Collision Tumor Composed of Nonfunctioning Pituitary Adenoma and Meningioma in the Sellar Region: Report of a Case and Literature Review

Sachin Baldawa1 Abhay Raikhailkar2

1 Department of Neurosurgery, SS Baldawa Neurosciences and Women’s Care Hospital, Solapur, Maharashtra, India
2 Department of Pathology, SS Baldawa Neurosciences and Women’s Care Hospital, Solapur, Maharashtra, India

Abstract

Background The coexistence of two histologically distinct neoplasms in the same area without histological admixture or an intermediate cell population zone represents a rare tumor type called collision tumor. Collision tumor of pituitary adenoma and meningioma has been reported years later following irradiation to pituitary adenoma. However, collision tumor of pituitary adenoma and meningioma in absence of irradiation therapy is extremely uncommon.

Case Description We report an unusual case of collision tumor involving diaphragma sella meningioma and pituitary adenoma in a 50-year-old lady without prior radiation therapy. She presented with visual blurring and impaired field of vision. Her preoperative magnetic resonance imaging (MRI) was suggestive of pituitary adenoma. Total excision of the lesion was performed through endoscopic transsphenoidal route. Histological diagnosis was consistent with collision tumor of pituitary adenoma and meningioma.

Keywords ► collision tumor ► meningioma ► pituitary adenoma ► sellar region ► endoscopic transsphenoidal

Conclusion Collision tumor comprising of nonfunctioning pituitary adenoma and meningioma is extremely rare. Preoperative MRI may not always be able to distinguish these histologically distinct neoplasms. Hence, histopathological examination is necessary to establish the diagnosis. Endoscopic transsphenoidal approach may suffice in excision of these collision tumors. Close follow-up is necessary to detect tumor recurrence. Though the association of these tumors can be coincidental, casual relationship between the occurrence of collision tumors cannot be totally excluded.

Introduction

Collision tumor is an uncommon entity representing the coexistence of two histologically distinct neoplasms in the same area without histological admixture or an intermediate cell population zone.1 The term “collision tumor” needs to be distinguished from two other similar terms—“mixed tumor” and “coexisting tumor.” A mixed tumor represents two different neoplasms with histologically admixed cell types.1
Collision Tumor of Sellar Region  Baldawa, Raikhailkar

Coexisting tumor represents two different neoplasms existing in two different locations not abutting against each other. Meningioma and pituitary adenoma are two most common tumors of the central nervous system. Collision tumor of these two tumors in absence of previous radiation therapy is extremely uncommon. We report a rare case of “collision tumor” of nonfunctioning pituitary adenoma and diaphragma sella meningioma in a 50-year-old lady diagnosed on histological examination.

Illustrative Case

A 50-year-old lady without previous history of cranial irradiation was referred to our institution following complaints of bifrontal headache and progressive diminution of vision over 3 years’ duration. On physical examination she had marked reduction of visual acuity (6/18) and bitemporal hemianopia. Her blood hormonal levels of anterior pituitary were normal; prolactin (PRL) 6.5 ng/mL (normal range <25 ng/mL), growth hormone (GH) 0.1 ng/mL (normal range <10 ng/mL), insulin-like growth factor 1 (IGF-1) 84 ng/mL (normal range: 80–209 ng/mL), adrenocorticotropic hormone (ACTH) 18 pg/mL (normal range: 10–60 pg/mL), cortisol 21 mcg/dL (normal range: 5–25 mcg/dL), thyroid-stimulating hormone 2.4 UI/mL (normal range: 0.5–5 UI/mL), thyroxine 1.74 ng/dL (normal range: 0.8–1.8 ng/dL), follicle-stimulating hormone 3.5 mUI/mL (normal range: 2.7–21.5 mUI/mL), luteinizing hormone 1.9 mUI/mL (normal range: 1.1–11.6 mIU/mL), and testosterone 4.1 ng/mL (normal range: 2–70 ng/mL). Magnetic resonance imaging (MRI) of the sellar region performed on 3.0 tesla unit (Siemens Lumina) showed an isointense sellar mass on T1-weighted (T1W) imaging (3 cm × 3 cm × 2 cm) and isointense to hypointense mass on T2W imaging with homogenous contrast enhancement. The sellar component was extending in the suprasellar region causing chiasmatic compression. The suprasellar component was broad based and was reaching till the tuberculum sella (Fig. 1). Based on hormonal tests and radiological imaging, diagnosis of nonfunctioning pituitary adenoma was agreed upon. Endoscopic transsphenoidal resection of pituitary adenoma was performed. Intraoperatively, the sellar component was soft while the suprasellar component was firm with rich vascularity. Histological diagnosis confirmed the presence of pituitary adenoma in collision with fibrous meningioma (Fig. 2). Close follow-up of 1 year revealed no tumor recurrence.

