Pituitary Adenoma Coexistent with Sellar Clear Cell Meningioma Unattached to the Dura: Case Report and Treatment Considerations

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Abstract

Collision tumors involving the sella are rare. Intrasellar collision tumors are most commonly composed of a combination of pituitary adenomas and pituitary neuroendocrine tumors; however, collision tumors consisting of a pituitary adenoma and intrasellar meningioma are exceedingly rare. The authors present the case of a 47-year-old man who presented with progressive right eye vision loss. Magnetic resonance imaging showed a large, heterogeneously enhancing sellar mass with suprasellar extension. Using a transcranial approach with a right subfrontal craniotomy, near-total resection of the mass was achieved. Histologic analysis confirmed a diagnosis of a gonadotroph adenoma with concomitant clear cell meningioma (CCM). This patient was discharged with improvement in visual acuity and no signs of diabetes insipidus. Given the indistinguishable radiographic characteristics of pituitary adenoma and CCM, a preoperative diagnosis of a collision tumor was difficult. This case was uniquely challenging since the CCM component lacked the classic dural attachment that is associated with meningiomas on neuroimaging. CCMs are classified as central nervous system (CNS) World Health Organization (WHO) grade 2 tumors and tend to behave more aggressively, therefore warranting close surveillance for signs of tumor recurrence. This is the first case to report a collision tumor consisting of pituitary adenoma and CCM.

Keywords
- collision tumors
- meningioma
- pituitary adenoma
- sella

Introduction

Coexistent tumors of the sellar region are rare. A recent study that reviewed 16,283 autopsy and surgical pathology cases found that double or triple pituitary adenomas/pituitary neuroendocrine tumors (PitNETs) were the most frequent combination, comprising 40/232 (17.2%) of cases presenting with dual lesions.[1] The combination of meningioma and PitNET was one of the rarest combinations (6/232 [2.6%]), far behind PitNET + gangliocytoma (34/232 [14.7%) or PitNET + sellar metastasis (12/232 [5.2%]).[1] Individually, both PitNETs and sellar meningiomas can attain large sizes and cause similar symptoms due to compromise of the optic apparatus,

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making the diagnosis and treatment of coexisting sellar tumors, or "collision tumors," difficult.

Herein we present the case of a 47-year-old man who underwent a right frontal craniotomy for resection of a large sellar/suprasellar mass, found only after histological examination to be a dual PitNET and suprasellar meningioma. A transcranial surgical approach was performed due to the extent of suprasellar extension of the tumor and the relative narrowing hourglass deformity seen on magnetic resonance imaging (MRI) at the level of the diaphragma sella. It was only intraoperatively that two separate tumors were identified. Interestingly, intraoperative recognition of the suprasellar lesion as a meningioma was challenging due to the lack of typical dural attachment or vascular pedicle, as is seen for most meningiomas. Histological examination revealed two noncontiguous tumors, a clear cell meningioma, central nervous system (CNS) World Health Organization (WHO) grade 2, and a gonadotroph adenoma. The nondural attachment characteristic of the clear cell meningioma, as well as its occurrence in an atypical anatomical site for this meningioma type, is discussed in conjunction with review of the literature on meningioma and PitNET.

Case Description

History and Examination

The patient is a 47-year-old otherwise healthy man who presented to the optometrist with a 2-year history of progressive vision loss of the right eye. Formal visual acuity testing revealed sensitivity to hand-waving only, and an OS 20/60 with visual fields testing showing complete loss of vision in all four quadrants and sparing of the inferior nasal quadrant only in the left eye. An urgent MRI brain demonstrated a large homogeneously enhancing sellar mass with suprasellar extension (►**Fig. 1**). It was noted to cause significant mass effect on adjacent neural structures including the optic chiasm/nerves with significant cavernous sinus invasion. Following admission to the neurosurgery service, a complete pituitary workup was obtained. The data at the time were the following: cortisol, 8 µg/dL; adrenocorticotropic hormone (ACTH), 11.0 pg/mL; follicle stimulating hormone (FSH), 9.1 mIU/mL; luteinizing hormone (LH), 2.1 mIU/mL; prolactin (PRL), 11.1 ng/mL; free T4, 1.55 ng/dL; thyroid-stimulating hormone (TSH), 1.41 mIU/L; insulinlike growth factor 1 (IGF-1), 208 ng/mL.

