Coexisting Non-functioning Pituitary Macroadenoma and Sellar-Suprasellar Lipoma: A Case Report and Literature Review

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ABSTRACT

Intracranial lipomas are benign tumors that may occasionally be found in the suprasellar cistern while pituitary adenomas are far more common brain tumors. Pituitary adenomas may rarely coexist with other intracranial tumors in the sellar-suprasellar region. We share a unique case of a patient with coexisting non-functioning pituitary adenoma and sellar-suprasellar lipoma presenting with blurring of vision.

We report a 55-year-old male presenting with a two-year history of blurring of vision with findings of a 2.7 x 3.0 x 3.2 cm homogeneously enhancing lobulated isointense mass on the sellar-suprasellar region. Hormonal workups revealed low cortisol and mildly elevated prolactin. He initially underwent endonasal transsphenoidal excision of the tumor which revealed to be a lipoma on histopathology. Due to minimal improvement of vision from the subtotal excision, he underwent repeat surgery through the transcranial approach which in turn showed a pituitary adenoma.

The co-occurrence of two sellar-suprasellar tumors with different histology is rare, as most of the evidence is based on only a handful of case series. Intracranial lipomas result from persistence and abnormal differentiation of the meninx primitiva during the development of the subarachnoid cisterns. On the other hand, pituitary tumorigenesis is still largely unclear but appears to involve multiple tumor suppressor genes, oncogenes, cell cycle deregulation factors, and miRNAs. Given the differing pathogenesis of each tumor type, the coexistence may only be coincidental. The best surgical approach in this situation is unknown but the focus is on complete excision of the adenoma.

Keywords: pituitary adenoma, suprasellar lipoma, coexistence

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INTRODUCTION

Intracranial lipomas are rare benign tumors with an estimated incidence of 0.06-0.46% of all intracranial tumors.[1] Most commonly, it occurs in the midline in the pericallosal region but may occasionally be found in the suprasellar cistern, which accounts for 15% of all intracranial lipomas and 0.4% of all tumors that may be found in the said area.[1-3] In contrast, pituitary adenomas are far more common tumors with an approximate prevalence of 16.7%.[4] These tumors may be non-functioning or hormonally-secreting and may present with symptoms relating to mass effect and/or hormonal hypersecretion and deficiency.[5]

Pituitary adenomas may rarely coexist with other intracranial tumors such as meningiomas, glial neoplasms, medulloblastomas, lymphomas, and schwannomas.[6,7] Unfortunately, the pathogenesis is still poorly understood but may be weakly attributed to common genetic linkages.[6] Even more infrequent is the coincidence of a functioning or non-functioning pituitary adenoma with another distinct tumor

particularly in the sellar-suprasellar area, such as lymphoma[7], craniopharyngioma[8-10], meningioma[11-16], schwannomas and gangliocytomas[16], Rathke's cleft cyst[16], and chondroma[16]. To the authors' knowledge, we have not encountered any published literature reporting the coexistence of a histopathology-confirmed pituitary adenoma and sellar-suprasellar lipoma.

In this study, we report a very rare case of a patient with non-functioning pituitary adenoma (NFPA) and coincidental sellar-suprasellar lipoma initially presenting with blurring of vision.

CASE PRESENTATION

History and Clinical Presentation

A 55-year-old male seafarer, with known hypertension for five years maintained on losartan, presented in the outpatient department with a two-year history of progressive bitemporal blurring of vision. He did not complain of headache, dizziness, vomiting, weakness, fatigue, and sensorial changes. Moreover, there was no clinical manifestation of hormonal hypersecretion nor deficiency. During a routine medical clearance for employment a year prior to admission, he was advised to undergo a cranial magnetic resonance imaging (MRI) which revealed a 2.7 x 3.0 x 3.2 cm homogeneously enhancing lobulated sellar-suprasellar mass isointense to gray matter in T1 (T1W) and T2 weighted (T2W) images expanding the sella. The mass compresses the optic chiasm and there is abutment of the cavernous segments of both internal carotid arteries without evidence of encasement. Moreover, there was dehiscence of the left sphenoid sinus roof resulting in minimal intrasinus tumoral extension (Figure 1).

On physical examination (PE), he was seen awake, alert, conversant, follows commands, and not in distress. He had stable vital signs and unremarkable systemic PE. Neurologic PE showed a visual acuity of 20/70 with correction to 20/50 on the right and 20/50 with correction to 20/40 on the left. Likewise, there were bitemporal visual field cuts. On fundoscopy, there was a note of temporal optic disc pallor on both eyes. The rest of the neurologic exam was normal.

