DHEAS Levels in Obese Patients with Hashimato Thyroiditis

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Introduction

Dehydroepiandrosterone (DHEA) and its sulfate ester (DHEAS) are the most abundant steroid hormones in the human bloodstream. DHEAS is produced primarily in the adrenal gland, where it reflects adrenal androgen production (Labrie et al., 2005). Although it has been proposed that reduced DHEA levels contribute to increased insulin resistance, it has been shown that insulin increases DHEA clearance, suggesting that low DHEA levels may in fact be a consequence of insulin resistance (Tchernof and Labrie, 2004).

DHEA and DHEAS are precursors in the biosynthesis of the sex steroid hormones. Multiple studies have found that serum levels of these steroids are much higher in male than in female inidividuals, and that the gender-specific associations exist between DHEA/DHEAS and health or survival. However, the latter findings are often contradictory, with some studies suggesting that such associations exist only in male individuals, other studies finding no significant differences between genders, and other studies (Berr et al., 1996; Glei et al., 2004) reporting stronger associations among female than male individuals.

Despite extensive research on DHEA and DHEAS, little is known about their specific functions, or about the mechanisms by which they affect health (Celec and Starka, 2003). Research suggests that DHEA may counterbalance the immunosuppressive effects of glucocorticoids, although the receptor for DHEA has not been fully characterized (Butcher and Lord, 2004). Moreover, DHEA has been shown to regulate processes as diverse as hemostasis, cell proliferation, lipid metabolism, stress response, and immune function (Feldman et al., 2001; Thijssen and Nieuwenhuyse, 1999).

Hashimoto's thyroiditis is the most common autoimmune endocrine disorders and often leads to hypothyroidism. It is an inherited condition and 7 times more common in women than in men. The disease is characterized by the production of immune cells and autoantibodies by the body's immune system, which can damage thyroid cells and compromise their ability to make thyroid hormone. The aim of this study was to investigate the DHEAS levels in obese patients with Hashimato thyroiditis.

Material and Methods

This study was approved by the institutional ethics and research committee of Pamukkale University. Informed consent was obtained from all participants. In this study 42 obese patient with Hashimato thyroiditis and 42 obese controls were included to the study. Anthropometric, biochemical and hematological findings were examined in both groups.

Height, weight, BMI, systolic and diastolic blood pressures were measured was calculated. Plasma total and high-density lipoprotein cholesterol, triglycerides, GH, IGF-1, IGFBP3, thyroid stimulating hormone (TSH) and insulin levels were measured.

Results

We observed significantly higher anti-throglobulin antibodies, anti-microsomal antibodies, fasting blood glucose, FSH, TSH and DHEAS levels in patient with Hashimato thyroiditis while decreased prospandial insulin levels. This study showed that blood glucose regulation effected in Hashimato thyroiditis. The results were shown in Table 1.

Discussion

The present study was performed to compare DHEAS levels, fatness, hormone, and blood parameters in obese individuals with Hashimato thyroidis. Compared with obese healthy individuals. Obese individuals with Hashimato thyroidis had higher DHEAS, TANT, MANT, fasting glucose, insulin and TSH levels. Collectively, our data support the notion that DHEAS levels vary according to physical condition.

DHEA, which is produced in the adrenal glands, gonads, and brain, is the most abundant circulating steroid in humans, and functions predominantly as a metabolic intermediate in the biosynthesis of the androgen and estrogen sex steroids (Mo et al., 2006). In addition, DHEA has been postulated to bind to nuclear and cell surface receptors (Webb et al., 2006) to exert a variety of biological effects in its own right, such as in the brain (Friess et al., 2000). DHEAS functions as a modulator of glucose metabolism in humans. The protective action of DHEAS against insulin resistance has been attributed to a variety of mechanisms, including reducing fat accumulation, counteracting glucocorticoid action, or direct stimulation of glucose uptake (Abbasi et al., 1998). Consistent with this, we found that insulin levels were higher in obese individuals with Hashimato thyroiditis than healthy individuals. Our results also suggest a relationship between thyroid disease and DHEAS levels.

Table 1: Baseline and demographic characteristics of obese patients with Hashimato Thyroiditis and control group

	Obese patient with out Hashimato throiditis (n=42)	Obese patient with Hashimato throiditis (n=42)	P
Anti-microsomal antibodies (IU/ml)	19.49±2.24	212.87±448.15	<0.001***
Anti-throglobulin antibody (IU/ml)	11.95±3.51	175.94±273.93	<0.001***
Ages (Years)	52.73±6.03	52.59±6.14	>0.05
Height (cm)	154.40±4.56	154.14±5.15	>0.05
Weight (kg)	85.90±13.89	84.88±12.05	>0.05
BMI (kg/m^2)	35.95±5.65	34.79±7.65	>0.05
Waist circumference (cm)	99.00±10.27	97.32±9.82	>0.05
Hip circumference (cm)	108.97±8.87	107.21±7.44	>0.05
Waist Hip Ratio (cm)	0.89±0.08	0.89±0.09	>0.05
Fasting blood glucose (mg/dl)	108.66±21.59	116.11±37.22	<0.05*
Postprandial blood glucose (mg/dl)	125.39±44.33	129.51±51.69	>0.05
Total cholesterol (mg/dl)	211.16±36.03	204.59±29.29	>0.05
Trigliserides (mg/dl)	152.35±81.87	141.64±71.50	>0.05
LDL (mg/dl)	132.30±33.18	128.76±51.26	>0.05
HDL (mg/dl)	50.70±12.96	55.80±13.68	>0.05
Fasting insulin (µIU/ml)	13.91±6.77	15.67±26.18	>0.05
Postprandial insulin (µIU/ml)	55.76±48.72	47.47±36.19	<0.05*
TSH (µIU/ml)	2.29±1.82	2.82±2.65	<0.05*
FT3(pg/ml)	2.56±0.46	2.46±0.58	>0.05
FT4 (ng/dl)	1.02±0.28	1.03±0.42	>0.05
Thyroglobulin (ng/ml)	29.32±45.36	28.61±112.59	>0.05
FSH (mIU/ml)	68.28±45.85	90.15±45.84	<0.001***
LH (mIU/ml)	26.75±13.33	32.87±17.87	>0.05
E2 (Pg/ml)	74.52±76.62	45.84±34.70	>0.05
SHBG (nmol/l)	44.65±34.87	48.53±25.70	>0.05
Total testosterone (mg/ml)	59.15±39.49	63.01±40.13	>0.05
DHEAS (mg/dl)	89.47±54.68	102.31±70.02	<0.05*
PRL (ng/ml)	12.06±8.68	12.35±8.99	>0.05
Systolic blood pressure (mmHg)	130.48±19.06	129.69±26.50	>0.05
Diastolic blood pressure (mmHg)	84.26±11.48	82.16±13.84	>0.05

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