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Case Report

Coexisting Intracranial Tumors with Pituitary Adenomas: Genetic Association or Coincidence?

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Abstract

Coexisting pituitary lesions may range from clinically non-functioning adenomas to hormonally active tumors such as prolactinomas and growth hormone (GH) or thyrotropin (TSH)-secreting adenomas. A 22-year-old male presented with a two-month history of generalized tonic-clonic seizures, accompanied by signs of intracranial hypertension, including intermittent frontal headaches resistant to analgesics and a sudden decrease in visual acuity. He also reported a 20 kg weight gain and an increase in shoe size from 40 to 44 over a seven-month period, without any decline in libido. Clinical examination revealed normal blood pressure and heart rate, and no dysmorphic features, particularly no acromegaloid characteristics. The patient had moderate obesity (BMI: 34 kg/m²), bilateral gynecomastia, mild violaceous striae, no galactorrhea, and was classified as Tanner stage G5P5. Hormonal evaluation showed hyperprolactinemia at 278 ng/mL, central hypothyroidism (TSH: 0.6 mIU/L; free T4: 8 pmol/L), and central hypogonadism (FSH: 1.37 IU/L; LH: 1.1 IU/L; total testosterone: 1.80 ng/mL). IGF-1 was within the normal range (275.8 ng/mL; reference: 120–338). Morning cortisol was 15 µg/dL, with an appropriate suppression after a 1 mg overnight dexamethasone test (0.7 µg/dL). A 24-hour urinary free cortisol measurement was also normal (75 µg/24h). HbA1c was 5.5%. Ophthalmologic examination revealed a normal fundus, but visual field testing showed nasal isopter narrowing. Pituitary MRI demonstrated a well-defined intra- and suprasellar lesion measuring 19 × 16 × 19 mm, consistent with a pituitary macroadenoma. Additionally, an infiltrative cortical and subcortical lesion in the fronto-cingulate region (36 × 24 × 47 mm) suggested a low-grade glioma. The patient was started on cabergoline 0.5 mg twice weekly and levothyroxine 25 µg daily. Neurosurgical intervention for the glioma was performed with gross total resection. Histopathological analysis confirmed a low-grade glial proliferation. Postoperative clinical and biochemical follow-up showed favorable outcomes. This case highlights the need for comprehensive neuroimaging in patients diagnosed with pituitary adenomas who present with atypical neurological symptoms, such as seizures.

Keywords: Glioma, pituitary adenomas, coexisting, hyperprolactinemia.

INTRODUCTION

Gliomas account for approximately 50% of primary intracranial neoplasms. Pituitary adenomas (PAs) have been reported to co-occur with glial tumors of varying grades. Coexisting pituitary lesions may range from clinically non-functioning adenomas to hormonally active tumors such as prolactinomas and growth hormone (GH) or thyrotropin (TSH)-secreting adenomas. We present a rare case of synchronous PA and glioma, and explore potential underlying mechanisms, including previously reported genetic abnormalities that might link these two distinct pathologies [1,2,4,5].

OBSERVATION

A 22-year-old male presented with a two-month history of generalized tonic-clonic seizures, accompanied by signs of intracranial hypertension, including intermittent frontal headaches resistant to analgesics and a sudden decrease in visual acuity. He also reported a 20 kg weight gain and an increase in shoe size from 40 to 44 over a seven-month period, without any decline in libido. Clinical examination revealed normal blood pressure and heart rate, and no dysmorphic features, particularly no acromegaloid characteristics. The patient had moderate obesity (BMI : 34 kg/m²), bilateral gynecomastia, mild violaceous striae, no galactorrhea, and was classified as Tanner stage G5P5.Hormonal evaluation showed hyperprolactinemia

