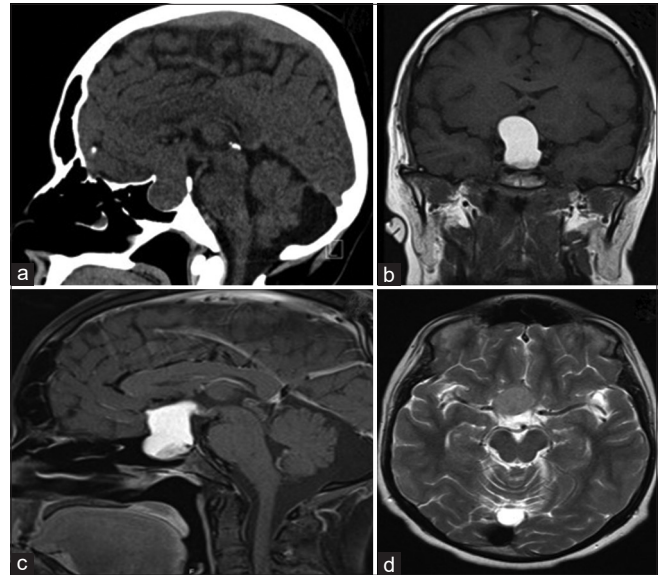


Figure 1: A 23-year-old man who presented with progressive headache, bitemporal hemianopsia, polydipsia and polyuria, erectile dysfunction, cold intolerance, and constipation for 6 months. Hormonal profile showed low thyroid-stimulating hormone, follicle-stimulating hormone, luteinizing hormone, and testosterone. Sagittal CT scan of the brain demonstrates a large sellar and suprasellar isodense mass with the expansion of the sellar floor (a). Coronal MRI T1 without contrast shows a hyperintense sellar and suprasellar mass compression the third ventricle inferiorly (b). Sagittal MRI T1 with contrast demonstrates a similar hyperintense pattern of the lesion (c). Axial MRI T2 demonstrates an isointense pattern of the lesion and abutting the right clinoid segment of the internal carotid artery (d).

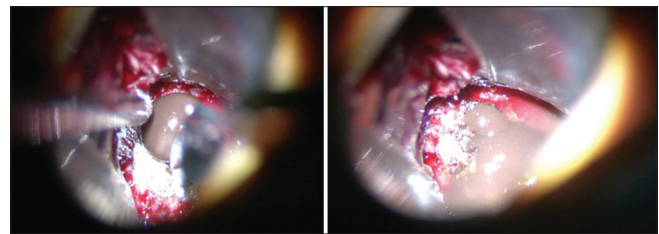


Figure 2: Intraoperative view with grayish necrotic material and hemosiderin after a dural incision was made.

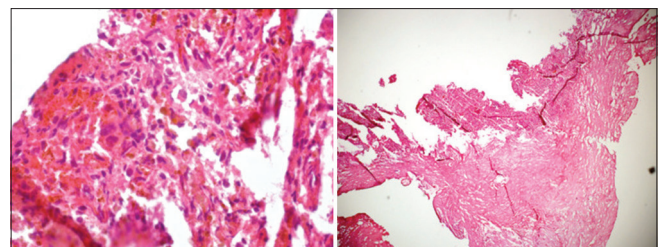


Figure 3: Frozen section with higher (left) and lower (right) magnification showing necrosis and hemorrhage with hemosiderin-laden macrophages.

[Table 3]. Of the 13 patients who are dependent on hormonal replacement therapy, 11 had hemorrhagic RCC apoplexy (85%). The most common type of hormonal replacement therapy postmanagement was thyroid hormone in 7/13 (54%), followed by gonadal hormone replacement such as testosterone in 5/13 (38%), desmopressin due to diabetes

