

What's in the Image? Pituitary Metastasis from Papillary Carcinoma of the Thyroid: A Case Report and a Comprehensive Review of the Literature

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What Is Known about This Topic?

- Sellar masses most commonly represent pituitary adenoma, and only rarely metastatic tumors, usually originating from breast or lung carcinoma. Accelerated growth of a sellar mass, rapid onset of diabetes insipidus or ophthalmoplegia indicate a metastatic nature of the lesion, particularly in the presence of primary malignancy. Papillary thyroid carcinoma (PTC) rarely metastasizes to the central nervous system.

What This Case Report Adds

- This case report highlights difficulties in diagnosing sellar masses when the primary malignancy is undiagnosed. Not only was the primary malignancy not known but there were no other distant metastases either. Of particular challenge was the management of the sellar metastasis. Due to proximity of vulnerable structures (compression of the optic nerve) and rapid deterioration of visual field acuity, the only possible solution in this case was surgical decompression of the optic nerves. Malignant tumors are known to be highly vascular and prone to bleeding, as was the case in our patient, which may lead to difficulties with the transsphenoidal approach. In our patient and in only 2 previously reported cases, the diagnosis of the sellar metastasis of PTC was confirmed by immunohistochemistry.

Key Words

Sellar mass · Papillary thyroid carcinoma · Sellar metastasis

Abstract

A 67-year-old female patient presented with visual field impairment and hyperprolactinemia. Imaging revealed a sellar and suprasellar mass and during the evaluation of the sellar

lesion, papillary thyroid carcinoma (PTC) was diagnosed by fine-needle aspiration biopsy in a long-standing euthyroid multinodular goiter. The patient did not have a previous history of PTC. Total thyroidectomy confirmed the diagnosis of PTC. Due to progressive visual loss, she underwent transcranial surgery for decompression of the optic chiasm. Pituitary metastasis from PTC was confirmed, histologically and immunohistochemically. In literature, overall 13 cases, includ-

ing ours, with PTC metastasis to the sellar region have been reported. Most were women, with a median age of 56 years. Two thirds of patients were previously diagnosed with PTC. The presence of other distant metastases was confirmed in less than half of the patients. Only 2 and our patient had immunohistochemical confirmation of PTC metastasis to the sellar region. The presenting signs and symptoms included: visual field defects, ophthalmoplegia, and anterior pituitary hormone deficiencies. In conclusion, this is a rare case of metastatic PTC to the sellar region unequivocally confirmed by immunohistochemistry in whom the disease first presented with a sellar and suprasellar mass.

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Introduction

Papillary thyroid carcinoma (PTC) is a common malignancy with a rising incidence possibly, but not solely, due to early diagnosis. PTC generally has a favorable prognosis, until distant metastases appear [1]. Distant metastases, primarily to lungs and bones, are rare and only exceptionally are the first manifestation of the disease. Central nervous system metastases comprise only a minor fraction, with the sellar region reported in a small number of individual cases [2]. The prognosis of a disseminated PTC is mostly influenced by the localization of metastasis and its proximity to vital and vulnerable structures. Sellar metastases present diagnostic and therapeutic challenges.

An intrasellar mass most likely represents a pituitary adenoma [3, 4]. Metastatic tumors represent a small fraction of sellar masses, but must always be considered. In most cases the nature of a metastasis to the sellar region is suggested by the primary tumor, but when the primary tumor is occult, sellar metastasis is usually diagnosed postmortem. Breast and lung cancers are the most common sources of metastases, but many other malignancies including PTC must also be considered.

Case Report

A 67-year-old female patient was referred to the neuroendocrine department for evaluation of a suspected pituitary tumor. Eight months earlier, she complained of visual field impairment and a CT scan reported an intrasellar and suprasellar tumor, 15 mm in diameter. Another small lesion in the anterior horn of the left lateral cerebral ventricle was reported as well. MRI confirmed the CT scan findings. She was then referred to a local endocrine specialist, who registered hyperprolactinemia (1,270 mU/l) and

initiated treatment with dopamine agonists. While on treatment for 6 months, symptoms persisted, the patient further felt unwell and there was no change in the tumor size. On repeated hormone evaluation while serum prolactin levels normalized, she developed anterior pituitary hormone deficiencies, with low levels of FSH, LH, and IGF-1, while fT₄, TSH and cortisol levels were reported normal. She was then referred to us for further evaluation.

