

Acta endocrinologica

Supplementum 184

ADVANCE ABSTRACTS OF PAPERS

20. Symposium Deutsche Gesellschaft für Endokrinologie
Tübingen
February 27 - March 2, 1974

PERIODICA · COPENHAGEN 1974

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II. Medizinische Klinik¹
und I. Frauenklinik² der Universität München

PROLACTIN SECRETION IN PATIENTS WITH PITUITARY TUMORS*

K. v. Werder¹, C. R. Pickardt¹, B. Glöckner¹,
M. Gottsmann¹, H. K. Rjosk² and P. C. Scriba¹

Problem: To elucidate the dynamics of prolactin (hPR) secretion in patients with pituitary tumors we set up a double antibody hPR-radioimmunoassay using the V.L.S.-reagents (National Pituitary Agency, NIAMD, USA).

Methods: 1 µg of Lewis-hPR was labelled with the chloramine-T-method and purified by one column chromatography on Sephadex G-100. Pooled pregnancy serum calibrated with the research standard A-71/222 (MRC, Division of Biolog. Stand., Holly Hill, London) served as standard: 1 ml of this standard serum contained 4000 µU of hPR-71/222, which was equivalent to 200 ng hPR-V.L.S. All data were expressed in µU hPR-72/222. There was only 0.1% crossreactivity with hGH and none with hPL, LH, TSH. The sensitivity of the assay ($B_0 \pm 3 SD$) was 12 µU/ml, the between assay coefficient of variation was 9.1%.

Results: The basal hPR-levels in normal males ranged from 170 to 500 µU/ml ($N = 21$), in females up to 650 µU/ml ($N = 20$). 55% of patients with *active acromegaly* ($N = 22$) had elevated basal hPR-levels ranging from 720 to 5400 µU/ml, whereas only 3 of 15 patients with inactive acromegaly had still elevated hPR-levels. No correlation existed between tumor size, hGH-secretion, and the hPR-level. The response to TRH was blunted in most patients tested. — The basal hPR-levels of 8 patients with amenorrhea, galactorrhea and pituitary tumor (*Forbes-Albright syndrome*) ranged from 2300 to 85000 µU/ml, the patients with the larger tumors having the higher hPR-levels. TRH injection caused only little further increase of the hPR-level. The hPR-levels could be effectively lowered by the removal of the tumor or by administration of CB 154, with concomitant disappearance of galactorrhea and amenorrhea. Three patients with galactorrhea without pituitary tumors had normal hPR-levels with a normal rise after TRH. — A dissociation of the TSH and hPR-response to TRH was seen in several of the patients with *disorders of thyroid function*. hPR-levels below the normal range with nonresponsiveness to TRH were only found in patients with complete anterior pituitary failure.

Conclusions: These results demonstrate that hyperprolactinemia is not only found in patients with hPR-producing tumors, though in these patients the hPR-levels may be extremely elevated, correlating with the tumor's size. Whether hyperprolactinemia in acromegaly is due to hypothalamic dysfunction, i. e. lack of PIF, or is rather a result of autonomous hypersecretion of hPR is still unclear. The blunted response to TRH in most cases, however, and the fact that small tumors can be accompanied by elevated hPR-levels, would favour an autonomous hPR-hypersecretion.

* Supported by Deutsche Forschungsgemeinschaft (SFB 51)