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Case Report

Collision of Craniopharyngioma and Pituitary Adenoma: A Rare Case-Report for a Challenging Neuroendocrine Condition

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Abstract

Collision tumors are defined as ≥2 histologically distinct tumors existing in the same anatomic location. Generally, collision tumors are rare, and if they occur in the sellar region, common combinations are pituitary adenoma with Rathke cleft cyst (most common), however the association of pituitary tumors with hypopthalamic tumors remains extremely rare. We report in an observation the case of a pituitary adenoma diagnosed after 5 years of a surgical cure of a craniopharyngioma. Although described in a few observations through the medical literature, it is an extremely rare condition illustrating the possibility of having 2 different neoplasias, within the hypothalamic-pituitary region. The chronology of the appearance of the 2 histological entities (or more than 2) is decisive first on a semantic level, this then allowing to better classify into one of collision, composite, or in coexistence forms. The management of these tumors of the pituitary region involves effective surgery but also a good endocrine evaluation in order to treat pituitary hormone deficiencies, sometimes with a vital prognosis (replacement with corticosteroids and thyroxin), or functional (GH, sexual steroids and gonadotrophins). The prognosis of these tumors that occurred in a collision mode remains less good compared to solitary tumors of the hypothalamic and hypopjhyseal region.

Keywords: Craniopharyngioma- Prolactinoma – Collision.

INTRODUCTION

Pituitary adenomas are the most common tumors in the sellar/ parasellar region, comprising 10% to 15% of all intracranial tumors, whereas craniopharyngiomas are uncommon, accounting for 3% of all intracranial tumors [1]. The first ever description of a craniopharyngioma dates back more than a century and a half (in 1856) by Carl Von Rokitnski, based on his gigantesque series of more than 30 000 autopsies [2]. No doubt, the advent of modern magnetic resonance imaging (MRI) in the 1990s dramatically increased the recognition of PCPs, prompting specific study of this histological type [3]. Historically, the treatment of choice was surgical excision, note that PCPs have willingly been considered more amenable to total removal than ACPs based on lower tendency to infiltrate the adjacent hypothalamic tissue and/or to induce a strong gliotic/inflammatory reaction in this vital brain region [4][5]. The collision of pituitary adenoma and craniopharyngioma is extremely rare, and a few cases including our institutional case were interested in the study of this condition [6]. which complicates the analytical study of the characteristics related to this pathological entity. We review in this paper the diagnostic aspects of collision tumors especially between craniopharyngioma and pituitary adenoma.

CLINICAL CASE

It is about a young man of 22 years old, without any notable history apart from a craniopharyngioma revealed at the age of 16 by a puberty delay. The initial presentation was a typical central hypogonadism with slight elevation of prolactin at 52 ng/mL, then considered as hyperprolactinemia of pituitary disconnection. The imaging revealed a tumor of the anterior stage of the skull with double fleshy and cystic component and microcalcifications; orienting the initial diagnosis towards a craniopharyngioma of the young teenager. He underwent a neurosurgical intervention, and the anatomopathological examination confirmed the histological diagnosis of papillary craniopharyngioma. The surgery was complicated by permanent adrenotropic and thyrotrophic insufficiencies for which he was put on substitutive treatment, also by a transient diabetes insipidus having marked remission later. In general, the evolution was good, notably a spontaneous onset of

puberty that was seen without recourse to androgen therapy. Then the patient was lost to sight. 5 years later he came for a bilateral gynecomastia grade 3. We find hyperprolactinemia this time with a tumor rate higher than 2000 ng/mL. The MRI shows a geant sellar process, infiltrating both cavernous sinuses, measuring at 86*40*66 mm. A compatible aspect rather of a pituitary adenoma than a recurrence of the craniopharyngioma. We also find thickened calcified bands occupying the roof of V3, and extending towards the brain peduncles related to craniopharyngioma residues (image 1). Characteristics that allowed us to decide on the diagnosis of a giant macroprolactinoma are both intra sellar localization and prolactin rate. He was put on dopaminergic analogues with gradual titration of the dose up to 2mg per day. The control assessment after 06 months demonstrated the following; biologically a prolactin rate of 160 ng/mL vs 2000. 8H cortisol = 268 nmol/L, FT4= 11.4 pmol/L (N:12-22), Testosterone= 9.56 nmol/L, LH= 2.6 and FSH= 1.8. While morphologically we assist to considerable reduction in the tumor volume which consolidates our initial hypothesis that it is a pure prolactinoma, this time measuring 38*30* 40 (Vs 86*40*66 mm) (image 2 and 3). The invasive character remained unchanged with bilateral invasion of the cavernous sinuses classified KNOSP4 at doite and KNOSP3 on the left, with a filling of the carotids reaching 180 degrees on the right side.