On examination, a goiter with a palpable nodule in the left lobe, with no local lymphadenopathy, was found. Physical examination was otherwise unremarkable. The patient had a long-standing euthyroid multinodular goiter since the age of 20. She reported no compressive symptoms and no past ultrasound (US) reports were available regarding the size of the thyroid nodules during previous follow-up. Fine-needle aspiration biopsy (FNAB) was previously not performed. Family history was negative for thyroid malignancy. She was diagnosed with type 2 diabetes mellitus 5 years prior to this investigation and was well controlled by oral therapy.

Our patient did not have diabetes insipidus (DI). Hormonal evaluation revealed partial hypopituitarism with preserved thyroid and adrenal functions (fT₄ 9.6 ng/l, TSH 1.64 mIU/l, cortisol 289 nmol/l, ACTH 27.4 ng/l), but low FSH (14.8 mIU/l) and LH (2.1 mIU/l) and low IGF-1 (58.1 ng/ml). CEA and calcitonin were normal. Low-dose ACTH test (1 µg Synacthen) performed to assess the HPA axis showed adequate cortisol response (baseline cortisol 405 nmol/l, peak cortisol 709 nmol/l).

Neck US confirmed a large multinodular goiter, with a dominant nodule of 27 mm in the left lobe. No lymphadenopathy was found. US-guided FNAB of the dominant nodule reported PTC. CT of the lungs was unremarkable. MRI of the brain and the sellar region, 4 months after the initial imaging, revealed a sellar-suprasellar mass and a mass in the left lateral ventricle (fig. 1a–c). Visual field assessment disclosed bilateral defects (fig. 2). It was only after the diagnostic procedure – FNAB of the dominant thyroid nodule – which revealed PTC, that the possibility of a distant metastasis to the sella was considered. However, a coexisting pituitary lesion, which may be found in 2% of patients harboring a malignant disease, could not have been excluded.

The patient was referred to the endocrine surgeon for total thyroidectomy. Final histology after thyroidectomy confirmed the diagnosis of PTC. Macroscopically the left thyroid lobe was 19 g in weight, measured 50 × 30 × 30 mm, and was harboring a solid nodule 30 mm in diameter, encapsulated, light brown in color, of soft consistency. The right thyroid lobe was 12 g in weight, measured 40 × 30 × 20 mm, pink, with colloid glow, and was harboring a solid nodule 7 mm in diameter, light brown in color, elastic in consistency. Microscopical analysis of the left thyroid lobe revealed PTC, follicular variant, stage pT2, pNx, pMx, 30 mm in diameter, encapsulated, exhibiting signs of tumor-capsule invasion, harboring psammoma bodies. In the remainder of the lobe tissue, colloid-cystic, partially hyperplastic goiter was identified. The thyroid capsule was intact. Microscopical analysis of the right lobe revealed colloid-cystic partially hyperplastic thyroid goiter, and one focus of PTC was identified, 7 mm in diameter.

After thyroid surgery, while awaiting radioiodine scan, the patient complained of progressive visual loss, and she was without delay referred to the neurosurgeon for decompression of the optic chiasm. Thyroglobulin (TGB) levels after surgery were not measured. It would have been helpful if TGB was measured and a postoperative radioiodine scan was done; however, although

