Case report

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Two collision sellar lesions: Rathke's cleft cyst with non-functional pituitary adenoma and Rathke's cleft cyst with plurihormonal adenoma

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Abstract

Introduction: Collision lesions of the sella involving pituitary adenomas and Rathke's cleft cysts (RCC) are considered a rare entity. Pre-operative diagnosis is challenging due to the variable MRI findings associated with RCC and similar clinical presentations as pituitary adenomas.

Case report: We report two collision lesions involving a non-functional pituitary adenoma with RCC and a plurihormonal adenoma producing prolactin and adrenocorticotrophic hormone with RCC. Both lesions were diagnosed with histopathological analysis and successfully treated with endoscopically-guided transnasal-transphenoidal surgery.

Conclusion: The diagnosis of pituitary adenomas with coexisting RCC should be considered when a cyst is visualized within, or adjacent to, pituitary adenomas detected by MRI, or when cystic contents with varying viscosities and color are discovered intraoperatively.

Keywords: Brain tumor, collision sellar lesions, rathke's cleft cyst, pituitary adenoma, prolactinoma

Introduction

Collision lesions refer to the uncommon merging of two histologically different pathological conditions from potentially different etiologies, including vascular, inflammatory, infectious and neoplastic [1]. Pre-operative diagnosis of collision sellar lesions is difficult due to their clinical and radiological resemblance to pituitary adenomas. For this reason, an accurate diagnosis is made with histopathological analysis. The underlying pathogenesis of collision sellar lesions is yet to be determined; however, common embryonic ancestries have been suggested as possible causal factors [2].

Noh *et al.*, identified 32 cases of co-existing pituitary adenoma and Rathke's cleft cyst (RCC) in 17 articles in 2007 [2]. Out of the 32 cases, 13 patients presented with a combination of RCC with prolactinomas and 5 patients had the combination of RCC with non-functional adenomas, 2 of whom presented with hypopituitarism. Five more collision lesions involving pituitary adenomas and RCCs have since been reported by Karavitaki *et al.*, [3], Koutourousiou *et al.*, [1], and Radhakrishnan *et al.*, [4]. Plurihormonal adenomas with concomitant RCC are extremely rare collision sellar lesions, of which only one true case has been reported in the English language [5]. We add to this body of literature by describing two cases of collision sellar lesions, one involving a non-functional pituitary adenoma with a RCC,

and the other a prolactin- and adrenocorticotrophic (ACTH)-secreting plurihormonal adenoma with a RCC.

Clinical series

Case 1

A 65-year-old African-American male with a past medical history of arteriosclerotic cardiovascular disease presented with complaints of decreased libido for 2 years. The patient noted minor visual changes, including blurred vision, over the past couple of years, but denied loss of vision and double vision. He denied headaches, nausea, vomiting, loss of consciousness, and dysarthria, as well as heat or cold intolerance. The patient's vital signs were normal. Neurologic examination was remarkable for mild bitemporal hemianopsia. The medical exam and the remainder of the neurologic exam were without focal deficits. Laboratory findings were significant for decreased testosterone, but prolactin levels were within normal limits.

Initial MR imaging with and without contrast of the brain revealed a large, heterogeneously enhancing mass arising from the sella turcica (Figure 1). The lesion extended superiorly into the suprasellar cistern, compressing and displacing the optic chiasm. It also extended laterally into the cavernous sinuses, intruding in the left sinus more than the right, and causing partial encasement of the cavernous portions of the internal carotid arteries. The overall size of

Figure 1. Precontrast axial T1-weighted MRI (A,B). Precontrast axial T2-weighted MRI (C,D). Postcontrast sagittal T1-weighted MRI (E,F). Postcontrast coronal T1-weighted MRI (G,H) image showing a large, heterogeneously enhancing mass arising from the sella turcica and extending superiorly into the suprasellar cistern, compressing the optic chiasm.

Figure 2. Postoperative day 1 postcontrast axial T1-weighted MRI (A). Precontrast sagittal T1-weighted MRI (B) showing absence of any residual mass. Three-month follow up axial FLAIR MRI (C). Postcontrast sagittal T1-weighted MRI (D) showing mild residual soft tissue density in the inferior aspect of the sella turcica.

the lesion was approximately 2.6 x 2.2 x 2.6 cm in transverse, anterior-posterior, and craniocaudal diameters. There was an irregular area of increased T1 signal intensity measuring 1.2 x 1.7 x 1.9 cm within the lesion just to the right of midline.

