"IMMUNOHISTOCHEMICAL CHARACTERISTICS OF PATIENTS WITH MACRO AND GIANT INACTIVE PITUITARY TUMORS"

Yulduz Makhkamovna Urmanova*; Mirtukhtaeva Malika Bakhtiyarovna**

*Professor, Doctor of Medical Sciences (DSc), Department of Endocrinology, Pediatric Endocrinology, TashPMI, Tashkent, UZBEKISTAN Email id: yulduz.urmanova@mail.ru

**Endocrinologist, Department of Neuroendocrinology with Pituitary Surgery, RSNPMCCE of the Ministry of Health of the Republic, Uzbekistan name of acad. Y.H. Turakulov, Employment Contract, Tashkent, UZBEKISTAN DOI: 10.5958/2278-4853.2023.00067.8

ABSTRACT

The assessment of the KI-67 and P53 using immunohistochemistry, usually with monoclonal antibodies of MIB1, is mandatory for evaluating proliferation in patients subjected to transnasal adenomectomy of the pituitary gland.

Goal. To study the prognostic significance of invasion and markers of proliferation in patients with macro and giant inactive pituitary tumors.

Material and Methods

In total, 272 patients with macro and giant naga were examined. Of the 272 patients with the naga in the study, 151 patients (men and women) took part in the study)

Research methods included: 1) general clinical (study of endocrine, neurological statuses), 2) instrumental (perimetry for all colors, eye bottom, visual acuity, 3) ECG, CT/MRI of the Turkish saddle and adrenal glands, 4) ultrasound of the internal and genitals, etc.), 5) hormonal blood tests (STH, IFR-1, LG, FSG, PRL, TSL, ACTH, prolactin, testosterone, estradiol, progesterone, cortisol and immunohymph hand-chemical studies.

Results

The observed frequency of immunoexpression of proliferation markers was 40%/50% for P53 ($\geq 3+$), 50%/60% for Ki-67 ($\geq 2+$). Tumors with immunoexpression of at least 2 markers with a high proliferation index were observed in 54% cohorts and regarded as proliferative adenomas.

Conclusion

Giant inactive pituitary adenomas of the pituitary gland are often accompanied by invasive growth in the surrounding anatomical structures (more than 80% of cases), which is the main factor that limits the radicality of surgical intervention and increases the number of relapses.

KEYWORDS: NFPA, Giant Pituitary Adenomas.

BIBLIOGRAPHY

- **1.** Molitch ME. Diagnosis and treatment of pituitary adenomas: a review. JAMA. 2017; 317:516-524.
- **2.** Freda PU, Beckers AM, Katznelson L, et al. Pituitary incidentaloma: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2011; 96:894-904.
- **3.** Greenman Y, Stern N. Non-functioning pituitary adenomas. Best Prac Res Clin Endocrinol Metab. 2009; 23:625-638.
- **4.** Drange MR, Fram NR, Herman-Bonert V, et al. Pituitary tumor registry: a novel clinical resource. J Clin Endocrinol Metab. 2000; 85:168-174.
- **5.** Lloyd RV, Osamura YR, Kloppel G, Rosai J. WHO Classification of Tumors of Endocrine Organs. Geneva, Switzerland: WHO Press; 2017:78-80
- **6.** Huan W, Molitch M.E. Management of nonfunctioning pituitary adenomas (NFAs): observation. Pituitary. 2018;21:162-167
- 7. Olsson DS, Hammarstrand C, Bryngelsson L, et al. Incidence of malignant tumors in patients with a non-functioning pituitary adenoma. Endocr Relat Cancer. 2017; 24:227-235.
- **8.** Lee M, Lupp A, Mendoza N, et al. SSTR3 is a putative target for the medical treatment of gonadotroph adenomas of the pituitary. Endocr Relat Cancer. 2015; 22:111-119.
- **9.** Dai C, Sun B, Liu X, et al. O-6-Methylguanine-DNA methyltransferase expression is associated with pituitary adenoma tumor recurrence: a systematic meta-analysis. oncotarget. 2017; 8:19674-19683.
- **10.** Lenders N, McCormack A. Malignant transformation in non-functioning pituitary adenomas (pituitary carcinoma). Pituitary. 2018; 21:217-229.
- **11.** Pernicone PJ, Scheithauer BW, Sebo TJ, et al. Pituitary carcinoma: a clinicopathologic study of 15 cases. Cancer. 1997; 79:804-812.
- **12.** Hansen TM, Batra S, Lim M, et al. Invasive adenoma and pituitary carcinoma: a SEER database analysis. Neurosurgical Rev. 2014; 37:279-285.
- **13.** Van der Zwan JM, Mallone S, van Dijk B, et al. Carcinoma of endocrine organs: results of the RARECARE project. Eur J Cancer. 2012; 48:1923-1931.
- **14.** Lopes MBS. The 2017 World Health Organization classification of tumors of the pituitary gland: a summary. Acta Neuropathologica. 2017; 134:521-535.
- **15.** Daly AF, Tichomirowa MA, Beckers A. The epidemiology and genetics of pituitary adenomas. Best Pract Res Clin Endocrinol Metab. 2009; 23:543-554.
- **16.** Pei L, Melmed S, Scheithauer B, Kovacs K, Prager D. H-ras mutations in human pituitary carcinoma metastases. J Clin Endocrinol Metab. 1994; 78:842-846.

- **17.** Rickert CH, Scheithauer BW, Paulus W. Chromosomal aberrations in pituitary carcinoma metastases. Acta Neuropathol. 2001;102:117-120
- **18.** Molitch, ME Pituitary incidentalomas//Endocrmetab clin north am.- 2013. Vol. 26. P. 725-740.
- **19.** Nagasaka, T. / Sarcomatous transformation of pituitary adenoma after bromocriptine therapy. // Hum pathol. -1998.-Vol. 29, No. 2.-P. 3-190.
- **20.** Lloyd RV, Kovacs K., Young WF, Jr., et al. Pituitary tumours: introduction. In: DeLellis R. 20/A., Lloyd RV, Heitz PU, Eng C., editors. //Tumours of the Pituitary, Chapter 1. Pathology and Genetics of Tumors of Endocrine Organs. Lyon, Paris: World Health Organization Classification of Tumors. IARC Press; 2004.pp. 10–13
- **21.** Mccormack AI, Wass JAH, Grossman AB Aggressive pituitary tumours: the role of temozolomide and the assessment of MGMT status. // European Journal of Clinical Investigation. 2011; 41(10):1133–1148. doi: 10.1111/j.1365-2362.2011.02520.x.