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ABOUT COVER

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CASE REPORT

Coexistence of meningioma and other intracranial benign tumors in non-neurofibromatosis type 2 patients: A case report and review of literature

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Abstract

BACKGROUND

The coexistence of meningioma and other intracranial primary benign tumors is rare, especially in non-neurofibromatosis type 2, and there is limited guidance for the management of such patients. Here, we report a series of 5 patients with concomitant meningioma and other intracranial benign tumors, including subependymoma and pituitary adenoma.

CASE SUMMARY

Five non-neurofibromatosis type 2 patients with simultaneous occurrence of meningioma and other intracranial benign tumors were retrospectively reviewed. The patients had no history of previous irradiation. The clinical features, pre- and postoperative imaging, surgical procedure and pathological findings were extracted from electronic medical records. There were 4 female patients (80%) and 1 male patient (20%). The mean age was 42.8 years (range: 29-52 years). The coexisting tumors included subependymoma in 1 case (20%) and pituitary adenoma in 4 cases (80%). The most common clinical symptom was headache (3/5, 60%). Four patients (80%) underwent craniotomy. One patient (20%) underwent transsphenoidal surgery followed by transcranial operation. All tumor diagnoses were confirmed by histopathological examination. The mean follow-up

was 38.8 mo (range: 23-96 mo), and all 5 patients were in a stable condition at the last follow-up.

CONCLUSION
The simultaneous occurrence of meningioma and other intracranial benign tumors is a rare clinical event. Histological examination is necessary for the accurate diagnosis. Neurosurgeons should select the appropriate surgical strategy according to the clinical features of each patient, which may provide a more favorable prognosis for individual patients.

Key Words: Meningioma; Sub-ependymoma; Pituitary adenoma; Coexisting tumors; Case report

Core Tip: The simultaneous occurrence of meningioma and other intracranial primary benign tumors is rare, especially in non-neurofibromatosis type 2, and there is limited guidance for the management of such patients. In this study, we report a series of 5 patients with coexistence of meningioma and other intracranial benign tumors, including subependymoma and pituitary adenoma.

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INTRODUCTION

Meningioma is the most frequent intracranial benign tumor, accounting for 37.1% of tumors overall[1]. However, the concomitant occurrence of meningioma and other intracranial benign tumors is extremely rare[2]. Only a few coexisting meningioma and other intracranial benign tumor cases have been reported in previous publications, such as pituitary adenoma[3,4], craniopharyngioma[5] and vestibular schwannoma[6-9]. It is well-recognized that the occurrence of multiple nervous system tumors in the same patient is a characteristic of neurofibromatosis type 2 (NF2)[8]. Patients are diagnosed with NF2 when they meet the Manchester criteria (Supplementary Table 1)[10,11]. However, clinical and pathological features of non-NF2 patients with collision-tumors remain unclear. Here we report the clinical presentation, radiological features, surgical management and outcomes in our series of 5 non-NF2 patients with concomitant meningioma and other intracranial benign tumors, including one subependymoma, which was reported for the first time, and four pituitary adenomas. In addition, we also reviewed the available literature.

CASE PRESENTATION

Chief complaints

Case 1: A 45-year-old female patient presented with a sudden night epileptic seizure 10 d prior.

Case 2: A 40-year-old female patient presented with dizziness and headache for 1 mo.

Case 3: A 48-year-old male patient presented with frontal and bilateral temporal headache for 1 mo.

Case 4: A 52-year-old female patient presented with left progressive blurred vision for 6 mo and headaches for 2 mo.

Case 5: A 29-year-old female patient presented with menstrual disorder for 2 years and intermittent headache for 3 mo.

History of present illness

Case 1: The patient suffered a sudden epileptic seizure 10 d ago at night, which lasted for a few minutes.

Case 2: The patient had dizziness and headaches for 1 mo.

Case 3: The patient had a headache for 1 mo. The headache took place at night and affected his sleep.

Case 4: The patient had progressive vision loss in the left eye for 6 mo. She suffered from headache from 2 mo.

Case 5: The patient had menstrual disorder for 2 years. She developed amenorrhea and intermittent headaches 3 mo ago.

History of past illness

All patients had no history of specific illnesses.

Personal and family history

All patients had no special personal and family history.

Physical examination

Case 1, 2, 3 and 5: Neurological examination of the patient found no positive signs.

Case 4: There was only slight sensation in her left eye when she was admitted.

Laboratory examinations

Case 1, 3 and 4: Preoperative endocrine examination showed no abnormal changes.

Case 2: Endocrine examination indicated that prolactin levels were moderately elevated (61.19 ng/mL).

Case 5: Preoperative endocrine examination showed that prolactin levels were slightly elevated (59.17 ng/mL).

Imaging examinations

Case 1: Brain computed tomography at the local hospital suggested an intracranial space-occupying lesion in the left parietal lobe. Brain contrast-enhanced magnetic resonance imaging (MRI) showed a well-circumscribed mass (3.6 cm × 2.7 cm × 2.7 cm) in the left parietal parafalcine and a mass (1.9 cm × 1.2 cm × 1.1 cm) in the left lateral ventricle.

Case 2: MRI showed a mass (3.0 cm × 2.3 cm × 2.5 cm) in the right middle cranial fossa and a mass (0.5 cm × 0.5 cm × 0.5 cm) in the sellar region.

Case 3: Brain MRI showed lesions located in the planum sphenoidale and sellar regions.

Case 4: There was a well-circumscribed mass (3.5 cm × 3.2 cm × 2.7 cm) surrounded with brain edema in the left sphenoid ridge and a mass (2.8 cm × 2.5 cm × 2.5 cm) encasing the internal carotid artery in the sellar and suprasellar regions, as determined by radiological examination.

