

Lower serum concentration of dehydroepiandrosterone sulphate in patients suffering from chronic idiopathic urticaria

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Key words: chronic urticaria; dehydroepiandrosterone.

As the hormonal profile of patients with chronic idiopathic urticaria (CIU) is poorly characterized, it is interesting to evaluate the behaviour of circulating hormones in urticaria patients, in order to relate the urticarial processes to possible hormonal alterations. Adrenal androgens, dehydroepiandrosterone (DHEA) and its sulphate derivate, DHEA-S, have been suggested to be involved in and important to immune and inflammatory responses (1, 2).

Previously, we examined the behaviour of circulating DHEA-S in female CIU patients (3), assuming that women were more frequently affected by this disease and develop some autoimmune diseases more often than males. Despite of an interesting observation proving significantly lower concentration of circulating DHEA-S in female patients than in healthy controls (3), we did not answer the question whether the decline in DHEA-S concentration was a phenomenon generally accompanying CIU. Therefore, the present study examined serum concentration of DHEA-S in both symptomatic and non-symptomatic male population suffering from CIU.

The study group consisted of 31 males (mean age 39 ± 11 years) with CIU. The patients were divided into two groups: I – 13 non-symptomatic patients, treated with anti-histamine drugs, and II – 18 symptomatic patients, untreated. The control group consisted of 36 healthy males (mean age 36 ± 12.5 years). Serum DHEA-S concentration was measured by electrochemiluminescence immunoassay 'ECLIA' (Roche Diagnostics, Mannheim, Germany).

The unpaired Student's *t*-test was used to compare results from urticaria patients (as a whole) and the controls. Analysis of variance (one-way ANOVA) was used to screen differences among the three groups, followed by the appropriate *post hoc* test.

In male CIU patients, serum concentration of DHEA-S was significantly lower than in healthy men (259.7 ± 131 and 373.5 ± 126 µg/dl; mean $\pm$ SD; $P = 0.001$) (Fig. 1). DHEA-S concentration was similar in symptomatic patients and non-symptomatic, treated CIU patients. Both patient groups showed significantly lower DHEA-S values with respect to controls.

The present findings demonstrate that statistically, serum concentration of DHEA-S is significantly lower in male

patients suffering from CIU than in healthy men. Decreased DHEA-S concentration has been recognized in different chronic diseases (2). In particular, it has been suggested that DHEA-S deficiency is a permanent feature of some autoimmune diseases and may contribute to their aetiology and/or pathophysiology (4, 5). However, no direct evidence is available to indicate whether reduced circulating concentration of the hormone contributes to the diseases (2, 5).

We do not know whether lower serum concentration of DHEA-S observed in CIU patients is the cause or rather the effect of urticarial processes. It has been suggested that, during inflammatory response, the adrenal steroid production is changed in the direction of cortisol, relative to DHEA and DHEA-S, such hormonal changes being probably necessary to achieve adequate cortisol levels at the expense of adrenal androgens (6). It is interesting to speculate that similar abnormalities in steroidogenesis might occur during chronic urticarial inflammation.

How does lower circulating DHEA-S actually contribute to CIU pathophysiology? The question proves both, difficult and complex as the specific molecular mechanism of action for

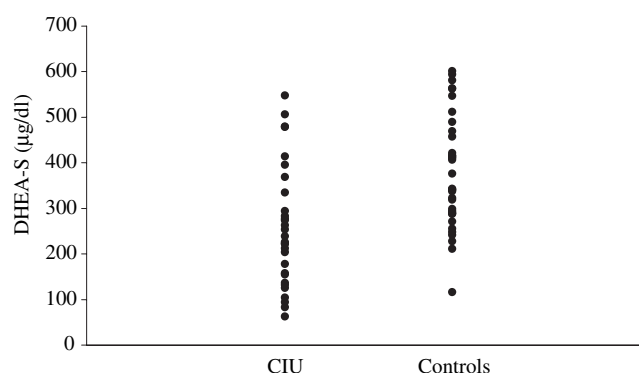


Figure 1. Individual serum concentration of DHEA-S in CIU male patients in comparison to healthy subjects.

DHEA-S, including mechanisms of DHEA-S effect upon the immunological function and inflammation, is unclear (2).

In conclusion, on the basis of the present study as well as our earlier investigations, it appears probable that circulating DHEA-S concentration may be lower in CIU patients, regardless of their gender. It seems that declining circulating concentration of DHEA-S is a phenomenon accompanying CIU, yet its significance and underlying mechanisms remain unknown. As adrenal androgens have been reported to exert immunoregulatory effects, it is interesting to speculate on DHEA-S deficiency role in urticarial inflammation irrespective of its primary or just secondary character.

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References

1. Daynes RA, Dudley DJ, Araneo BA. Regulation of murine lymphokine production in vivo. II. Dehydroepiandrosterone is a natural enhancer of interleukin 2 synthesis by helper T cells. *Eur J Immunol* 1990;**20**:793–802.
2. Dillon JS. Dehydroepiandrosterone, dehydroepiandrosterone sulfate and related steroids: their role in inflammatory, allergic and immunological disorders. *Curr Drug Targets Inflamm Allergy* 2005;**4**:377–385.
3. Kasperska-Zajac A, Brzoza Z, Rogala B. Serum concentration of dehydroepiandrosterone sulphate in female patients with chronic idiopathic urticaria. *J Dermatol Sci* 2006;**41**:80–81.
4. Barry NN, McGuire JL, van Vollenhoven RF. Dehydroepiandrosterone in systemic lupus erythematosus: relationship between dosage, serum levels, and clinical response. *J Rheumatol* 1998;**25**:2352–2356.
5. Schwartz KE. Autoimmunity, dehydroepiandrosterone (DHEA), and stress. *J Adoles Health* 2002;**30S**:37–43.
6. Straub RH, Schuld A, Mullington J, Haack M, Schölmerich J, Pollmacher T. The endotoxin-induced increase of cytokines is followed by an increase of cortisol relative to dehydroepiandrosterone (DHEA) in healthy male subjects. *J Endocrinol* 2002;**175**:467–474.