Discussion

Pituitary adenomas are one of the most common sellar suprasellar lesions accounting for 10 to 15% of cases seen on cranial autopsy and 23% of cases seen on MRI. Meningiomas are one of the most frequent primary intracranial neoplasms accounting for 15 to 25% of all intracranial neoplasms. Both sellar meningioma and pituitary adenoma show female preponderance and manifest in adults. Coexistence of pituitary adenoma and other sellar mass like craniopharyngioma, gangliocytoma, schwannoma, and meningioma is extremely uncommon with very few studies published. The term “collision tumor” needs to be distinguished from similar terms “coexistent/concomitant tumor” and “mixed tumor.” “Coexistent/concomitant” tumors are two different tumors at two separate locations.

A “mixed tumor” represents neoplasm in which endocrine and nonendocrine components are strictly admixed with no shared border. Finz et al have reported a case of mixed pituitary adenoma/cranioopharyngioma in a 75-year-old woman and has reviewed five other papers of mixed pituitary adenoma/cranioopharyngioma reported in English literature. A “collision tumor” represents two histologically distinct primary neoplasms occurring in the same anatomic position with a shared border. The concurrent occurrence of collision tumor involving ganglioglioma and ependymoma and glioblastoma and meningioma have been reported.

Collision tumor involving meningioma and pituitary adenoma in the sellar region is extremely uncommon. Very few articles regarding collision tumors composed of pituitary adenoma and meningioma have been published. Table 1 depicts all cases of collision tumors of pituitary adenoma and meningioma. Articles with “mixed” or “coexistent” tumors of pituitary adenoma and meningioma have been excluded from the review. Honegger et al have reported three patients with “coexistent” pituitary adenoma and meningioma. In all three patients the pituitary adenoma and meningioma coexisted; however, both these tumors were never in close approximation to each other to strictly label them as collision tumors—the first patient had temporal pole meningioma, the second a falci meningioma, and the third parietal convexity meningioma.

The type of pituitary adenoma in a collision tumor may vary from nonfunctioning to functioning adenoma. Though prolactinomas represent the most common type of pituitary adenomas in adults, GH-secreting pituitary adenoma is most commonly found in concurrence with meningiomas as a component of collision tumor. Meningioma when coexist are generally found in proximity to pituitary adenoma—suprasellar, parasellar, and sphenoid wing location.

Amirjamshidi et al have reported two cases of coexisting pituitary adenoma and suprasellar meningioma. The authors believed these coexisting tumors to be coincidental and not collision tumors. Hence, these cases were not included in the table. Zentner and Gilsbach reported the first case of collision tumor of pituitary adenoma and meningioma in a 61-year-old lady which was resected transphenoidally as early as 1989. In 1984, Banik et al reported collision tumor of pituitary adenoma and meningioma and another adrenal gland collision tumor in a patient with MEN1 syndrome on postmortem examination. Karsy et al have reported the case study of a 70-year-old lady presenting with altered mental status, mutism, and incontinence. Radiological diagnosis was of pituitary adenoma. The tumor was resected through transphenoidal route. Pathological examination of the resected tumor revealed coexistent meningioma along with pituitary adenoma. Zhao et al have reported two patients with collision tumors of GH-secreting adenoma and meningioma. The GH-secreting adenoma was resected through transphenoidal route. Craniotomy was performed to excise the residual tumor.
which was diagnosed to be a meningioma.\textsuperscript{1} Gezer et al have reported a collision tumor of corticotroph-secreting pituitary adenoma and meningioma in a 34-year-old Caucasian lady. MRI revealed only tuberculum sella meningioma. Histopathological examination confirmed a corticotroph-secreting adenoma infiltrated by meningioma.\textsuperscript{5}

The diagnosis of collision tumor is most often based on histopathological examination. Preoperative MRI may
Table 1 Literature review of “collision tumor” of pituitary adenoma with meningioma isointense sellar mass on T1W imaging (3 cm × 3 cm × 2 cm), isointense to hypointense mass on T2W imaging with homogenous contrast enhancement

