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Case Report

Pituitary lymphoma appearing 9 years after pituitary adenoma resection

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ABSTRACT

Background: Pituitary lymphomas (PLs) are very rare, accounting for <0.1% of all intracranial tumors. Of which, PL that is associated with PL is even rarer. Here, we describe a case of PL of a 51-year-old woman that appeared 9 years after pituitary adenoma resection.

Case Description: A 51-year-old woman presented with visual disturbance. She had a history of pituitary adenoma resected through endoscopic trans-sphenoidal surgery (eTSS) 9 years before. Although her previous annual follow-up did not show any signs of recurrence, she noticed visual disturbance. One month later, her visual acuity rapidly worsened with headache and fatigue, being referred to our hospital. On examination, she had bilateral quadrantanopia. Her laboratory data showed slightly increased prolactin levels. Magnetic resonance images showed a mass in the sella with suprasellar extension, so she underwent eTSS. The tumor had a fibrous, hard part and a soft gray part, and it was mostly resected. Visual symptoms improved transiently, but ophthalmoplegia appeared 2 weeks after surgery, indicating intrathecal dissemination. Histological analysis confirmed the diagnosis of T-lymphoblastic lymphoma. Positron emission tomography showed tracer accumulation at the pancreas, confirmed as lymphoma through biopsy. However, we could not determine which site of lymphoma was the primary site. She underwent chemotherapy, including cyclophosphamide, vincristine sulfate, doxorubicin hydrochloride, dexamethasone, and methotrexate. The patient died despite several months of treatment.

Conclusion: Recurrence of pituitary adenoma cannot be carelessly assumed from a pituitary growing mass after pituitary adenoma resection. PLs have poor prognosis due to their aggressive character. Immediate biopsy and confirmation of the diagnosis are necessary for the treatment of pituitary masses with aggressive features.

Keywords: Endoscopic trans-sphenoidal surgery, Pituitary adenoma, Pituitary lymphoma, Recurrence, T-lymphoblastic lymphoma

INTRODUCTION

Pituitary tumors account for 15% of all intracranial tumors.[28] Various tumors arise in the seller region, including pituitary adenoma, craniopharyngioma, teratoma, meningioma, glioma, and germ cell and metastatic tumors.[2,9] Of all the tumors that arise in the pituitary gland, pituitary lymphoma (PL) is very rare, accounting for <0.1% of all sellar tumors. Interestingly, few PLs arise coexistent with or after pituitary adenoma resection.[10] Only eight PL associated pituitary adenoma (PLPA) cases have been reported. So far, this tumor is described only in

case reports or case series, and only three cases occurred after resection of pituitary adenoma. Therefore, its etiology, pathophysiology, clinical course, and prognosis are not well known.[3,4,13,17,18,22,24,25] The manifestation of the disease mimics ordinary pituitary adenomas, and histological examination is essential for diagnosis and treatment. Here, we describe a very rare case of PL in the Sellar region that occurred 9 years after the resection of a pituitary adenoma.

CLINICAL SUMMARY

A 51-year-old woman presented to our hospital with blurred vision and a visual field defect. Nine years before the presentation, she visited the hospital due to visual disturbance. Her magnetic resonance (MR) images showed a mass in the sellar region compressing the chiasma [Figure 1]. She underwent endoscopic trans-sphenoidal surgery (eTSS), resulting in total resection of the sellar mass. Her vision completely recovered. Histological analysis of the tumor confirmed the diagnosis of nonfunctioning pituitary adenoma. Since then, she has been followed up with MR images annually.

A year before the presentation, she did not have any symptoms, and her MR images did not show tumor recurrence [Figure 1]. However, at this time, she experienced blurred vision and a visual field defect. A visual field test showed bitemporal quadrantanopia. Her blood test showed normal blood count and biochemistry. Her hormonal test showed slightly elevated prolactin (PRL) (38.4 ng/mL) and low insulin-like growth factor-1 (101 ng/mL, −1 Standard deviation) levels. A triple stimulation test produced normal responses. She was immunocompetent and had no history of irradiation or chemotherapeutic drug use. Her MR images showed a sellar mass extending to the suprasellar region, compressing the optic chiasma upward. The mass was heterogeneously enhanced with gadolinium [Figure 1] but no other brain lesions were found. A pituitary adenoma recurrence was suspected so she underwent tumor resection through eTSS. The tumor was heterogenous, having a fibrous, hard part and a soft gray part. Although the tumor was mostly removed, the lesion attached to the right cavernous sinus remained. The intraoperative histological analysis of the resected specimen suggested a pituitary adenoma. After surgery, her visual symptoms transiently improved. Since her postoperative laboratory findings suggested panhypopituitarism, hormone supplementation was started. In contrast to intraoperative histological findings, the histological analysis demonstrated tumor cell invasion into the normal pituitary gland. Tumor cells had different nuclear sizes with numerous mitotic figures. The immunological analysis showed that tumor cells were positive for CD3, CD4, CD8, and terminal deoxynucleotidyl transferase, which