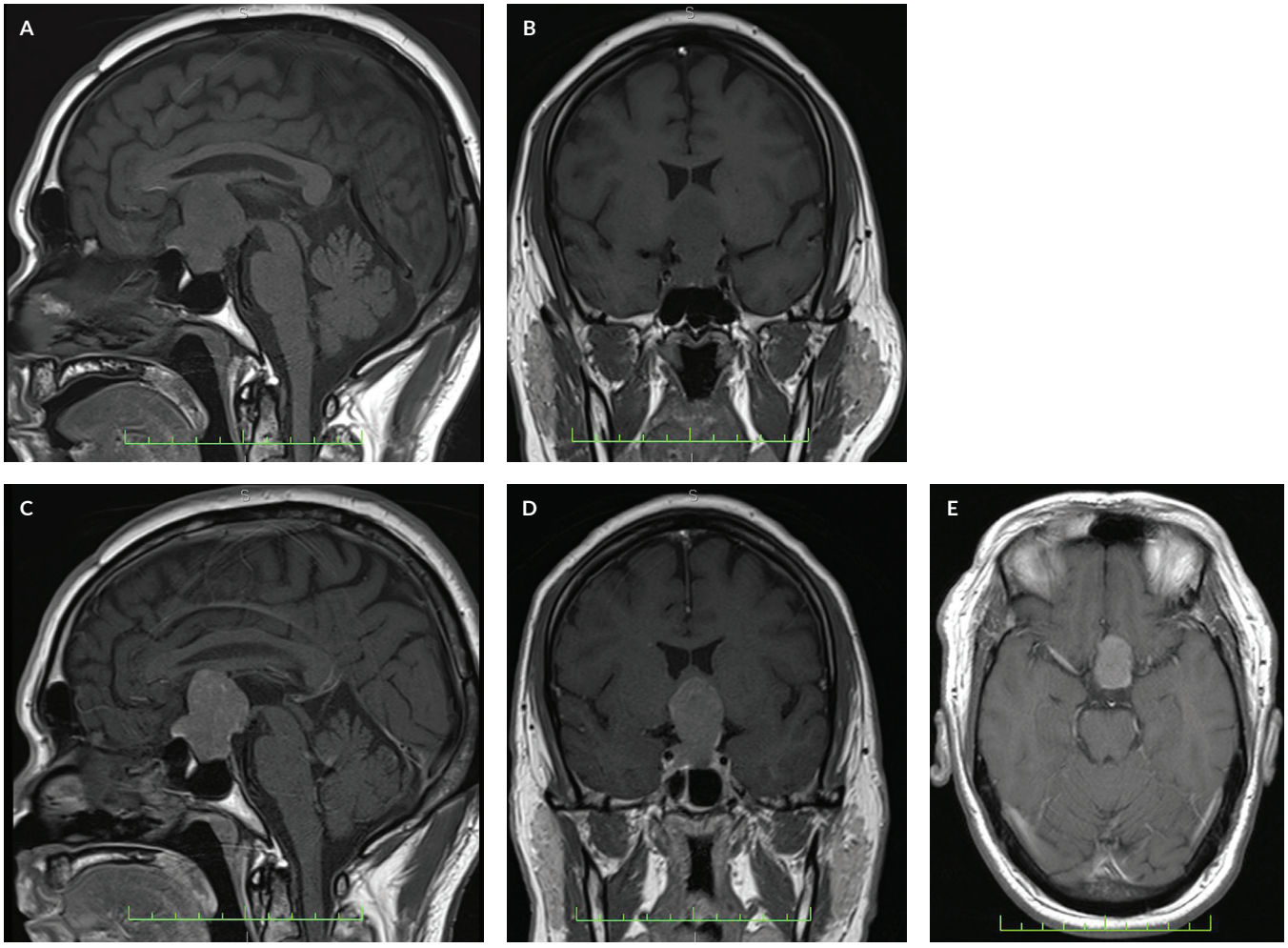

Figure 1. ***T1 weighted sequences of the patient's cranial MRI with and without contrast.*** **(A)** Sagittal without contrast, **(B)** Coronal without contrast, **(C)** Sagittal with contrast, **(D)** Coronal with contrast, **(E)** Axial with contrast.

Hormonal Work-ups

Due to this sellar-suprasellar mass, hormonal work-ups were facilitated prior to planned admission for surgery to assess baseline pituitary function. These revealed slightly elevated prolactin, normal free T4, normal TSH, normal free T3, low cortisol, elevated FSH, normal LH, normal testosterone, and normal IGF-1 for age and sex (Table 1). Likewise, visual perimetry using a central 24-2 threshold test was done which showed good reliability OU, severe generalized defects OU and contiguous points of depression at the temporal visual field affecting fixation OU, consistent with bitemporal hemianopsia. Furthermore, an optical coherence tomography scan revealed thinning of the temporal retinal nerve fiber layer below the fifth percentile OU.

