

CORRESPONDENCE

Serum concentration of dehydroepiandrosterone sulfate (DHEA-S) in patients suffering from acute urticaria

Dehydroepiandrosterone (DHEA) and its sulphated compound – dehydroepiandrosterone sulphate (DHEA-S) – are the major androgens secreted by human adrenal glands, shown to be involved in many biological functions. As DHEA(-S) are capable of controlling the activity of immune and inflammatory cells, it has been suggested that they have a potential role in mediating inflammatory responses [1, 2]. Previously, we observed that serum DHEA-S concentrations may be lower in chronic urticaria patients, regardless of their gender and response to autologous serum skin tests (ASST), as compared with healthy subjects. Stress or hormonal shift might play an important role in this phenomenon [2, 3]. To investigate further the behaviour of DHEA-S in urticaria, in this study we asked whether there are any changes in serum DHEA-S concentrations in patients suffering from acute urticaria (AU). The study comprised twenty five female symptomatic severe AU patients (median age 35 years, range 21–42), mainly with unidentified cause of the disease. We may only presume that in 12 patients the symptoms were probably caused by non-steroidal anti-inflammatory drugs [acetylsalicylic acid (ASA), diclofenac, ketoprofen, naproxen]. The specific challenge test to confirm ASA hypersensitivity was performed in only 3 patients. In 6 patients the symptoms occurred during an infectious period. In the remaining 7 patients, no apparent reason for AU was observed.

Blood samples were taken during the first day, before treatment started. The control group consisted of 19 women (median age 34 years, range 23–40). Serum concentration of dehydroepiandrosterone sulfate was measured by automated electrochemiluminescence immunoassay “ECLIA” (Roche Diagnostics, Mannheim, Germany). Comparisons between groups were made using Mann-Whitney’s unpaired rank sum test. P values lower than 0.05 were considered significant.

In AU patients, DHEA-S serum concentrations did not differ significantly from the healthy controls (*table 1*). In premenopausal chronic urticaria patients, the serum concentration of DHEA-S was significantly lower compared to the healthy subjects. In addition, it has been demonstrated that lower DHEA-S serum concentration may be a transient and reversible phenomenon, accompanying chronic urticaria, occurring during the active period and disappearing upon spontaneous remission of the disease [3]. The lack of persistence of hormonal alterations in the clinically inactive phase of the disease suggests that the mechanisms responsible for such changes perform

Table 1. Serum DHEA-S concentration in patients suffering from acute urticaria and in healthy controls.

Analysed parameters (unit)	Healthy controls (n = 19) median range	AU patients (n = 25) median range
DHEA-S (ug/dL)	211.6 123.5–277.6	225.6 133.4–310.0

n: number of subjects; AU: acute urticaria.

only throughout the active period of the disease and are most probably associated with distress, caused *e.g.* by itching intensity [4]. Moreover, we speculate that, in chronic urticaria, the adrenal steroid production is changed in the direction of cortisol relative to DHEA and DHEA-S [2, 3], similarly to other chronic inflammatory diseases [5, 6]. Contrary to chronic urticaria patients, in the present study no changes in the peripheral blood of DHEA-S concentrations were observed in patients suffering from AC as compared with the healthy controls. Taken together, we speculate that, at least in the initial phase (tests were performed during the first day), acute symptoms might not lead to any hormonal shift. Also, distress may not be long and/or strong enough to significantly influence the DHEA-S concentration, as suggested previously in chronic urticaria symptoms. As a matter of fact, we do not know if acute urticaria lasting longer than a few days, *e.g.* 5 weeks, leads to DHEA-S concentration changes similar to those recorded in chronic urticaria.

In conclusion, it seems that chronic urticaria exhibits a pattern of DHEA-S behaviour different from acute urticaria (at least in the initial phase). ■

Disclosure. Financial support: none. Conflicts of interest: none.

¹ Department of Internal Diseases
Allergy and Clinical
Immunology,
Medical University of Silesia,
ul. Ceglana 35,
41-800 Katowice, Poland
² Department of Pediatrics,
Zabrze Medical University of Silesia
³ Department of Internal Diseases,
Medical University of Silesia
⁴ Department of Dermatology,
Bytom, Poland
<kasperska@plusnet.pl>

Alicja
KASPERSKA-ZAJAC¹
Edyta MACHURA²
Alicja GRZANKA³
Ewa HADAS³
Ewa KOCZY-BARON⁴

1. Kroboth PD, Salek FS, Pittenger AL, Fabian TJ, Frye RF. DHEA and DHEA-S: a review. *J Clin Pharmacol* 1999; 39: 327–48.

2. Kasperska-Zajac A, Brzoza Z, Rogala B. Dehydroepiandrosterone and dehydroepiandrosterone sulphate in atopic allergy and chronic urticaria. *Inflammation* 2008; 31: 141-5.
3. Kasperska-Zajac A. Does dehydroepiandrosterone influence the expression of urticaria? A mini review. *Inflammation* 2010.(in press).
4. Yosipovitch G, Ansari N, Goon A, Chan YH, Goh CL. Clinical characteristics of pruritus in chronic idiopathic urticaria. *Br J Dermatol* 2002; 147: 32-6.
5. 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: 467-74.
6. Schwartz KE: Autoimmunity, dehydroepiandrosterone (DHEA), and stress. *J Adolesc Health* 2002; 30S: 37-43.

doi:10.1684/ejd.2011.1430

IN PRESS