Case Report

Apoplexy in sellar metastasis from papillary thyroid cancer: A case report and literature review

Masahiro Hirayama¹, Atsushi Ishida¹, Naoko Inoshita¹, Hideki Shiramizu¹, Haruko Yoshimoto¹, Masataka Kato¹, Satoshi Tanaka¹, Seigo Matsuo¹, Nobuhiro Miki², Masami Ono², Shozo Yamada²

¹Department of Neurosurgery, Moriyama Memorial Hospital, ²Hypothalamic and Pituitary Center, Moriyama Neurosurgical Center Hospital, Tokyo, Japan.

E-mail: *Masahiro Hirayama - hiramasa0701@gmail.com; Atsushi Ishida - v2danyon@gmail.com; Naoko Inoshita - inonao3939@gmail.com; Hideki Shiramizu - h.shiramizu@gmail.com; Haruko Yoshimoto - jjsbach1music@yahoo.co.jp; Masataka Kato - gmstkg@gmail.com; Satoshi Tanaka - satoshishionanmn@yahoo.co.jp; Seigo Matsuo - sergio5679700@yahoo.co.jp; Nobuhiro Miki - miki-nob@tb3.so-net.ne.jp; Masami Ono - miki-masa@abox.so-net.ne.jp; Shozo Yamada - syamadays11@hotmail.com

ABSTRACT

Background: Pituitary metastasis from papillary thyroid cancer (PTC) is rare and only a few cases have been reported.

Case Description: We report the case of a patient who presented with visual dysfunction and panhypopituitarism. Magnetic resonance imaging revealed a pituitary tumor and hydrocephalus. Transphenoidal surgery had been indicated, but his surgery had been postponed due to COVID-19 pandemic. During that waiting period, he showed pituitary apoplexy with consciousness disturbance, resulting in acute adrenal insufficiency and diabetes insipidus. He was urgently hospitalized and underwent transphenoidal surgery. Rapid and permanent pathological examinations have confirmed metastasis of PTC to the pituitary. The patient also underwent serial thyroidectomy. He was also suspected to have secondary hydrocephalus and underwent lumbo-peritoneal shunting after excluding cerebrospinal fluid metastasis. Thereafter, his cognitive dysfunction and performance status improved dramatically.

Conclusion: To the best of our knowledge, this is the first patient with PTC who developed pituitary apoplexy secondary to metastasis.

Keywords: Case report, Papillary thyroid cancer, Pituitary apoplexy, Pituitary metastasis, Transphenoidal surgery

INTRODUCTION

Thyroid carcinoma accounts for only 2–4.1% of primary tumors.¹¹,¹⁴,²¹ Recently, the number of patients with cancer metastasis has been increasing due to prolonged survival time after diagnosis.¹⁰ Papillary thyroid cancer (PTC) metastasizes in 8% of cases,²⁷ but metastasis to pituitary gland is extremely rare. Such patients develop symptoms such as pituitary insufficiency, visual impairment, cranial nerve palsy, or diabetes insipidus (DI). However, to the best of our knowledge, there have been no reports that metastatic pituitary tumor of PTC is associated with apoplexy. We present a case of pituitary metastasis from PTC whose course was unusual as it caused apoplexy and hydrocephalus during the period when surgery was postponed due to COVID-19.
CASE REPORT

A 74-year-old man without notable medical history and family history who noticed a visual disturbance 3 years prior had extreme tiredness and sought medical care at a different hospital. After a detailed examination, bitemporal hemianopia and panhypopituitarism without DI were noted. Magnetic resonance imaging (MRI) revealed a dumbbell-shaped tumor (22 × 17 × 11 mm) pushing the optic chiasm upward and unobstructed hydrocephalus [Figures 1a and b]. He was referred to our hospital and scheduled to undergo surgery, but it was postponed due to the COVID-19 pandemic under appropriate hormone replacement treatment. Four months later, he developed persistent fever, headache, and vomiting and he was transferred to a nearby medical center. His Glasgow Coma Scale (GCS) score was 12 (E3V3M6) and he became somnolent. Computed tomography (CT) showed a high-density area on the pituitary gland and MRI showed enlargement of the upper part of the tumor (24 × 18 × 18 mm) [Figures 1c and d]. A previous doctor suspected pituitary apoplexy. The patient was treated with steroids and transferred to our hospital. He developed DI and was administered cortisol, thyroid hormones, and vasopressin. CT scan on admission for COVID-19 screening showed an enlarged thyroid gland. His consciousness gradually recovered after hormone treatment and correction of hyponatremia. Then, his GCS score improved to 14 (E4V4M6) and fever subsided. At the time, pituitary function tests were typical for hypopituitarism (growth hormone: 0.16 ng/mL; prolactin: 4.37 ng/mL; thyroid-stimulating hormone: <0.005 mIU/mL; free T4: 1.13 ng/dL; adrenocorticotropic hormone: <1.5 pg/mL; cortisol: <0.3 mIU/mL; luteinizing hormone: <0.3 mIU/mL; follicle-stimulating hormone: <0.3 mIU/mL; and testosterone: <0.003 ng/mL).