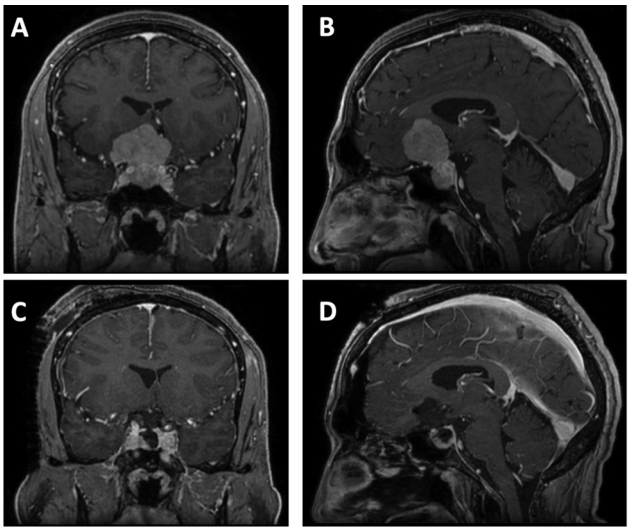

Fig. 1 (**A**,**C**) Coronal and (**B**,**D**) sagittal T1-weighted postcontrast brain magnetic resonance imaging (MRI). (**A**,**B**) Preoperative images demonstrating a heterogeneously enhancing mass ($3.8 \times 5.1 \times 3.5$ cm) expanding the sella turcica and extending superiorly into the suprasellar cisterns. Compression of optic apparatus is noted along with encasement of the paraclinoid internal carotid artery (ICA). A thin hyperintense layer of signal is seen between the intrasellar and suprasellar compartment. (**C**,**D**) Postoperative images illustrate the near total resection of the sellar and suprasellar mass with minimal residual enhancing tissue along the floor of the sella turcica.

Operation and Postoperative Course

Due to the radiographical evidence of significant suprasellar extension of the tumor toward the tuberculum sella along with a relative narrowing of the tumor at the level of the diaphragma sella, a transcranial approach was chosen over a transsphenoidal surgery. Although experienced with both the microscopic and endoscopic transnasal transsphenoidal approaches, we were concerned with our ability to safely access the suprasellar portion of the tumor, as well as the portion extending laterally over the right internal carotid artery. Therefore, the patient underwent a right frontal craniotomy and the dissection was carried out subfrontally to access the tumor. Intraoperative gross pathological findings revealed a well-circumscribed encapsulated suprasellar tumor that was dissected from of the brain parenchyma without obvious dural attachments. A frozen specimen section was suggestive of meningioma. As expected from preoperative imaging, it was significantly displacing the optic nerves and chiasm laterally and posteriorly (►**Fig. 2**). Following resection of the suprasellar lesion, the neurosurgeons were able to clearly visualize the pituitary stalk displaced to the left leading to the diaphragma sella. At this point, as no tumoral connection was seen between the resected suprasellar tumor and the diaphragma sella, a decision was made to open the diaphragma sella adjacent to the pituitary stalk on the right (►**Fig. 2**). At that point, intrasellar tumor was encountered, which was classic for a pituitary adenoma. Tumor was removed from within the sella using pituitary curettes through a transdiaphragmatic approach. Two separate tumor samples were sent for permanent pathology.

Histopathology

Microscopically, there were two distinct tumors. The meningioma showed numerous bands of collagen, with cells manifesting clear cytoplasm (►**Fig. 3A**). Immunohistochemical (IHC) staining for somatostatin receptor confirmed a meningothelial tumor (►**Fig. 3B**), while histochemical staining for periodic acid–Schiff (PAS) highlighted the glycogen-rich cytoplasm (►**Fig. 3C**, left). Diastase digestion proved the presence of glycogen by the absence of PAS staining after treatment, pathognomonic for clear cell meningioma (►**Fig. 3C**, right). The other, separate component of the