Case 5: MRI showed a mass (5.7 cm × 3.3 cm) encasing the internal carotid artery located in the left petroclival region and a mass (maximum diameter 1.0 cm) located in the sellar region.

FINAL DIAGNOSIS

Case 1: Postoperative histopathological examination showed meningioma and subependymoma (Figure 1).

Case 2: Histopathological examination showed a meningioma and non-functioning pituitary adenoma (Figure 2).

Case 3: Histopathological examination showed a meningioma and non-functioning pituitary adenoma (Figure 3).

Case 4: Histopathological examination showed a meningioma and non-functioning pituitary adenoma (Figure 4).

Case 5: Postoperative histopathological examination showed a meningioma and non-functioning pituitary adenoma (Figure 5).

TREATMENT

Case 1: After this discovery, she underwent craniotomy, and the two neoplasms were removed in one session.

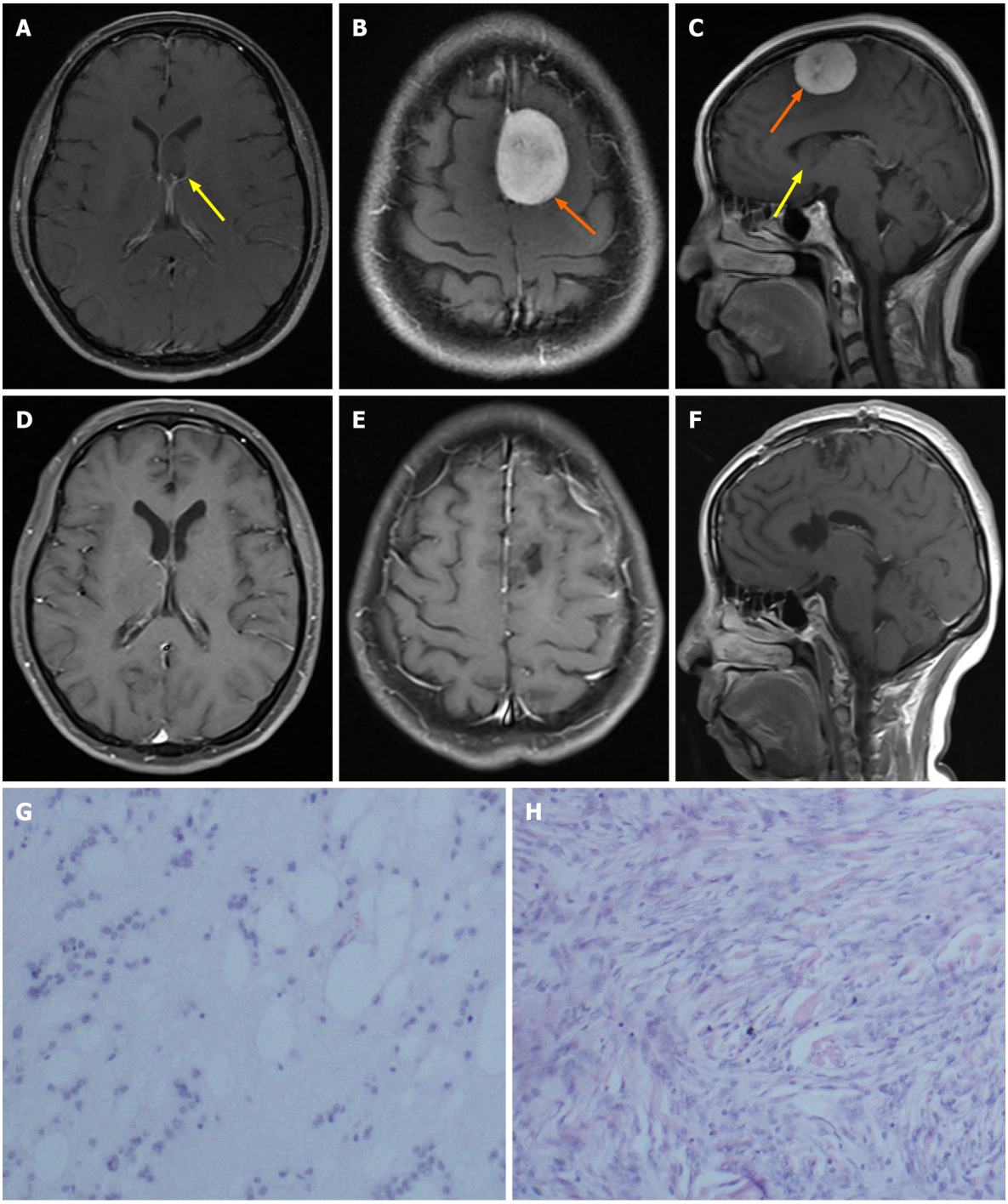

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Figure 1 Histopathological examination showed a meningioma and subependymoma. A-C: Preoperative axial and coronal contrast-enhanced magnetic resonance imaging (MRI) showed a mass in the left lateral ventricle (yellow arrow) and a mass in the left parietal parafalcine (orange arrow); D-F: Postoperative axial and coronal MRI showed that gross total resection of the tumors was achieved; G: Histological examination revealed subependymoma; H: Histological examination revealed meningioma.

Case 2: First, endonasal transsphenoidal surgery was performed for resection of the intrasellar mass. Then, the right middle cranial fossa mass was removed (Simpson grade II) by transcranial surgery.

Case 3: The patient underwent transcranial resection for the two tumors through the right transpterional approach.

Case 4: A single transcranial procedure was performed for removal of the sphenoid ridge mass (Simpson grade II) and sellar region mass. The pituitary tumor underwent subtotal resection.

