

Collision tumors composed of meningioma and growth hormone-secreting pituitary adenoma in the sellar region

Case reports and a literature review

Yi Zhao, MD^a, Hui Zhang, MD^{a,b}, Wei Lian, MD^{a,*}, Bing Xing, MD^a, Ming Feng, MD^a, Xiaohai Liu, MD^a, Renzhi Wang, MD^a

Abstract

Rationale: Collision tumor is a rare disease that represents the coexistence of two histologically distinct neoplasms in the same area without histological admixture or an intermediate cell population zone. To our best knowledge, 13 cases besides our 2 cases have been reported till now, and our report represents the first publication regarding a collision tumor composed of growth hormone (GH)-secreting pituitary adenoma and sellar meningioma.

Patient concerns: We collected two cases of collision tumors composed of meningioma and GH-secreting adenoma in the sellar region from 2014 to 2015 at Peking Union Medical College Hospital (PUMCH).

Diagnosis: Two cases were diagnosed with solid sellar tumors, and two tumor types were suspected with magnetic resonance imaging (MRI). Blood hormone tests revealed increased insulin-like growth factor 1 (IGF-1) and GH levels.

Interventions: Both cases underwent transsphenoidal microsurgical resection of pituitary adenoma.

Outcomes: The tumor was completely resected, and the pathological examination after the operation revealed meningioma and GH-secreting pituitary adenoma.

Lessons: Collision tumors consisting of pituitary adenomas with other sellar neoplasms are rare. Histological examination is necessary because preoperative studies cannot guarantee an accurate diagnosis. If a collision tumor is suspected prior to operation, a craniotomy may need to be considered before other operation methods to avoid reoperation.

Abbreviations: ACTH = adrenocorticotrophic hormone, FSH = follicle-stimulating hormone, GH = growth hormone, IGF-1 = insulin-like growth factor 1, LH = luteinizing hormone, MRI = magnetic resonance imaging, PRL = prolactin, PUMCH = Peking Union Medical College Hospital, TSH = thyroid-stimulating hormone.

Keywords: characteristics, collision tumor, therapy, transsphenoidal microsurgical resection

1. Introduction

Collision tumor is a rare disease that represents the coexistence of 2 histologically distinct neoplasms in the same area without histological admixture or an intermediate cell population zone.^[1] Different histogenesis and tumorigenic pathways represent the

mosaic of 2 concurrent tumors. Notably, the term “collision tumor” should be distinguished from another similar term, mixed tumor, which is 2 different neoplasms with histologically admixed cell types. Here, we report 2 cases of cerebral collision tumors composed of 2 benign components in the sellar region. To the best of our knowledge, this study represents the first report of a collision tumors consisting of meningioma and growth hormone (GH)-secreting pituitary adenoma. A detailed medical history is important for choosing the best treatment for collision tumors.

2. Case reports

2.1. Case 1

A 58-year-old female developed an acromegalic appearance and snoring over 15 years. Nine years ago, the patient complained of a headache with no dizziness or vision loss. Her past medical history included thyroid adenoma, hypertension, and a varicose vein in a lower limb. After admission, physical examination revealed enlargement of her fingers and toes, mandibular protrusion, and a broadened nose and lips. Blood hormone tests revealed increased insulin-like growth factor 1 (IGF-1) (991 ng/mL) and GH (15.9 ng/mL) levels, but a glucose suppression GH test could not be obtained. Other anterior pituitary hormones were within the normal range. Enhanced magnetic

Editor: Sheyu Li.

YZ and HZ are co-first authors.

ORCID: None.

This retrospective study was not supported by any funding.

The authors declare that they have no conflicts of interest.

^aDepartment of Neurosurgery, Peking Union Medical College Hospital, ^bTsinghua University School of Medicine, Beijing 100084, China.

* Correspondence: Wei Lian, Department of Neurosurgery, Peking Union Medical College Hospital, Beijing 100730, China (e-mail: lwpumch@hotmail.com).