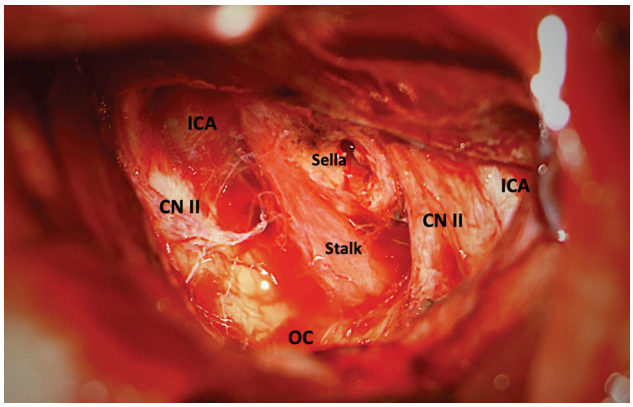

Fig. 2 Intraoperative image of the transcranial subfrontal approach illustrating the suprasellar region during tumor resection and the surrounding anatomy. Note the sella opening through which the pituitary adenoma was resected using ring curettes. CN II, cranial nerve II (optic nerve); ICA, internal carotid artery; OC, optic chiasm

resection showed pituitary adenoma/PitNET with patternless sheets of cells (►**Fig. 3D**), diffuse nuclear immunostaining for steroidogenic factor 1 (SF-1; ►**Fig. 3E**), and the typical patchy immunostaining for FSH (►**Fig. 3F**) indicative of gonadotroph adenoma/PitNET.

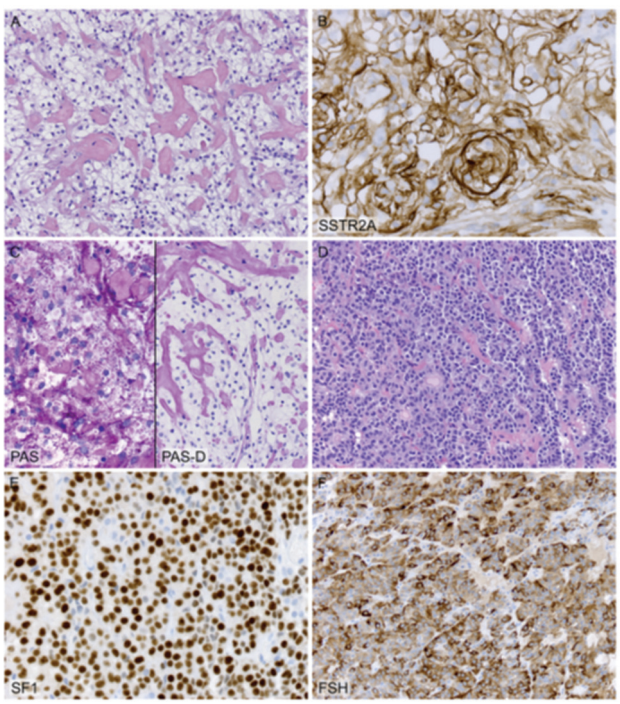

Fig. 3 Microscopically, there were two distinct tumors. (**A**) The meningioma showed numerous bands of collagen, with cells manifesting clear cytoplasm. (**B**) Immunohistochemical (IHC) staining for somatostatin receptor confirmed a meningothelial tumor, (**C**) while IHC staining for periodic acid–Schiff (PAS) highlighted the glycogen-rich cytoplasm. Diastase digestion proved the presence of glycogen by absence of PAS staining after digestion, which is pathognomonic for clear cell meningioma (**C**). (**D**) The other separate component of the resection showed pituitary adenoma/pituitary neuroendocrine tumors (PitNET) with patternless sheets of cells, (**E**) diffuse nuclear immunostaining for steroidogenic factor 1, and (**F**) the typical patchy immunostaining for follicle stimulating hormone, which is indicative of a gonadotroph adenoma.

Literature Search Strategy

A literature review was conducted using PubMed to identify available case reports and case series on collision tumors occurring in the sellar or suprasellar region of the brain from 1986 to 2022, linking "collision tumor" and "sella" or "suprasellar" or "parasellar" or "pituitary." The search regarding clear cell meningioma utilized the search terms "clear cell meningioma" and "without dural attachment." After independent evaluation for content and relevance, we identified a total of 30 articles published from the years 1986 to 2022 that describe collision tumors comprising PitNETs and various co-occurring tumors, and 6 articles published from 2009 to 2021 describing the diagnosis of clear cell meningioma without a dural attachment. The results of this literature review are shown in ►**Tables 1**–[1–30] and **2**.[31–37]

