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.

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. 1](A,B) Coronal and (B,D) sagittal T1-weighted postcontrast brain magnetic resonance imaging (MRI). (A,B) Preoperative images demonstrating a heterogeneously enhancing mass (3.8 × 5.1 × 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 paracns 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.
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.

**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. 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. 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. 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. 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...
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<th>Study</th>
<th>Patient age (y)</th>
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<tr>
<td>Yamada et al&lt;sup&gt;2&lt;/sup&gt;</td>
<td>52</td>
<td>F</td>
<td>Headache with disturbances in visual acuity and galactorrhea</td>
<td>PA</td>
<td>Meningioma</td>
<td>Frontal osteoplastic craniotomy</td>
<td>Complete relief of headache and visual disturbance, with galactorrhea controlled with bromocriptine</td>
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<td>Tajika et al&lt;sup&gt;3&lt;/sup&gt;</td>
<td>56</td>
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<td>Prevedello et al&lt;sup&gt;4&lt;/sup&gt;</td>
<td>52</td>
<td>F</td>
<td>Continuous headache with right eye temporal visual field loss</td>
<td>PA</td>
<td>Meningioma</td>
<td>Endoscopic transsphenoidal excision</td>
<td>Postoperative resolution of headache, complete resolution of visual loss, and preservation of preoperative pituitary function</td>
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<td>Karavitaki et al&lt;sup&gt;5,a&lt;/sup&gt;</td>
<td>50 54</td>
<td>M</td>
<td>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</td>
<td>Gonadotrophic PA</td>
<td>Adamantinomatous craniopharyngioma Rathke’s cleft cyst</td>
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<td>Moshkin et al&lt;sup&gt;6&lt;/sup&gt;</td>
<td>12</td>
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<td>Incidental finding</td>
<td>Silent PA subtype 3</td>
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<td>Koutouroussi et al&lt;sup&gt;7,a&lt;/sup&gt;</td>
<td>42 60 47 38 52 49 76 46</td>
<td>F M F M F M F</td>
<td>Cushing’s disease Hypogonadism and hyperprolactinemia Acromegaly and hypopituitarism Acromegaly, headache, and decreased libido Acromegaly, amenorrhea, and hyperprolactinemia Hypopituitarism Acromegaly and headache</td>
<td>ACTH-secreting PA Nonfunctioning PA GH-secreting PA</td>
<td>Rathke’s cleft cyst Neurosarcoi-dosis Gangliocytoma Schwan-noma Gangliocytoma Prolactinoma Rathke’s cleft cyst Gangliocytoma</td>
<td>Transsphenoidal excision</td>
<td>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</td>
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<td>Rivera et al⁸</td>
<td>58</td>
<td>M</td>
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<td>Sahli et al⁹</td>
<td>74</td>
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<td>Jin et al¹⁰</td>
<td>37</td>
<td>F</td>
<td>Intermittent left eye blurring and headache</td>
<td>PA</td>
<td>Craniopharyngioma</td>
<td>Transsphenoidal excision (primary tumor) Interhemispheric transcallosal approach (residual tumor)</td>
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<td>Mahvash et al¹¹</td>
<td>36</td>
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<td>Frontal headache with visual field disturbance in the right eye</td>
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<td>Endoscopic transsphenoidal excision</td>
<td>Gross total resection with sufficient decompression of the optic apparatus</td>
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<td>Karsy et al¹²</td>
<td>70</td>
<td>F</td>
<td>Altered mental status, mutism, and incontinence</td>
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<td>Matyja et al¹³,¹⁴</td>
<td>51, 59, 58, 63</td>
<td>F, M, F</td>
<td>Acromegaly and menstrual irregularities, Headache and visual disturbances, Headache and diplopia, Acromegaly, headache, and sleep apnea syndrome</td>
<td>Somatotroph PA, Somatotroph PA, Somatotroph PA, Somatotroph PA</td>
<td>Gangliocytoma, Gangliocytoma, Gangliocytoma, Gangliocytoma</td>
<td>Transsphenoidal excision with adjuvant radiosurgery, Endoscopic transsphenoidal excision, Transsphenoidal excision, Transsphenoidal excision</td>
<td>Persistent postoperative acromegaly with hypopituitarism, N/A, N/A, N/A</td>
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<td>Lim et al¹⁴</td>
<td>65</td>
<td>F</td>
<td>Visual disturbances and vertigo</td>
<td>Nonfunctioning PA</td>
<td>Meningioma</td>
<td>Endoscopic transsphenoidal excision</td>
<td>Improvement in visual symptoms with normal postoperative hormonal studies</td>
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<td>Ban et al15</td>
<td>74</td>
<td>M</td>
<td>Bilateral retro-orbital pain, left-sided ptosis, diplopia, headache, and nausea</td>
<td>FSH-secreting PA</td>
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<td>Endoscopic transsphenoidal excision (PA) Chemotherapy (DLBCL)</td>