Copyright © 2017 the Author(s). Published by Wolters Kluwer Health, Inc. This is an open access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

Medicine (2017) 96:50(e9139)

Received: 21 June 2017 / Received in final form: 15 November 2017 /

Accepted: 16 November 2017

<http://dx.doi.org/10.1097/MD.0000000000009139>

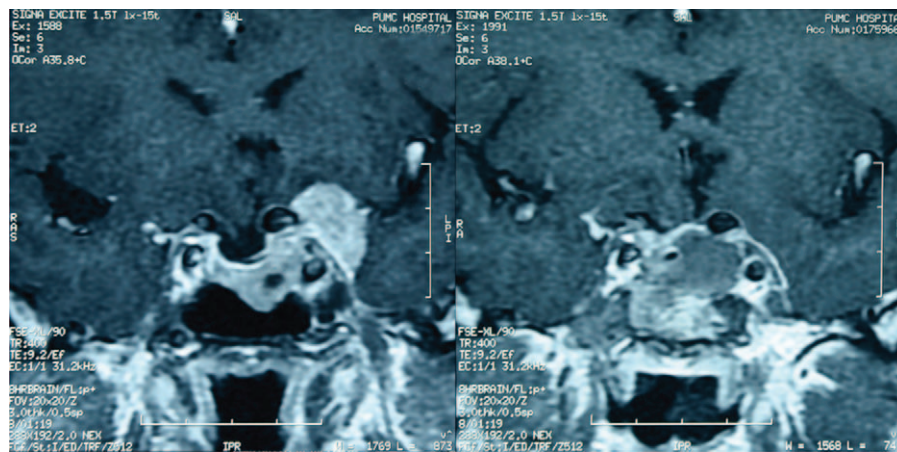


Figure 1. MRI scan before the first operation and after the second operation of case 1.

resonance imaging (MRI) of the sellar region showed a slightly hyperintense mass ($3.5 \times 1.9 \times 2.0$ cm in size) on T1-weighted images and an iso-hyperintense mass on T2-weighted imaging, with heterogeneous contrast enhancement near the sellar floor (Fig. 1). The left cavernous sinus was completely surrounded by the mass (Knosp 4), while the right side was not invaded, and the optic chiasm was not compressed. The patient was diagnosed with GH-producing pituitary adenoma and underwent transsphenoidal pituitary resection surgery. The tumor was gray and heterogeneous, mostly soft and fragile, and partly tough with a rich blood supply. After successful resection of the tumor, the saddle decreased to a normal size. The sellar floor was reconstructed with artificial dura mater, and no cerebrospinal fluid leakage was observed. A postoperative histopathologic examination confirmed the diagnosis of GH-secreting pituitary adenoma, and immunohistochemical results showed adrenocorticotropic hormone (ACTH) (–), follicle-stimulating hormone (FSH) (+), GH (+), luteinizing hormone (LH) (+), prolactin (PRL) (+), thyroid-stimulating hormone (TSH) (–), P53 (–), and Ki-67 (index approximately 1%) (Fig. 2). GH and IGF-1 levels

decreased to normal immediately after surgery. However, postoperative MRI revealed a remnant of the tumor that could not be removed transsphenoidally, and the remainder of the tumor was diagnosed as meningioma according to MRI. Based on the postoperative imaging result, the patient was treated with a craniotomy within 3 months after the transsphenoidal pituitary resection operation because the headache symptom returned. During the operation, the tumor exhibited a dark red color and was rubbery with an abundance of blood vessels. The tumor was completely resected, and the pathological examination after the operation revealed meningioma. The postoperative imaging is shown in Figure 1. The pathology result is shown in Figure 3.

2.2. Case 2

One year ago, a 58-year-old female detected enlargement of her superciliary ridge, fingers, and toes, as well as broadening of her nose and lips. Meanwhile, she suffered from severe snoring. No special medical history was reported except for cured tuberculosis and hepatitis. Blood hormone tests revealed increased IGF-1

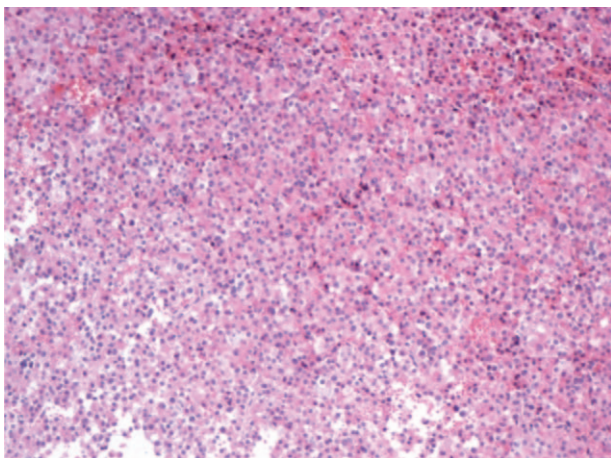


Figure 2. The pathological examination showed a pituitary adenoma; immunohistochemical results revealed ACTH (–), FSH (+), GH (+), LH (+), PRL (+), TSH (–), P53 (–), and Ki-67 (index approximately 1%). HE staining with an amplification factor of 20.