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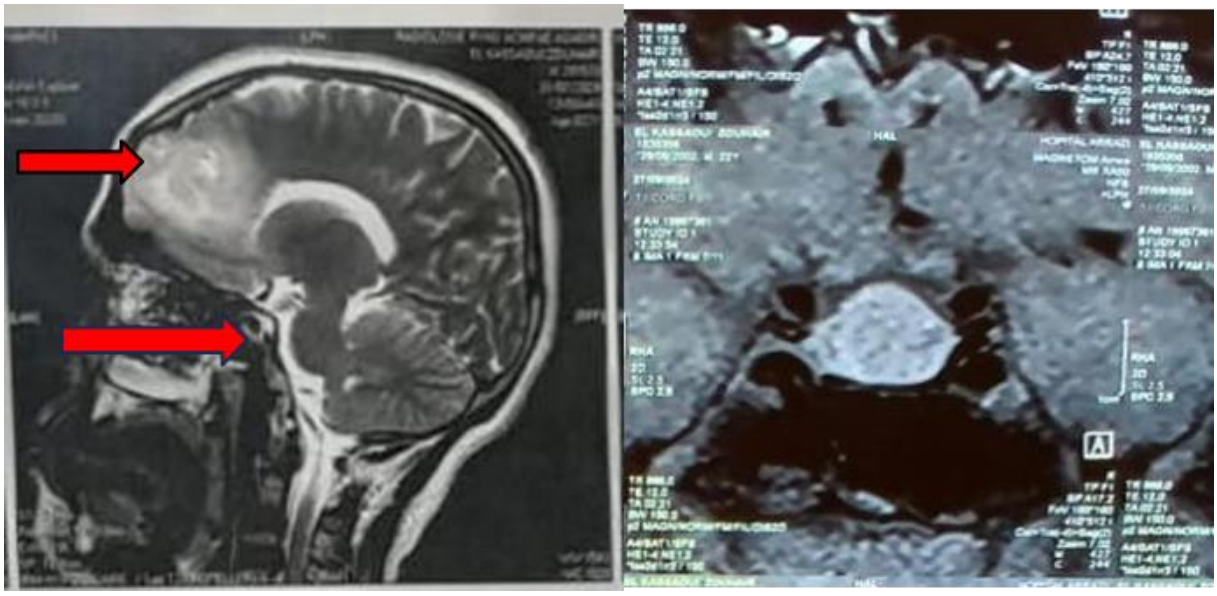
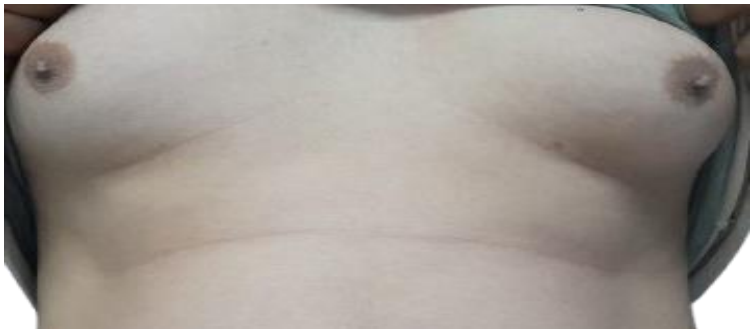


Pituitary Macroadenoma Associated with a Frontal Glioma



Bilateral Gynecomastia

DISCUSSION

The synchronous occurrence of a pituitary macroadenoma and a low-grade glioma, as seen in our patient, is exceedingly rare. To date, only 13 similar cases have been reported in the literature [4,5,8]. This rarity raises the question of whether such an association is coincidental or underpinned by a shared pathogenic mechanism. Some authors, including Russell and Rubinstein [4], have proposed that the coexistence of these tumors in non-syndromic patients is merely coincidental. However, repeated reports of gliomas occurring alongside pituitary adenomas—particularly prolactinomas— have prompted speculation about common molecular or genetic factors. Several chromosomal abnormalities have been reported in both tumor types, including deletions on chromosomes 1p, 4, 5, 6, 11q, 13q, and 18q, and gains on 17p, 19, and 20q [9]. Gliomas, in particular, have been linked to deletions involving chromosome 19p13.3 [10]. This overlap, especially involving chromosome 19, may point to a shared genetic predisposition. Furthermore, fibroblast growth factor receptor-1 (FGFR1), expressed in both pituitary adenomas and gliomas, may contribute to mitogenic activity and tumor proliferation [11]. The presence of hormone receptors—such as estrogen and progesterone receptors—in both tumor types also suggests potential endocrine influences in tumor development [5,12]. It is important to note that no

genetic or immunohistochemical analyses were performed in this case to confirm a shared molecular etiology. The typically indolent nature of both tumor types may lead to underdiagnosis in asymptomatic or mildly symptomatic patients, contributing to underreporting. This case highlights the need for comprehensive neuroimaging in patients diagnosed with pituitary adenomas who present with atypical neurological symptoms, such as seizures. Future molecular studies may shed light on possible genetic links between these tumors and enhance our understanding of their concurrent development.