<table>
<thead>
<tr>
<th>No.</th>
<th>Author</th>
<th>Year</th>
<th>Age/sex</th>
<th>Presenting complaint</th>
<th>Collision type 1</th>
<th>Collision type 2</th>
<th>Radiological findings</th>
<th>Surgical approach</th>
<th>Intraoperative findings</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Present case</td>
<td>2023</td>
<td>50/F</td>
<td>Visual impairment</td>
<td>Nonfunctioning pituitary adenoma</td>
<td>Meningioma</td>
<td>Sellar lesion was isointense on T1W, iso-to hypointense on T2W with homogenous enhancement. Suprasellar component was broad based</td>
<td>Transsphenoidal</td>
<td>Sellar component was soft, suprasellar component was firm with rich vascularity</td>
<td>No recurrence 1 year</td>
</tr>
<tr>
<td>2</td>
<td>Zhao et al</td>
<td>2017</td>
<td>58/F</td>
<td>Acromegaly</td>
<td>GH-secreting adenoma</td>
<td>Meningioma</td>
<td>T1W-hyperintense T2W-isointense, heterogeneous enhancement</td>
<td>Transsphenoidal followed by craniotomy</td>
<td>Pituitary tumor was gray, soft, fragile, partly tough with rich blood supply. Meningioma was dark red, rubbery, vascular</td>
<td>Not available</td>
</tr>
<tr>
<td>3</td>
<td>Karsy et al</td>
<td>2015</td>
<td>70/F</td>
<td>Altered mental status, incontinence, mutism</td>
<td>Nonfunctioning pituitary adenoma</td>
<td>Meningioma</td>
<td>CT-isodense spherical mass MRI T1W-homogenous enhancement T2W-isointense mass</td>
<td>Transsphenoidal/ VP shunt for hydrocephalus</td>
<td>Not available</td>
<td>Not available</td>
</tr>
<tr>
<td>4</td>
<td>Gezer et al</td>
<td>2020</td>
<td>34/F</td>
<td>Cushing’s disease</td>
<td>ACTH secreting pituitary adenoma</td>
<td>Meningioma</td>
<td>Solid mass with well-defined margin in anterior part of gland suggestive of meningioma</td>
<td>Extended transsphenoidal</td>
<td>Not available</td>
<td>No recurrence 11 months</td>
</tr>
<tr>
<td>5</td>
<td>de Vries et al</td>
<td>2023</td>
<td>75/F</td>
<td>Hypopituitarism, bitemporal visual field deficits</td>
<td>Nonfunctioning pituitary adenoma</td>
<td>Meningioma</td>
<td>Sellar suprasellar lesion with dural tail, sphenoid sinus extension. Inhomogeneous intensity of tumor</td>
<td>Transsphenoidal</td>
<td>Suprasellar tumor was firm and completely separated from the sellar tumor by diaphragm sella</td>
<td>Not available</td>
</tr>
<tr>
<td>6</td>
<td>Ruiz-Juretschke et al</td>
<td>2015</td>
<td>61/F</td>
<td>Progressive visual loss, bitemporal hemianopia</td>
<td>Nonfunctioning pituitary adenoma</td>
<td>Meningioma</td>
<td>Homogenously enhancing sellar-suprasellar tumor</td>
<td>Transsphenoidal followed by redo surgery</td>
<td>Sellar component was soft while the suprasellar component was firm</td>
<td>Not available</td>
</tr>
</tbody>
</table>
Table 1 (Continued)

<table>
<thead>
<tr>
<th>No.</th>
<th>Author</th>
<th>Year</th>
<th>Age/sex</th>
<th>Presenting complaint</th>
<th>Collision type 1</th>
<th>Radiological findings</th>
<th>Surgical approach</th>
<th>Intraoperative findings</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>Prevedello et al(^{10})</td>
<td>2007</td>
<td>52/F</td>
<td>Headache, right temporal visual field loss</td>
<td>Nonfunctioning pituitary adenoma</td>
<td>Meningioma</td>
<td>Sellar lesion was enhancing homogeneously with T1 shortening, Planum sphenoidal lesion was broad based, enhancing homogenously</td>
<td>Extended transsphenoidal</td>
<td>Sellar tumor was soft, suckable. Planum sphenoidal tumor was debulked followed by extracapsular dissection</td>
</tr>
<tr>
<td>8</td>
<td>Cannavò et al(^{11})</td>
<td>1993</td>
<td>47/F</td>
<td>Acromegaly</td>
<td>GH-secreting pituitary adenoma</td>
<td>Meningioma</td>
<td>Sellar lesion showed nonhomogeneous enhancement with peripheral bright signal rim, retro sellar component showed bright homogenous enhancement</td>
<td>Transcranial</td>
<td>Not available</td>
</tr>
<tr>
<td>9</td>
<td>Zentner and Gilsbach(^{12})</td>
<td>1989</td>
<td>61/F</td>
<td>Headache, visual field loss</td>
<td>Nonfunctioning pituitary adenoma</td>
<td>Meningioma</td>
<td>CT demonstrated large sellar suprasellar hyperdense lesion</td>
<td>Transsphenoidal followed by craniotomy</td>
<td>Pituitary adenoma was soft and fragile, meningioma was more firm</td>
</tr>
<tr>
<td>10</td>
<td>Banik et al(^{13})</td>
<td>1984</td>
<td>56/F</td>
<td>Asymptomatic MEN 1 syndrome Adrenal collision tumor</td>
<td>Nonfunctioning pituitary adenoma</td>
<td>Meningioma</td>
<td>Not available</td>
<td>Diagnosed on postmortem</td>
<td>Not available</td>
</tr>
</tbody>
</table>