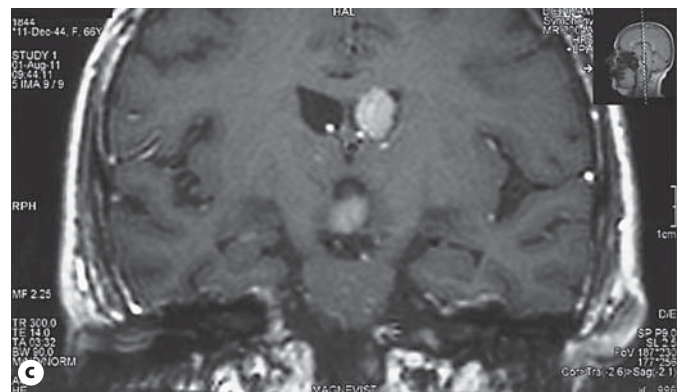
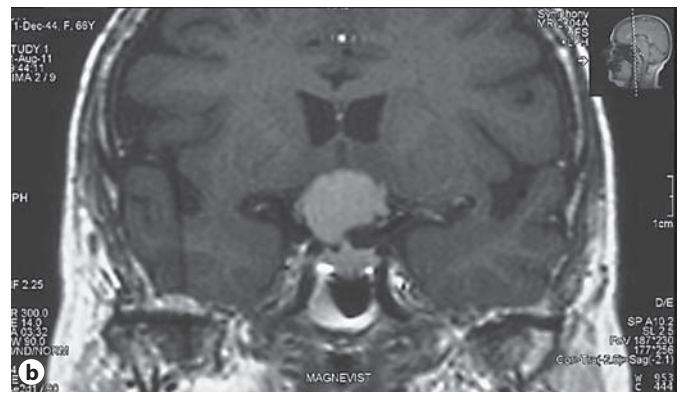
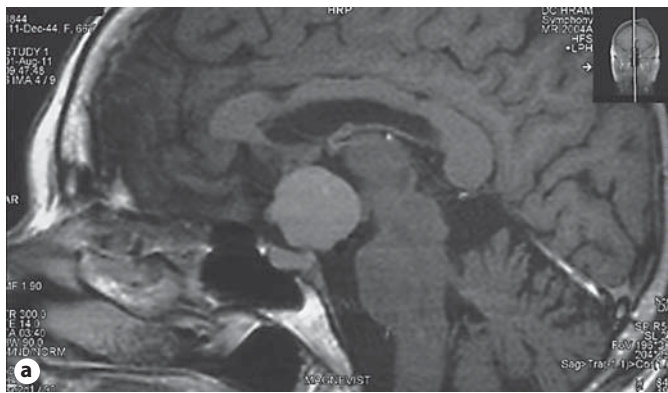


Fig. 1. MRI of the brain and the sellar region disclosing a narrow-waisted sellar-suprasellar mass and a mass in the left cerebral ventricle.