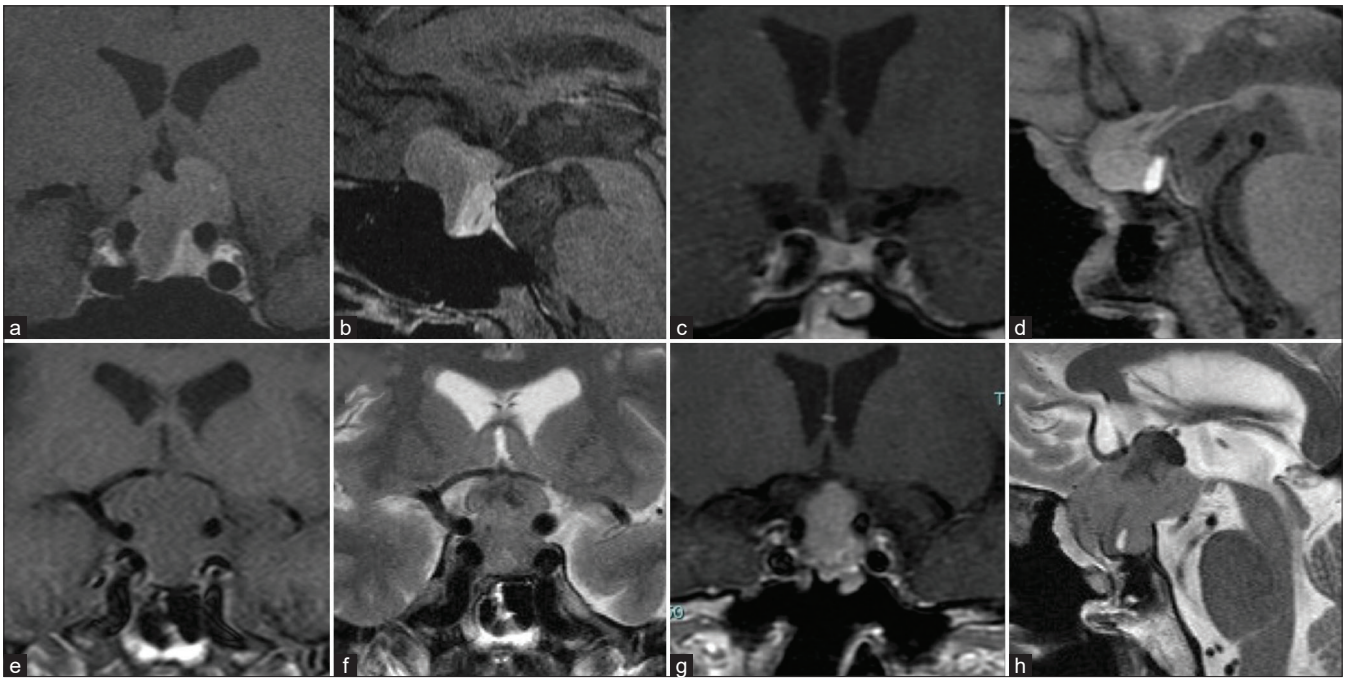

Figure 1: (a-d) Gadolinium-enhanced magnetic resonance (MR) images before the 1st operation and 1 year before the presentation. The heterogeneously enhanced tumor is observed in the sella with suprasellar extension before surgery. No tumor recurrence is apparent at 8 years after surgery. MR images at presentation. An extensively enlarged tumor in the sellar and suprasellar portion with optic nerve compression. (e-h) The tumor is shown isointense on T1-weighted images (WI), having iso-low mixed intensity on T2-WI, and heterogeneously enhanced by gadolinium.

confirmed the diagnosis of T-lymphoblastic lymphoma (T-LBL) [Figure 2].