The patient underwent cardiac revascularization after thorough cardiac risk stratification and pre-operative assessment. An endoscopically-assisted transnasal-transsphenoidal (TNTS) surgery was successfully performed. The patient's dura was intact at the end of the case. The

Figure 3. Cyst of Patient 1 consisting of a single layer of columnar and ciliated cells x 100 (A) and x 400 (B).

post-operative FLAIR showed post-operative bleeding along the cerebral hemispheres at the level of the superolateral convexities in the parietal region bilaterally, as well as in the basal cisterns in the interpeduncular fossa cistern and the perimesencephalic cistern. This complication was managed conservatively without further complication. Post-operative CT angiogram of the cerebral vessels demonstrated no focal aneurysm. Immediate post-operative MRI, with and without contrast of the suprasellar cistern, displayed no sign of overt large residual mass and revealed almost complete obliteration of the soft tissue planes (Figure 2). The hemorrhage regressed by post-operative day 4 and the patient was discharged with only mild complaints of headache.

Histopathological evaluation reported two lesions. Frozen section analysis was undertaken on multiple fragments of tan, hemorrhagic mucoid tissue measuring 3.5 x 2.2 x 0.5 cm in aggregate. The mass consists of patternless sheets of uniform cells with basophilic or clear cytoplasm and relatively uniform, round to oval, and a delicate "salt-and-pepper" chromatin pattern. The mass lacks the acinar pattern of normal pituitary tissue. Immunohistochemistry analysis did not show immunoreaction to any pituitary hormones. The final pathological diagnosis was pituitary adenoma accompanied by RCC with columnar ciliated lining cells (Figure 3).

Follow-up non-contrast and contrast brain MRIs showed no residual tumor at 1-month, 2-month, and 6-month intervals. Six months after surgery, the patient stated that his libido had returned and his headaches were decreasing in severity and frequency.

Case 2

A 45-year-old Hispanic female, a practicing Jehovah's Witness whose faith prohibits blood transfusions, presented with intermittent sharp and pressure-like headaches for 6 months and galactorrhea for 1 year. There were multiple episodes of vomiting associated with the headaches and the patient denied having any history of headaches in the past. She also reported diplopia and decreased peripheral vision for 3 months. The patient's vital signs were normal and her BMI was 39.7. The medical and neurologic exams were normal. The patient did not display a cushingoid appearance. Laboratory findings showed elevated prolactin

Figure 4. Precontrast coronal T1-weighted MRI (A), postcontrast coronal T1-weighted MRI (B), precontrast sagittal T1-weighted MR (C), postcontrast sagittal T1-weighted MRI (D) showing an atypical heterogeneously enhancing appearing mass.

Figure 5. Postoperative day 1 precontrast coronal T1-weighted MRI (A), postcontrast coronal T1-weighted MRI (B), precontrast sagittal T1-weighted MRI (C), postcontrast sagittal T1-weighted MRI (D) showing good resection without evidence of residual tumor.

levels.

MR imaging of the sella demonstrated a pituitary macroadenoma measuring 0.7 x 1.3 x 1.8 cm in the transverse, anterior-posterior and craniocaudal diameters (Figure 4). This

Figure 6. Specimen A: tumor cells show multifocal, strongly Prolactin positivity and strongly ACTH positivity in some cells (A,C). Specimen B: tumor cells show multifocal, strongly Prolactin positivity and strongly ACTH positivity in some cells (B,D).

is seen on the right laterally along the sella turcica bordering the medial wall of the right cavernous carotid artery. Right infrasellar extension into the right sphenoid sinus was noted. This lesion measured 0.6 x 0.5 x 1.1 cm in the transverse, anterior-posterior and craniocaudal diameters. Contrast-enhancement helped delineate normal pituitary tissue and macroadenoma segments. No abnormal hyperintensities were seen on the axial FLAIR or T2 images intra-axially. The patient failed medical management with Cabergoline and wished for surgical resection of her pituitary tumor, with the condition that no blood transfusions would take place. The patient was amenable to the use of a cell saver.

An endoscopically-assisted TNTS surgery was successfully performed. Post-operative imaging showed satisfactory resection without evidence of residual tumor (Figure 5).