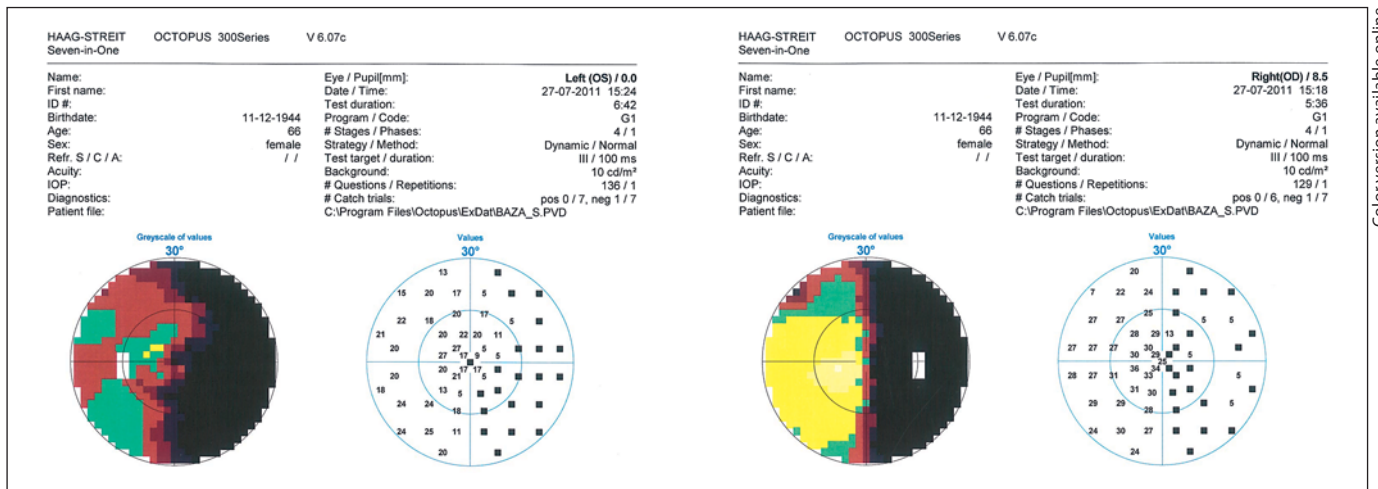


Fig. 2. Computerized perimetry disclosing bilateral visual field defects.

planned, due to the inability to obtain recombinant TSH (rhTSH) required for the expected central hypothyroidism, the scan was not performed. Furthermore, this would not have influenced the decision to proceed with pituitary surgery in order to decompress the optic nerves because of rapidly deteriorating visual loss.

Transcranial operation was performed and a large, fragile, highly vascular intrasellar tumor was removed. Suprasellar expansion into the third ventricle was observed and tumor compression of the pituitary stalk, anterior optical chiasm, and the right optical nerve was reported.