We performed an extended transsphenoidal surgery. The pituitary gland was thin and located in front of the tumor. The upper part of the tumor showed hemorrhagic changes consistent with apoplexy. The tumor was highly vascularized, unlike pituitary adenoma, and adhered tightly to the ventral part of the optic chiasm. The floor of the third ventricle was torn and the contents had leaked. Rapid diagnosis during surgery was suspicious of PTC, and due to the position of the tumor, we decided to retain the tumor capsule. MRI after surgery showed satisfactory decompression [Figures 1e and f].

After surgery, his visual symptoms improved. He underwent whole-body CT scan screening, which detected small lesions in the lung other than the thyroid mass. His serum thyroglobulin was abnormally high (1540 ng/mL) before surgery and decreased to 36.3 ng/mL after surgery. He then developed meningitis and hyponatremia due to cerebral salt-wasting syndrome, which was medically treated.

Histological examination of the resected tumor confirmed PTC. An immunohistochemical test reported positive TTF1 and 25% MIB-1 [Figures 2a and b]. Part of the lesion showed hemorrhagic changes. Since the cell block of cerebrospinal fluid (CSF) was negative for TTF1 and thyroglobulin, we concluded that there was no dissemination of the tumor to the CSF.

Figure 1: Gadolinium-enhanced T1-weighted MR images (a and b) before apoplexy, (c and d) after pituitary apoplexy, and (e and f) after surgery.
He was transferred to another hospital for further treatment of thyroid cancer. He underwent left-sided thyroidectomy and lymphadenectomy, and near-total resection was achieved. The pathological diagnosis was PTC (31 × 22 mm) that had invaded the surrounding muscle (pEx1, pT3b, and pN1b).

123I-iodoamphetamine single-photon emission computed tomography (IMP-SPECT) was performed for further evaluation of his cognitive dysfunction, which showed decreased signal mainly in the frontal lobe and was suspicious of hypothyroidism, hyponatremia, or other metabolic dysfunction disorders and not typical for hydrocephalus or Alzheimer’s disease.

The patient returned to our hospital with prolonged cognitive dysfunction. The tap test was performed. The CSF opening pressure was 19 cm H₂O and he showed mild cognitive improvement the following day. The patient underwent lumboperitoneal (LP) shunt placement. Thereafter, his cognitive function recovered dramatically [Table 1]. He visited the outpatient clinic after 18 months without complications (modified Rankin scale 1).

DISCUSSION

Thyroid cancer is the most common endocrine malignancy; PTC accounts for 80% and is known for its good prognosis. Distant metastasis occurs in about 8% of PTC cases, but pituitary metastasis is extremely rare. Patients exhibit symptoms due to mass effects, such as visual impairment from compression of the optic nerve, ophthalmoplegia of the oculomotor nerve, trochlear nerve, abducens nerve invasion, anterior pituitary insufficiency, and DI.[14] The posterior lobe of the pituitary gland is more susceptible to hematogenous spread,[32] as the blood supply differs between the anterior and posterior lobes, which are mainly supplied by the portal vessels and the inferior hypophyseal arteries, respectively.

The reported poor prognostic factors of distant metastatic PTC are advanced age, distant metastasis from areas other than the lung, and degree of extrathyroidal invasion of the primary neoplasm. To the best of our knowledge, 15 cases of pituitary metastasis from PTC have been reported. Excluding postmortem cases, 50% of these patients died within 13 months of the first presentation [Table 2].

Treatment options for pituitary metastasis from PTC include transcranial surgery, transsphenoidal surgery, radiotherapy, and iodotherapy, usually for local symptom management because there is no evidence of improvement in life expectancy.[26] Gross total resection for metastatic pituitary tumors is difficult to achieve because of their highly vascularized structure.