The pathological examination included a histologic and immunohistochemical analysis. Review of hematoxylin and eosin stains revealed small nests and a large fragment of neoplastic cells with a variable appearance. The tumor cells range from demonstrating small and round nuclei with dispersed chromatin to having large and irregular nuclei with prominent nucleoli. Some of the more atypical tumor cells had eccentric nuclei and contained eosinophilic cytoplasmic inclusions while other tumor cells had clear to pale eosinophilic cytoplasm. Mitoses were inconspicuous. Immunohistochemistry results revealed strong reactivity with ACTH in some cells and strong, multifocal reactivity with prolactin (Figure 6). The final pathological diagnosis was pituitary adenoma, immunoreactive for prolactin and ACTH (plurihormonal adenoma), accompanied by RCC with ciliated columnar epithelium and goblet cells (Figure 7).

The patient was discharged on postoperative day 2 without any complications, no clinical or laboratory evidence of diabetes insipidus, and no evidence of CSF leak. The patient is without complaints 6 months after surgery.

Table 1. Reported cases of collision sellar lesions involving a Rathke's Cleft Cyst (RCC) and pituitary adenoma.

No.	Author (ref)	Sex/Age	Clinical Presentation	MRI Findings (size, localization)	Histology	Surgical Results	Adjuvant Treatment	Follow-up (months)
2	Koutourousiou (1)	F/42	Cushing Disease	12 mm, intrasellar	ACTH adenoma + RCC	Remission of disease, hyocortisolism: substitution therapy	N/R	55
3	Koutourousiou (1)	M/76	Hypopituitarism	25 mm, intrasella/suprasellar/ parasellar	NFPA + RCC	Persisting hypopituitarism: substitution therapy	N/R	27
4	Noh (2)	F/62	GH tumor, Acromegaly	2.1 cm intrasella/suprasellar	GH pituitary adenoma + RCC	Remission of diabetes	N/R	6
1	Karavitaki (3)	M/54	Osteoporosis, decreased libido, ACTH Cushing syndrome	12 mm, suprasellar	Corticotroph adenoma + RCC	Persistent panhypopituitarism	Replacement therap: hydrocortisone, thyroxine, desmopressin	60
6	Ikeda (5)	M/50	Loss of libido, acromegaly	1 cm, suprasellar	Plurihormonal adenoma + RCC	N/R	N/R	24
7	Nishio (10)	F/21	Amenorrhea, galactorrhea, headache	8 mm	Pituitary adenoma + RCC	Successful pregnancy 11 mo postop	N/R	N/R
8	Nishio (10)	F/23	Amenorrhea, galactorrhea	9 mm	Pituitary adenoma + RCC	Decreased galactorrhea, menses resumed	N/R	N/R
9	Nishio (10)	F/24	Amenorrhea, galactorrhea	8 mm	Pituitary adenoma + RCC	No galactorrhea, menses resumed	N/R	N/R
10	Nishio (10)	F/25	Amenorrhea, galactorrhea	9 mm	Pituitary adenoma + RCC	Menses resumed	N/R	N/R
11	Nishio (10)	F/29	Irregular menses	7 mm	Pituitary adenoma + RCC	Menses resumed	N/R	N/R
12	Nishio (10)	F/30	Amenorrhea, galactorrhea	8 mm	Pituitary adenoma + RCC	Menses resumed, no galactorrhea	N/R	N/R
13	Nishio (10)	F/31	Amenorrhea, galactorrhea, headache	12 mm	Pituitary adenoma + RCC	Menses resumed, no galactorrhea	N/R	N/R
14	Nishio (10)	M/31	Galactorrhea, decreased libido	10 mm	Pituitary adenoma + RCC	No galactorrhea, wife successful pregnancy	N/R	N/R
15	Nishio (10)	F/34	Lethargy, amenorrhea, galactorrhea	12 mm	Pituitary adenoma + RCC	Amenorrhea persistent, no galactorrhea	N/R	N/R