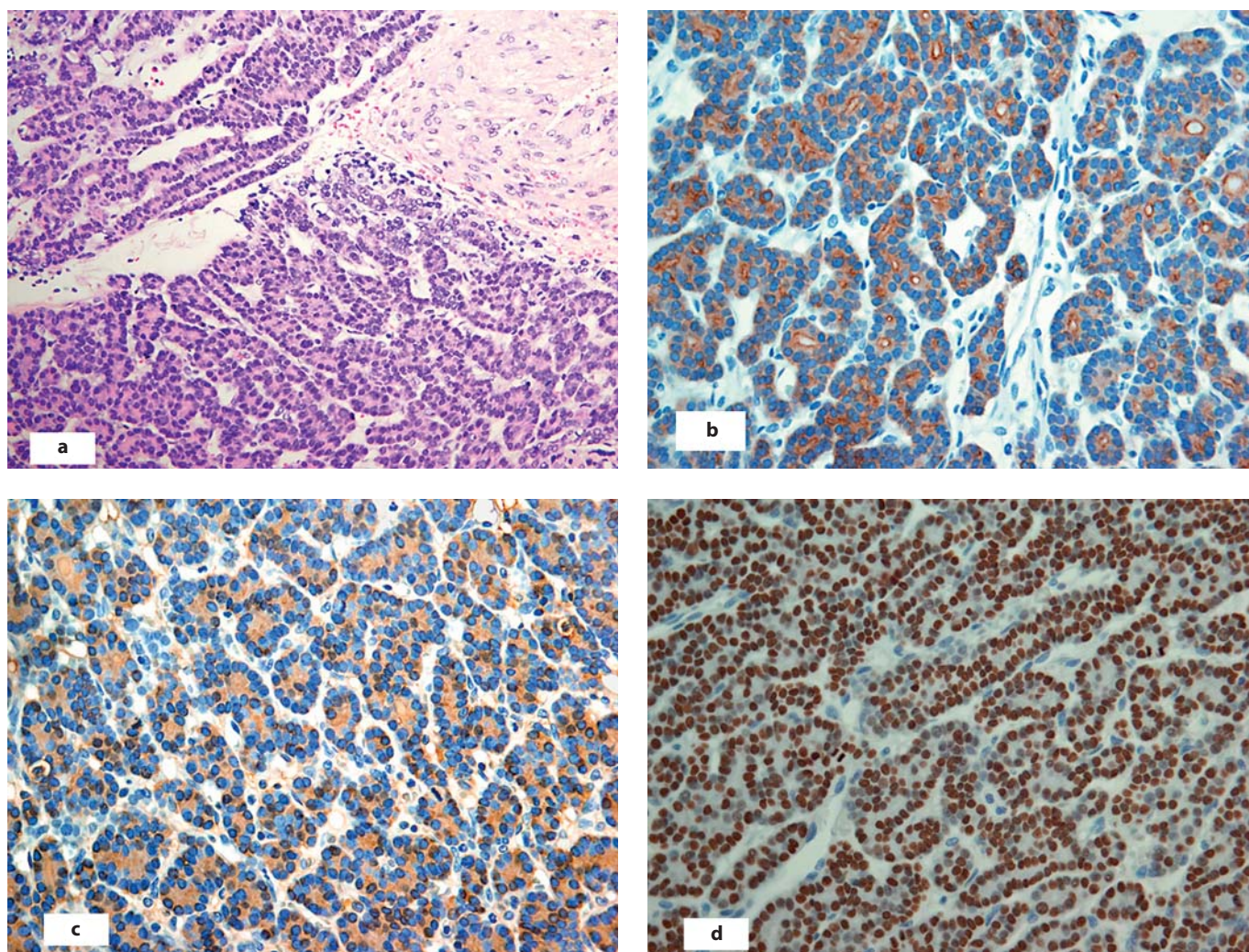


Fig. 3. Metastatic deposit of follicular variant of PTC. **a** Tumor tissue with irregular follicular structures and clearing of tumor cell nuclei. A fragment of leptomeninges is present in the right upper portion of the field. HE. $\times 250$. **b** Strong immunoreactivity of tumor cells for CK19. $\times 400$. **c** Tumor cells and colloid within follicles are immunoreactive for TGB. $\times 400$. **d** Typical nuclear TTF-1 positivity of all tumor cells. Immunoperoxidase stain. $\times 400$.

Histology of the removed sellar mass unequivocally confirmed metastasis of the follicular variant of PTC. No pituitary tissue was found in the specimen. Macroscopically the removed tissue was of soft consistency, grayish-brown color and measured $20 \times 10 \times 10$ mm. Microscopical analysis was performed on formalin-fixed, paraffin-embedded and hematoxylin and eosin (HE)-stained tissue. For immunohistochemistry, the following primary (Dako) antibodies were used: cytokeratin AE1/AE3, cytokeratin (CK)19, TGB, synaptophysin, chromogranin A and thyroid transcription factor-1 (TTF-1). Microscopic examination revealed tumor tissue composed of neoplastic cells arranged mostly in small follicles with a variable amount of colloid (fig. 3a). The majority of tumor cells showed typical nuclear clearing. Some mi-

totic figures were atypical. There was no necrosis. Immunohistochemistry showed strong immunostaining for CK19 (fig. 3b), TGB (fig. 3c) and TTF-1 (fig. 3d). Tumor cells were negative for synaptophysin and chromogranin A.

The immediate postoperative course was complicated by a spontaneous (pathological) fracture of the right humerus. Bone scan was previously postponed due to urgent pituitary surgery, and this skeletal event could have been attributed to thyroid cancer metastasis, but this was never confirmed. Postoperatively, the patient's general condition deteriorated. Progressive loss of consciousness was most probably caused by intracerebral bleeding. The patient died 2 weeks after the operation. Autopsy was not performed.

Discussion

Metastatic Tumors in the Sellar Region

An intrasellar and parasellar mass, in more than 90% of cases, represents a pituitary adenoma, with Rathke and arachnoidal cysts, craniopharyngioma, benign non-pituitary tumors and inflammatory lesions accounting for the remaining 10% [3, 4]. Metastatic tumors occur in 0.3% of all sellar masses, but a rise in incidence is observed due to longer life expectancy in the malignant disease and improvements in imaging precision. In most malignancies, the incidence of sellar metastases is significantly higher in autopsy studies than during lifetime (28 vs. 3–5%) [5]. The most frequent sources of metastases are: breast carcinoma (53% of pituitary metastatic lesions) and lung carcinoma (19%) [5]. Pituitary metastases are more often identified in the late stage of the malignant disease, although cases of sellar masses as the first manifestation of malignancy have also been reported [6].