Discussion

Collision tumors affecting the sellar and suprasellar regions are rare. Due to indistinguishable radiographical characteristics, the preoperative diagnosis of a sellar collision tumor is difficult to differentiate from an isolated tumor type and is often preemptively diagnosed as a pituitary adenoma. Dual PitNETS are the most frequent type of sellar collision tumor, some of which are separate and some of which are contiguous.[1,38] However, the second most common combination of tumor types varies significantly. Secondary tumors that have been described as co-occurring with pituitary adenomas include gangliocytomas, pilocytic astrocytomas, craniopharyngiomas, spindle cell astrocytomas, pituicytomas, granular cell tumors, and Rathke's cleft cysts.[19–21,25] Collision tumors consisting of a pituitary adenoma and parasellar meningioma are exceedingly rare. There are several case reports and series describing the coexistence of a pituitary adenoma and sellar/suprasellar meningioma, although none have reported a specific diagnosis of clear cell meningioma.[1,2,4,11,12,14,17,18,23,26,28,30] Therefore, this case report represents the first description of a collision tumor consisting of a gonadotroph adenoma with a coexisting clear cell meningioma without a dural tail, the latter of which represented a significant diagnostic challenge.

Sellar or suprasellar meningiomas can closely mimic pituitary adenomas on neuroimaging.[39] Therefore, a definitive preoperative diagnosis of a pituitary adenoma and parasellar meningioma is not often possible on MRI. In considering our patient's preoperative MRI, in retrospect it may have been possible to appreciate a small layer of hyperintense signal on T1 postcontrast sequences between the intrasellar and suprasellar tumor components, which may have delineated the distinct border of both the adenoma and meningioma. However, this layer, representing the diaphragma sella, was not found to be completely contiguous on the sagittal MRI, and was therefore considered an unreliable measure for confirming the radiographical appearance of a collision tumor.

Definitive diagnosis of a collision tumor requires histologic evaluation of the resected tumor specimen by the

Table 1 Literature review of case reports and case series on sellar and suprasellar collision tumors consisting of pituitary adenomas with various secondary neoplasm

Study	Patient age (y)	Sex	Clinical presentation	Tumor type 1	Tumor type 2	Surgical approach	Additional comment
Yamada et al[2]	52	F	Headache with disturbances in visual acuity and galactorrhea	PA	Meningioma	Frontal osteoplastic craniotomy	Complete relief of headache and visual disturbance, with galactorrhea controlled with bromocriptine
Tajika et al[3]	56	F	Mild acromegaly	GH-secreting PA	Gangliocytoma	Transsphenoidal excision	N/A
Prevedello et al[4]	52	F	Continuous headache with right eye temporal visual field loss	PA	Meningioma	Endoscopic transsphenoidal excision	Postoperative resolution of headache, complete resolution of visual loss, and preservation of preoperative pituitary function
Karavitaki et al[5,a]	50 54	M M	Headache, sleep difficulties, decreased libido, hot flashes Proximal muscle weakness, central weight gain, back/hip pain, easy bruising, hypertension, decreased libido, erectile dysfunction, insomnia, and nocturia	Gonadotropic PA Corticotropic PA	Adamantinomatous craniopharyngioma Rathke's cleft cyst	Endoscopic transsphenoidal excision Endoscopic transsphenoidal excision	Postoperative hypogonadism requiring testosterone replacement therapy Postoperative diabetes insipidus and panhypopituitarism
Moshkin et al[6]	12	M	Incidental finding	Silent PA subtype 3	Adamantinomatous craniopharyngioma	Right craniotomy	N/A
Koutourousiou et al[7,a]	42 60 47 38 52 49 76 46	F M F M F F M F	Cushing's disease Hypogonadism and hyperprolactinemia Acromegaly and hypopituitarism Acromegaly, headache, and decreased libido Acromegaly Acromegaly, amenorrhea, and hyperprolactinemia Hypopituitarism Acromegaly and headache	ACTH-secreting PA Nonfunctioning PA GH-secreting PA GH-secreting PA GH-secreting PA GH-secreting PA Nonfunctioning PA GH-secreting PA	Rathke's cleft cyst Neurosarcoidosis Gangliocytoma Schwannoma Gangliocytoma Prolactinoma Rathke's cleft cyst Gangliocytoma	Transsphenoidal excision Transsphenoidal excision Transsphenoidal excision Transsphenoidal excision Transsphenoidal excision Transsphenoidal excision Transsphenoidal excision Transsphenoidal excision	Postoperative hypocortisolism Persistent hypogonadism and tumor recurrence requiring reoperation Persistent hypopituitarism N/A Tumor recurrence requiring somatostatin analog treatment and radiotherapy Required dopamine agonist for persistent hyperprolactinemia Required hormone