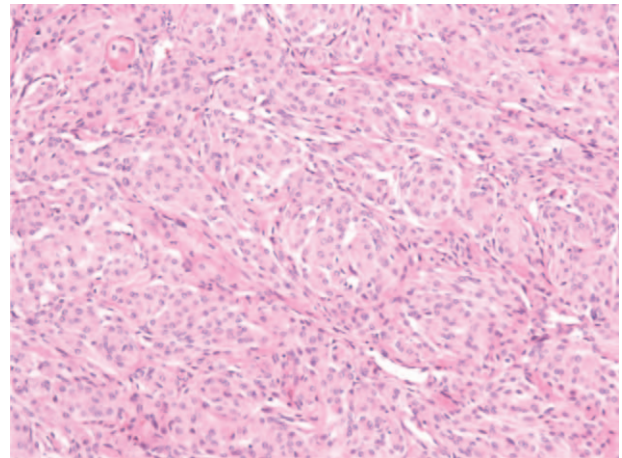


Figure 3. The pathological examination after the second operation of case 1 revealed a meningioma; immunohistochemical results showed CD34 (–), EMA (+), PR (+), Ki-67 (index approximately 3%), GFAP (–), and vimentin (++) HE staining with an amplification factor of 100.

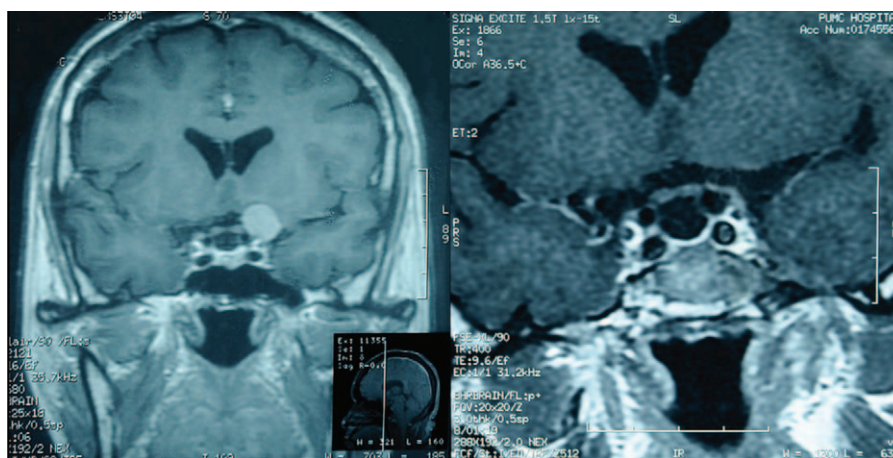


Figure 4. MRI scan before the first operation and after the second operation of case 2.

(1291ng/mL) and GH (21.2ng/mL) levels, but a glucose suppression GH test could not be obtained. Enhanced sellar MRI showed a mass (1.12 × 0.54 × 1.07 cm in size) with a short signal on T1-weighted imaging and a short signal on T2-weighted imaging, with a low signal surrounding the high signal on T2-weighted imaging. The right cavernous sinus was completely surrounded (Knosp 4) by the mass, while the left side was not invaded (Fig. 4), and the optic chiasm was not compressed. The patient was diagnosed with GH-producing pituitary adenoma and underwent transsphenoidal pituitary resection surgery. The tumor was gray and heterogeneous, mostly soft and fragile, and partly tough with a rich blood supply. After successful resection of the tumor, the sellar floor was reconstructed with artificial dura mater, and no cerebrospinal fluid leakage was observed. A postoperative histopathologic examination confirmed the diagnosis of GH-secreting pituitary adenoma (Fig. 5). GH and IGF-1 levels decreased to normal immediately after the surgery. However, postoperative MRI revealed a remnant of the tumor that could not be removed transsphenoidally, and the rest of the tumor was suspected to be a meningioma based on imaging. Four

months after the first operation, the patient experienced frequent headaches. Based on the postoperative imaging result, the patient was treated with a craniotomy. During the operation, the tumor exhibited a dark red color and was rubbery with a rich blood supply. In addition, the postoperative pathological examination revealed meningioma. The postoperative imaging result is shown in Figure 4, and the pathology result is shown in Figure 6.