Preoperative diagnosis of metastatic tumors is challenging. Imaging can support the diagnosis of pituitary tumors. Dutta et al. reported that metastatic lesions tend to destroy the sellar floor and are less likely to destroy the sellar diaphragm.[9,14] Habu et al. reported that the upward extension of pituitary metastases was longer than that of nonfunctioning adenomas, although the difference was not statistically significant. In contrast, the downward extension of pituitary metastasis is significantly shorter than that of nonfunctioning adenoma.[10] In the report, the rate of constriction tumors in the diaphragmatic hiatus in pituitary metastatic cases was 44.7%. In our case, a dumbbell-shaped tumor was also observed.

DI is a rare manifestation of benign pituitary gland tumors. DI occurs in 3–23% of patients with pituitary apoplexy[9,25,34] and was previously reported as a common manifestation in patients with pituitary metastasis, as approximately 70% of metastatic patients were affected.[20,22] However, since the 2000s, several reports indicated the rate of DI from metastatic pituitary lesions ranged from 26% to 45%.[10,11,14,33] much lower than the previous reports.

Verrees et al. postulated that DI occurred as a result of impingement of the inferior hypophyseal artery, which caused diminished perfusion to the posterior lobe, or kinking or pressure on the infundibulum by edematous, hemorrhagic material that impeded transit of antidiuretic hormones from the hypothalamus to the posterior lobe.[39] Interestingly, our patient developed DI only after experiencing apoplexy. Based on the images obtained before and after apoplexy, the size of the upper part of the tumor increased and expanded backward to the ventral hypothalamus. In addition to

---

**Table 1: Results of tests before and after shunting.**

|                | On admission | Pretap test | Posttap test | Postshunting |
|----------------|--------------|-------------|--------------|--------------|
| Mini-Metal State Examination Frontal assessment battery | 11/30 | 15/30 | 20/30 | 27/30 |
| Timed up and go | - | - | 26 s | 17 s |

---

**Figure 2**: Carcinoma cells from pituitary tumor (a) Tumor tissue with papillary structures, intranuclear inclusions, and nuclear grooving are observed. HE. (b) Tumor cells showed strong immunopositivities to TTF-1.
impingement of the inferior hypophyseal artery and effects on the infundibulum, direct damage to the hypothalamus due to the tumor mass effect may be contributed to development of DI. DI frequency due to PTC is lower than that of other types of metastases. Based on the reported 16 cases of pituitary metastasis from PTC, only two other cases besides ours developed DI.

Pituitary apoplexy is a life-threatening event resulting from an acute hemorrhagic or ischemic event. Four theories regarding the precipitants of pituitary apoplexy are proposed: rapid tumor growth exceeding arterial supply, acute increase in blood flow, endocrine stimulation of the pituitary gland, and coagulation disturbance. Considering pituitary apoplexy from pituitary metastasis, intratumoral hemorrhage due to vasculopathy would be an additional factor. Pituitary apoplexy typically occurs in macroadenomas and rarely in metastatic lesions; 14 cases have been reported. Reports on the incidence of symptomatic pituitary apoplexy in the pituitary adenoma vary from 0.6% to 9.1%. Komninos et al. reported that pituitary apoplexy can be seen about 5% of patients with pituitary metastasis. To the best of our knowledge, this is the first reported case of metastatic PTC with pituitary apoplexy.

The patient's hydrocephalus was evident before apoplexy. IMP-SPECT can support the diagnosis of idiopathic normal-pressure hydrocephalus (iNPH). Ohmichi et al. reported in

