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Endocrinology & Metabolic Syndrome

Commentary

Applications of Cushing's syndrome: A Commentary

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DESCRIPTION

In 79-97 percent of cases, Cushing's syndrome is associated with typical central (visceral) adipose tissue redistribution, as well as a distinct "moon face" and "buffalo hump." The mechanism underlying normal fat redistribution in hypercortisolism is unknown. The site-specific regulation of intracellular lipolysis enzymes (hormone-sensitive lipase) and intravascular lipolysis enzymes is most likely one of the reasons (lipoprotein lipase). Catecholamines are hormones that play an important role in the regulation of lipolysis [1].

Several studies have found that patients with Cushing's syndrome have significantly increased lipolysis in subcutaneous adipose tissue, as well as increased local concentrations of catecholamines and their metabolites. Glucocorticoids promote gluconeogenesis in the liver, protein degradation to free amino acids in muscle, and muscle atrophy in addition to lipolysis. The main function of 1113-HSD1 is to increase the local concentration of active glucocorticoids in tissues where the steroids play an important regulatory role, such as the liver. A number of studies have emerged that support this hypothesis, including phenotypic analysis of 1113-HSD1-null mice [2,3].

Because of impaired glucocorticoid feedback, these mice have elevated corticosterone levels but are resistant to hyperglycemia caused by stress or overfeeding. Furthermore, they have higher levels of high-density lipoprotein cholesterol, lower levels of low-density lipoprotein cholesterol, and lower triglycerides. The metabolic responses appear to be driven by key changes that reduce gluconeogenesis and (3-oxidation of lipids in the liver and possibly also attenuate glucocorticoid-dependent functions in visceral adipose tissue by preventing amplification of the local corticosterone concentration. When fed a high fat diet, 1 1P-I-ISD 1-null mice gain less weight than wild type mice and tend to deposit fat in subcutaneous rather than visceral sites associated with metabolic disease.

Transgenic mice overexpressing 1113-HSD 1 selectively in adipose tissue, similar to that found in obese human adipose tissue, had increased adipose corticosterone levels and developed visceral obesity, which was exacerbated by a high-fat diet. These mice also had insulin resistance, hyperlipidemia, and, surprisingly, hyperphagia despite hyperleptinemia. Increased 1113-HSD1 activity in adipocytes may be a common molecular aetiology of visceral obesity and the metabolic syndrome. Chronic hypercortisolism has also been linked to increased GR binding sites and GR mRNA expression, as well as lower affinity in intraabdominal adipose tissue samples compared to subcutaneous adipose tissue samples. Furthermore, Cushing's syndrome beta isoform GR mRNA expression was increased when compared to controls. As a result, Cushing's syndrome is associated with a reversible decrease in GR affinity, which may be related to increased GR beta expression as a compensatory mechanism for GC excess. Excess glucocorticoids raise blood pressure for Cushing's syndrome through the interaction of several pathophysiological mechanisms that regulate plasma volume, peripheral cardiovascular resistance, and cardiac output. Cortisol acts on the distal nephron in the kidney to cause sodium retention and potassium loss (mediated by the MR), resulting in an increase in blood volume. Dexamethasone treatment increased mean arterial pressure, urine flow rate, and sodium and urea excretion in the urine.

These changes in body water observed with glucocorticoid excess could be due to impaired urinary concentrating capacity, downregulation of urea transporters UT-A1 and UT-A3, and increased urea excretion. In addition to 1 113-HSD2 inhibition, factors that contribute to an increase in plasma volume include mineralocorticoid co-secretion (deoxycorticosterone, corticosterone); sodium redistribution to extracellular compartments; increased cardiac output mediated. A second mechanism involves increased systemic vascular resistance as a result of vasopressor responses to angiotensin II, catecholamines, AVP, and erythropoietin being potentiated [4,5].

The increased response to angiotensin H is due to glucocorticoids inducing angiotensin II receptors. Furthermore, increased systemic vascular resistance occurs as a result of the inhibition of vasodilatory systems such as nitric oxide, kinin/kallikrein, and prostacyclin, inhibition of peripheral catabolism of catecholamines, particularly norepinephrine, by the direct action of glucocorticoids on cardiovascular receptors, increased calcium uptake and calcium channel antagonist binding in vascular smooth muscle cells, and decreased atrial natriuretic

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Although glucocorticoids have no effect on the number or affinity of adrenergic receptors, they do increase the strength of downstream -adrenergic signalling. Localized vasoreactivity changes may contribute to the beneficial effects of glucocorticoids and 132-agonists in asthma patients. This exemplifies one of the potential benefits of inhaled glucocorticoids, which have been designed to target lung tissue and thus reduce the negative effects of systemic delivery.

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