3. Discussion

The coexistence of pituitary adenoma and intracranial tumor outside the sella is not uncommon, especially in patients who have received radiotherapy for a pituitary mass.^[2] However, only a few publications have described collision tumors consisting of pituitary adenoma and a different sellar neoplasm. Neither of the patients described in this article received any radiotherapy before their intrasellar tumors were detected, which made their cases rarer. We carefully reviewed cases in which pituitary adenoma coexisted with another type of neoplasm, and we found only reports of craniopharyngiomas,^[3,4] gangliocytomas,^[5-8] schwannomas,^[7]

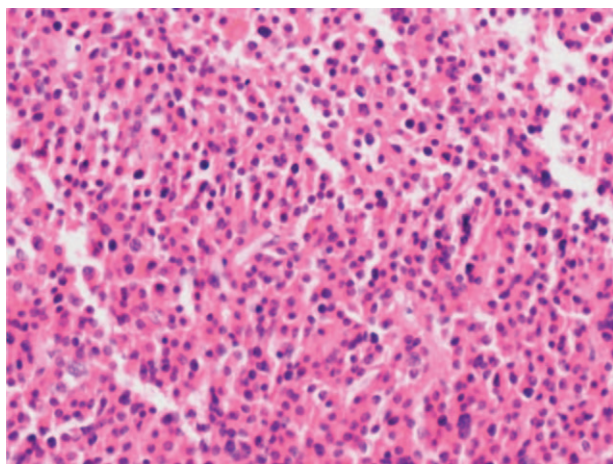


Figure 5. The pathological examination showed a pituitary adenoma; immunohistochemical results revealed ACTH (-), FSH (+), GH (+), LH (-), PRL (-), TSH (-), P53 (-), and Ki-67 (index approximately 1%). HE staining with an amplification factor of 200.

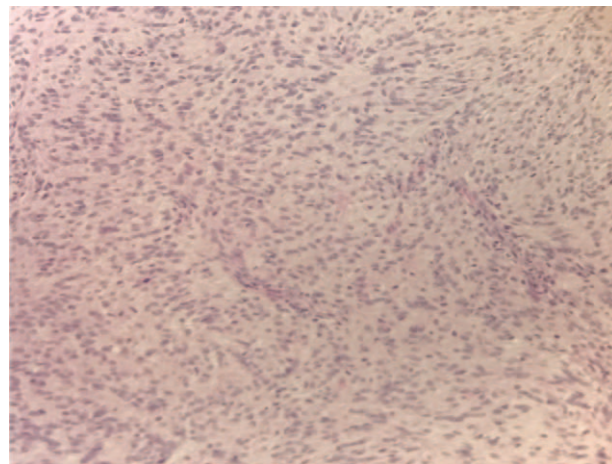


Figure 6. The pathological examination after the second operation of case 2 revealed a meningioma; immunohistochemical results showed CD34 (+), EMA (+/-), Ki-67 (index approximately 3%), P53 (-), S-100 (-), and vimentin (+). HE staining with a medium amplification factor.

Table 1 Literature review for collision tumor of pituitary adenoma with a different neoplasm.