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<td>Heng et al16</td>
<td>46</td>
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<td>Zhao et al17,a</td>
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<td>Amirjamsheidi et al18,a</td>
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<td>Oligomenorrhea, headache, diplopia, and progressive visual impairment Acromegaly, decreased visual acuity with bitemporal hemianopsia</td>
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<td>Levitus and Charitou19</td>
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<td>Incidental finding following head injury</td>
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<td>Transient postoperative central adrenal insufficiency and permanent diabetes insipidus No tumor recurrence</td>
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<td>Malli et al20</td>
<td>64</td>
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<td>Pilocytic astrocytoma</td>
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<td>Endoscopic transsphenoidal excision with subfrontal craniotomy</td>
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<td>Miyazaki et al21</td>
<td>48</td>
<td>M</td>
<td>Memory disturbance, depression, and hemiplegia</td>
<td>PA</td>
<td>Adamantinomatous craniopharyngioma</td>
<td>Transsphenoidal excision (PA) Left frontoparietal craniotomy with tumor resection and cyst drainage (craniopharyngioma)</td>
<td>Improvement in all symptoms</td>
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<td>Snyder et al22</td>
<td>49</td>
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<td>Headache, dizziness, blurred vision, and nausea</td>
<td>Corticotropic PA</td>
<td>Craniopharyngioma</td>
<td>Endoscopic transsphenoidal excision (primary tumor) Bifrontal inter-hemispheric</td>
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<td>de Vries et al&lt;sup&gt;23&lt;/sup&gt;</td>
<td>75</td>
<td>F</td>
<td>Depression, fatigue, unintended weight loss</td>
<td>Nonfunctioning PA</td>
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<td>Bteich et al&lt;sup&gt;24&lt;/sup&gt;</td>
<td>35</td>
<td>M</td>
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<td>N/A</td>
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<td>de Almeida Verdolín et al&lt;sup&gt;25,a&lt;/sup&gt;</td>
<td>Median = 60 (33–78)</td>
<td>3 F 2 M</td>
<td>Progressive visual field loss and/or headache</td>
<td>PA</td>
<td>Rathke's cleft cyst</td>
<td>Endoscopic transsphenoidal excision</td>
<td>N/A</td>
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<td>Gezer et al&lt;sup&gt;26&lt;/sup&gt;</td>
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<td>Menstrual irregularities, proximal muscle weakness, and rapid weight gain</td>
<td>Corticotropic PA</td>
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<td>Shareef et al&lt;sup&gt;27&lt;/sup&gt;</td>
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<td>M</td>
<td>Prior history of PA with resection, nonremitting bitemporal visual deficit</td>
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<td>Adamantinomatous craniopharyngioma</td>
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<td>Bao et al&lt;sup&gt;28,a&lt;/sup&gt;</td>
<td>62 56</td>
<td>F  F</td>
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<td>Nonfunctioning PA Nonfunctioning PA</td>
<td>Meningioma Meningioma</td>
<td>Endoscopic transsphenoidal excision Transmaxillary-transpterygoid approach</td>
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<td>Ren et al&lt;sup&gt;29&lt;/sup&gt;</td>
<td>41</td>
<td>M</td>
<td>Intermittent headache</td>
<td>Lactotroph PA</td>
<td>DLBCL</td>
<td>Endoscopic transsphenoidal excision (PA) Chemotherapy (DLBCL)</td>
<td>No tumor recurrence</td>
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<td>Schöning et al&lt;sup&gt;1,a&lt;/sup&gt;</td>
<td>Mean = 53.8 ± 18.5</td>
<td>N/A</td>
<td>N/A</td>
<td>Double PitNET (n = 38) Triple PitNET (n = 2) PitNET (n = 34) PitNET (n = 6) PitNET (n = 5) PitNET (n = 12)</td>
<td>Gangliocytoma Meningioma Posterior lobe tumor Metastasis</td>
<td>N/A</td>
<td>N/A</td>
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<td>Lu et al&lt;sup&gt;30&lt;/sup&gt;</td>
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<td>F</td>
<td>Progressive decline of binocular vision</td>
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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.

<sup>a</sup>Case series
Although more common in young adults and children, sellar region clear cell meningioma was diagnosed in 11- to 79-year-old patients.\textsuperscript{33,42–46} Additionally, in all but one case, a dural attachment was seen,\textsuperscript{42–46} while a single case described the occurrence of an intrasellar clear cell meningioma without a dural attachment.\textsuperscript{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 parasellar 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.\textsuperscript{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 macroadeno- noma, GH excess can induce meningioma growth resulting in collision tumor formation, although this has never been confirmed.\textsuperscript{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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