Table 1. Baseline Hormonal Work-ups prior to Surgery

Hormone	Result	Normal Value	Unit
Prolactin	27.34	3.46-19.4	ng/mL
Free T4	11.5	9.01-19.05	pmol/L
Free T3	4.78	2.89-4.88	pmol/L
TSH	1.0933	0.35-4.94	uIU/mL
8AM Cortisol	51.14	154-638	nmol/L
FSH	42.03	0.95-11.95	mIU/mL
LH	1.61	0.57-12.07	mIU/mL
Testosterone	12.81	7.66-24.82	nmol/L
IGF-1	126.35	56-201	ng/mL

Initial Impression

With these findings, he was presumptively diagnosed with a non-functioning pituitary macroadenoma with compressive optic neuropathy, central hypocortisolism, and hyperprolactinemia from stalk effect. He was maintained on prednisone 7.5 mg/day divided into morning and afternoon doses.

Course during the First Admission

Due to limitations on operating room schedules brought about by the COVID-19 pandemic, he was only able to undergo endonasal transsphenoidal excision of the mass on the sixth hospital day. Intraoperatively, there was a note of yellowish fatty non-suctionable lobulated tumor extruding out of the durotomy site, grossly compatible with a lipoma (Figure 2). Subtotal tumor excision was done until the diaphragma sella descended. The aggregate diameter of the surgical specimen was 4 cm with the largest fragment measuring 3.0 x 2.0 x 0.5 cm. Histopathology of the tissue confirmed lipoma (Figure 3).

Post-operative course was unremarkable without note of postoperative diabetes insipidus, infection or cerebrospinal fluid leak. However, there was only minimal subjective improvement of vision in the left eye without resolution of the visual field cuts on both eyes. Because of this, a cranial computed tomography (CT) scan with contrast was done on the eighth hospital day (second day post-surgery) (Figure 4) which revealed a significant residual tumor at the sellar-suprasellar area measuring 3.9 x 2.7 x 3.8 cm with extensions and mass effects: (1) superiorly: compresses the third ventricle; (2) inferiorly: abuts and causes thinning of the sellar floor and posterior clinoid with erosion of the anterior clinoids; (3) anteriorly: extends into the interhemispheric fissure; and (4) bilaterally: cannot be clearly delineated from the cavernous sinuses. At this point, the consideration was a persistent sellar-suprasellar lipoma with compressive optic neuropathy. Repeat surgery through a transcranial approach was offered as an attempt to completely resect the mass during the same admission but the patient did not consent. Repeat 8AM cortisol on the ninth hospital day (third day post-surgery) was normal at 481.7 nmol/L (NV: 154-638). Prednisone was withheld and he was sent home the next day.

Course during the Second Admission

Eventually, he gave consent to do the planned repeat surgery and he was readmitted a month later. Preoperatively, he was given intravenous hydrocortisone as stress dose coverage. He underwent left pterional craniotomy and subtotal excision of tumor on the second hospital day.

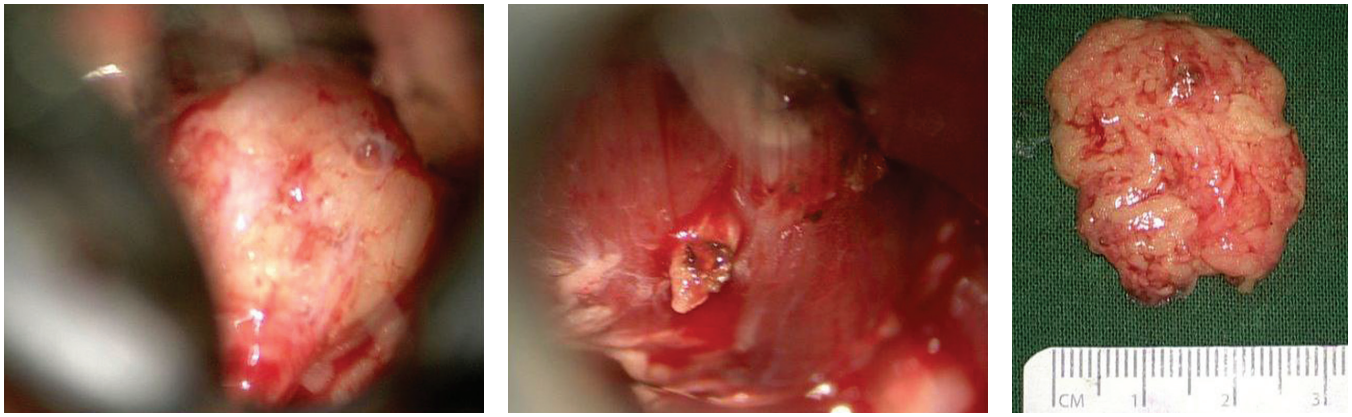

Figure 2. Gross features of the tumor during durotomy and upon excision.