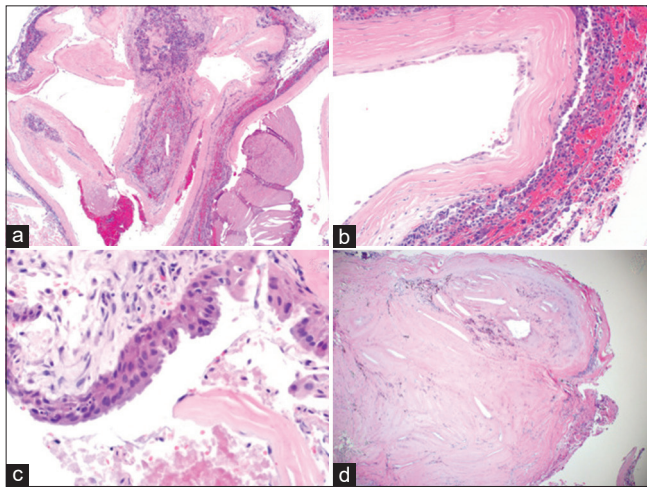


Figure 4: Permanent section showing cystic lesion with fibrous capsule surrounding nonneoplastic adenohypophysis (a). Higher magnification shows squamous epithelial metaplasia (b). Higher magnification of a different section shows a pseudostratified ciliated columnar epithelium (c). A lower magnification shows fibrosis with cholesterol cleft and old hemorrhage (d).

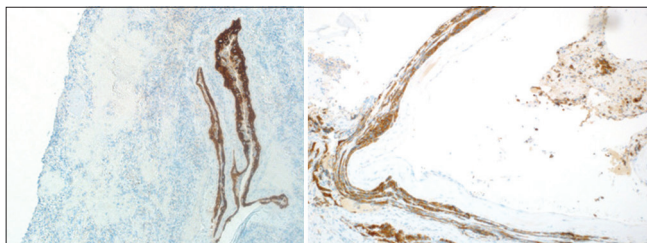


Figure 5: Special stains with cytokeratin 7 (left) and synaptophysin (right) showing normal adjacent pituitary gland tissue.

insipidus in 3/13 (23%), and corticosterone replacement in 2/13 (15%) [Figure 8].

DISCUSSION

RCC

RCC is an uncommon cystic lesion that is commonly asymptomatic. The prevalence of RCC among sellar lesions can range from 2% to as high as 7%.^[8,14] Patients who manifest clinical symptoms complain of headache (44–100%) and visual acuity and field deficits or diplopia (11–56%).^[5,9,14,16,22,23] Hormonal status abnormalities are present in 19–81% of newly diagnosed patients; the most commonly affected hypothalamic-pituitary axis is hyperprolactinemia, followed by hypogonadism, hypocortisolism, hypothyroidism, and diabetes insipidus.^[8,11,12,18,22] The most common location is combined sellar and suprasellar (87%), followed by sellar (13%), with a diameter between 1–2 cm (55%), 2–3 cm (35%), and >3 cm (10%).^[23]

Its appearance in MRI can be difficult to differentiate from other sellar lesions. Around 70% are hyperintense on T2 with little to no cystic wall enhancement on gadolinium administration.^[11,22] Cystic content can be discerned based on MRI signal intensity, however, such as water-like content with hypointensity on T1 and hyperintensity on T2, mucoid-like content appearing hyperintense on T1 and isointense on T2, or cystic hemorrhage appearing hyperintense on T1 and T2.^[11] A case series by Aho *et al.*^[2] showed that 31% of patients had progression of cyst size with resultant visual or endocrinologic deterioration if left untreated. Conversely, Amhaz *et al.*^[3] concluded that 31% of cysts spontaneously resolve without surgical management. Therefore, natural history may lean toward a benign course. Recurrence after surgical resection can be predicted if the histopathology shows epithelial squamous metaplasia (9–39% recurrence), contrary to the common columnar or cuboidal epithelial appearance.^[2,22] As many as, 24% of patients required long-term hormonal replacement postsurgery.^[9]

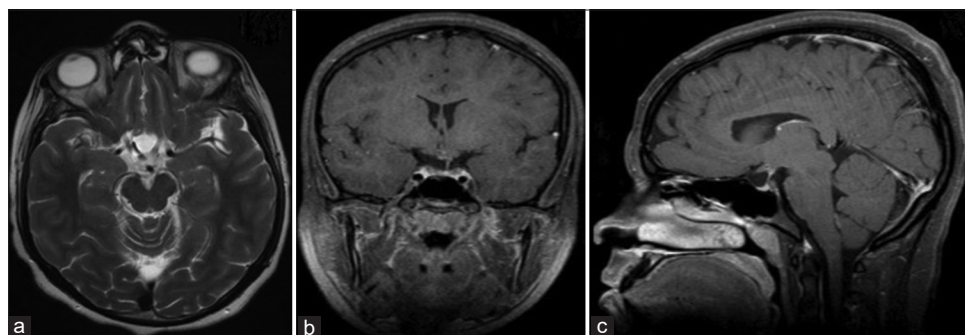


Figure 6: Postoperative axial MRI T2 (a), coronal T1 without contrast (b), and sagittal T1 with contrast (c) showing decompression of the sella and optic chiasm with drainage of the hemorrhagic cyst. Follow-up after 3 months of surgery demonstrated that the patient has complete improvement of the hormonal profile and visual field.