Two weeks after surgery, she experienced ptosis on her right eyelid, ophthalmoplegia, dysarthria, and dysphagia. MR images showed pial enhancement in the brain stem in a fluid-attenuated inversion recovery image, diagnosed as tumor meningeal dissemination [Figure 3]. Full body positron emission tomography (PET) showed tracer accumulation in the pancreas [Figure 4], also confirmed as the same type of lymphoma by biopsy through endoscopic ultrasound-guided fine-needle aspiration. She underwent chemotherapy, including cyclophosphamide, vincristine sulfate, doxorubicin hydrochloride, dexamethasone, methotrexate, and 40 gray of whole brain irradiation. She incompletely recovered from ptosis and ophthalmoplegia. Despite several months of treatment, she suffered from paraparesis due to spinal dissemination of the tumor and died from respiratory failure 10 months after the second surgery.

DISCUSSION

PL, whether primary or metastatic, is very rare, accounting for <1% of all intracranial tumors. PL can arise *de novo* or in association with pituitary adenomas or lymphocytic hypophysitis.[15] Primary PL (*de novo*) (PPL) reportedly has a slight female predominance among people in their 50s.[28] However, it has male predominance in immunocompromised patients.[11] Histologically, a predominance of B-cell against T-cell origin of 5.5:1 is reported for PPL, similar to peripheral lymphomas.[5] However, PLPA has different characteristics from PPL. PLPA has no sex preference, and the mean age at onset is 62.7 ± 9.8 years old. Five cases were reported as a collision tumor with pituitary adenoma, and four cases after adenoma resection. The latency to lymphoma presentation varies from 4 to 25 years. Most importantly, the histology of tumor cell type is balanced between T-cell and B-cell origin with a ratio of 5:4, which is a clearly different characteristic from PPL or peripheral lymphomas [Table 1].

The neurological manifestation of PL resembles pituitary adenoma due to its dependency on tumor location and its extension. For example, common physical manifestations include visual acuity decline, temporal hemianopsia, diplopia, and headache. Pituitary insufficiency can also be commonly seen. Cranial nerve (CN) palsies retroorbital pain may be observed in case of extension into the cavernous sinus or orbital apex. Due to anatomical localization, CN II