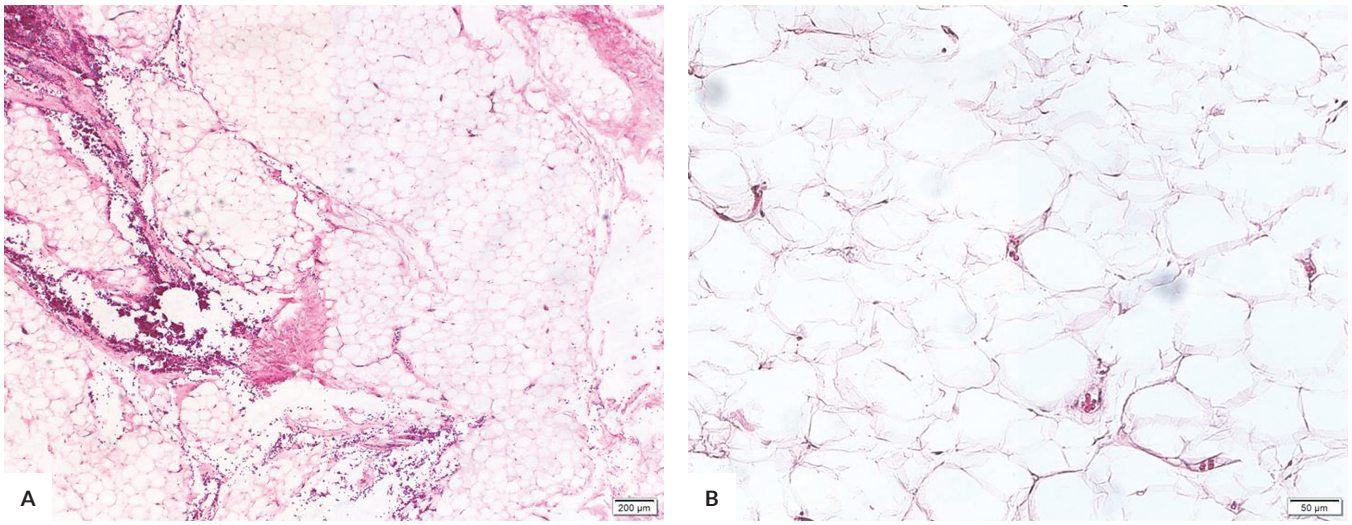

Figure 3. ***Histopathology of the excised tumor after initial surgery.*** **(A)** Low Power and **(B)** High Power Objective view. *[Hematoxylin and Eosin (H & E) staining]*

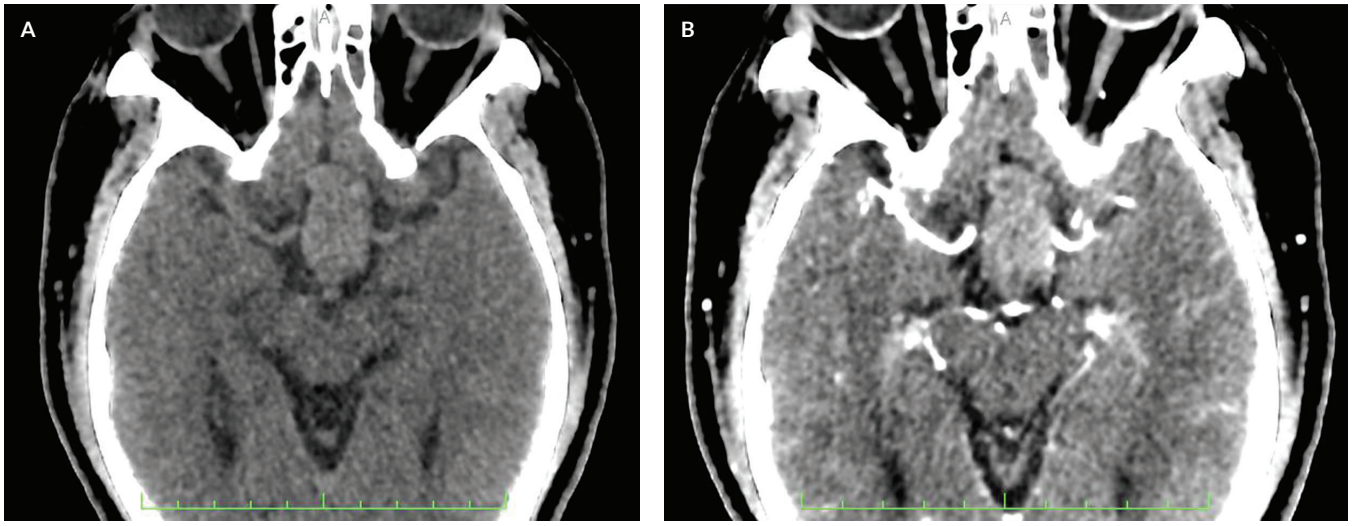

Figure 4. ***Post-operative cranial CT scan (2nd day post-surgery).*** **(A)** Axial without contrast, **(B)** Axial with contrast.