Authors	Year	Age, y	Sex	Clinical presentation	Collision type 1	Collision type 2	Surgical approach(s)	Follow-up
Case 1 in this article	2014	58	F	Acromegaly	GH-secreting PA	Meningioma	First transsphenoidal approach for resection, second craniotomy	N/A
Case 2 in this article	2015	58	F	Acromegaly	GH-secreting PA	Meningioma	First transsphenoidal approach for resection, second craniotomy	N/A
Karsy et al ^[9]	2015	70	F	Altered mental status, mutism, and incontinence	Non-secreting PA	Meningioma	Transnasal, transsphenoidal approach for resection	N/A
Koutourousiou et al ^[11]	2009	38	N/A	Acromegaly	GH-secreting PA	Schwannoma	Sub-labial transsphenoidal approach for resection	No evidence of recurrence of the adenoma 2 y after surgery
Tanriover et al ^[8]	2014	39	F	Acromegaly	GH-secreting PA	Gangliocytoma	Transsphenoidal approach for resection	No recurrence, and the patient is clinically and biochemically in remission.
Prabhakar et al ^[12]	1971	29	M	Bifrontal headache, vision loss, acromegaly	PA	Craniopharyngioma	Right frontal craniotomy	Died 4 d after surgery with uncontrolled diabetes insipidus; necropsy not performed
Wheatley et al ^[13]	1986	61	M	Deteriorating vision	Prolactin-secreting PA	Craniopharyngioma	Subfrontal approach	Fatal postoperative cardiac arrest; PA confirmed by autopsy
Yoshida et al ^[14]	2008	29	M	Atrial fibrillation	PA	Adamantinomatous craniopharyngioma	Transsphenoidal	N/A
Gokden et al ^[15]	2009	47	M	Headache, vision loss	PA	Adamantinomatous craniopharyngioma	Transsphenoidal	Uneventful and no recurrence in 1 y
Moshkin et al ^[16]	2009	12	M	Partial hypopituitarism	PA	Adamantinomatous craniopharyngioma	Right frontal craniotomy	Uneventful and no recurrence in 10 mo
Sargis et al ^[17]	2009	59	M	Progressive vision loss	Gonadotropin-secreting PA	Adamantinomatous craniopharyngioma	Subtotal transcranial resection	N/A
Jin et al ^[3]	2013	47	F	Deteriorating vision	Non-secreting PA	Adamantinomatous craniopharyngioma	First transsphenoidal resection of the tumor, second right frontal craniotomy using an interhemispheric transcallosal approach	3-mo follow-up MRI confirmed complete resection of the tumor
Finzi et al ^[4]	2013	75	F	Diplopia and slight increase in serum prolactin	Typical pituitary silent subtype 2 ACTH cell adenoma	Adamantinomatous craniopharyngioma	Endoscopic endonasal transsphenoidal tumor resection	Complete recovery, no recurrence in 10 mo

F = female, M = male, N/A = not available, PA = pituitary adenoma.

and meningioma.^[9] Finzi et al^[4] propose that the term “mixed tumor” should be limited because of neoplasms in which endocrine and non-endocrine components are strictly admixed; this admixture distinguishes “mixed pituitary adenoma/craniopharyngioma” from collision tumors. We summarize all reported cases of collision tumors with pituitary adenoma in Table 1 (only cases that could be strictly defined as “collision” were included). Although Amirjamshidi et al^[10] reported 2 cases of coexisting PA and suprasellar meningioma, these cases were not included in the table since histological evidence was not provided to confirm the diagnosis of collision tumor. Moreover, the authors claimed that the coexisting tumors in their cases were not collision tumors and were likely coincidental. Although articles have provided many explanations for the pathogenetic relationship between concomitant tumors, none have provided clear evidence. In both of our cases, GH-secreting pituitary adenoma combined with meningioma was diagnosed after pathological examinations. To the best of our knowledge, this report represents the first publication regarding a collision tumor composed of GH-secreting pituitary adenoma and sellar meningioma.

3.1. Pituitary adenoma and sellar meningioma

The association between pituitary adenomas and meningiomas has been widely explored. Pituitary adenoma is the first diagnosis considered for sellar lesions, and 10% to 15% of central nervous system tumors identified as pituitary adenoma are found through autopsy and 23% through thin-section MRI.^[8] Meningioma is one of the most common benign cranial neoplasms. Thus, the coexistence of pituitary adenoma and intracranial meningioma is not rare.^[18–21] Meanwhile, giant intrasellar meningiomas mimicking pituitary adenomas have been well described in previous studies.^[22,23] However, few articles regarding collision tumors composed of pituitary adenoma and meningioma have been published. To date, Karsy et al^[9] reported the only case study of coincident pituitary adenoma and sellar meningioma in which mental changes were the chief complaint and a non-secreting pituitary adenoma was identified by pituitary laboratory tests and postoperative pathology. In addition, a pathological examination revealed a coexisting, microscopic fibroepithelial meningioma.