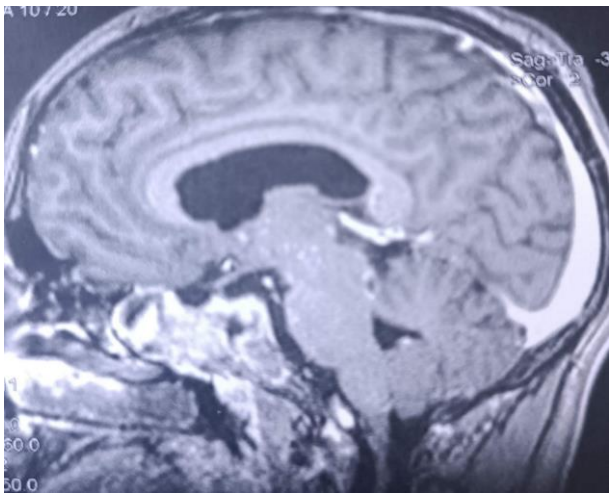

Image 1: Sagittal MRI section in our case-report, showing the typical location of an intra- and supra-sellar process (macro prolactinoma), with surgical residues from a craniopahyrngioma and calcification of the di-encephalon

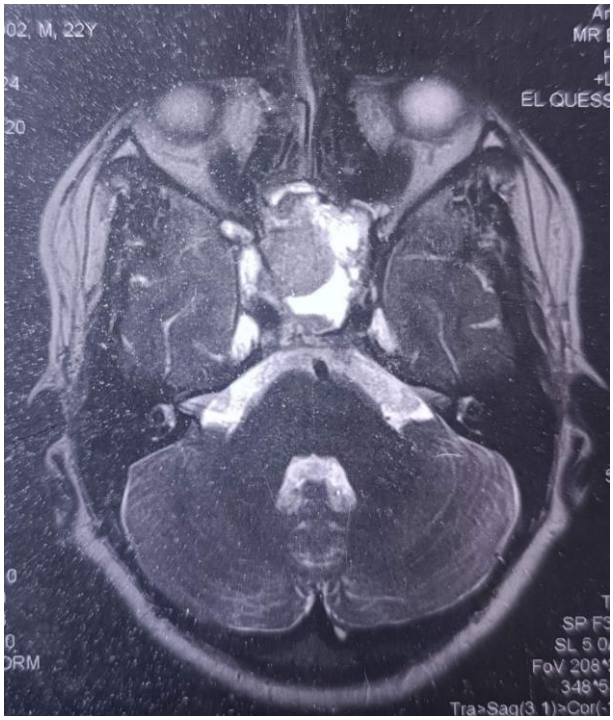

Image 2: Axial section of the control MRI in our patient demonstrates the reduction in pituitary adenoma size after dopmainergic analogues, appears heterogeneous on this section with intimate relations with the cavernous sinuses bilaterally.

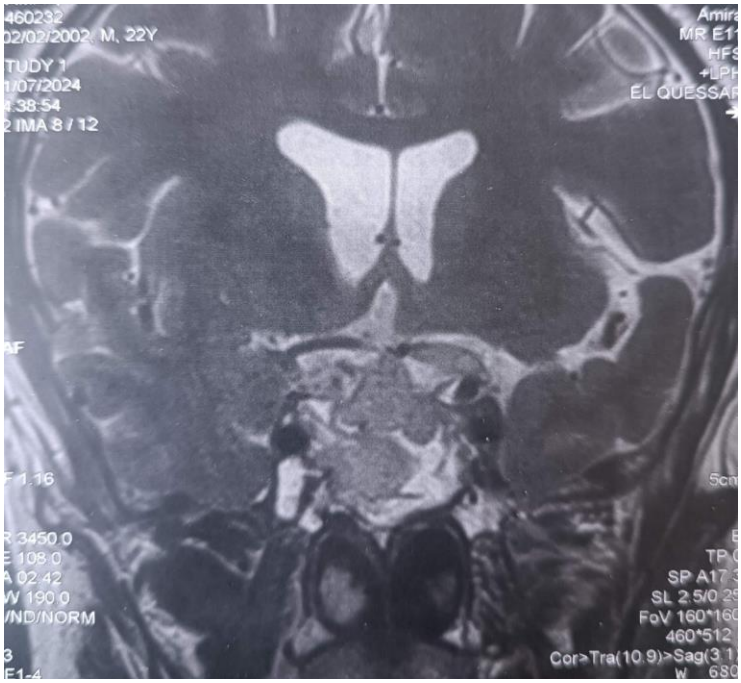

Image 3: Cross-sectional MRI through the di encephalon, showing the pituitary tumor process and its relationships with carotid siphons and the roof of V3.