Intraoperative findings revealed a predominantly cream soft white suctionable fairly vascular tumor and yellowish tumor with some areas that are firm and fatty. Histopathology of the provided specimen with aggregate diameter of 1.7 cm later revealed a pituitary adenoma (Figure 5). Note that the excised tissue during the second surgery was distinct from the one removed during the first surgery. Postoperatively, he reported improvement in visual fields and visual acuity on both eyes. However, he developed transient diabetes insipidus necessitating intermittent administration of desmopressin 50 mcg during episodes of polyuria. On the sixth hospital day (fourth day post-surgery), repeat 8AM serum cortisol yielded low results at 24.45 nmol/L (NV: 154-638) necessitating resumption of prednisone 5 mg/day. He was discharged stable on the seventh hospital day.

DISCUSSION

The co-occurrence of two sellar-suprasellar tumors with different histology is rare. Most of the evidence is based on a handful of case reports/series (Table 2).[7-14] Although non-functioning pituitary adenomas are relatively common, co-existence with an already rarely encountered sellar-suprasellar lipoma makes this case unique.

Intracranial lipomas are uncommon tumors that are brought about by persistence and abnormal differentiation of the meninx primitiva during the development of the subarachnoid cisterns.[1-3] Moreover, they are sometimes associated with congenital brain malformations such as dysgenesis of the corpus callosum and dysgenesis of the septum pellucidum, among others.[2,3] A genetic link has

Table 2. Case Reports / Series Involving Co-occurrence of Pituitary Adenoma and another Sellar-Suprasellar Tumor

Case Report / Series	Age / Sex	Imaging	Type of Pituitary Adenoma	Co-occurring Tumor	Proposed Mechanism of Co-occurrence
Ban et al. (2017)[7]	74/M	2.7 x 2.5 x 2.0 cm soft tissue mass in the pituitary fossa with suprasellar extension	FSH-producing	Diffuse large B-cell Non-Hodgkin's lymphoma	Coincidental or development of lymphoma triggered by hormonal effects of the adenoma.
Jin et al. (2013)[8]	47/F	Enhancing mass in the sellar and suprasellar areas with a cystic mass in the prepontine cistern	Non-functioning	Craniopharyngioma	Tumorigenesis unclear but may be explained by metaplastic mechanisms.
Hasegawa et al. (2021)[9]	51/M	2.7 x 2.5 x 3.5 cm enhancing sellar-suprasellar mass compressing the optic chiasm	Non-functioning	Craniopharyngioma	Coincidental; craniopharyngioma may be derived from a metaplastic change in a pituitary adenoma; hybrid cells containing characteristics of adenoma and craniopharyngioma
Snyder et al. (2019)[10]	49/F	Heterogeneous lobulated solid suprasellar mass with an inferior hyperintense superior hyperintense components on T1 contrast sequences	ACTH-secreting	Craniopharyngioma	Coincidental; abnormal embryogenic differentiation; paracrine growth effects from pituitary hormones; possible presence of hybrid cells containing characteristics of adenoma and craniopharyngioma.
Mahvash et al. (2014)[11]	36/F	Intrasellar lesion with compression of optic chiasm with a contrast-enhancing extra-axial lesion with a dural tail	Non-functioning	Meningioma	Cause unknown but higher incidence is detected after radiation therapy.
Lim et al. (2016)[12]	65/F	1.6 x 1.3 x 2.0 cm pituitary lesion with adjacent 1.5 x 1.3 x 1.3 cm homogeneously enhancing lesion and 0.6 x 0.5 cm lesion with a dural tail in the olfactory groove	Non-functioning	Meningioma	No explanation
Bao et al. (2021)[13]	62/F	Homogeneously enhancing sellar mass with adjacent homogeneous enhancing extra-axial lesion with dural tail	Non-functioning	Meningioma	Coincidence or caused by activation of the signal pathway of receptor tyrosine kinases.
Bao et al. (2021)[13]	56/F	2.8 x 2.0 x 1.6 cm moderate inhomogeneously enhancing intra- and suprasellar mass involving the right cavernous sinus			
Amirjamshidi et al. (2017)[14]	37/F	3.0 x 2.5 x 2.0 cm well-delineated round tumor, T1W isointense and T2W hyperintense within the sella and another suprasellar lesion with the same intensity	Prolactinoma	Meningioma	Activation of signaling pathways of receptor tyrosine kinases or one tumor may secrete a growth factor that stimulates growth of the other.
Amirjamshidi et al. (2017)[14]	42/M	Intrasellar lesion isointense in T1W and T2W images enhancing homogeneously with a suprasellar lesion 3.0 x 3.0 x 2.0 cm with same intensity in T1W and T2W enhancing after contrast	GH-secreting	Meningioma	
Zhao et al. (2017)[15]	58/F	3.5 x 1.9 x 2.0 cm slightly hyperintense on T1W and iso to hyperintense in T2W with heterogeneous enhancement	GH-secreting	Meningioma	Growth hormone secretion may induce meningioma growth.
Zhao et al. (2017)[15]	58/F	1.12 x 0.54 x 1.07 cm short signal on T1W and T2W imaging with low signal surrounding a high signal on T2W	GH-secreting	Meningioma	
Gezer et al. (2020)[16]	34/F	1.2 x 0.75 cm solid well-defined mass in the anterior part of the pituitary with an enhancing mass with a dural tail within the sellar region	ACTH-secreting	Meningioma	Paracrine growth effects of other pituitary hormones due to hormone receptor immunoreactivity found within the meningiomatous component.