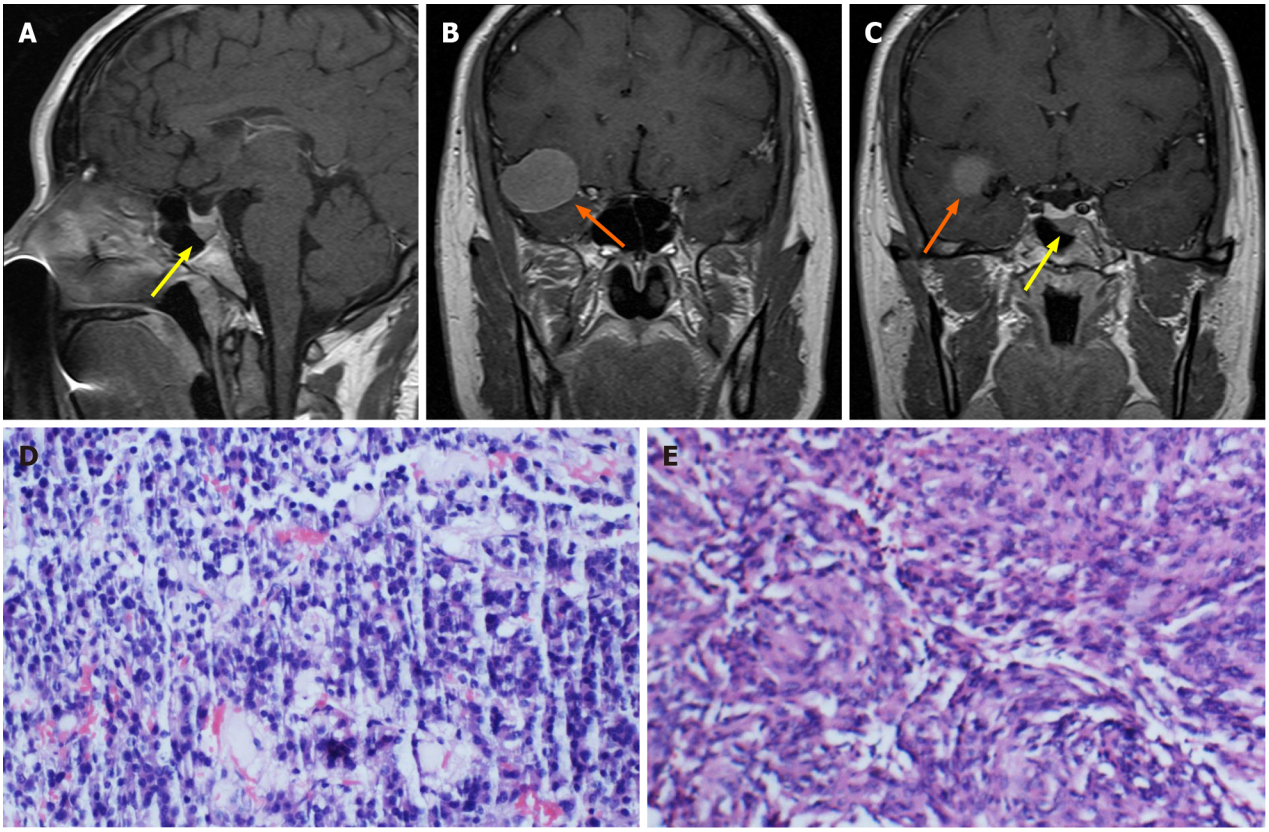

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Figure 2 Histopathological examination showed a meningioma and non-functioning pituitary adenoma. A: Sagittal contrast magnetic resonance imaging showed an intrasellar mass (yellow arrow); B and C: Sagittal coronal contrast magnetic resonance imaging showed an intrasellar mass (yellow arrow) and a mass in the right middle cranial fossa (orange arrow); D: Histological examination revealed pituitary adenoma; E: Histological examination revealed meningioma.

Case 5: The patient underwent craniotomy for the two tumors. The petroclival tumor was hard in consistency and rich in blood supply with internal carotid artery encasement. The tumor was also close to cranial nerves II-VI and compressed the brain stem. A subtotal resection was performed (Simpson grade IV) for the petroclival tumor. Then the intrasellar tumor was removed.

OUTCOME AND FOLLOW-UP

Between January 2011 and January 2019, 2922 consecutive patients were diagnosed with meningioma in our institution. There were 5 meningioma patients (0.17%) with different intracranial benign tumors, and none of them were diagnosed with NF2. They had no history of previous irradiation. Clinical data were obtained and analyzed through retrospective medical history reviews, neuroimaging information, histopathological examination and follow-up. This retrospective study was approved by the institutional review board. Written consent was obtained from each patient for the use of their clinical data for research.

There were 4 female patients (80%) and 1 male patient (20%) with concomitant meningioma and other intracranial benign tumors. The mean age was 42.8 years (range: 29-79 years). The coexisting tumors included subependymoma in 1 case (20%) and pituitary adenoma in 4 cases (80%, four non-functional pituitary adenomas). The mean follow-up time was 38.8 mo (range: 23-96 mo). The clinical information of these cases is summarized in Table 1.

Case 1: The patient was followed up for 27 mo with no evidence of recurrent disease.

Case 2: The patient was followed up for 24 mo, and she was in good health.

Case 3: He was treated by hormone replacement therapy because of postoperative hypopituitarism. The patient was followed up for 23 mo. He took LT4 regularly and is currently in good health.