Table 1: Summary of cases from the literature.

Reference	Age/sex	Hemorrhage	Hormonal deficits	Clinical presentation	Management	Outcome
Current case	23 years/male	Yes	Low gonadotropin and thyroid hormones	Headache, visual acuity deficits, decreased libido, erectile dysfunction	Trans-sphenoidal surgery	Improved headache and vision, dependent on hormonal replacement (thyroid and testosterone)
Schooner <i>et al.</i> (2019) ^[12]	38 years/male	Yes	Low gonadotropin	Headache and visual field deficits	Trans-sphenoidal surgery	Improved
Schooner <i>et al.</i> (2019) ^[12]	33 years/female	Yes	Low gonadotropin, thyroid, and corticosteroids	Headache	Trans-sphenoidal surgery	Improved, except for hormonal replacement (thyroid)
Schooner <i>et al.</i> (2019) ^[12]	36 years/female	Yes	Low gonadotropin and corticosteroids	Headache	Trans-sphenoidal surgery	Improved
Schooner <i>et al.</i> (2019) ^[12]	37 years/female	Yes	Low gonadotropin and thyroid	Headache	Trans-sphenoidal surgery	Improved, except for hormonal replacement (thyroid)
Schooner <i>et al.</i> (2019) ^[12]	72 years/male	Yes	Panhypopituitarism	Headache, visual field deficits	Trans-sphenoidal surgery	Improved, except for hormonal replacement (thyroid, corticosteroids, and testosterone)
Schooner <i>et al.</i> (2019) ^[12]	49 years/male	Yes	Panhypopituitarism and low sodium	Headache	Trans-sphenoidal surgery	Stable, except for hormonal replacement (thyroid, corticosteroids, testosterone)
Kim <i>et al.</i> (2012) ^[24]	62 years/female	Yes	High prolactin, low thyroid and corticosteroid hormones, and low sodium	Headache	Trans-sphenoidal surgery	Improved, except hormonal replacement (thyroid)
Binning <i>et al.</i> (2008) ^[10]	20 years/male	No	Low gonadotropin hormone	Headache, nausea, vomiting, and diplopia	Trans-sphenoidal surgery	Improved, except for hormonal replacement (testosterone)
Binning <i>et al.</i> (2008) ^[10]	23 years/female	No	High prolactin	Headache, visual acuity deficit	Trans-sphenoidal surgery	Improved
Binning <i>et al.</i> (2008) ^[10]	49 years/male	No	None	Headache	Trans-sphenoidal surgery	Improved
Binning <i>et al.</i> (2008) ^[10]	21 years/female	No	Low thyroid hormone	Headache	Trans-sphenoidal surgery	Improved, except for hormonal replacement (thyroid)
Binning <i>et al.</i> (2008) ^[10]	24 years/female	Yes	None	Headache	Trans-sphenoidal surgery	Improved
Binning <i>et al.</i> (2008) ^[10]	54 years/female	Yes	None	Headache, visual acuity deficits, and meningism	Trans-sphenoidal surgery	Improved
Rosales <i>et al.</i> (2004) ^[25]	34 years/male	Yes	High prolactin and low thyroid hormone	Headache and diplopia	Trans-sphenoidal surgery	Improved, except for hormonal replacement (thyroid)

(Contd...)

Table 1: (Continued)

Reference	Age/sex	Hemorrhage	Hormonal deficits	Clinical presentation	Management	Outcome
Pawar <i>et al.</i> (2002) ^[26]	19 years/male	Yes	None	Headache, blurred vision	Trans-sphenoidal surgery	Improved
Kurisaka <i>et al.</i> (1998) ^[27]	8 years/female	Yes	None	Headache	Trans-sphenoidal surgery	Improved
Kleinschmidt-DeMasters <i>et al.</i> (1995) ^[8]	51 years/female	Yes	NA ^b	Visual acuity deficits	Trans-sphenoidal surgery	NA ^b
Onesti <i>et al.</i> (1990) ^[28]	25 years/female	Yes	None	Headache	Trans-sphenoidal surgery	Improved
Nishioka <i>et al.</i> (1990) ^[29]	46 years/female	Yes	None	Headache, visual acuity deficits	Trans-sphenoidal surgery	Improved

^aChaiban *et al.*^[7] didn't list the details of each patient, and thus, patients were not added in the table
^bThe information was not mentioned in the article

Table 2: Descriptive statistics of Preoperative findings.

