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Endocrine Hormonal Changes as the Consequence of Obesity

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Introduction

Obesity is a metabolic disease with increasing prevalence even in childhood, in which etiopathogenesis the adipose tissue plays a crucial role as endocrine and metabolically active organ. More than 95% cases of obesity is caused by unhealthy lifestyle (food abundance and lack of physical activity), while only less than 5% of obesity is caused by endogenous etiological factors (monogenic or syndromic forms of obesity, hypothyreosis, hypercorticism) [1]. Untreated obesity may be associated with many chronic diseases, negatively influencing the quality of life, such as hypertension, cardiovascular diseases, arthrosis, type 2 diabetes and its complications, psychologic disorders or fatty liver disease [2,3]. Obese individuals have significantly higher concentrations of pro-inflammatory cytokines and altered profile of secreted adipokines, leading in particular to insulin resistance, dyslipidemia and subclinical inflammation with many implications including endocrine hormonal changes.

Obesity is associated with changes in the concentration of thyroid hormones, mainly increased thyrotropin (TSH) and free triiodothyronine (fT3) with normal concentration of free thyroxine (fT4) and negative autoantibodies. Pathogenetic factors include reduced tissue response to circulating thyroid hormones (with subsequent compensatory increase in TSH), increased leptin concentration (which, in addition to its other functions, stimulates TRH secretion and deiodinase activity) or the influence of pro-inflammatory cytokines inhibiting thyroid uptake of iodine. According to current information, an isolated TSH elevation is the consequence of obesity [4], not its cause, so the substitution treatment with levothyroxine is not indicated. Moreover, some studies indicate the link between obesity and autoimmune thyroiditis, as well as other autoimmune diseases.

Obesity can contribute to the early onset of puberty in girls, while data from boys are not entirely uniform. A possible factor in this etiology is decreased concentration of liver sex hormone-binding globulin (SHBG) with consequent increased free sexual hormones; hyperandrogenism due to stimulation of proinflammatory cytokines; and leptin, which centrally stimulates the pulsatile secretion of gonadotropins. As fat tissue is a source of enzyme aromatase converting androgens into estrogens, in obese men excessive aromatase activity can lead to gynecomastia and to an obesity-related infertility [5].

In women with polycystic ovary syndrome, obesity may be associated with dysregulation of gonadotropins - with increased LH/FSH ratio, which may contribute to ovarian hypersecretion of androgens and to secondary infertility [6]. Obese women have usually insulin resistance leading to compensatory hyperinsulinism. Interestingly, ovaries are not insulin resistant, but insulin acts as co-gonadotropin and stimulates ovarian production of androgens. Changes in the prolactin concentration in obese individuals are not completely clear.

Discussion and Conclusion

In obese subjects, a lower growth hormone concentration is observed, flattened response of growth hormone in stimulation tests and higher IGF-1 concentrations. These hormonal changes are, in some studies, associated with the atherogenic plasma profile and with higher cardiovascular risk [7]. Information about cortisolemia in obese subjects is not uniform; however, most studies claim the elevated concentration of free urinary cortisol. Despite obesity is thought to be related to increased sympathoneural activity, another possible feature is adrenal medullary dysfunction and reduced adrenal secretion and storage of epineprine [8].

Better insight into these hormonal changes is convenient to distinguish between endocrine changes due to obesity (which are usually initially reversible) and possibly associated endocrinopathies. Weight reduction can lead to adjustment of hormonal imbalance, to prevention of permanent organ changes and to improvement of overall quality of life.

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