Case 4: Four months after the surgery, MRI reexamination revealed the growth of residual pituitary tumor. Subsequently, she underwent radiotherapy. The patient was in good health at the 24-mo follow-

Table 1 Clinical features of 5 meningioma patients in the study

Case No.	Sex/age	Symptom	Meningioma location	WHO grade of meningioma	Second primary tumor	Operation	Surgical approach	Simpson grade	Recurrence	Postoperative treatment	Follow-up, mo
1	F/45	Seizure	Left frontal parafalcine	I	Subependymoma	Synchronous	TC	II	None	None	27
2	F/40	Headache	Right middle cranial fossa	I	Pituitary adenoma	Synchronous	TS + TC	II	None	None	24
3	M/48	Headache	Tuberculum sellae	I	Pituitary adenoma	Synchronous	TC	II	None	None	23
4	F/52	Blurred vision, headache	Left sphenoid ridge	I	Pituitary adenoma	Synchronous	TC	II	Growth of residual pituitary tumor after 4 mo	Radiotherapy	24
5	F/29	Amenorrhea and headache	Left petroclival	I	Pituitary adenoma	Synchronous	TC	IV	None	None	96

F: Female; M: Male, TC: Transcranial surgery; TS: Transsphenoidal surgery; WHO: World Health Organization.

up.

Case 5: As of this writing, the patient had been in stable condition for 96 mo.

DISCUSSION

The coexistence of meningioma and other intracranial benign tumors is a rare phenomenon that nevertheless deserves our attention. The most frequent coexistence of simultaneous benign tumors is pituitary adenoma with meningioma and schwannoma with meningioma[12]. The co-occurrence of meningioma and schwannoma is more likely to occur in patients with NF2, which has been well described previously. However, the clinical characteristics of non-NF2 patients with coexisting tumors are largely unknown.

To the best of our knowledge, we present the first case of concomitant meningioma and intracranial subependymoma. Subependymomas are rare, benign, slow-growing tumors and represent only 0.2% to 0.7% of intracranial tumors[13-16]. These tumors most often arise in the fourth ventricle (50%-60%) and the lateral ventricles (30%-40%)[14,15]. Most patients present with hydrocephalus as a consequence of ventricular obstruction or less commonly focal neurological dysfunction and seizures caused by mass effects[14,17]. The main purpose of surgery is to maximize the removal of the tumor[18]. In the published case series, there were satisfactory postoperative mortality and morbidity rates from supratentorial subependymomas[13-18]. In our case 1, resection of the left parietal meningioma and supratentorial subependymoma was accomplished *via* a single procedure. In a recent long-term outcome study of subependymoma, no patients exhibited a deterioration of performance status or tumor recurrence at medium to long-term follow-up[16]. As with the single subependymoma, the patient in our case also had a good prognosis after operation, and there was no evidence of recurrence at the last follow-up. Although the extremely rare coexistence of meningioma and subependymoma in our case may be an incidental event, the intrinsic relationship of these two tumors might require future investigation.

Until now, our understanding of the coexistence of meningioma and pituitary adenoma is based on occasional case reports[2-4,12,19-54]. We reviewed all the reported cases that were available to us, and the information was summarized in Table 2. The mean age of patients was 54.6 years (range: 26-82 years), and there were 39 women and 14 men among the published cases (female:male = 2.79:1). Our 4 cases also showed a female tendency (female:male = 3:1), and the mean age was 42.8 years (range: 29-52 years). A preference for parasellar, suprasellar and sphenoid ridge localization was found with 27 reported cases (50.94%)[2-4,20-23,25,26,30,32-37,42,47-49,51,54]. Consistently, the meningioma of our cases was in the petroclival, sellar and sphenoid wing regions.

The most common type of pituitary adenoma with coexisting meningioma among reported cases was a non-secreting tumor (21/53, 39.62%) followed by growth hormone-producing tumor (17/53, 32.08%). In this paper, all 4 cases were non-functioning pituitary adenoma, representing the most common type. Although prolactinomas are the most frequent pituitary adenomas in general, the higher prevalence of

Table 2 Summary of coexisting meningioma and pituitary adenoma

No.	Ref.	Age	Sex	Symptom	Size of meningioma, cm	Type of meningioma	Location of meningioma	Type of pituitary adenoma	History of radiation	Treatment	Postoperative therapy
1	Love *et al*[19], 1955	65	F	NA	NA	Meningothelial	Sylvian fissure	Non-secreting	NA	NA	NA
2	O'Connell[20], 1961	47	F	Failing vision in right eye	NA	Meningothelial	Tuberculum sellae	Non-secreting	No	TC (MN + PA)	No
3	Kitamura *et al* [21], 1965	66	F	Headache and impaired vision	NA	Meningothelial	Sphenoid wing	Non-secreting	No	TC (MN + PA)	No
4	Probst[22], 1971	48	F	NA	NA	Meningothelial	Suprasellar	ACTH-producing	NA	NA	NA
5	Brennan *et al* [23], 1977	36	M	Blurred vision of right eye	NA	Transitional	Sphenoid wing	Non-secreting	No	TC (MN + PA)	Radiotherapy
6	Bunick *et al*[24], 1978	57	M	Intermittent right-sided headache, acromegaly	6 × 6	Fibrous	Right frontal lobe	GH-producing	No	TC (MN + PA)	No
7	Hainer *et al*[25], 1978	72	M	NA	NA	NA	Suprasellar	GH-producing	NA	NA	NA
8	Deen *et al*[26], 1981	75	F	Chronic dementia, only had postmortem examination	NA	NA	Right sphenoid ridge	NA	NA	Autopsy	No
9	Hyodo *et al*[27], 1982	52	F	Acromegaly, diabetes mellitus, headache, right hemiparesis and right impaired visual acuity	NA	Fibrous and meningothelial	Left parietal region	GH-producing	No	TC (MN) + TS (PA, 4 mo later)	No
10	Irsy *et al*[28], 1985	59	F	NA	NA	NA	Centro-parietal	GH-producing	NA	NA	NA
11	Ohata[29], 1985	50	F	Acromegaly, visual disturbance, headache, vomiting and a floating sensation	4 × 4 × 4	Transitional	Falcotentorial junction	GH-producing	No	TS (PA) + TC (MN, 2 mo later)	Bromocriptine, acetyl-cortisone, desiccated thyroid
12	Yamada *et al* [30], 1986	52	F	Headache, disturbance of visual acuity and galactorrhea	NA	Meningothelial	Sphenoid ridge (parasellar)	Non-secreting	No	TC (PA + MN)	Bromocriptine
13	Honegger *et al* [31], 1989	37	F	Marked alopecia	2.5	Meningothelial	Right temporal pole	PRL-producing	No	TC (MN)	Bromocriptine
14	Honegger *et al* [31], 1989	49	F	Acromegaly persisted after radiotherapy	3	Meningothelial	Left parasagittal	GH-producing	Yes	TS (PA) + TC (MN, 2 mo later)	No
15	Honegger *et al* [31], 1989	74	M	Recurrence of pituitary adenoma	1.5	NA	Left parietal	Non-secreting	Yes	TS (PA) + TC (PA, 5 yr later)	No
16	Zentner *et al*	46	M	CT demonstration of a large	1.5	Transitional	Planum sphenoidale	PRL-producing	No	TC (PA + MN)	No