CONCLUSION

Although no definitive causative factors have been established, the rare coexistence of non- syndromic pituitary adenomas and gliomas may indicate a shared, yet unidentified, genetic or molecular mechanism. Recognizing such associations can improve diagnostic vigilance and potentially guide future research on tumorigenesis.

Compliance with Ethical Standards:

Acknowledgments: We would like to thank the teams of endocrinology, hematology, radiology and biology departments of Mohammed VI University hospital of Marrakech.

Disclosure of Conflict of Interest: The authors declare no conflict of interest.

Statement of Ethical Approval: The present research work does not contain any studies performed on animals/human's subject by any of the authors.

Statement of Informed Consent: Informed consent was obtained from all individual participants included in the study.

BIBLIOGRAPHIE

1. Curto L, Squadrito S, Almoto B, Longo M, Granata F, Salpietro F, *et al.,* MRI finding of simultaneous coexistence of growth hormone-secreting pituitary adenoma with intracranial meningioma and carotid artery aneurysms: report of a case. Pituitary 2007; 10:299-305.
2. da Costa LB, Riva-Cambrin J, Tandon A, Tymianski M. Pituitary adenoma associated with intraventricular meningioma: case report. Skull Base 2007; 17:347-51.
3. Amit A, Achawal S, Dorward N. Pituitary macro adenoma and vestibular schwannoma: a case report of dual intracranial pathologies. Br J Neurosurg 2008; 22:695-6.
4. Furtado SV, Dadlani R, Ghosal N, Mahadevan A, Shankar SK, Hegde AS. Co-existing thyrotropin secreting pituitary adenoma and low-grade glioma: clinical considerations and literature review. J Neurosurg Sci 2009; 53:71-5.
5. Mathuriya SN, Vasishta RK, Dash RJ, Kak VK. Pituitary adenoma and parasagittal meningioma: an unusual association. Neurol India 2000 ;48 :72-4.
6. Samaras V, Samaras E, Stergiou I, KonstantopoulouP, Arnaoutoglou C, Arnaoutoglou M, *et al.,* Simultaneous occurrence of cerebellar medulloblastoma and pituitary adenoma : A case report. Cases J 2008 ;1 :175.
7. Fernandez A, Karavitaki N, Ansorge O, Fazal-Sanderson V, Wass JA. Acromegaly and anaplastic astrocytoma: coincidence or pathophysiological relation? Pituitary 2008 ;11 :325-30
8. Szymas J, Schluens K, Liebert W, Petersen I. Genomic instability in pituitary adenomas. Pituitary 2002; 5:211-9.
9. Ritland SR, Ganju V, Jenkins RB. Region-specific loss of heterozygosity on chromosome 19 is related to the morphologic type of human glioma. Genes Chromosomes Cancer 1995 ;12 :277-82.
10. Ueba T, Takahashi JA, Fukumoto M, Ohta M, Ito N, Oda Y, *et al.,* Expression of fibroblast growth factor receptor-1 in human glioma and meningioma tissues. Neurosurgery 1994 ;34 :221-5.
11. Pravdenkova S, Al-Mefty O, Sawyer J, Husain M. Progesterone and estrogen receptors: opposing prognostic indicators in meningiomas. J Neurosurg 2006 ;105 :163-73.
12. Bello MJ, de Campos JM, Kusak ME, Vaquero J, Sarasa JL, Rey JA. Chromosomal abnormalities in pituitary adenomas. Cancer Genet Cytogenet 2001 ;124 :76-9.
13. Bettio D, Rizzi N, Giardino D, Persani L, Pecori-Giraldi F, Losa M, *et al.,* Cytogenetic study of pituitary adenomas. Cancer Genet Cytogenet 1997; 98:131-6.