| References | Age, Sex | VI | CN | AI | DI | PA | Other symptoms | Prior diagnosis years | Other metastasis | Treatment | Outcome |
|------------|----------|----|----|----|----|----|-----------------|---------------------|------------------|-----------|---------|
| McCarthy and Karsner, 1912 | 57, M | - | - | - | - | - | None | 0 | Lungs | None | Postmortem |
| Johnson and Atkins, 1965 | 56, F | - | ○ | - | - | - | None | 7 | None | RT, IT | Alive at 20 months f/u |
| Kistler and Pribram, 1975 | 69, F | - | ○ | - | - | - | Unknown | 9 | Unknown | TCS | Died 1 year a/p |
| Pelosi et al., 1977 | 32, M | - | ○ | ○ | ○ | x | Unknown | 0 | Unknown | TCS | Died 1 month a/p |
| Ozanne et al., 1982 | 66, M | o | o | x | x | x | Unknown | 2 | Unknown | None | Died 2 months a/p |
| Siklas et al., 1985 | 44, M | - | - | ○ | x | x | Mediastinum, skull, chest wall | 25 | Lungs, bone | IT | Died 13 months a/p |
| Masiukiewicz et al., 1999 | 56, M | x | x | o | x | x | Unknown | 5 | Lungs | IT | Alive at the time of reporting |
| Bell et al., 2001 | 35, F | o | - | ○ | o | x | Lungs | 25 | Lungs | RT, surgery, IT | Alive at the time of reporting |
| Simmons et al., 2010 | 48, M | - | ○ | x | x | Seizure | 0 | None | IT | Alive at 3 years f/u |
| Xia and Wang, 2010 | 56, F | o | o | o | x | x | N.A. | 7 | None | TSS | Alive at 12 months f/u |
| Madronio and Frances, 2011 | 53, F | o | - | - | - | - | None | 0 | None | TSS | Alive at 12 months f/u |
| Stojanović et al., 2012 | 67, F | o | - | ○ | o | x | Possible: intracerebral, bone | 0 | Lungs | TSS | Alive at 13 months f/u |
| Chikani and Lambie, 2013 | 70, F | x | x | o | x | x | Nausea | 3 | Manubrium | TSS, RT, IT | Died 5 years a/p |
| Ardhaoui et al., 2020 | 70, F | o | o | - | - | x | Headache | 1 | Sphenoid sinus | TSS | Alive at 6 months f/u |
| Popławska-Kita, 2020 | 68, F | o | o | x | x | x | Headache | 2 | None | TSS, IT | Died 3 years a/p |
| Present case | 74, M | o | x | o | o | o | Headache, confusion | 0 | Lung | TSS, Surgery | Alive at 18 months f/u |

VI: Visual impairment, CN: Cranial nerve, AI: Anterior pituitary insufficiency, DI: Diabetes insipidus, PA: Pituitary apoplexy, RT: Radiotherapy, IT: Iodotherapy, TSS: Transsphenoidal surgery, TCS: Transcranial surgery, f/u: Follow-up, a/p: After presentation

Table 2: Review of reported cases of sellar metastases of the PIC including the presented case.
80% of cases, IMP-SPECT for a typical iNPH patient shows convexity apparent hyperperfusion (CAPPAH) sign.[27] In our case, IMP-SPECT did not reveal a typical CAPPAH sign. We concluded his hydrocephalus was likely secondary NPH (sNPH). Daou et al. reported that intracranial malignancy is the third main etiology of sNPH, for which shunting is effective in 86.4% of patients.[21]

After the tap test, the patient's cognitive decline became milder. We implanted an LP shunt. Before surgery, CSF tests showed no signs of metastasis. Considering his cognitive improvement and performance status after LP shunt placement, CSF production or absorption seemed to be affected. Mitsuya et al. reported palliative CSF shunting for leptomeningeal metastasis-related hydrocephalus. In the study, CSF shunt surgery yielded rapid improvement in the performance status of 90.3% of patients.[21] Considering these results, CSF shunting is a good choice for improving the quality of life of end-stage patients regardless of CSF metastasis.

CONCLUSION

We present the case of a patient with pituitary metastases from papillary thyroid carcinoma, who did not have a prior diagnosis of and developed pituitary apoplexy. To the best of our knowledge, this is the 16th case of pituitary metastasis from PTC, and the first case of apoplexy reported in the literature. His performance status improved dramatically after extended transphenoidal surgery, hormonal treatment, and shunting. Finally, it should be noted that this pituitary apoplexy occurred during the period when surgery was postponed due to COVID-19 pandemic.

Acknowledgment

We also would like to thank Editage (www.editage.com) for English language editing.