	[32], 1989			intrasellar and suprasellar space-occupying lesion							
17	Zentner *et al* [32], 1989	63	F	Ataxia	NA	Meningothelial	Sphenoid wing	Non-secreting	No	TS (PA) + TC (MN, 1 mo later)	No
18	Zentner *et al* [32], 1989	61	F	Frontal headache	NA	Meningothelial	Infradiaphragmatic	Non-secreting	No	TS + TS (PA + MN) + TC (PA, 1 d later)	No
19	Partington *et al* [2], 1989	26	M	Evaluation of persistent symptom of Cushing's disease; left temporal hemianopsia (10 yr later)	NA	Meningothelial	Tuberculum sellae	ACTH-secreting	Yes	TS (PA) + TS (PA, 8 yr later) + TC (1 yr later)	No
20	Uno *et al*[33], 1991	70	F	Headache and acromegaly	NA	Meningothelial	Sphenoid ridge	GH-producing	No	TC (MN + PA)	No
21	Cannavo *et al* [34], 1993	47	F	Acromegaly, diminished visual acuity, weakness and headache	NA	Meningothelial	Right latero- and retrosellar	GH-producing	No	TC (PA + MN)	No
22	Abs *et al*[35], 1993	47	F	Aphasia and temporary right-sided hemiparesis	NA	Meningothelial	Tuberculum sellae	PRL-producing	No	TC (MN) + TS (PA, 3 mo later)	No
23	Abs *et al*[35], 1993	61	F	A toxic thyroid adenoma, Cushing's disease	NA	Meningothelial; transitional	Frontal convexity; occipital convexity	ACTH-producing	No	TC (MN)	No
24	Abs *et al*[35], 1993	45	F	Amenorrhea and galactorrhea	NA	NA	Temporal fossa	PRL-producing	No	No surgery	No
25	Abs *et al*[35], 1993	45	F	A toxic multinodular goiter and suspicion of acromegaly	NA	NA	Parietal convexity	GH-producing	No	TC (Schwannoma) + TS (PA)	No
26	Abs *et al*[35], 1993	82	F	Bitemporal hemianopsia	NA	NA	Sphenoid ridge; parasellar	Non-secreting	No	TS (PA)	No
27	Abs *et al*[35], 1993	61	F	Headache	2.6	NA	Choroid plexus (right lateral ventricle)	PRL-producing	No	No surgery	No
28	Abs *et al*[35], 1993	51	F	Unilateral palpebral edema and exophthalmos	NA	NA	Sphenoid wing	Non-secreting	No	Embolization (MN)	No
29	Gorge *et al*[36], 1993	53	M	Progressive impotence, decrease of libido and left-sided defective vision	NA	NA	Para- and suprasellar	PRL-producing	NA	TC (PA + MN)	No
30	Laun *et al*[37], 1993	61	F	Deteriorating vision of the left eye and bitemporal hemianopia	NA	Meningothelial	Tuberculum sellae	Non-secreting	NA	NA	No
31	Mathuriya *et al* [38], 2000	58	F	Acromegaly	NA	NA	Parasagittal	GH-producing	No	TC (PA + MN)	Radiotherapy
32	Maiuri *et al*[39], 2005	49	M	Acromegaly, right hemiparesis	3.5	NA	NA	GH-producing	No	TC (MN) + TS (PA, 3 mo later)	No
33	Maiuri *et al*[39], 2005	63	F	Left hemiparesis, bitemporal visual field defect	4	NA	NA	Non-secreting	No	TC (MN) + TS (PA, 1 mo later)	No