MRI is commonly used to diagnose sellar masses. However, due to their similar imaging characteristics, preoperative differential diagnosis of pituitary adenoma and intrasellar meningioma or a collision tumor composed of both is not possible with MRI. Our patients had a slightly hyperintense or low signal on T1-weighted imaging and an iso-hyperintense or high signal on T2-weighted imaging. Due to the same signals obtained during imaging, these 2 tumors appear to be a single tumor. Thus, postoperative pathological examination is necessary for a final diagnosis.

The formation of a collision tumor composed of pituitary adenoma and meningioma is difficult to explain. In terms of the tumorigenesis of these neoplasms, one possible explanation is that GH secretion in GH-secreting pituitary adenoma induces meningioma growth^[24]; the coexistence of these neoplasms in the same anatomic position can be considered incidental.

Transsphenoidal surgery was the first operation performed on our patients, but the postoperative pathologic examination results revealed another tumor in the parasellar area. Thus, a craniotomy was performed. When a collision tumor is suspected, only 1 operation should be performed if possible.

3.2. Craniopharyngioma with pituitary adenoma

Although both are common pathologies in the sellar or suprasellar areas, collision tumors composed of craniopharyngioma and pituitary adenoma components are rare. To date, 14 cases of collision tumors composed of pituitary adenoma and craniopharyngioma have been reported.^[3] Prolactin type were the most frequently reported, with 8 cases, and 2 cases of ACTH and a case of TSH were also reported; the remaining 3 cases were silent. Adamantinomatous-type craniopharyngioma is the most well documented, and the main clinical manifestations include deteriorating vision and abnormal secreting hormone symptoms, which is similar to the diagnosis of pituitary adenoma or craniopharyngioma alone. However, CT and MRI cannot identify the coexistence of pituitary adenoma and craniopharyngioma. Due to the non-distinguishing clinical and imaging features of craniopharyngioma, a collision tumor composed of craniopharyngioma and pituitary adenoma is difficult to differentiate from pituitary adenoma alone before operation; histological studies are required for diagnosis.

3.3. Gangliocytomas of the sellar region

Gangliocytomas account for 0.5% of all brain tumors and rarely occur in the sellar region, with an incidence of 0.52% to 1.26% within clinical sellar tumor series.^[7] Reports of collision tumors composed of gangliocytomas and pituitary adenomas are rare. Koutourousiou et al reported 3 cases of gangliocytoma associated with GH-secreting pituitary adenoma. However, the association was actually an admixture of 2 cell types, which does not fit the definition of collision tumor that we mentioned earlier in this article.^[6,7]

MRI and other radiological examinations cannot provide an accurate diagnosis of this tumor type, and the diagnosis should ultimately be based on histopathologic examinations. However, the use of pathological examinations in differential diagnosis is still controversial.

Some therapies have been identified during the study of the underlying mechanism of collision tumors composed of gangliocytomas and pituitary adenoma. During early embryogenesis, hypothalamic neurons are thought to migrate abnormally in the anterior hypophyseal parenchyma and result in pituitary adenoma. Another theory suggests that ganglion cells can release pituitary hormones, thus promoting adenoma formation.^[7] A third theory suggests neuronal transformation of pituitary adenoma cells.^[25,26] Nevertheless, this theory is still a topic of debate; Koutourousiou et al^[7] stated that this theory challenges the understanding of embryology. In a recent study, Kontogeorgos et al^[6] argued that neuronal and adenomatous parts of these tumors may share a common progenitor cell that may differentiate into distinct cell types.