Declaration of patient consent

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

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Ardhaoi H, Halily S, Elkrimi Z, Rouadi S, Roubal M, Mahtar M. Thyroid papillary carcinoma with an insular component metastasizing to the sella turcica and sphenoid sinus: Case report. Int J Surg Case Rep 2020;67:254-7.
2. Bell CD, Kovacs K, Horvath E, Smythe H, Asa S. Papillary carcinoma of thyroid metastatic to the pituitary gland. Arch Pathol Lab Med 2001;125:935-8.
3. Bioussé V, Newman NJ, Oyesiku NM. Precipitating factors in pituitary apoplexy. Neurol Neurosurg Psychiatry 2001;71:542-5.
4. Bobinski M, Greco CM, Schrot RJ. Giant intracranial medullary thyroid carcinoma metastasis presenting as apoplexy. Skull Base 2009;19:359-62.
5. Chhiber SS, Bhat AR, Khan SH, Wani MA, Ramzan AU, Kirmani AR, et al. Apoplexy in sellar metastasis: A case report and review of literature. Turk Neurosurg 2011;21:230-4.
6. Chikani V, Lambie D, Russell A. Pituitary metastases from papillary carcinoma of thyroid: A case report and literature review. Endocrinol Diabetes Metab Case Rep 2013;2013:130024.
7. Daou B, Klinge P, Tjoumakaris S, Rosenwasser RH, Jabbour P. Revisiting secondary normal pressure hydrocephalus: Does it exist? A review. Neurosurg Focus 2016;41:E6.
8. Dutta P, Bhansali A, Shah VN, Walia R, Bhadada SK, Paramjeet S, et al. Pituitary metastasis as a presenting manifestation of silent systemic malignancy: A retrospective analysis of four cases. Indian J Endocrinol Metab 2011;15 Suppl 3:5242-3.
9. Glezer A, Bronstein MD. Pituitary apoplexy: Pathophysiology, diagnosis and management. Arch Endocrinol Metab 2015;59:259-64.
10. Habu M, Tokimura H, Hirano H, Yasuda S, Nagatomo Y, Iwai Y, et al. Pituitary metastases: Current practice in Japan. J Neurosurg 2015;123:998-1007.
11. He W, Chen F, Dalm B, Kirby PA, Greenlee JD. Metastatic involvement of the pituitary gland: A systematic review with pooled individual patient data analysis. Pituitary 2015;18:159-68.
12. Johnson PM, Atkins HL. Functioning metastasis of thyroid carcinoma in the sella turcica. J Clin Endocrinol Metab 1965;25:1126-30.
13. Kistler M, Pribram HW. Metastatic disease of the sella turcica. Am J Roentgen Radium Ther Nucl Med 1975;123:13-21.
14. Komininos J, Vlassopoulou V, Protopapa D, Korfias S, Kontogeorgos G, Sakas DE, et al. Tumors metastatic to the pituitary gland: Case report and literature review. J Clin Endocrinol Metab 2004;89:789-80.
15. Kruljic I, Cerina V, Pecina HI, Prazanin L, Matic T, Bozikov V, et al. Pituitary metastasis presenting as ischemic pituitary apoplexy following heparin-induced thrombocytopenia. Endocr Pathol 2012;23:264-7.
16. Luu ST, Billing K, Crompton JL, Blumbergs P, Lee AW, Chen CS. Clinicopathological correlation in pituitary gland metastasis presenting as anterior visual pathway compression. J Clin Neurosci 2010;17:790-3.
17. Madronio E, Frances L. The tale of two tumors: An undiagnosed case of papillary thyroid carcinoma. BMJ Case Rep 2011;2011:bcr0820114604.
18. Masukiewicz US, Nakhchbandi IA, Stewart AF, Inzucchi SE. Papillary thyroid carcinoma metastatic to the pituitary gland. Thyroid 1999;9:1023-7.
19. Masui K, Yonezawa T, Shinji Y, Nakano R, Miyamae S. Pituitary apoplexy caused by hemorrhage from pituitary metastatic melanoma: Case report. Neurol Med Chir (Tokyo) 2013;53:695-8.
20. Max MB, Deck MD, Rottenberg DA. Pituitary metastasis: Incidence in cancer patients and clinical differentiation from pituitary adenoma. Neurology 1981;31:998-1002.
21. McCarthy DJ, Karsner HT. Adenocarcinoma of the thyroid with metastasis to the cervical glands and to the pituitary: A contribution to the pathology of abnormal fat formation. Am J Med Sci 1912;144:834-47.
22. McCormick PC, Post KD, Kandji AD, Hays AP. Metastatic carcinoma to the pituitary gland. Br J Neurosurg 1989;3:71-9.