Table 1 (*Continued*)

Study	Patient age (y)	Sex	Clinical presentation	Tumor type 1	Tumor type 2	Surgical approach	Additional comment
							replacement therapy for persistent panhypopituitarism N/A
Rivera et al[8]	58	M	Rapid-onset diplopia	Prolactinoma	Plasmacytoma (multiple myeloma)	Stereotactic radiosurgery + cabergoline (prolactinoma) Nonoperative treatment regimen (plasmacytoma): Thalidomide Dexamethasone Pamidronate Bone marrow transplant	Postoperative secondary hypogonadism and hypothyroidism
Sahli et al[9]	74	M	Progressive neuro-ophthalmologic deterioration	Gonadotropic PA	Osteochondroma	Endoscopic transsphenoidal excision with adjuvant radiotherapy	Partial residual pituitary insufficiency with persistent FSH level elevation
Jin et al[10]	37	F	Intermittent left eye blurring and headache	PA	Craniopharyngioma	Transsphenoidal excision (primary tumor) Interhemispheric transcallosal approach (residual tumor)	Transient postoperative diabetes insipidus and hyponatremia
Mahvash et al[11]	36	F	Frontal headache with visual field disturbance in the right eye	PA	Meningioma	Endoscopic transsphenoidal excision	Gross total resection with sufficient decompression of the optic apparatus
Karsy et al[12]	70	F	Altered mental status, mutism, and incontinence	PA	Meningioma	Endoscopic transsphenoidal excision	Discharged home with permanent ventriculoperitoneal shunt and no significant neurological deficits
Matyja et al[13,a]	51 59 58 63	F M F F	Acromegaly and menstrual irregularities Headache and visual disturbances Headache and diplopia Acromegaly, headache, and sleep apnea syndrome	Somatotroph PA Somatotroph PA Somatotroph PA Somatotroph PA	Gangliocytoma Gangliocytoma Gangliocytoma Gangliocytoma	Transsphenoidal excision with adjuvant radiosurgery Endoscopic transsphenoidal excision Transsphenoidal excision Transsphenoidal excision	Persistent postoperative acromegaly with hypopituitarism N/A N/A N/A
Lim et al[14]	65	F	Visual disturbances and vertigo	Nonfunctioning PA	Meningioma	Endoscopic transsphenoidal excision	Improvement in visual symptoms with normal postoperative hormonal studies

(*Continued*)

Table 1 (*Continued*)

Study	Patient age (y)	Sex	Clinical presentation	Tumor type 1	Tumor type 2	Surgical approach	Additional comment
Ban et al[15]	74	M	Bilateral retro-orbital pain, left-sided ptosis, diplopia, headache, and nausea	FSH-secreting PA	DLBCL	Endoscopic transsphenoidal excision (PA) Chemotherapy (DLBCL)	N/A
Heng et al[16]	46	F	Headache and decrease in visual acuity	Nonfunctioning PA	Gangliocytoma	Endoscopic transsphenoidal excision	Required Gamma Knife radiosurgery due to tumor recurrence
Zhao et al[17,a]	58 58	F F	Acromegaly Acromegaly	GH-secreting PA GH-secreting PA	Meningioma Meningioma	Transsphenoidal excision (PA) Craniotomy (meningioma) Transsphenoidal excision (PA) Craniotomy (meningioma)	N/A N/A
Amirjamshidi et al[18,a]	37 42	F M	Oligomenorrhea, headache, diplopia, and progressive visual impairment Acromegaly, decreased visual acuity with bitemporal hemianopsia	Prolactinoma GH-secreting PA	Meningioma Meningioma	Right pterional craniotomy with tumor resection (meningioma) Cabergoline (PA) Transsphenoidal excision (failed) with subsequent right pterional craniotomy with tumor resection	Continuous postoperative treatment with cabergoline Postoperative improvement in vision with no evidence of tumor recurrence
Levitus and Charitou[19]	44	F	Incidental finding following head injury	GH-secreting PA	Gangliocytoma	Endoscopic transsphenoidal excision	Transient postoperative central adrenal insufficiency and permanent diabetes insipidus No tumor recurrence
Malli et al[20]	64	M	Bitemporal hemianopsia	Prolactinoma	Pilocytic astrocytoma	Endoscopic transsphenoidal excision with subfrontal craniotomy	N/A
Miyazaki et al[21]	48	M	Memory disturbance, depression, and hemiplegia	PA	Adamantinomatous craniopharyngioma	Transsphenoidal excision (PA) Left frontoparietal craniotomy with tumor resection and cyst drainage (craniopharyngioma)	Improvement in all symptoms
Snyder et al[22]	49	F	Headache, dizziness, blurred vision, and nausea	Corticotropic PA	Craniopharyngioma	Endoscopic transsphenoidal excision (primary tumor) Bifrontal interhemispheric	Postoperative CSF leak