34	Curto *et al*[40], 2007	61	F	Acromegaly and visual field impairment	3.5	NA	Right frontal	GH-producing	No	NA	No
35	da Costa *et al* [41], 2007	45	M	Generalized malaise and weight loss for a few months. Progressive headache, vomiting and gait ataxia for 2 wk	NA	NA	The fourth ventricle	PRL-producing	No	TC (MN)	Bromocriptine
36	Prevedello *et al* [42], 2007	52	F	Unremitting headache and profound right temporal visual field loss	1.0 × 0.6	NA	Right planum sphenoidale	Non-secreting	No	TS (PA + MN)	No
37	Basu *et al*[43], 2010	39	M	Frontal headaches and erectile dysfunction with loss of libido	NA	Meningothelial	Left cavernous sinus	PRL-secreting	No	TC (MN)	No
38	Furtado *et al* [12], 2010	53	M	Headache for 2 yr and altered sensorium for 2 wk	6.0 × 5.5 × 5.0	Meningothelial	Parasagittal	Non-secreting	No	TS (PA) + TC (MN)	No
39	Guaraldi *et al* [44], 2012	46	F	Acromegaly	2.3 × 2.6 × 2.6	NA	Left parasagittal	GH-producing	No	TS (PA) + TC (MN, 10 yr later) + TS (Recurrence PA, 8 mo later)	No
40	Ramirez *et al* [45], 2012	61	F	Headache and acute hydrocephalus for 2 mo	1.0 × 1.0	Psammomatous	Left anterior clinoidal	Silent corticotroph adenoma subtype II	No	TC (MN) + TS (PA, 1 mo later)	No
41	Masoodi *et al* [46], 2013	65	M	Acromegaly	NA	NA	Frontal parasagittal	GH-producing	No	TS (PA) + TC (MN)	No
42	Mahvash *et al* [47], 2014	36	F	Frontal headache and extended right visual field loss	2.0 × 2.0 × 2.5	NA	Tuberculum sellae	Non-secreting	No	TS (PA + MN)	No
43	Karsy *et al*[48], 2014	70	F	Altered mental status, mutism and incontinence	3.6 × 4.1 × 4.5	Fibroepithelial	Tuberculum sellae	Non-secreting	No	TS (PA + MN)	No
44	Ruiz-Juretschke *et al*[49], 2015	61	M	Progressive visual loss and bitemporal hemianopia for 6 mo	2.0 × 2.2 × 2.2	Meningothelial	Tuberculum sellae and planum sphenoidale	Non-secreting	No	TS (PA + MN) + TS (residual tumor)	No
45	Ben Nsir *et al* [50], 2016	61	F	Loss of vision for 3 yr and occasional episode headache	NA	NA	Foramen magnum	Non-secreting	No	TC (MN) + [TS + TC] (PA)	No
46	Lim *et al*[51], 2016	65	F	Visual symptoms and an episode of self-resolving vertigo	1.5 × 1.3 × 1.3; 0.6 × 0.5	NA	Tuberculum sellae and olfactory groove	Non-secreting	No	TS (PA + MN)	No
47	Amirjamshidi *et al*[3], 2017	37	F	Oligomenorrhea for 8 mo, headache, diplopia and progressive visual impairment	3.0 × 2.5 × 2.0	NA	Suprasellar	PRL-secreting	No	TC (MN)	No
48	Amirjamshidi *et al*[3], 2017	42	M	Acromegaly, visual acuity decreased and bitemporal hemianopia	3.0 × 3.0 × 2.0	NA	Suprasellar	Non-secreting	No	TS + TC (coexisting tumors)	No
49	Herrero-Ruiz *et al*[52], 2017	35	F	Chagas disease and acromegaly	2.0 × 2.0 × 1.2	NA	Left parietal parasagittal	GH-producing	No	TS (Only PA)	No

50	Kumaria *et al* [53], 2017	46	F	Lethargy, sleep disturbance, personality change and mild daily right-sided headache for 18 mo	NA	Transitional; Atypical	Right frontal and left temporal regions	Mammosomatotroph cell adenoma (PRL- and GH-secreting)	Yes	TC (MN) + TC (MN) + TS (PA)	No
51	Zhao *et al*[54], 2017	58	F	Acromegaly and headache	NA	NA	Sellar	GH-producing	No	TS (PA) + TC (MN, 3 mo later)	No
52	Zhao *et al*[54], 2017	58	F	Acromegaly	NA	NA	Sellar	GH-producing	No	TS (PA) + TC (MN, 4 mo later)	No
53	de Vries *et al*[4], 2019	75	F	Depression, fatigue and unintended weight loss	NA	Meningothelial	Suprasellar	Non-secreting	No	TS (PA + MN)	No