In a postmortem study, isolated involvement of the posterior pituitary lobe was more common (52%) than the involvement of both lobes (27%) or isolated anterior pituitary lesion (21%) [7]. Predilection for the posterior lobe involvement was explained by the direct arterial supply and larger area of contact with adjacent dura.

Signs and symptoms of sellar metastases are non-specific, and common for any sellar mass regardless of its nature. The presentation most frequently includes: polyuria and polydipsia, headache, hypopituitarism, visual field defects and cranial nerve lesions. The metastatic nature might be indicated by: accelerated growth of the mass, recent ophthalmoplegia, sudden onset of DI and hypopituitarism disproportionate to the size of the mass [6].

Imaging can sometimes assist in the differential diagnosis of the sellar lesions. On plain profile craniogram, pituitary adenomas, as slow-growing lesions, result in a gradual expansion of the sella, while metastatic lesions rather cause the destruction of the sellar floor [6]. On an MRI the metastatic nature could be indicated by: pituitary stalk infiltration, cavernous sinus invasion, and posterior lobe invasion. Metastases are less likely to destroy the sellar diaphragm, and are often represented as tumors with a narrow waist (shaped like number 8 or dumbbell) [6].

Particular challenge is the coexistence of pituitary incidentaloma in a patient with malignancy. The frequency of finding an unrelated pituitary adenoma in a patient with other malignancy is estimated to be 1.8% [5]. Fur-

thermore, in rare cases a malignant tumor may metastasize to a pituitary adenoma [8].

Neurosurgical exploration may lead to significant symptomatic relief, to confirmation of the diagnosis, and to improvement in quality of life, while increased life expectancy has not been demonstrated. Tumor invasiveness reduces the effectiveness of resection and frequency and severity of bleeding is increased. Overall life expectancy in patients with sellar metastases is 6–22 months [9].

Papillary Thyroid Carcinoma

PTC is the most common thyroid malignancy, constituting 80–85% of well-differentiated thyroid carcinoma [1]. PTC is one of the malignancies with best prognosis. Women are more frequently affected, with a peak incidence in the fourth and fifth decades. PTC most often presents with a thyroid nodule (90%), rarely first presents as cervical lymphadenopathy with occult thyroid cancer (10%) and extremely rarely are distant metastases alone the first presentation of the disease [10]. Ultrasonography and FNAB are the most important tools in diagnosing PTC. The most frequent histological variants of PTC are the classical, follicular variant, and tall cell variant, but a large number of less frequent variants are described [11]. Immunohistochemical analysis is necessary to confirm thyroid origin in metastatic lesions. TTF-1 and TGB are markers of thyroid origin, while the markers for distinction of follicular variant of PTC from the follicular thyroid carcinoma include: cytokeratin, HBME-1, CITED and others.

Metastatic lesions from PTC most frequently involve the regional lymph nodes, resulting in a higher local recurrence incidence but not altering the overall prognosis except in older patients. Distant metastases are rare, occurring in the late stage of the disease and represent the most important negative prognostic factor [12]. Distant metastases are found in 1–9% of newly diagnosed PTC while in 7–23% of patients during follow-up [12]. They are most often identified by whole-body radioiodine scintigraphy (¹³¹I-WBS). In cases of low iodine affinity, ¹⁸F-FDG PET represents an alternative, and a promising high precision new method is ¹³¹I-SPECT/CT. The most common sites for PTC metastases are lungs alone (45–49%), or bone alone (24–39%). Other localizations are most often found jointly with lung or bone metastases (12%), while only in 4% isolated distant lesions in the brain, liver or skin are found. Cerebral metastases from PTC, with a very low incidence of 0.4–1.2%, are mostly located in cerebral hemispheres or cerebellum [2]. The prognosis of

Table 1. Review of reported cases of sellar metastases of PTC including the presented case

