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The endocrine background of meningioma carcinogenesis

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Abstract

Meningiomas, being mostly benign tumors, are derived from the arachnoid cap cells, their etiopathogenesis is based on various factors. The etiology of sporadic meningiomas is not yet known. Many factors have been identified as possible causes of the development of intracranial meningiomas. These include head trauma, viral infections, deletion in the NF2 gene, the use of cell phones, and sex hormones. The review is based on a endocrine factors, playing a role in meningioma carcinogenesis.

The carcinogenesis of meningioma appears to be profoundly dependent from hormonal factors. Mayor ones, usually underlined in according to their prognostic significance, are female sex hormones. Due to this, meningiomas are twice as more likely to occur in female than in male patients. The other group of hormones appointed to play a role in meningioma carcinogenesis are adipokines in general – and leptin in particular. Leptin secretion correlates with BMI elevation, what may explain the confirmed linking between obesity and brain tumors. The scientific literature has documented the occurrence of meningioma in five patients with CAH, but the role of cortical axis and/or ACTH secretion impairments is still

under consideration. Authors didn't find any publication about the role of thyroid disorders in meningioma carcinogenesis.

The carcinogenesis of meningioma appears to be profoundly dependent from hormonal factors. The effects of female sex hormones and adipokines are under a significant consideration, and may be useful in severity prediction. Basic science research should be focused on ACTH secretion in meningioma and possible common genetic etiopathogenesis of meningioma and CAH.

Keywords: meningioma, female sex hormones, adipokines, ACTH, congenital adrenal hyperplasia, neurosurgery, endocrinology

Introduction

Meningiomas, being mostly benign tumors, are derived from the arachnoid cap cells, their etiopathogenesis is based on various factors. Meningiomas are most commonly found on the convexity (19% to 34%) and in parasagittal locations (18% to 25%), followed by the sphenoid wing and middle cranial fossa (17% to 25%), anterior skull base (10%), posterior fossa (9% to 15%), cerebellar convexity (5%) and clivus (<1%)[1]. They are more common in the older age group (incidence over age 85 years: 52.95 per 100,000 persons) and female patients (incidence: 11.25 per 100,000 females vs 5.15 per 100,000 males). Five-year survival of patients with typical meningiomas exceeds 80% but decreases for malignant and atypical meningiomas. Among poor prognostic factors are large tumor sizes, deletions and loss of heterozygosity, high mitotic index, absence of progesterone receptors, and papillary and hemangiopericytic tumor morphology [1–3]. The etiology of sporadic meningiomas is not yet known. Many factors have been identified as possible causes of the development of intracranial meningiomas. These include head trauma, viral infections, deletion in the NF2 gene, the use of cell phones, and sex hormones[3]. The review is based on a endocrine factors, playing a role in meningioma carcinogenesis.

Methods

To access necessary articles, the literature review was performed using two databases – PubMed and GoogleScholar. Used keywords included “meningioma” and particular hormones names. Articles written in languages other than Polish and English were rejected.

State of the art

The carcinogenesis of meningioma appears to be profoundly dependent from hormonal factors. Mayor ones, usually underlined in according to their prognostic significance, are female sex hormones. Due to this, meningiomas are twice as more likely to occur in female than in male patients [2]. Progesterone receptors are detected in 79,5% of meningiomas, and correlate with better WHO Grade [4,5]. This hormonal status may also be a key to an unexplained (and questionable) linking between meningioma and breast cancer[2,6].

The other group of hormones appointed to play a role in meningioma carcinogenesis are adipokines in general – and leptin in particular [7]. Leptin secretion correlates with BMI elevation, what may explain the confirmed linking between obesity and brain tumors[8]. The patients with meningioma have significantly higher concentration of plasma leptin, which strengthen the hypothesis of leptin impact on brain tumors etiopathogenesis[9].

The subject of impairments in ACTH-cortisol axis in meningioma patients was not under particular scientific consideration yet. This topic in scientific literature is usually considered in context of meningioma and pituitary adenoma coexistence – which may result with Cushing disease[10]. On the other hand, rapid growth of meningioma, may result up with hypoglycemia[11]. The scientific literature has documented the occurrence of meningioma in five patients with CAH. Their demographic and clinical features are compared in Table 1.

	Case 1	Case 2	Case 3	Case 4
Year	1995	2008	2016	2021
Sex	“46,XX male (severe female hermaphrodite raised as a male)”	46, XX male	female	genetically born female (46,XX)
Age	43	47	45	38
Meningioma size	-	-	-	4.3 × 4.6 cm
Meningioma Localization	the right medial sphenoid area and partly in the cavernous sinus	mostly extra axial, right frontal/temporal and also in the right orbital and in the intra-orbital fossa on the right site	centered upon the right greater wing of sphenoid, expanding into the middle cranial fossa, lateral orbit and infra-temporal fossa	centered on the left petrous apex with involvement of left cavernous sinus and Meckel’s cave
17-Hydroxyprogesterone	-	552 nmol/l	>180 nmol/L	-
Plasma Cortisol	82.6 nmol/L	105 nmol/l	-	-
Height	163 cm	150 cm	148 cm	-
Reference	[12]	[13]	[14]	[15]

Tab. 1. Comparison of demographical and clinical data of patients with CAH, diagnosed with meningioma.

Authors didn’t find any publication about the role of thyroid disorders in meningioma carcinogenesis.

Conclusion

The carcinogenesis of meningioma appeals to be profoundly dependent from hormonal factors. The effects of female sex hormones and adipokines are under a significant consideration, and may be useful in course severity prediction. However congenital adrenal

hyperplasia (or in general ACTH secretion impairments) may be a possible rare cause of meningioma, this correlation needs further research. Basic science research must be focused on ACTH secretion in meningioma and possible common genetic etiopathogenesis of meningioma and CAH.

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