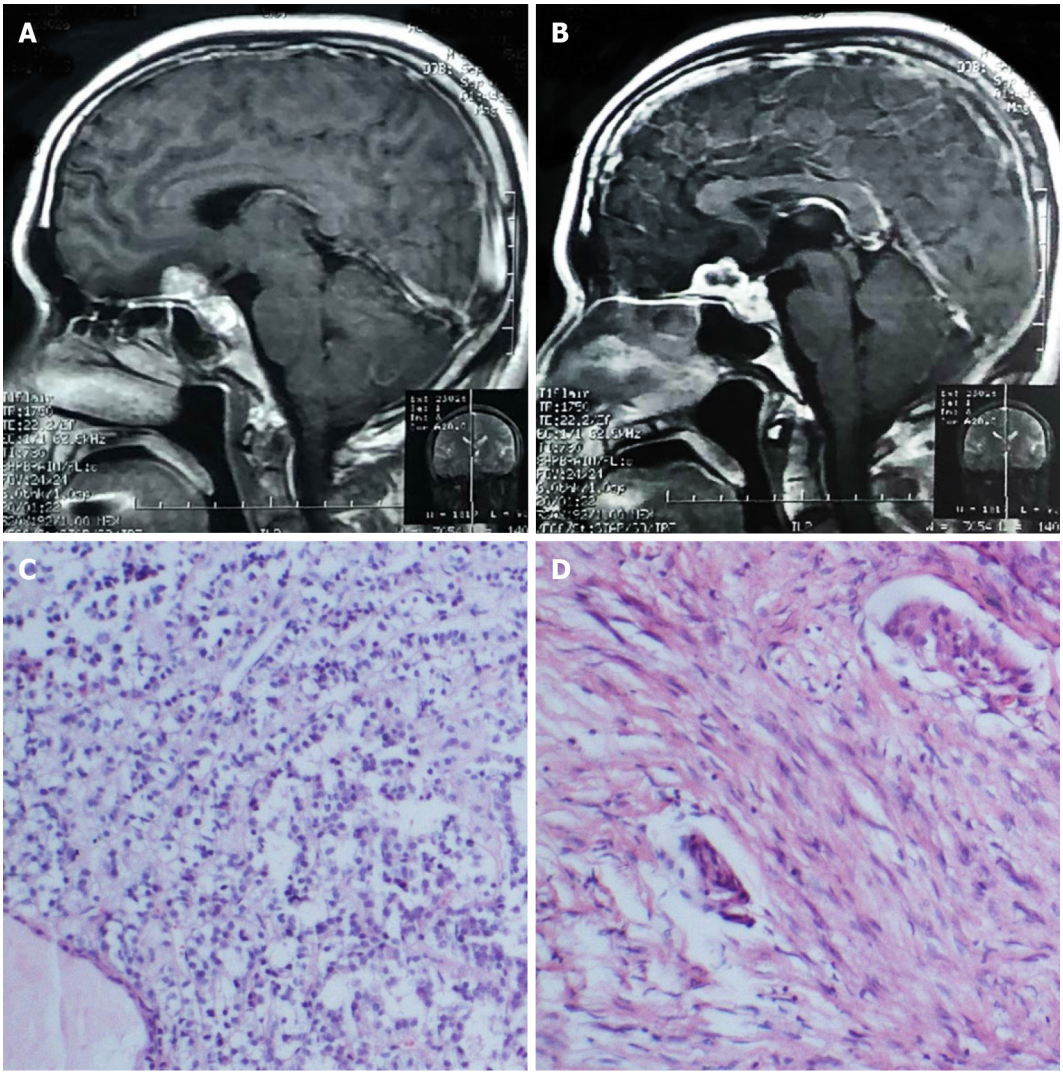
ACTH: Adrenocorticotropic hormone; CT: Computed tomography; GH: Growth hormone; F: Female; M: Male; TC: Transcranial surgery; TS: Transsphenoidal surgery; MN: Meningioma; NA: Not available; PA: Pituitary adenoma; PRL: Prolactin.

acromegaly in patients with coexisting meningioma has led some authors to propose an association between growth hormone-producing adenomas and meningioma[29,34,40,42,49]. They suggested that persistently elevated growth hormone might stimulate arachnoid cap cells to play a role in the development of meningioma[24,39,51]. A recent study suggested that patients with acromegaly were at increased risk of meningioma[55].

Some researchers have tried to explore the association between meningiomas and pituitary tumors. The early reports suggested that this phenomenon was related to history of irradiation for pituitary tumors[2]. However, many cases with no history of radiotherapy were reported as well[24,27,29,48,49], including our cases. Therefore, Curto *et al*[40] suggested that the coexistence of meningioma and pituitary adenoma was a coincidental phenomenon. However, there was a higher proportion of involvement of chromosome 14 and 22 in estrogen receptor positive de novo meningiomas[56]. Similar genetic changes shared by two unrelated tumors found on the same chromosome may explain their coexistence[12].

Moreover, due to the indolence of benign tumors, a significant portion of this coexisting tumor population may remain undiagnosed[12]. MRI is useful for the diagnosis of the coexistence of two intracranial tumors but has limited significance for adjacent pituitary adenoma and meningioma[4]. Histological results are necessary for diagnosis because other preoperative findings cannot support accurate diagnosis[54]. For example, some reported cases were coexisting sellar meningioma and pituitary adenoma[4,42,47-49,54]. Because of the close location of the two tumors, it is difficult to produce an accurate diagnosis by preoperative imaging, as in our case 3. It deserves special attention because the two different types of tumors were not definitely diagnosed before surgery but later when the pathologist's results were obtained. The patient in case 3 developed hypopituitarism after the operation, and this complication was also reported in other similar cases[47,49].

Traditionally, the treatment of these two coexisting tumors required one craniotomy[30,31,33] or two separate operations using two different approaches[2,32,35,39]. Prevedello *et al*[42] performed a single endoscopic expanded endonasal approach in patients with coexisting tuberculum sellae meningiomas and pituitary adenoma. In our opinion, surgical strategies should be decided according to the characteristics of the coexisting tumors (*e.g.*, location, size and adjacent neurovascular structures) and the clinical



