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Increased Serum High-Sensitivity C-Reactive Protein Levels in Adult Growth Hormone Deficient Patients with Non-Functioning Pituitary Tumors

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Background: Growth hormone (GH) deficiency, a common endocrine deficit in non-functioning pituitary tumors, causes visceral obesity and fatty liver and increases cardiovascular event risks. High-sensitivity C-reactive protein (hs-CRP) has been used as a useful marker to estimate cardiovascular event risks. Because GH supplementation therapy was reported to decrease serum hs-CRP levels in GH deficient patients, inflammatory processes might be activated in GH deficient state, however, the underlying mechanism has been still unknown. **Patients and Methods:** We retrospectively reviewed charts of 134 patients with non-functioning pituitary adenoma and Rathke's cysts who underwent preoperative GH-releasing peptide-2 (GHRP-2) tests and investigated the association between serum hs-CRP levels and background

characteristics. Patients who had a history of pituitary surgery, severe renal insufficiency or active inflammatory diseases or received GH supplementation therapy were excluded. GH secretion was determined by GHRP-2 tests. **Results:** Among 134 patients (94 NFPA and 40 Rathke's cysts), 46 (34%) presented severe GH deficiency, as diagnosed using GHRP-2 tests. Serum hs-CRP levels were significantly higher in the patients with severe GH deficiency than in those without severe GH deficiency (723 [299-1285] vs 278 [124-561] ng/mL, P < 0. 001). Serum hs-CRP levels were significantly higher in men (P = 0. 003) and in patients with diabetes mellitus (P = 0. 040) and were significantly correlated with age (r s = 0.19, P = 0. 039), body mass index (r s = 0.37, P < 0. 001), serum levels ofgamma-glutamyl transpeptidase(r s = 0.28, P = 0. 001), creatinine (r s = 0.30, P < 0. 001), low-density lipoprotein cholesterol (r s = 0.21, P = 0. 013), triglyceride (r s = 0.38, P < 0. 001) and free thyroxine (r s = -0.30, P= 0. 001), blood hemoglobin A1c levels (r s = 0.20, P = 0. 018), peak GH response to GHRP-2 (r s = -0.47, P < 0. 001) and IGF-1 SD score (r s = -0.18, P = 0. 040). In the multiple regression analysis, peak GH response to GHRP-2 was a significant variable for determining serum hs-CRP levels (β = -0.340, P = 0. 003) after adjustment with age, sex, BMI, smoking, alcohol consumption, hypertension, diabetes mellitus, serum levels ofgamma-glutamyl transpeptidase, creatinine,triglyceride and free thyroxine and adrenal function. **Conclusion:** We observed a significant association between GH deficiency and increased serum hs-CRP levels independent to BMI and liver dysfunction. GH deficient state might cause inflammation independent to development of visceral obesity and fatty liver.

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