Age (range) ^a	36.87 years (8-72)
Males, n (%)	11 (36%)
Females, n (%)	20 (64%)
Male to Female ratio	1:1.8
Hemorrhagic apoplexy, n (%)	25/29 (86%)
Non-hemorrhagic apoplexy, n (%)	4/29 (14%)
Hemorrhagic to Non-hemorrhagic apoplexy ratio	6.5:1
Headache, n (%)	27/29 (93%)
Visual deficits ^c , n (%)	9/29 (31%)
Diplopia, n (%)	3/29 (10%)
Decreased level of consciousness ^d , n (%)	3/29 (10%)
Hormonal deficits, n (%)	15/29 (52%)
Low gonadotropin, n (%)	11/15 (73%)
Low corticosteroid, n (%)	9/15 (60%)
High prolactin, n (%)	6/15 (40%)
Low thyroid, n (%)	7/15 (47%)
Low sodium, n (%)	2/15 (13%)

^aThe standard deviation wasn't calculated due to missing individual patients' age in Chaiban *et al.*^[7]

^bKleinschmidt-DeMasters *et al.*^[8] was not added in the analysis due to missing hormonal profile and outcome post-surgery

^cVisual deficits include acuity and field deficits

^dGCS of $\leq 14/15$

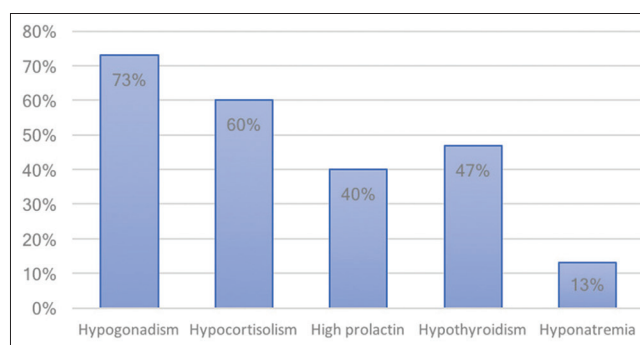
RCC apoplexy demographics and pathogenesis

RCC apoplexy is extremely rare, and only a few case reports have been published in the literature. The term RCC apoplexy was coined by Chaiban *et al.*^[8] who identified 20% occurrence of RCC apoplexy among all RCC patients. Pituitary adenoma apoplexy, commonly associated with null cell type, is more prevalent in males (65.5%), whereas RCC apoplexy, much like RCC, is more prevalent in females

Table 3: Descriptive statistics of Postoperative findings.

Improved, n (%)	15/29 (52%)
Improved with hormonal replacement therapy, n (%)	11/30 (38%)
Stable, n (%)	1/30 (3%)
Stable with hormonal replacement therapy, n (%)	2/30 (7%)
Hemorrhagic apoplexy with hormonal deficits, n (%)	11/30 (85%)
Non-hemorrhagic apoplexy with hormonal deficits, n (%)	2/13 (15%)
Thyroid replacement post-operation, n (%)	7/13 (54%)
Gonadal hormone replacement post-operation, n (%)	5/13 (38%)
Corticosteroid replacement post-operation, n (%)	2/13 (15%)
Desmopressin due to diabetes insipidus, n (%)	3/13 (23%)

^aAt last follow up

**Figure 7:** Presenting hormonal status.

(64%).^[4,19] The mean age of presentation in pituitary adenoma apoplexy is 50.9 years (15–91), while RCC apoplexy is 36.87 years (8–72), indicating a younger mean age of presentation in RCC apoplexy.^[19] The pathogenesis of

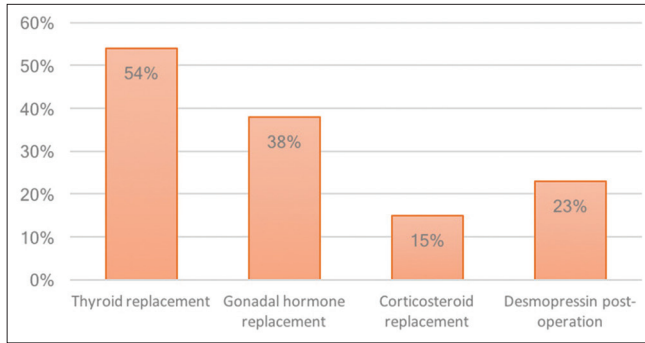


Figure 8: Postoperative hormonal replacement.