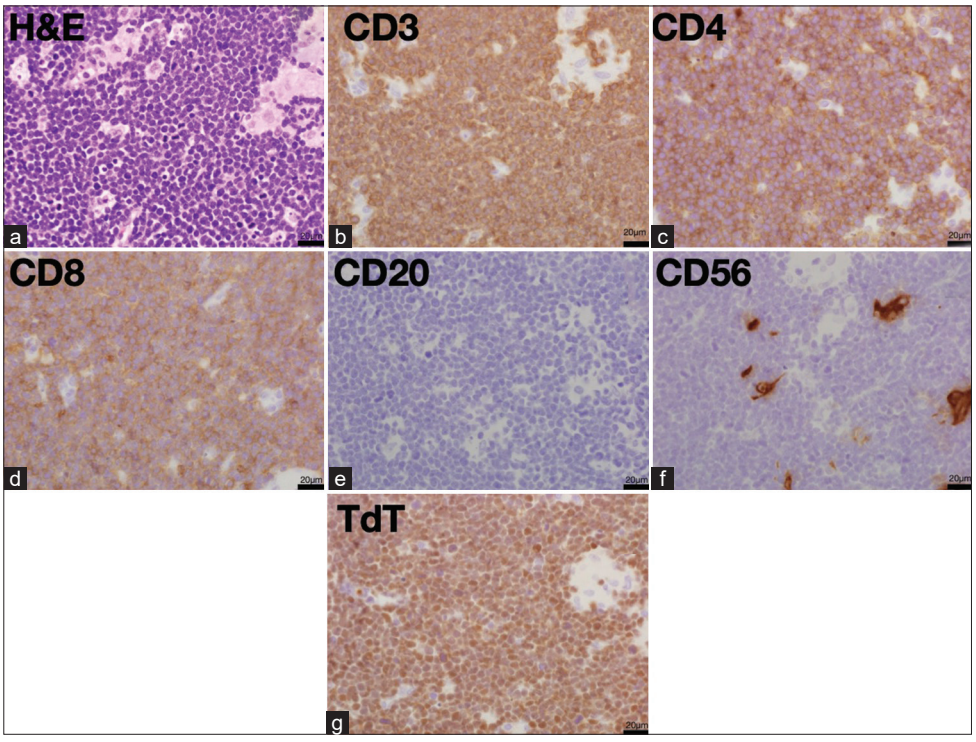

Figure 2: (a: Hematoxylin and eosin (H&E) stain, Magnifications×400) Histological analysis of resected tumor tissue. Tumor cells have round hyperchromatic nuclei and sparse eosinophilic cytoplasm. No apparent necrotic lesion is observed. (b: CD3, c: CD4, d: CD8, e: CD20, f: CD56, g: TdT) Immunohistochemical analysis reveals that tumor cells were positive for CD3, CD4, CD8, and terminal deoxynucleotidyl transferase (TdT) and negative for CD20 and CD56 Magnifications: ×400. Scale bars: 20 μm (a-g).

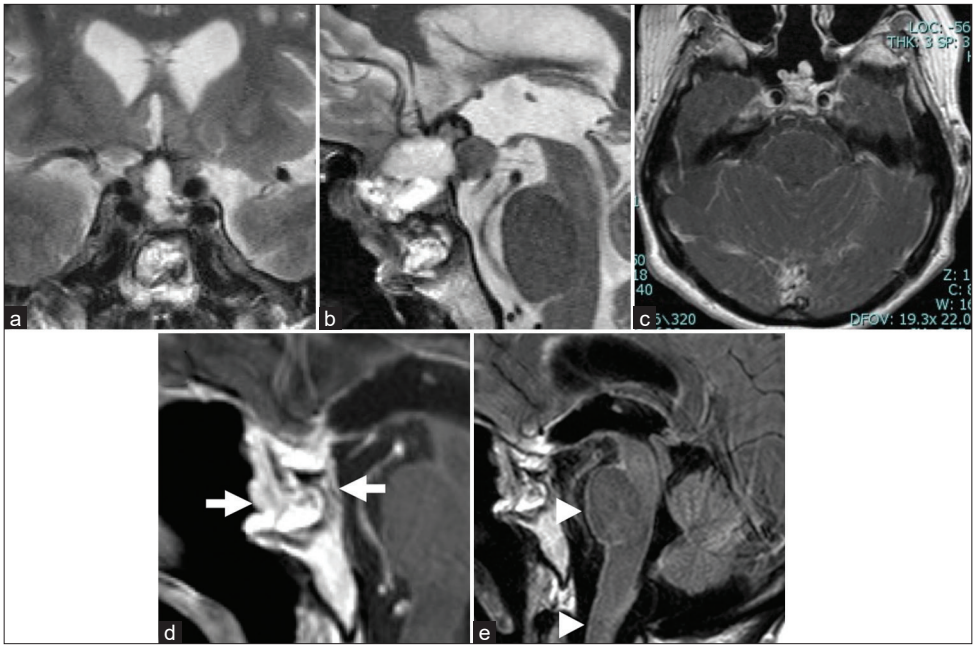

Figure 3: (a and b) Postoperative magnetic resonance (MR) images. T2-weighted image (WI) shows the residual tumor attached to the right cavernous sinus and suprasellar portion ([a] Coronal, [b] Sagittal). (c) Gadolinium-enhanced MR fluid-attenuated inversion recovery image taken 14 days after the second surgery shows pial enhancement on the brain stem and cerebellum and indicates intrathecal dissemination. (d and e) Gadolinium-enhanced T1-WI after chemoradiotherapy (11 months after the second surgery) shows an enhanced region in the sellar portion and clivus (arrows) and pial enhancement from the brain stem to the spinal cord (arrowheads).