3.4. Intrasellar schwannomas

Only 1 case of GH-secreting pituitary adenoma coexisting with an intrasellar schwannoma has been published.^[11] The authors did not find an interaction between GH-secreting pituitary adenomas and intrasellar schwannomas and considered the finding incidental. Intrasellar schwannomas are rare. To date, no more than 18 cases have been reported.^[27–39] Preoperative diagnosis through MRI or clinical presentation was not possible in all these cases. Regarding the origin of intrasellar schwannomas, several theories have been put forward. If cranial nerve symptoms are observed, a schwannoma may have extended into

the sella from a cranial nerve within the cavernous sinus.^[28,30] Other possible origins include the perivascular nerve plexus, multipotential mesenchymal cells, displaced neural crest cells, ectopic Schwann cells, and lateral sellar nerve plexus.^[34,40]

4. Conclusion

Collision tumors composed of pituitary adenomas and other sellar neoplasms are rare. Our report is the first regarding collision tumors composed of GH-secreting pituitary adenoma and sellar meningioma. Histological examination is necessary because preoperative studies cannot ensure an accurate diagnosis. Furthermore, the etiology of a collision tumor composed of meningioma and pituitary adenoma is unknown. If a collision tumor is suspected before operation, a craniotomy may need to be considered before other operation methods to avoid reoperation.

Acknowledgment

We are very grateful to the patients and caregivers who participated in this study for making this research possible.