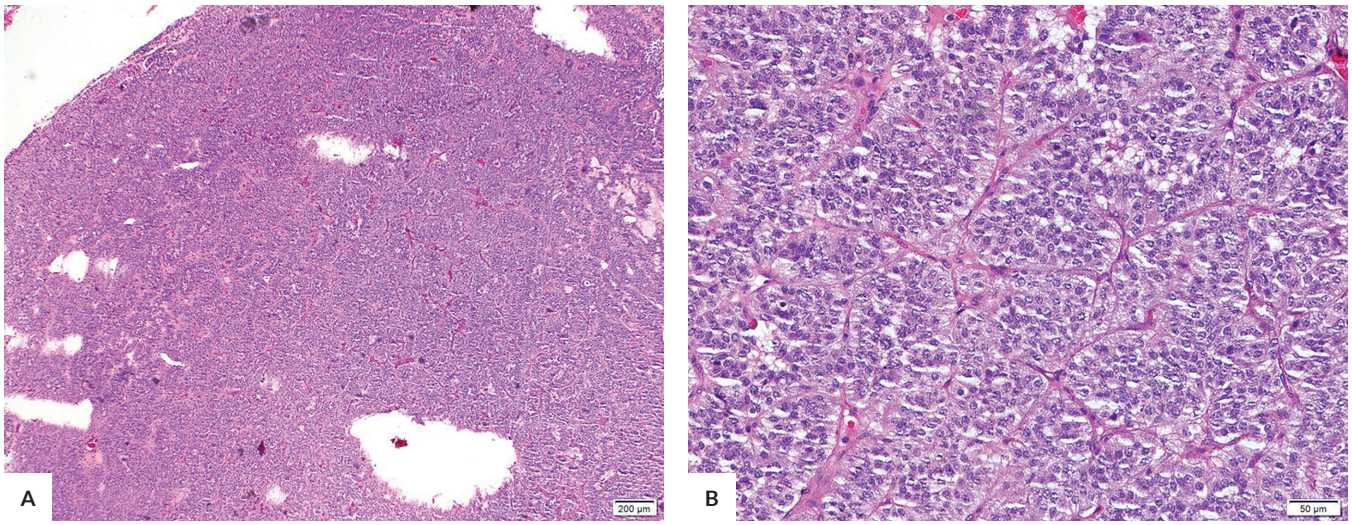

Figure 5. ***Histopathology of the excised tumor after reoperation.*** **(A)** Low Power and **(B)** High Power Objective view. *[Hematoxylin and Eosin (H & E) staining]*