Table 1 (*Continued*)

Study	Patient age (y)	Sex	Clinical presentation	Tumor type 1	Tumor type 2	Surgical approach	Additional comment
						transcallosal approach (residual tumor)	
de Vries et al[23]	75	F	Depression, fatigue, unintended weight loss	Nonfunctioning PA	Meningothelial meningioma	Extended endoscopic transplanum excision	Improvement of symptoms
Bteich et al[24]	35	M	Headache, progressive visual disturbance	Nonfunctioning PA	Papillary craniopharyngioma	Endoscopic transsphenoidal excision	N/A
de Almeida Verdolin et al[25,a]	Median = 60 (33–78)	3 F 2 M	Progressive visual field loss and/or headache	PA	Rathke's cleft cyst	Endoscopic transsphenoidal excision	N/A
Gezer et al[26]	34	F	Menstrual irregularities, proximal muscle weakness, and rapid weight gain	Corticotropic PA	Meningioma	Endoscopic transsphenoidal excision	Postoperative resolution of weight gain, menstrual irregularities, and proximal muscle weakness
Shareef et al[27]	60	M	Prior history of PA with resection, nonremitting bitemporal visual deficit	PA	Adamantinomatous craniopharyngioma	Endoscopic transsphenoidal excision	Transient postoperative diabetes insipidus
Bao et al[28,a]	62 56	F F	Progressive visual loss in left eye Headache with progressive bilateral visual loss	Nonfunctioning PA Nonfunctioning PA	Meningioma Meningioma	Endoscopic transsphenoidal excision Transmaxillary-transpterygoid approach	Improvement in visual acuity postoperatively
Ren et al[29]	41	M	Intermittent headache	Lactotroph PA	DLBCL	Endoscopic transsphenoidal excision (PA) Chemotherapy (DLBCL)	No tumor recurrence
Schöning et al[1,a]	Mean = 53.8 ± 18.5	N/A	N/A	Double PitNET ($n = 38$) Triple PitNET ($n = 2$) PitNET ($n = 34$) PitNET ($n = 6$) PitNET ($n = 5$) PitNET ($n = 12$)	Gangliocytoma Meningioma Posterior lobe tumor Metastasis	N/A	N/A
Lu et al[30]	61	F	Progressive decline of binocular vision	PA	Meningioma	Endoscopic transsphenoidal excision	Stable visual acuity without tumor recurrence

Abbreviations: ACTH, adrenocorticotropic hormone; DLBCL, diffuse large B-cell lymphoma; F, female; FSH, follicle stimulating hormone; GH, growth hormone; M, male; PA, pituitary adenoma; PitNET, pituitary neuroendocrine tumor.
[a]Case series

Table 2 Literature review of case reports on clear cell meningiomas without dural attachment