DISCUSSION

Pituitary adenomas are the most common tumors in the sellar/ parasellar region, dominated by prolactin adenomas and non-functional PTINETs [1]. Their revelation is most often noisy related to the hormone secreted in abundance (prolactin, GH, ACTH) thus giving typical symptomatic pictures (galactorrhoea amenorrhea, acromegaly, cuhsing). However, when the adenoma is non-functional, the symptomatology can be summarized to compression related signs. Craniopharyngiomas are sellar/parasellar tumors accounting for 2% to 5% of primary intracranial neoplasms [7]. The very first pathological PCP report provided the essential topographical concept; their specific location within the 3V and their origin from the infundibulo-tuberal region of the third ventricle floor (3VF) [8]. The papillary type accounts for only around 10% to 30% of all CPs, while this proportion only represents 1% to 4.5% of all intra- cranial tumors [9][10]. Macroscopically, PCPs are typically well-circumscribed le- sions that usually grow either as compact solid masses or as unilocular cysts with a mural cauliflower-like nodule projecting inwards, adjacent to the infundibulum [10]. Notably, macroscopic calcifications are conspicuously absent in practically all PCPs whereas they are common in ACPs [11]. these characteristics are of great importance regarding tumor collision cases, obviously coming in the second degree of importance compared to the immuno-histochemical study which allows the confirmation of 2 or more cellular subtypes.

- **Characteristics found in collision states between PiTNET and craniopahyrngioma:**

In a cohort of 22 case-reports about collision of pituitary adenomas with craniopharyngioma, we were able to list a certain number of characteristics forming the biological and radiological clinical profile of this rare condition; the median age was around 47.0 years, which seems in line with solitary craniopharyngiomas and pituitary adenomas [6]. To note that preferred age for craniopharyngiomas follows basically a double peak in incidence: a pediatric distribution (0-19 years old) and an adult distribution [12]. Our patient would therefore be among the cases in whom the discovery of the craniopharyngioma was made at a young age. There was also a moderate male predominance, which seems to be different from regular craniopharyngiomas, in which no strong gender difference has generally been reported [13], nor pituitary adenomas, in which there's often a mild female dominance [14].

In our case-report, the nature of secretion was lactotropic with an extremely high initial rate of more than 2000 ng/mL (more than 42.000 mUI/L), even in cases of lactotropic adenoma, in which strong female predominance is usually observed, 3 of 5 patients with collision were male [15]. However, and following the cohort mentioned above; somatotropic (n = 5) and lactotropic (n = 5) adenomas are the major collision counterparts [6]. Finally, the size of the adenoma found in our patient is the highest compared to the series of colliding adenomas (86×40×66 Vs 58 × 54 reported by Gokden [16], Vs 46 × 38 ×36 reported by Yoshida [17], as the largest sizes reported).

- **Treatment and prognosis for collision tumors of the sellar and para-sellar area:**

Following certain cohorts interested in pituitary adenomas, worsened pituitary function and diabetes insipidus after surgical complications count for 2% to

12% in patients with solitary pituitary adenoma [18]. In patients with solitary pituitary adenomas, overall postoperative remission is obtained in approximately 60% or a bit more of all functioning adenomas; around 65% to 85% of lactotropic, 40% to 69% of somatotropic, 74% to 90% of corticotropic, and 50% to 82% of thyrotropic adenomas [19] [20]. These remission rates seem to be in perpetual improvement, which is due to progress in pituitary surgery techniques and major innovations in terms of the instruments used, notably microscopes. Craniopharyngiomas when occurring in collision of tumors are associated to a substantially decreased overall survival, with mortality 3-fold to 6-fold higher than that of the general population [21], worse than for patients with solitary craniopharyngiomas, in whom the 3-year and 5-year survival is reported as 86% to 97% and 80% to 96%; the overall survival is respectively [13] [22]. This could be due the result of suboptimal tumor control rate.

CONCLUSION

The presence of 2 tumors with different histology affecting both hypothalamic and pituitary regions is an extremely rare condition. Imaging is the cornerstone that allows etiological guidance, guided by clinical presentation and hormonal dosages. These entity of tumors constitutes a diagnostic and therapeutic challenges for any clinician whether he/she is an endocrinologist radiologist or neurosurgeon.