been demonstrated in about two-thirds of lipomas with a mutation in the HMGA2 gene located on 12q14.3 seen in a subgroup of lipomas.[17] Moreover, structural rearrangements of chromosomes such as in 12q13-15 region, 6p21-23 region and 13q portion loss have also been associated with lipoma occurrence.[17] Intracranial lipomas often occur at specific locations that correspond to their types such as pericallosal lipoma (45%), quadrigeminal lipoma (25%), suprasellar cistern/hypothalamic lipoma (15%), cerebellopontine angle lipoma (10%), Sylvian fissure lipoma (5)%, and choroid plexus lipoma (rare).[18] Clinical manifestations depend on the location but the affected patients are usually asymptomatic.[1-3] Among those who are symptomatic, epilepsy is the most reported in about half of supratentorial lipomas.[1,3] Like most intracranial lipomas, suprasellar or hypothalamic lipomas are usually asymptomatic but larger-sized lesion may cause neurologic deficits.[18] In a retrospective review by Alkhaibiry and colleagues which included six patients with suprasellar lipomas out of 246 with documented intracranial lipomas, no one presented with endocrinologic dysfunction including pituitary or hypothalamic endocrinopathy.[18] However, their team cited some case reports that suprasellar lipomas may cause hypothalamic dysfunction such as precocious puberty, obesity, and hypothermia. In this same review, most of the lesions are smaller than 10 mm with only one patient presenting with a lesion measuring 11 mm at its largest dimension.[18] Since patients are often asymptomatic, they are usually discovered incidentally during work-up for another presumed illness as in the case of our patient.[1-3]

Intracranial lipomas have characteristic appearance on both CT scan and MRI. On one hand, on cranial CT scan, they are usually non-enhancing and have low attenuation ranging from -60 to -100 HU (Figure 6A).[3,19,20] In addition, they may also be accompanied by calcifications, have soft lobulated appearance, and conform to adjacent anatomy.[3] On the other hand, on cranial MRI, they usually present with hyperintensity on T1W sequencing (Figures 6B, 6C and 6D) and intermediate-to-low signal on T2W sequencing.[19,21] Moreover, they have low-signal intensity on fat-saturated sequences, have no enhancement on T1W with contrast, and can produce blooming on susceptibility weighted imaging due to susceptibility artifact.[3]

Since the patients are asymptomatic in most instances and since vessels and nerves may course through these lipomas making surgery dangerous and technically difficult, they are managed conservatively.[1-3,18] Rare exceptions include cosmesis due to a frontal lipoma and relief of hydrocephalus from a quadrigeminal cistern lipoma.[2]

NFPAs, on the other hand, are much more common sellar-suprasellar lesions. The pathogenic mechanisms of pituitary tumorigenesis are still largely unclear but appears to be a complex process involving extrinsic and intrinsic factors.[22,23] Recent evidence shows the vital roles of tumor suppressor genes, oncogenes, cell cycle deregulation, and miRNAs in pituitary tumorigenesis.[23] However, the cause or effect in tumor development remains largely unanswered. Usual signs/symptoms of NFPAs are caused by mass effect to surrounding structures such as visual impairment, headache, and cranial nerve dysfunction.[5] Occasionally, they may also present with clinical findings of hormonal deficiency.[5] Cranial MRI with gadolinium enhancement is currently the preferred imaging for diagnosis.[5,24] They typically appear isointense to gray matter on both T1W and T2W sequences.[24] However, larger lesions may present heterogeneously due to areas of cystic change, necrosis or hemorrhage.[24] Contrast enhancement demonstrates moderate to bright enhancement.[24] Surgery is the primary therapeutic modality for symptomatic pituitary adenomas.[5]

To our knowledge, there has been no prior published report on the co-occurrence of a pituitary adenoma and