is most involved, followed by CN III, VI, and, less frequently, CN V, IV, and VII. Endocrinological manifestations such as hypopituitarism and diabetes insipidus (DI) appear to be more common in PLs than in pituitary adenomas.[11] The radiological features of PLPA consist of an isointense lesion in T1-weighted image (WI), a hypointense lesion in T2-WI, and inhomogeneous enhancement with gadolinium. The tumor character is often aggressive as it frequently extends into the suprasellar region and cavernous and sphenoid sinus in most cases reported.[3,4,13,17,18,22,24,25] In our case, the tumor extended into the suprasellar region, compressing the optic nerve as in the other reported cases, and also extended into the sphenoid and cavernous sinus and even into the clivus. However, it is difficult to completely differentiate PLPA from pituitary adenoma recurrence by radiologic image alone. The important point is that this tumor rapidly extended into neighboring structures in <1 year, which may have been an indication of a different tumor type rather than adenoma recurrence. A biopsy is essential to diagnose this tumor. We reviewed MR images yearly for 9 years but mistakenly assumed that the growing sellar mass was a tumor recurrence and not a *de novo* PL. However, a new malignant tumor or metastasis should be considered as a differential diagnosis if the tumor has different characteristics compared with the original tumor.

The pathophysiology of lymphoma arising in the Sella is still controversial; it is unclear whether PL is derived from adjacent meningeal lymphoid tissue or is caused by the malignant transformation of normal lymphocytes migrating into the sella during inflammation.[17] There are some hypotheses on how lymphocytes migrate into the pituitary gland. First, the development of lymphoma in pituitary adenoma may have been induced by its own secreted hormones. Pituitary hormones such as PRL,[20] growth hormone,[12] thyroid-stimulating hormone,[6] and follicle-stimulating hormone (FSH) are reported to have mitogenic effects on lymphoma or lymphocytes through its correspondent receptors.[7] Abdelbaset-Ismail *et al*. reported that pituitary sex hormones, luteinizing hormone, FSH, and PRL show positive effects on proliferation, adhesion, and chemotaxis. In fact, PRL elevation due to stalk displacement was seen in most cases of PLPA since the suprasellar extension of the tumor was often prominent.[1] Furthermore, FSH, which is increased with aging and menopause, is reported to induce the proliferation of T-cells *in vitro*.[7] In two out of three case reports of PLPA that showed increased secretion of FSH, the histology of the tumor was confirmed as T-LBL.[25] Second, modulation of adhesion molecules in adenoma during inflammation may play a key role. Adhesion molecules such as L-selectin, CD44, integrins,

Table 1: Summary of reported cases of pituitary lymphoma associated with pituitary adenoma.

	Sex	Age	Histology	Secreted hormone	Timing of lymphoma presentation from pre-existing adenoma	Radiation before presentation	Postoperative radiation	Postoperative chemotherapy
Kuhn (1999)	F	67	T-LBL	FSH	25 years	-	+	-
Au (2000)	M	82	DLBCL	TSH	Simultaneous	-	+	-
Romeike (2008)	M	64	T-LBL	FSH	17 years	-	+	+
Martinez (2011)	F	71	DLBCL	GH	Simultaneous	-	+	-
Morita (2012)	M	60	DLBCL	None	4 years	+	-	+
Ban (2017)	M	74	DLBCL	FSH	Simultaneous	-	-	-
Gupta (2017)	F	55	T-LBL	ACTH	Simultaneous	-	-	-
Ren (2022)	M	41	DLBCL	PRL	Simultaneous	-	-	+
Present case	F	51	T-LBL	None	9 years	-	+	+

T-LBL: T-lymphoblastic lymphoma, DLBCL: Diffuse large B-cell lymphoma, FSH: Follicle-stimulating hormone, TSH: Thyroid-stimulating hormone, GH: Growth hormone, ACTH: Adrenocorticotropic hormone, PRL: Prolactin

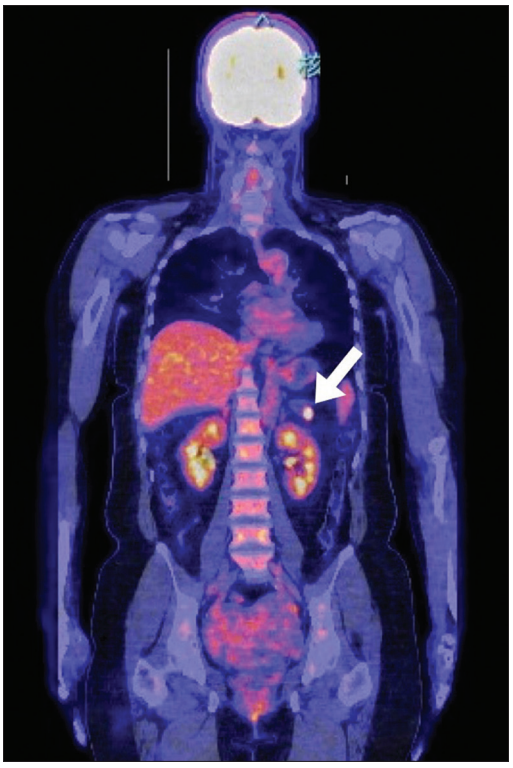

Figure 4: Positron emission tomography. Tracer accumulation is seen in the pancreas (arrow). No other tracer accumulation is apparent elsewhere.