References

- Willis RA. *Pathology of Tumors*. 4th ed. Butterworth, London:1967.
- Partington MD, Davis DH. Radiation-induced meningioma after treatment for pituitary adenoma: case report and literature review. *Neurosurgery* 1990;26:329–31.
- Jin G, Hao S, Xie J, et al. Collision tumors of the sella: coexistence of pituitary adenoma and craniopharyngioma in the sellar region. *World J Surg Oncol* 2013;11:178.
- Finzi G, Cerati M, Marando A, et al. Mixed pituitary adenoma/craniopharyngioma: clinical, morphological, immunohistochemical and ultrastructural study of a case, review of the literature, and pathogenetic and nosological considerations. *Pituitary* 2014;17:53–9.
- Bodi I, Martin AJ, Connor SE, et al. Mixed pituitary gangliocytoma/adenoma (prolactinoma) with histogenetic implications. *Neuropathol Appl Neurobiol* 2002;28:252–5.
- Kontogeorgos G, Mourouti G, Kyrodimou E, et al. Ganglion cell containing pituitary adenomas: signs of neuronal differentiation in adenoma cells. *Acta Neuropathol* 2006;112:21–8.
- Koutourousiou M, Kontogeorgos G, Wesseling P, et al. Collision sellar lesions: experience with eight cases and review of the literature. *Pituitary* 2010;13:8–17.
- Tanriover N, Aydin O, Kucukyuruk B, et al. Endoscopic approach to a collision tumor of growth hormone-secreting adenoma and gangliocytoma in the pituitary gland. *J Craniofac Surg* 2014;25:1277–9.
- Karsy M, Sonnen J, Couldwell WT. Coincident pituitary adenoma and sellar meningioma. *Acta Neurochir (Wien)* 2015;157:231–3.
- Amirjamshidi A, Mortazavi SA, Shirani M, et al. Coexisting pituitary adenoma and suprasellar meningioma—a coincidence or causation effect: report of two cases and review of the literature. *J Surg Case Rep* 2017;2017:rjx039.
- Koutourousiou M, Seretis A, Kontogeorgos G. Intra-sellar schwannoma co-existing with GH-secreting pituitary adenoma. *Acta Neurochir (Wien)* 2009;151:1693–7.
- Prabhakar V, Rao BD, Subramanyam MV. Pituitary adenoma associated with craniopharyngioma. *J Pathol* 1971;103:185–7.
- Wheatley T, Clark JD, Stewart S. Craniopharyngioma with hyperprolactinaemia due to a prolactinoma. *J Neurol Neurosurg Psychiatry* 1986;49:1305–7.
- Yoshida A, Sen C, Asa SL, et al. Composite pituitary adenoma and craniopharyngioma?: an unusual sellar neoplasm with divergent differentiation. *Am J Surg Pathol* 2008;32:1736–41.
- Gokden M, Mrak RE. Pituitary adenoma with craniopharyngioma component. *Hum Pathol* 2009;40:1189–93.
- Moshkin O, Scheithauer BW, Syro LV, et al. Collision tumors of the sella: craniopharyngioma and silent pituitary adenoma subtype 3: case report. *Endocr Pathol* 2009;20:50–5.
- Sargis RM, Wollmann RL, Pytel P. A 59 year-old man with sellar lesion. *Brain Pathol* 2009;19:161–2.
- Honegger J, Buchfelder M, Schrell U, et al. The coexistence of pituitary adenomas and meningiomas: three case reports and a review of the literature. *Br J Neurosurg* 1989;3:59–69.
- Abs R, Parizel PM, Willems PJ, et al. The association of meningioma and pituitary adenoma: report of seven cases and review of the literature. *Eur Neurol* 1993;33:416–22.
- Cannavò DS, Curtò L, Fazio R, et al. Coexistence of growth hormone-secreting pituitary adenoma and intracranial meningioma: a case report and review of the literature. *J Endocrinol Invest* 1993;16:703–8.
- Curto L, Squadrito S, Almoto B, et al. MRI finding of simultaneous coexistence of growth hormone-secreting pituitary adenoma with intracranial meningioma and carotid artery aneurysms: report of a case. *Pituitary* 2007;10:299–305.
- Yin S, Zhou P, Li Q, et al. Intracellular clear cell meningioma mimicking invasive pituitary adenoma: a case report and review of the literature. *Turk Neurosurg* 2015;25:976–9.
- Bang M, Suh JH, Park JB, et al. Pure intrasellar meningioma mimicking pituitary macroadenoma: magnetic resonance imaging and review of the literature. *World Neurosurg* 2016;91:675.e1–4.
- Furtado SV, Dadlani R, Ghosal N, et al. Co-existing thyrotropin secreting pituitary adenoma and low grade glioma: clinical considerations and literature review. *J Neurosurg Sci* 2009;53:71–5.
- Vidal S, Horvath E, Kovacs K, et al. Reversible transdifferentiation: interconversion of somatotrophs and lactotrophs in pituitary hyperplasia. *Mod Pathol* 2001;14:20–8.
- Horvath E, Kovacs K, Scheithauer BW, et al. Pituitary adenoma with neuronal choristoma (PANCH): composite lesion or lineage infidelity? *Ultrastruct Pathol* 1994;18:565–74.
- Goebel HH, Shimokawa K, Schaaqe T, et al. Schwannoma of the sellar region. *Acta Neurochir (Wien)* 1979;48:191–7.
- Perone TP, Robinson B, Holmes SM. Intrasellar schwannoma: case report. *Neurosurgery* 1984;14:71–3.
- Wilberger J Jr. Primary intrasellar schwannoma: case report. *Surg Neurol* 1989;32:156–8.
- Guenot M, Bataille B, Wager M. Intrasellar neurinoma. Apropos of a case and review of the literature. *Neurochirurgie* 1994;40:263–6.
- Civit T, Pinelli C, Klein M, et al. Intrasellar schwannoma. *Acta Neurochir (Wien)* 1997;139:160–1.
- Bhagat S, Smith C, Teasdale GM, et al. Nerve sheath tumors of the sellar region. *J Neuroophthalmol* 2002;22:275–8.
- Whee SM, Lee JI, Kim JH. Intrasellar schwannoma mimicking pituitary adenoma: a case report. *J Korean Med Sci* 2002;17:147–50.
- Maartens NF, Ellegala DB, Vance ML, et al. Intrasellar schwannomas: report of two cases. *Neurosurgery* 2003;52:1200–5.
- Esposito F, Cappabianca P, Del Basso De Caro M, et al. Endoscopic endonasal transsphenoidal removal of an intra-suprasellar schwannoma mimicking a pituitary adenoma. *Minim Invasive Neurosurg* 2004;47:230–4.
- Honegger J, Koerbel A, Psaras T, et al. Primary intrasellar schwannoma: clinical, aetiopathological and surgical considerations. *Br J Neurosurg* 2005;19:432–8.
- Bernreuther C, Flitsch J, Lüdecke DK, et al. A 61-year-old man with hyponatremia. *Brain Pathol* 2008;18:283–7.
- Mohammed S, Kovacs K, Munoz D, et al. A short illustrated review of sellar region schwannomas. *Acta Neurochir (Wien)* 2010;152:885–91.
- Kong X, Wu H, Ma W, et al. Schwannoma in sellar region mimics invasive pituitary macroadenoma: literature review with one case report. *Medicine* 2016;95:e2931.
- Bleys RL, Janssen LM, Groen GJ. The lateral sellar nerve plexus and its connections in humans. *J Neurosurg* 2001;95:102–10.