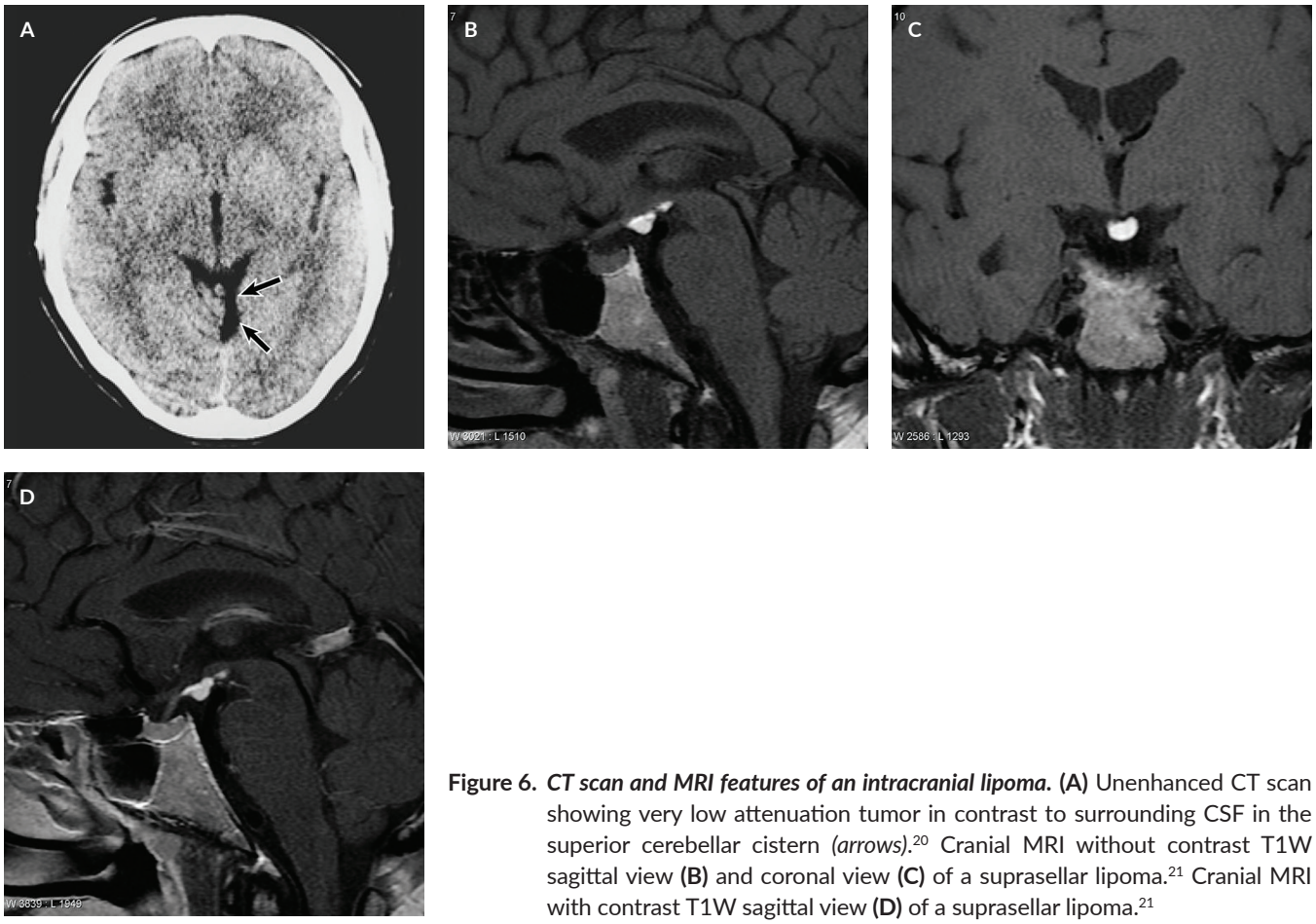

Figure 6. ***CT scan and MRI features of an intracranial lipoma.*** **(A)** Unenhanced CT scan showing very low attenuation tumor in contrast to surrounding CSF in the superior cerebellar cistern *(arrows)*.[20] Cranial MRI without contrast T1W sagittal view **(B)** and coronal view **(C)** of a suprasellar lipoma.[21] Cranial MRI with contrast T1W sagittal view **(D)** of a suprasellar lipoma.[21]

sellar-suprasellar lipoma or their potential association. Given the differing pathogenesis of both tumors, we infer that this coexistence of a pituitary adenoma and sellar lipoma may be simply coincidental.

Nothing in the initial presentation alerted us of the peculiarity of the case as there was absence of any evidence pointing to a diagnosis of a lipoma. Going back to our patient, he presented solely with progressive blurring of vision. Since intracranial lipomas are usually asymptomatic, this was mainly attributed to the classic mass effect of NFPA. Furthermore, comparing the patient's MRI results to the usual MRI appearance of both NFPAs and intracranial lipomas, the predominantly isointense presentation without a distinct area of hyperintensity points to a clear diagnosis of a solitary NFPA (Figures 1 and 4) rather than a coexisting NFPA and lipoma. Hence, the finding of fatty tissue during endoscopic excision was truly surprising.

The ideal surgical approach of these coexisting tumors is unknown. However, given the conservative management recommendation for most intracranial lipomas, the main consideration in surgical planning must be the total excision of the pituitary adenoma if possible. Our patient underwent an initial endoscopic transsphenoidal surgery followed by a transcranial approach during the subsequent admission. Had we caught on to the presence of the coexisting tumors on preoperative imaging, it may have been possible to have minimized the surgery to just one procedure, focusing on the excision of the pituitary adenoma and not stopping with the removal of the lipoma.

CONCLUSION

We reported a very rare case of coexisting NFPA and sellar-suprasellar lipoma. The pathogenic mechanism for this occurrence and possible association is unclear given the differing pathogenesis of each tumor type. Intracranial lipomas are managed conservatively while surgery is still the first-line treatment for symptomatic pituitary adenoma. The best surgical approach in coexisting disease is unknown but the focus may be on complete excision of the pituitary adenoma.

Statement of Authorship

All authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

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