GH = growth hormone

ACTH = adrenocorticotrophic hormone

NFPA = non-functioning pituitary adenoma

N/R = not reported

Discussion

In this report, we describe two cases of collision sellar tumors: a non-functional pituitary adenoma with a RCC and a prolactin- and ACTH-secreting plurihormonal adenoma with a RCC. The incidence of asymptomatic RCC in autopsy studies of normal pituitary glands ranges from 13-33% [6-9]. The coexistence of RCC with a pituitary adenoma, however, is rare. A 2010 study by Koutourousiou *et al.*, of 548 transsphenoidally resected pituitary adenomas reported 8 collision cysts (1.46%), 2 of which were RCC

combined with pituitary adenomas (0.36%) [1]. In 2007, Noh *et al.*, identified 4 RCCs in 782 (0.51%) patients [2] and in 1987, Nishio *et al.*, reported 9 RCC in 464 (1.9%) patients with a pituitary adenoma [10]. A summary of all reported collision sellar lesions involving RCCs with pituitary adenomas is provided in (Table 1). Plurihormonal adenomas are pituitary adenomas that produce more than one hormone. There is only one other report of a plurihormonal adenoma with associated RCCs [5]. This patient was a 50-year-old male who clinically presented with acromegaly and immunohistochemical studies revealed positive immunoreactivity for growth hormone, prolactin, and thyroid-stimulating hormone. Other cases of pituitary tumors with features transitional between those of anterior pituitary cells and RCCs (*e.g.*, secretory granules) have been reported as "transitional cell tumors" of the pituitary [11,12]. Coexisting sellar tumors are best diagnosed based on histological studies, as they share similar clinical and imaging characteristics as pituitary adenomas.

The differential diagnoses for incidental cysts in the sellar region include RCC, pituitary apoplexy, arachnoid cyst, cystic craniopharyngioma, and hemorrhagic pituitary adenoma [2]. Some characteristics that help differentiate

Craniopharyngiomas from RCC are their larger size and increased tendency to calcify [13]. Transitional cell tumors are thought to be developed from the wall of a RCC and should be kept in mind in patients with suspected RCC. Transitional cell pituitary tumors are composed of squamous metaplasia cells, mucus-producing cells and anterior lobe cells of the pituitary which display intercellular bridges, intracellular mucus droplets and intracytoplasmic secretory granules-features resembling the early developmental stage of the anterior pituitary gland [14]. Rathke's pouch, the remnant from which RCCs are derived, however, have a free surface of pseudostratified columnar epithelium and lack squamous metaplastic cells, which is more consistent with our pathological findings in both cases presented. RCCs vary in size from 3-40 mm and usually expand into the suprasellar cistern through the cleft of the diaphragm sellae [15]. When asymptomatic RCCs measure less than 3 mm, they are referred to as intermediate lobe cysts, which are colloid-filled cysts that stem from Rathke's pouch after degeneration of the pars intermedia (intermediate lobe) [16,17]. RCCs are commonly asymptomatic, but may present with headaches, visual impairment, and hypopituitarism, among other symptoms [18]. Patients with sellar collision lesions involving pituitary adenomas may also present with the same foregoing symptoms; however, these groups of patients are more likely to demonstrate hormonal symptoms from the adenoma [2]. Most symptoms stem from hypersecretory etiologies, the manifestations of which depend on the hormone secreted, while other hormonal symptoms may be attributed to varying degrees of pituitary insufficiency.

Traditionally, coexisting sellar pituitary adenomas and RCC were rarely diagnosed before surgery, but with the aid of MRI technology, the ability to detect RCC has greatly improved [19]. MRI features of RCC are variable, but this pathology should be suspected when lesions show the following characteristics: located in the intrasellar region or involving both intra and suprasellar regions, less than 1.5 cm in diameter, iso- or hyperintense on T1WI and no contrast enhancement [20,21]. In 2005, Binning *et al.*, reported a distinguishing MRI finding for RCCs in the form of intracystic nodules, which are more visible on T2WI [22]. Cysts with low signal intensity on T1WI may contain CSF-like fluid [2], while cysts with low protein content are typically isointense. As the cystic protein and mucopolysaccharide content rises, they become hyperintense on T1WI [23,24]. The cyst contents may have varying viscosities and color, ranging from clear and colorless to oily and milky [25].

Transnasal-transphenoidal surgery is an effective and safe surgical procedure for pituitary collision lesions. In a study of 8 patients with pituitary collision tumors, Koutourousiou *et al.*, reported recurrence of disease in 2 patients (25%, involving cases of non-functioning pituitary adenoma with sarcoidosis and GH adenoma with gangliocytoma) [1]. Five patients had undergone remission of their respective