23. Mitsuya K, Nakasu Y, Hayashi N, Deguchi S, Takahashi T, Murakami H, et al. Palliative CSF shunting for leptomeningeal metastasis-related hydrocephalus in patients with lung adenocarcinoma: A single-center retrospective study. PLoS One 2019;14:e0210074.
24. Mohr G, Hardy J. Hemorrhage, necrosis, and apoplexy in pituitary adenomas. Surg Neurol 1982;18:181-9.
25. Moller-Goede DL, Brändle M, Landau K, Bernays RL, Schmid C. Re-evaluation of risk factors for bleeding into pituitary adenomas and impact on outcome. Eur J Endocrinol 2011;164:37-43.
26. Ng S, Fomekong F, Delabar V, Jacquesson T, Enachescu C, Raverot G, et al. Current status and treatment modalities in metastases to the pituitary: A systematic review. J Neurooncol 2020;146:219-27.
27. Ohmichi T, Kondo M, Itsukage M, Koizumi H, Matsushima S, Kuriyama N, et al. Usefulness of the convexity apparent hyperperfusion sign in 123I-iodoamphetamine brain perfusion SPECT for the diagnosis of idiopathic normal pressure hydrocephalus. J Neurosurg 2019;130:398-405.
28. Ozanne P, Jedynak CP, Charbonnel B, Derome PJ. Pituitary and hypothalamic metastases: 5 cases (in French). Ann Med Interne (Paris) 1982;133:92-6.
29. Pearce JM. On the origins of pituitary apoplexy. Eur Neurol 2015;74:18-21.
30. Pelosi RM, Romaldini JH, Massuda LT, Reis LC, França LC. Pan-hypopituitarism caused by to sellar metastasis from papillary carcinoma of the thyroid gland associated with secondary thrombophlebitis of the cavernous sinus and purulent leptomeningitis (in Portuguese). AMB Rev Assoc Med Bras 1977;23:277-80.
31. Poplawska-Kita A, Wielogórska M, Poplawski L, Siewko K, Adamska A, Szumowski P, et al. Thyroid carcinoma with atypical metastasis to the pituitary gland and unexpected postmortem diagnosis. Endocrinol Diabetes Metab Case Rep 2020;2020:190148.
32. Ranavir S, Manash BP. Pituitary apoplexy. Indian J Endocrinol Metab 2011;15 Suppl 3:S188-96.
33. Schill F, Nilsson M, Olsson DS, Ragnarsson O, Berinder K, Engström BE, et al. Pituitary metastases: A nationwide study on current characteristics with special reference to breast cancer. J Clin Endocrinol Metab 2019;104:3379-88.
34. Shou XF, Wang YF, Li SQ, Wu JS, Zhao Y, Mao Y, et al. Microsurgical treatment for typical pituitary apoplexy with 44 patients, according to two pathological stages. Minim Invasive Neurosurg 2009;52:207-11.
35. Simmons JD, Pinson TW, Donnellan KA, Harbarger CF, Pitman KT, Griswold R. A rare case of a 1.5-mm papillary microcarcinoma of the thyroid presenting with pituitary metastasis. Am Surg 2010;76:336-8.
36. Stojanović M, Pekić S, Doknić M, Miljić D, Cirić S, Diklić A, et al. What’s in the image? Pituitary metastasis from papillary carcinoma of the thyroid: A case report and a comprehensive review of the literature. Eur Thyroid J 2013;1:177-84.
37. Sugitani I, Fujimoto Y, Yamamoto N. Papillary thyroid carcinoma with distant metastases: Survival predictors and the importance of local control. Surgery 2008;143:35-42.
38. Sziklas JJ, Mathews J, Spencer RF, Rosenberg RJ, Ergin MT, Bower BE. Thyroid carcinoma metastatic to pituitary. J Nucl Med 1985;26:1097.
39. Verrees M, Arafah BM, Selman WR. Pituitary tumor apoplexy: Characteristics, treatment, and outcomes. Neurosurg Focus 2004;16:e6.
40. Wakai S, Fukushima T, Teramoto A, Sano K. Pituitary apoplexy: Its incidence and clinical significance. J Neurosurg 1981;55:187-93.
41. Xia J, Wang Y. Papillary thyroid carcinoma metastatic to the pituitary gland: A case report and literature review. J Chin Clin Med 2010;2:116-9.

How to cite this article: Hirayama M, Ishida A, Inoshita N, Shiramizu H, Yoshimoto H, Kato M, et al. Apoplexy in sellar metastasis from papillary thyroid cancer: A case report and literature review. Surg Neurol Int 2022;13:253.