RCC apoplexy is still unknown; however, it can be attributed to the fragile epithelial wall vascular supply and the development of immature vascular endothelium from cystic wall granulation tissue.^[8,13] Nonhemorrhagic RCC apoplexy is hypothesized to cause neurological and hormonal deficits by the expanding infarcted tissue.^[5]

RCC clinical and hormonal presentation

The most common clinical presentation in RCC apoplexy is headache (93%), followed by visual acuity and field deficits (31%), diplopia (10%), and altered level of consciousness (10%). Pituitary adenoma apoplexy has a similar frequency of clinical presentation to RCC apoplexy, with headache being the most common (84–100%), followed by visual field deficits (34–70%), visual acuity deficits (56%), diplopia (45–57%), and decreased level of consciousness (13–30%).^[4,17]

From the literature review analysis, 52% of RCC apoplexy patients had hormonal abnormalities on presentation, compared to 70–80% of pituitary adenoma apoplexy patients.^[4] Therefore, patients with pituitary adenoma apoplexy have worse endocrinologic abnormalities than those with RCC apoplexy.^[8] Hypogonadism is the most common deficient hormone, occurring in 73% of patients, mainly translated in low testosterone level; low cortisol level is the second most common deficiency (60%), followed by low thyroid hormone (47%), hyperprolactinemia (40%), and hyponatremia (13%). Low cortisol level is the most common hormonal deficiency in pituitary adenoma apoplexy, occurring in 61% of patients, followed by low thyroid hormone (55%), low gonadotropin (40%), hyperprolactinemia (11%), and hyponatremia (< 5%).^[6,17] It is difficult to distinguish RCC apoplexy from pituitary adenoma apoplexy. Intraoperative diagnosis using histopathology is the gold standard method, showing hemosiderin, cholesterol crystals, and histiocytes, among others.^[16]

RCC clinical and hormonal outcome

The neurological outcome posttranssphenoidal surgery has improvement in 90% of cases, with 10% having stable

neurological deficits. Approximately 45% of patients required long-term hormonal replacement, with 38% improving neurologically. The neurological improvement rate posttranssphenoidal surgery in pituitary adenoma apoplexy is 53–89%; however, 58–83% of patients required long-term hormonal replacement – higher than for RCC apoplexy.^[4]

The most common long-term hormonal replacement in RCC apoplexy is thyroid hormone (54%), followed by testosterone replacement (38%), desmopressin for hyponatremia (23%), and cortisol replacement (15%). This is in contrast to pituitary adenoma apoplexy, with testosterone as the most common long-term hormonal replacement (63.6%), followed by thyroid replacement (62.7%), cortisol replacement (60%), and desmopressin (10%). In our case, the patient required long-term thyroid and testosterone replacement. The prevalence of hemorrhagic type RCC apoplexy is 86%, with 85% requiring long-term hormonal replacement.

Treatment guideline

No treatment protocol is designed for RCC apoplexy; however, we can apply the current management plan for pituitary adenoma apoplexy to RCC apoplexy. A review of management guidelines and outcomes of pituitary adenoma apoplexy recommended urgent transsphenoidal microscopic or endoscopic decompression of the RCC lesion for patients presenting with acute or progressive deterioration of their visual field or visual acuity; whereas, other clinical presentations, such as headache, mild decreased level of consciousness, or endocrine abnormalities, were not statistically significant in prompting urgent surgical intervention as compared to conservative management and a wait-and-see approach.^[1,7]

CONCLUSION

RCC apoplexy is a rare entity that is difficult to diagnose using standard radiological imaging. It has a higher female preponderance and commonly presents in a younger age group in comparison to pituitary adenoma apoplexy. Compared to pituitary adenoma apoplexy, the clinical and hormonal presentation is relatively less severe with a benign postoperative course. Although diabetes insipidus is comparatively more prominent in RCC apoplexy, it is recommended to apply pituitary adenoma apoplexy management guidelines to RCC apoplexy.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Elarjani T, Alhuthayl MR, Dababo M, Kanaan IN. Rathke cleft cyst apoplexy: Hormonal and clinical presentation. *Surg Neurol Int* 2021;12:504.