Abbreviation:

ACP: Adamantinomatous craniopharyngioma
PCP: Papillary craniopharyngioma
PiTNET: Pituitary Neuroendocrine Tumor
TSH: Thyroid stimulating hormone

REFERENCES

1. Hasegawa H, Jentoft ME, Young WF, *et al.,* Collision of craniopharyngioma and pituitary adenoma: comprehensive review of an extremely rare sellar condi- tion. World Neurosurg. 2021;149:e51ee62.
2. Pascual JM, Prieto R, Rosdolsky M, *et al.,* Joseph Engel (1816-1899), author of a meaningful dissertation on tumors of the pituitary infundibulum: his report on the oldest preserved whole craniopharyngioma specimen. *Virchows Arch*. 2020;476(5):773-782.
3. Crotty TB, Scheithauer BW, Young WF Jr, *et al.,* Papillary cranio- pharyngioma: a clinicopathological study of 48 cases. *J Neurosurg*. 1995;83(2):206-214.
4. Kahn EA, Gosch HH, Seeger JF, Hicks SP. Forty-five years experi- ence with the craniopharyngiomas. *Surg Neurol*. 1973;1(1):5-12
5. Adamson TE, Wiestler OD, Kleihues P, Yasargil MG. Correlation of clinical and pathological features in surgically treated cranio-pharyngiomas. *J Neurosurg*. 1990;73(1):12-17
6. Hirotaka Hasegawa1,3, Mark E. Jentoft4, William F. Young Jr2, Nikita Lakomkin1, Jamie J. Van Gompel1, Michael J. Link1, John L. Atkinson1, Fredric B. Meyer1Collision of Craniopharyngioma and Pituitary Adenoma: Comprehensive Review of an Extremely Rare Sellar ConditionWorld Neurosurg. (2021) 149: e51-e62https://doi.org/10.1016/j.wneu.2021.02.091
7. El-Bilbeisi H, Ghannam M, Nimri CF, Ahmad AT. Craniopharyngioma in a pa-tient with acromegaly due to a pituitary macroadenoma. Ann Saudi Med. 2010;30:485e488.
8. Pascual JM, Prieto R, Barrios L. Harvey Cushing's craniopharyng- ioma treatment: Part 1. Identification and clinicopathological characterization of this challenging pituitary tumor. *J Neurosurg*. 2018 ;131(3):949-963.
9. Castellanos LE, Gutierrez C, Smith T, Laws ER, Iorgulescu JB. Epidemiology of common and uncommon adult pituitary tumors in the U.S. according to the 2017 World Health Organization clas- sification. *Pituitary*. 2022;25(1):201-209.
10. Müller HL, Merchant TE, Warmuth-Metz M, Martinez-Barbera JP, Puget S. Craniopharyngioma. *Nat Rev Dis Primers*. 2019;5(1):75.
11. Prieto R, Barrios L, Pascual JM. Papillary craniopharyngioma: a type of tumor primarily impairing the hypothalamus-A compre- hensive anatomo-clinical characterization of 350 well-described cases. *Neuroendocrinology*. 2022;112(10):941-965.
12. Zacharia BE, Bruce SS, Goldstein H, Malone HR, Neugut AI, Bruce JN. Incidence, treatment and survival of patients with craniopharyngioma in the surveillance, epidemiology and end results program. Neuro Oncol. 2012; 14:1070-1078.
13. Bunin GR, Surawicz TS, Witman PA, Preston-Martin S, Davis F, Bruner JM. The descriptive epidemiology of craniopharyngioma. J Neurosurg. 1998;89:547-551.
14. Raappana A, Koivukangas J, Ebeling T, Pirila T. Incidence of pituitary adenomas in Northern Finland in 1992-2007. J Clin Endocrinol Metab. 2010; 95:4268-4275.
15. Agustsson TT, Baldvinsdottir T, Jonasson JG, *et al.,* The epidemiology of pituitary adenomas in Iceland, 1955-2012: a nationwide population- based study. Eur J Endocrinol. 2015; 173:655-664.
16. Gokden M, Mrak RE. Pituitary adenoma with craniopharyngioma component. Hum Pathol. 2009; 40:1189-1193
17. Yoshida A, Sen C, Asa SL, Rosenblum MK. Composite pituitary adenoma and craniophar-yngioma?: an unusual sellar neoplasm with divergent differentiation. Am J Surg Pathol. 2008; 32:1736-1741.
18. Berker M, Hazer DB, Yucel T, *et al.,* Complications of endoscopic surgery of the pituitary adenomas: analysis of 570 patients and review of the litera- ture. Pituitary. 2012; 15:288-300.

19. Losa M, Giovanelli M, Persani L, Mortini P, Faglia G, Beck-Peccoz P. Criteria of cure and follow-up of central hyperthyroidism due to thyrotropin-secreting pituitary adenomas. J Clin Endocrinol Metab. 1996; 81:3084-3090.
20. Dorward NL. Endocrine outcomes in endoscopic pituitary surgery: a literature review. Acta Neurochir (Wien). 2010; 152:1275-1279.
21. Zacharia BE, Bruce SS, Goldstein H, Malone HR, Neugut AI, Bruce JN. Incidence, treatment and survival of patients with craniopharyngioma in the surveillance, epidemiology and end results program. Neuro Oncol. 2012; 14:1070-1078
22. Muller HL. Craniopharyngioma. Endocr Rev. 2014; 35:513-543.