Reference (first author)	Sex	Age	Presentation	Prior PTC diagnosis years	Other distant metastases	PH of the sellar mass	Immuno-histo-chemistry	Treatment	Outcome
McCarthy 1912 [13]	M	57	postmortem	0	lungs	PTC	no	none	postmortem presentation
Johnson 1965 [14]	F	56	ophthalmoplegia	7	none	no	no	external cranial irradiation, ablative ¹³¹ I-WBS	alive at 20 month follow-up
Kistler 1975 [15]	F	69	ophthalmoplegia	9	unknown	thyroid adenocarcinoma	no	transcranial surgery	died 1 year after presentation
Pelosi 1977 [16]	M	32	ophthalmoplegia, DI, hypopituitarism	0	unknown	PTC	no	transcranial surgery	died 1 month after presentation
Ozanne 1982 [17]	F	66	ophthalmoplegia, hemianopsia	2	unknown	PTC	no	none	died 2 months after presentation
Sziklas 1985 [18]	M	44	hypopituitarism	25	mediastinum, skull, chest wall	PTC	no	transsphenoidal surgery, ablative ¹³¹ I-WBS	died 13 months after presentation
Masiukiewicz 1999 [19]	M	56	hypopituitarism	5	lungs, bone	no	no	ablative ¹³¹ I-WBS	alive at the time of reporting
	F	55	ophthalmoplegia	20	lungs	PTC	no	stereotactic radiosurgery, dexamethasone, surgical debulking, ablative ¹³¹ I-WBS	died 7 months after presentation
Bell 2001 [20]	F	35	hemianopsia, DI, amenorrhoea	25	lungs	PTC	yes	transsphenoidal surgery	alive at the time of reporting
Simmons 2010 [21]	M	48	visual field defect, seizures	0	none	papillary thyroid microcarcinoma	no	ablative ¹³¹ I-WBS	alive at 3 year follow-up
Xia 2010 [22]	F	56	ophthalmoplegia, visual field defect, amenorrhoea	7	none	PTC	yes	transsphenoidal surgery	alive at 12 month follow-up
Madronio 2011 [23]	F	53	headache, visual impairment	0	none	PTC	no	transsphenoidal surgery	alive at 13 month follow-up
Present case 2012	F	67	visual field defect	0	possible: intracerebral, bone	follicular variant of PTC	yes	transcranial surgery	died 10 months after presentation

the PTC metastases is most determined by their localization, and additionally by their iodine affinity or by patient's age. Progressively growing metastases, particularly in vitally sensitive locations, may require additional systemic therapy.

Sellar Metastases of Papillary Thyroid Carcinoma

A century ago the first case of a patient with sellar metastasis of PTC was reported [13] and since then an additional 12 cases, including ours, have been reported [14–23]. All reported patients are presented in table 1. Only two reports [20, 22] prior to ours had immunohistochemical confirmation of a PTC metastasis in the sellar region. Overall in the reported cases (including ours), women

were affected almost twice as often as men (8:5). The median age of the patients was 56 years with the span of 32–69. Most patients (8/13) have previously been diagnosed with PTC and an average of 8 years elapsed since the diagnosis. Interestingly, in 5 reported cases, PTC was not diagnosed until the sellar mass was discovered. The most frequent presenting signs and symptoms were: visual field defects or ophthalmoplegia (with equal prevalence), followed by anterior pituitary deficiencies with only 2 cases presenting with DI. Thus, while most other malignancies which metastasize to the hypothalamic-pituitary region cause DI, PTC presents without DI in most cases, including ours. In the reported cases of sellar metastasis from PTC, visual field impairment was equally

Table 2. Summary of clinical features of the reported cases of sellar metastases of PTC (including the present case)

Male:female	5:8
Age, years	32–69
Mean \pm SE	53.4 \pm 3.1
Median	56
Presenting signs ^a	1 visual field defects (6) 2 ophthalmoplegia (6) 3 anterior pituitary deficiencies (5) 4 DI (2)
Previous PTC diagnosis	61.5% (8/13)
Years since PTC diagnosis	
Mean \pm SE	8.3 \pm 2.8
Median	6
Other known distant metastases	38.5% (5/13)
Localization of other distant metastases ^a	1 lungs (4) 2 bone (2) 3 mediastinum (1)

^a Sometimes multiple in a single patient.

prevalent as ophthalmoplegia. In general, ophthalmoplegia is expected to be more prevalent in patients with pituitary metastases.