endocrine disease (4 acromegaly and 1 Cushing's disease) with resolved clinical signs and symptoms. The final patient in the case series presented with panhypopituitarism, which persisted after surgical management. Although there was no recurrence of the collision sellar lesions involving RCCs in the aforementioned study, the literature reports a recurrence rate of 16–42% for isolated RCCs [26–28]. Factors that were found to be associated with RCC recurrence were the use of a fat and/or fascial graft for closure and the presence of squamous metaplasia in the cyst wall [1,26]. The extent of cyst wall resection on recurrence rates remains equivocal at this point. Thus it is important for surgeons to obtain postoperative follow-up neuroimaging studies on patients with RCCs, regardless of whether the cyst is isolated or exists as part of a collision sellar lesion. In situations where large RCC recur despite TNS drainage, a traditional transcranial approach may be considered for complete cyst wall resection. In scenarios where the pituitary adenoma invades the sphenoid sinus, such as in our second case, it is important to differentiate the source of the columnar epithelium forming the cyst wall-it may represent RCC or borders of the sphenoid sinus. During surgery the sphenoid sinus mucosa and epithelium were stripped off completely before the pituitary fossa dura was opened. The entire tumor and corresponding cyst were then removed intradurally, supporting the diagnosis of RCC with concomitant pituitary adenoma. It is critical to completely remove all adenomatous tissue and not simply resect the cystic elements from RCCs coexisting with pituitary adenomas.

RCCs are thought to be derived from remnants of Rathke's pouch [7], the primordium of the anterior and intermediate lobes of the pituitary gland that arises from the oral ectoderm and the floor of the developing diencephalon [29]. Inductive signaling acting on Rathke's pouch from the ventral diencephalon is responsible for initiating and regulating the combinatorial patterns of transcription factor gene expression that give rise to the anterior pituitary lobe [30].

In 1839 Martin H. Rathke reported the origination of the anterior lobe of the pituitary gland from an evagination of the stomodeal epithelium [31]. Recent evidence suggests that the anterior pituitary originates from the anterior neural ridge, which is anterior to the neural plate cells that give rise to the hypothalamus and posterior pituitary lobe [30]. While Rathke's pouch typically closes early in development, its apical extremity persists as a cleft between the anterior and posterior pituitary lobes [7]. This residual lumen that is left between the anterior pituitary lobe and pars intermedia is what constitutes Rathke's cleft. Rathke's cleft is lined with cuboidal or columnar epithelium and may include cilia and mucous goblet cells [7]. This cleft provides a prime setup for cyst development.

There is evidence to suggest a common ectodermal origin in the primitive craniopharyngeal duct for many

cystic epithelial lesions of the sellar region [32]. Cystic epithelial lesions may comprise varying entities along a continuum of pathology, with the benign RCCs at one end of the spectrum and at the other end are craniopharyngiomas, which are neoplastic lesions arising from squamous epithelial cell rests occurring in the region of the remnant hypophyseal/pharyngeal duct [33]. The papillary subtype of craniopharyngioma is thought to arise from metaplasia of the squamous epithelial cell rests found in the adenohypophysis and infundibulum [34], but studies examining pituitary specimens in rats reported finding epithelial craniopharyngeal derivatives consisting of cuboidal or columnar epithelium with goblet cells or stratified squamous epithelium [35]. Furthermore, other cases of ciliated epithelial cells or mucin-containing goblet cells occurring in squamous-papillary craniopharyngiomas have been reported to further support the theory for a common ectodermal origin between RCCs and craniopharyngiomas [36-38].

The pathogenesis of coexisting sellar lesions is still controversial. Some interesting theories have been proposed which are elucidated elsewhere [5,39,40]. There is evidence to suggest that pituitary adenomas rise from clonal expansion of mutated somatic cells in the anterior pituitary gland [41,42] and Rathke's pouch gives rise to the anterior pituitary gland. Hence, it is not unreasonable to consider a shared embryonic origin between RCC and pituitary adenoma. This genetic link may be clarified with further studies and future progress in newer molecular biology technology.

Conclusions

Pituitary adenomas may uncommonly present with coexisting RCC. This diagnosis should be considered when a cyst is visualized within or adjacent to pituitary adenomas detected by MRI, or when cystic contents with varying viscosities and color are discovered intraoperatively. Diagnosis may be confirmed by histologic evaluation and the treatment of choice is endoscopic transnasal-transphenoidal surgery.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

J.M.S. performed literature searches, collected the necessary clinical information, and wrote the manuscript. M.S.W. performed literature searches and prepared the manuscript. Z.P. gathered and analyzed pathology information. K.B. gathered and analyzed pathology information. E.S.S. gathered the necessary clinical information and prepared and reviewed the manuscript.

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