Abbreviations: ACTH, adrenocorticotropic hormone; CT, computed tomography; F, female; GH, growth hormone; M, male; MRI, magnetic resonance imaging; T1W, T1-weighted; VP, ventriculoperitoneal. Note: The sellar component was extending in the suprasellar region causing chiasmatic compression. The suprasellar component was broad based and was reaching till the tuberculum sella.
seldom be able to diagnose dual sellar pathology, that is, two different histological entities of pituitary adenoma and meningioma as both the tumors may exhibit similar imaging characteristics. Ruiz-Juretschke et al have reported a patient with collision tumor of pituitary adenoma and meningioma. Preoperative MRI could not distinguish the two separate tumor components and the diagnosis of collision tumor was reached on histopathological examination. Prevedello et al, however, in their patient have reported identification of collision tumor of pituitary adenoma and meningioma based on MRI. In the present case, preoperative radiological diagnosis of collision tumor was not made as the two different components of the tumor showed the same signal intensity on all sequences on MRI. On reviewing the images retrospectively, the broad based suprasellar component reaching till the tuberculum sella could have been diagnosed as meningeval component of the collision tumor.

In the present case, the tumor was excised completely via endonasal transsphenoidal approach. Intraoperatively, the soft sellar component and firm suprasellar component with high vascularity could represent the pituitary adenoma and meningeval component of the collision tumor. de Vries et al and Ruiz-Juretschke et al have also reported total excision of such collision tumor by endoscopic transsphenoidal approach. Zhao et al have proposed a second surgery performed transcranially to resect residual tumor. Prevedello et al have described the extended endonasal transsphenoidal approach for extirpation of pituitary adenoma and meningioma, obviating the need for additional craniotomy.

One of the proposed hypotheses for coexistence of meningioma and pituitary adenoma is radiation therapy to pituitary adenoma. Meningiomas tend to occur after a latent period of 5 years of radiation and within the pathway of irradiation. Honegger et al have reported one such patient of meningioma coexisting following irradiation of pituitary adenoma. We did not find any such case in literature of collision tumor composed of pituitary adenoma and meningioma occurring after radiation of pituitary adenoma. On the other hand, coincidental meningioma occurring in patient with pituitary adenoma without prior radiation therapy is extremely uncommon. Meningiomas in such patients tend to occur in perisellar location at the planum sphenoidale, tuberculum sella, and sphenoid wing. It is postulated that pituitary adenomas are a causative factor in the development of meningiomas. Meningiomas express hormone receptors on the tumor surface, implying that their growth is under hormonal control. This is more likely in case of functioning pituitary adenoma especially if the adenoma is GH-secreting adenoma. GH secreted causes meningioma growth. Seventy-five percent of meningiomas express GH and IGF1 receptors. However, meningiomas are also found in association with nonsecreting, PRL–secreting, and ACTH-secreting pituitary adenoma. Hence, it is assumed that the coexistence of a meningioma with pituitary adenoma is a casual finding with no relationship between the two tumors. The formation of a collision tumor composed of pituitary adenoma and meningioma is difficult to explain in nonfunctioning pituitary adenoma. In the present case, the only possible explanation of coexistence of nonfunctioning pituitary adenoma and meningioma is coincidental. Progress in molecular genetics and further research will shed more light on tumorogenesis of collision tumors.

**Conclusion**

Collision tumor comprising of nonfunctioning pituitary adenoma and meningioma is extremely rare. Preoperative MRI may not always be able to distinguish these histologically distinct neoplasms. Hence, histopathological examination is necessary to establish the diagnosis. Endoscopic transsphenoidal approach may suffice in excision of these collision tumors. Close follow-up is necessary to detect tumor recurrence. Though the association of these tumors can be coincidental, casual relationship between the occurrence of collision tumors cannot be totally excluded.

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**Conflict of Interest**

None declared.

**References**