Study	Patient age (y)	Sex	Clinical presentation	Tumor location	Surgical approach	Additional comment
Miranda et al[31]	10	F	Neck pain, progressive right-sided hemiparesis, gait instability, somnolence, bilateral nystagmus, voice changes, lower cranial nerve dysfunction, left tongue/uvula deviation, (+) Hoffman's sign, and extensor plantar reflex	Craniocervical junction	Posterior fossa craniotomy, C1–C2 laminectomy with gross total resection	Complete recovery without signs of residual tumor
Ko et al[32]	34	F	Leg/hip pain with voiding difficulties	Cauda equina (L2–L3)	L1–L3 laminectomy with gross total resection	No evidence of tumor recurrence at 2 y
Yin et al[33]	55	M	Intermittent pulsatile headache, right eye vision loss + diplopia	Sella	Transsphenoidal approach with gross total resection	Local tumor recurrence at 4-mo follow-up requiring stereotactic radiosurgery
Gupta et al[34]	19	M	Back pain radiating to bilateral leg with diminished knee and ankle reflexes bilaterally	Lumbosacral spine (L5–S2)	L4–S2 laminectomy with gross total resection	Complete recovery without signs of residual tumor
Tsuchiya et al[35]	10	M	Worsening low back and bilateral lower extremity pain	Lumbar spine (L3)	L3 laminectomy with gross total resection	Complete recovery without signs of residual tumor
Zhang et al[36]	45	F	Episodic low back and bilateral upper leg pain	Lumbar spine (L3)	L3 laminectomy with gross total resection	Complete recovery without signs of residual tumor
Maamri et al[37]	58	F	Low back pain with bilateral sciatica	Lumbar spine (L3)	L2–L3 laminectomy with gross total resection	Complete recovery

pathologist, as was the case for our patient. While a diagnosis of a PitNET in the context of a sellar/suprasellar collision tumor in itself is relatively unsurprising, a concomitant diagnosis of clear cell meningioma represents a unique entity. Clear cell meningiomas have a proclivity for the cerebellopontine angle and spine, particularly in the cauda equina region, and tends to affect children and young adults, neither of which were the case in our patient (47-year-old, sellar region).[40] Clear cell meningiomas are unique in that both germline and somatic *SMARCE1* mutations are present, unlike other meningioma types. While the histopathologic diagnosis can be supported by a loss of nuclear *SMARCE1* expression by IHC, it is not required in the cases with archetypal histological features such as dense bands of collagen, glycogen-rich cytoplasm, and clear cell morphology, as in our case.[41] Importantly, clear cell meningiomas tend to exhibit more aggressive behavior, with higher rates of recurrence and occasional seeding of cerebrospinal fluid in comparison to other meningioma subtypes. As such, clear cell meningiomas have been designated a CNS WHO grade 2, thus warranting careful surveillance of our patient.

Few reports have described the diagnosis of a parasellar clear cell meningioma, all of which occurred as a single tumor.[33,42–46] Although more common in young adults and children, sellar region clear cell meningioma was diagnosed in 11- to 79-year-old patients.[33,42–46] Additionally, in all but one case, a dural attachment was seen,[42–46] while a single case described the occurrence of an intrasellar clear cell meningioma without a dural attachment.[33] Therefore, given the rarity of clear cell meningioma arising as a single-region tumor, this further emphasizes the diagnostic conundrum seen in our case where a gonadotropic adenoma was simultaneously diagnosed with a suprasellar clear cell meningioma without the classic dural attachment.

The association between pituitary adenomas and intracranial meningiomas has been widely described and are thought to arise as a delayed complication following radiotherapy for pituitary lesions.[47] However, collision tumors composed of simultaneously occurring pituitary adenoma and meningioma are difficult to explain, as the underlying etiology is not understood. One theory suggests that in patients with a growth hormone (GH) secreting macroadenoma, GH excess can induce meningioma growth resulting in collision tumor formation, although this has never been confirmed.[48,49] Nonetheless, the co-occurrence of a pituitary adenoma and parasellar meningioma is likely an incidental finding of two common lesions within the sellar region.

Conclusion

We describe the diagnosis and treatment of a collision tumor composed of a pituitary adenoma and sellar region meningioma in a 47-year-old-man. Collision tumors arising in the sellar/suprasellar region of the brain are exceedingly rare entities, currently with unclear etiologies. In the absence of reliable radiographic measures to diagnose collision tumors using neuroimaging, histological evaluation remains the gold standard. A multidisciplinary approach between neurosurgeons and neuropathologists is critical for the management of these patients.

Informed Consent

Informed consent was deemed unnecessary for this work by the Colorado Multiple Institutional Review Board.

Conflict of Interest

None declared.

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