The diagnosis of PTC metastasis to the sella was reported in 1 patient by postmortem analysis [13]. However, in a large autopsy series, sellar PTC metastases were absent [24]. This is contrary to other malignancies where a much higher incidence of sellar metastasis is found postmortem than in clinical reports. In a minority of reported cases (5/13), other distant metastases were also confirmed, primarily including lungs or bones (table 2). The gender distribution in patients with sellar PTC metastases (8:5 female vs. male) is different than for other distant metastases of PTC which were twice more often reported in male than in female patients (19.3 vs. 10.0%) [25]. Reports of PTC metastasis to the skull base or sphenoidal sinus represent further diagnostic difficulties in relation to intrasellar or parasellar tumors. The possibility of ectopic thyroid tissue in the sella harboring well-differentiated thyroid carcinoma has also been suggested in one report [26].

Distant PTC metastases may respond well to the radioiodine therapy but in the case of sellar localization, a multimodal approach may be needed (operative, radioiodine treatment, external cranial irradiation or stereotactic radiosurgery) with each option having caveats and

limitations. The surgical resectability is limited, and complications are more prevalent, particularly bleeding, as was the case in our patient. These lesions are highly vascular, which may lead to difficulties with transsphenoidal approach. After long discussions with our patient it was decided that surgery was necessary due to rapid visual deterioration. Surgery for the sellar mass was performed in 8/12 of reported patients [15, 16, 18–20, 22, 23] and it was more often transsphenoidal than transcranial. After total thyroidectomy in 2 patients, ablative radioiodine alone was the treatment of choice [19, 21], while in 2 other patients, surgery plus ablative radioiodine therapy was performed [18, 19]. As additional treatment modalities external cranial irradiation was performed in 1 patient [14] and stereotactic radiosurgery (gamma-knife) in another with parasellar localization of the metastatic mass [19]. Most patients (7/12) were alive 1 year after presentation, with the longest reported survival of 3 years.

Hypopituitarism caused either by metastatic lesion (as was the case with our patient) or by the treatment procedures impairs the diagnostic value of radioiodine. The problem with the absence of endogenous TSH rise after total thyroidectomy has been effectively bypassed with the use of rhTSH, which is recommended by the current guidelines [27]. While its use for diagnostic purposes is thought to be safe, its use for treatment needs caution due to reports of intracerebral hemorrhage probably caused by tumor growth stimulation by rhTSH [28]. Radioiodine treatment of cerebral metastases can be associated with cerebral edema and was associated in several reported cases with hemiplegia, particularly in the case of a large mass [29]. Steroid treatment prior to and during radioiodine therapy can reduce the risk. External radiotherapy prior to ¹³¹I therapy can cause a decrease in radioiodine uptake due to tumor fibrosis following radiotherapy. Stereotactic radiosurgery is an option for other cerebral metastasis of PTC, but its use is limited by proximity of vulnerable structures, such as the optic chiasm. Sellar metastases of PTC in particular are not addressed in the ATA guidelines and ETA management consensus regarding distant metastases from PTC [27, 30].

Conclusion

We present a patient with confirmed sellar metastasis from PTC, who did not have prior diagnosis of PTC. To the best of our knowledge, this is the 13th patient ever reported in the literature and only the 3rd patient who had immunohistochemical confirmation of PTC in the

seller region. The patient presented with signs and symptoms of sellar and suprasellar mass (visual field impairment, hyperprolactinemia and hypopituitarism) confirmed by the MRI. She also had a long-standing euthyroid multinodular goiter without cervical lymphadenopathy. During the evaluation of the sellar mass, papillary thyroid cancer was diagnosed and subsequently the sellar mass was unequivocally confirmed to be a PTC metastasis.

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Disclosure Statement

The authors have no conflicts of interest to disclose.

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