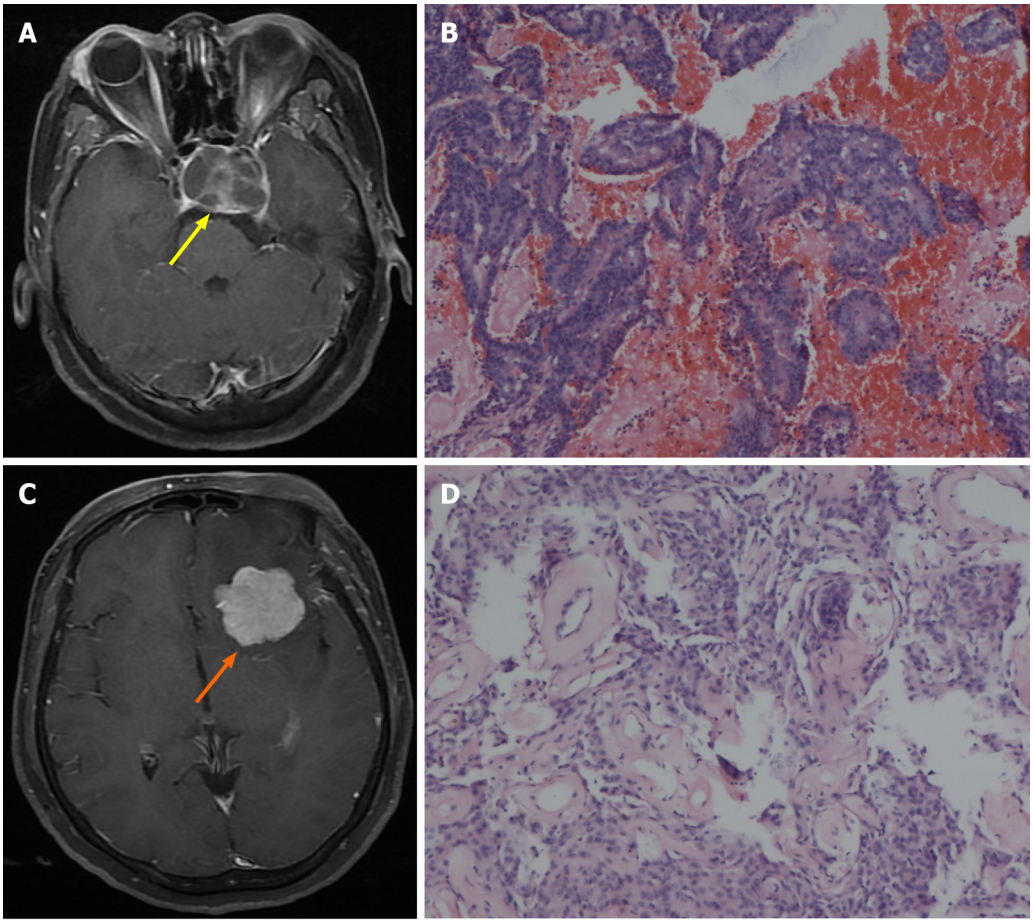
DOI: 10.12998/wjcc.v10.i13.4249

Figure 3 Histopathological examination showed a meningioma and non-functioning pituitary adenoma. A and B: Mid-sagittal contrast magnetic resonance imaging showed space-occupying lesions located in the planum sphenoidale and sellar region; C: Histological examination revealed pituitary adenoma; D: Histological examination revealed meningioma.

features of individual patients (*e.g.*, symptoms and systemic conditions). Like the single pituitary adenoma, the patient with coexisting meningioma and pituitary adenoma had a favorable prognosis using the retrospective case reports. Postoperative endocrine reexamination should be periodically monitored at the endocrinology outpatient department, especially in patients with postoperative hormone imbalance after surgery. However, as this study is a retrospective analysis and the case numbers are limited, we cannot draw strong conclusions.

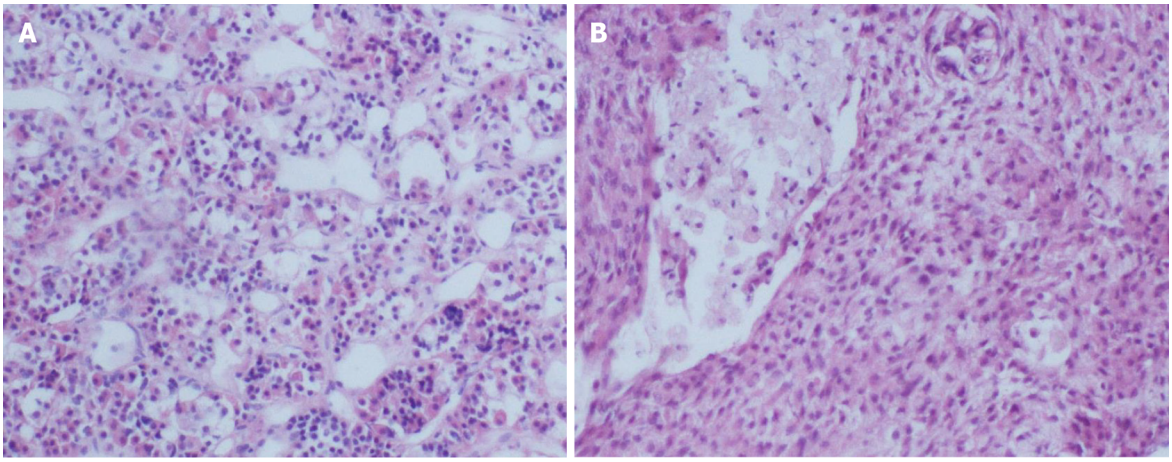
CONCLUSION

The simultaneous occurrence of meningioma and other intracranial benign tumors is a rare clinical event, and histological examination is necessary for their accurate diagnosis. Neurosurgeons should select the appropriate surgical strategy according to the clinical features of individual patients, which may provide the patient with a more favorable prognosis.



DOI: 10.12998/wjcc.v10.i13.4249

Figure 4 Histopathological examination showed a meningioma and non-functioning pituitary adenoma. A and C: Axial contrast magnetic resonance imaging showed a mass (yellow arrow) in the sellar and suprasellar region and a well-circumscribed mass (orange arrow) in the left sphenoid ridge; B and D: Postoperative histological examination revealed pituitary adenoma and meningioma.



DOI: 10.12998/wjcc.v10.i13.4249

Figure 5 Postoperative histopathological examination showed a meningioma and non-functioning pituitary adenoma. A and B: Histological examination revealed pituitary adenoma and meningioma.

FOOTNOTES

Author contributions: Hu TH, Wang R, Duan YZ, Liu T and Han S performed the data acquisition and prepared the figures; Hu TH, Wang HY and Song YF prepared the manuscript and followed up the patients; Yu JH and Wang ZX performed histopathology examination of the patients; Hu TH and Han S designed the report; All authors contributed to the article and approved the submitted version.

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