and chemokine receptors are reported to mediate central nervous system infiltration of leukocytes.[26,29] Kern *et al*. reported that CD56/NCAM expression in T-cells exhibited a striking predilection for unusual anatomic site involvement, including the pituitary gland.[16] In fact, CD56/NCAM is physiologically expressed by natural killer cells and a subset of mature T-cells, and they are expressed when cells undergo malignant transformation.[8] Furthermore, neurotransmitters, neuropeptides, and cognate receptors that are usually present in the central nervous system are expressed in T-LBL and could affect the development and migration of lymphocytes into the pituitary gland.[21] However, further analyses of these adhesion molecules in the pituitary gland are necessary. Third, destruction of the blood–brain barrier (BBB) by previous surgery may also induce lymphocyte infiltration into the sella. Including the present patient four out of nine patients developed lymphoma postoperatively [Table 1]; thus, the surgery itself may have been a risk for developing PL. Furthermore, among the entry routes for T-LBL cells in the central nervous system, the BBB has been proposed to serve as a primary entry point for the blasts.[19] Radiation can also result in the development of various tumors. In the present series, one patient developed PL 4 years after irradiation to the ocular adnexal region.[23] In the present case, the BBB may have been damaged due to previous resection of the pituitary adenoma through eTSS. This may have made it easier for lymphocytes to migrate into the pituitary gland. Inflammatory response may have taken place at some points during tumor growth which may also have exacerbated infiltration of leukocytes into the sella by increased expression of certain adhesion molecules or receptors mentioned above. After migrating into the pituitary gland, pituitary hormones such as PRL and FSH may have contributed to the differentiation of lymphocytes into T-cells.

Treatment of PLs often includes surgical resection, chemotherapy, and radiotherapy. In most cases, surgical resection through eTSS is performed. Total or subtotal tumor resection increases more overall survival and progression-free survival than biopsy alone.[30] There is no evidence of benefit from postoperative chemotherapy and radiotherapy.[11] Postoperative radiotherapy with/or chemotherapy, including methotrexate, rituximab, cyclophosphamide, vincristine, and prednisolone, can be used against residual tumors depending

on the tumor cell lineage.[4,22,24] Nonetheless, the prognosis is often poor, and two out of nine PLPA cases died from the disease within a year [Table 1].

The present patient had a single tumor in the pancreas, confirmed by biopsy to be the same type of lymphoma in the sella. We could not fully determine whether the PL in our case was a primary lesion or due to metastasis. However, metastasis in the pituitary gland occurs mostly in the posterior lobe with manifesting DI.[27] This is due to the anatomical fact that the hypophyseal artery supplies the posterior lobe; thus, it receives direct blood supply from the systemic circulation. Meanwhile, the anterior lobe is supplied by the portal vessel system, an indirect supply from the lower infundibular stem.[14] On the contrary, the present patient had lymphoma mainly in the anterior lobe of the pituitary gland and did not show DI at presentation. Further, no other tumor except that in the pancreas was identified in PET. These findings indicated that lymphoma of the pituitary gland, in our case, was more likely to be the primary site. The present tumor showed a very aggressive character with rapid growth and invasion into the suprasellar region, clivus, and sphenoid sinus. The patient underwent tumor removal followed by chemotherapy and radiation, but the tumor disseminated into the whole central nervous system. Reviewing adenoma recurrence cases with different characteristics than the original tumor may unveil overlooked PLPA cases.

CONCLUSION

Herein, we reported a case of PLPA. PLPA has a poor prognosis due to its aggressive character. Recurrence cannot be assumed from a pituitary growing mass after pituitary adenoma resection. Immediate confirmation of the diagnosis by biopsy and general examination is necessary in case of a pituitary mass with aggressive features. Further studies are necessary to clarify the pathogenesis of lymphoma cell's invasion into the pituitary gland.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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