Polycystic Ovary Syndrome: Implications of Corticotropin in the Regulation of Blood Pressure, Aldosterone, and Androgen Secretion

To the Editor:

In a recent issue of *Hypertension*, Chen et al. have described a correlation between androgen levels and blood pressure in young women with polycystic ovarian syndrome (PCOS), and this correlation was independent from age, insulin resistance, obesity, and dyslipidemia. We would like to discuss other factors involved in the interpretation of these data.

High adrenal androgen levels in women with PCOS can also be related to an increase of corticotropin (ACTH). The major question is why ACTH could be responsible for an increase of adrenal androgens in PCOS. Previous reports have documented that patients with PCOS have reduced activity of 11β-hydroxysteroid dehydrogenase type 1, the enzyme that activates cortisone to cortisol. 11β-Hydroxysteroid dehydrogenase type 1 may enhance the metabolic clearance rate of cortisol, thereby reducing negative feedback and increasing ACTH-dependent adrenal androgen production. It is interesting, at this point, that an extremely high increase of cortisone metabolites and of androgen concentrations are reported in some patients with PCOS and cortisol reductase deficiency.

In a recent paper by Cascella et al., a positive correlation was found between aldosterone and mean blood pressure in patients with PCOS and, more interestingly, between aldosterone and C-reactive protein, metabolic patterns, and intima-media thickness. Both androgens and aldosterone correlate with blood pressure, and these findings are consistent with chronic ACTH drive in adrenals of patients with PCOS.

In fact, whereas administration of high amounts of ACTH for several days or 11-hydroxylase deficiency is associated with suppression of aldosterone and increase of deoxycorticosterone, a chronic ACTH drive because of a partial defect of 11β-hydroxysteroid dehydrogenase type 1 could activate aldosterone synthase at the level of adrenal glomerulosa and androgen secretion by the adrenal reticularis. We, therefore, suggest that a relative chronic increase of ACTH related to a slight deficiency of 11β-hydroxysteroid dehydrogenase type 1 can explain the reported hormonal pattern of these subjects.

The correlation among aldosterone, oxidative stress, metabolism abnormalities, and blood pressure is consistent with a primary involvement of aldosterone and not of androgens in the increase of blood pressure. These data also support the possibility of therapy with spironolactone in patients with PCOS. The drug blocks both the proinflammatory status because of aldosterone binding to mineralocorticoid receptors and the clinical picture of hyperandrogenism because of the binding of androgens to androgen receptors. We have recently stressed these concepts in a study of women with PCOS.

Disclosures

None.

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