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Assessment of sympathetic skin response to acne vulgaris

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Abstract

Acne vulgaris (AV) is a common pilosebase unit disease characterized by peripheral inflammation. Because it is associated with the peripheral nervous system, the cranial nervous system and the autonomic nervous system, the skin is a neuroimmunoendocrine organ. Various neuroendocrine hormones and catecholamines are also released by keratinocytes, which stimulate inflammation in the pilosebase unit. In this study, sympathetic skin response (SSR) evaluating the sympathetic nervous system function as a reflex against internal and external stimuli in AV will be evaluated. 31 AV patients (mean age: 22.48 ± 2.35, 20 female, 11 male) and 29 healthy volunteers (mean age: 23.19 ± 1.27, 18 female, 11 male) between the ages of 18-30 were included in the study. All patient and control groups were questioned about detailed disease and medication intake history. Dermatologic examination was performed by a single experienced dermatologist, and global acne score (GAS) was calculated. For SSR measurement, both median nerve were warned separately on both wrists. The frequency filters is set to 0.5-2000 Hz and the analysis time is set to about 10 seconds. The SSR latencies were recorded in seconds (s) and the amplitudes were recorded in millivolts (mV). The mean age and sex of the groups were similar (p> 0,05, for each). There was no significant difference between AV and healthy control groups in terms of SSR latencies and amplitudes in both extremes ((p> 0.05, for each) AV patients had a mean GAS of 15.61. There was no correlation between GAS and latency and amplitude values of both extremities in AV patients (p> 0.05). As a result, it was found that SSR did not differ from normal people in AV cases. Changes in the autonomic nervous system in AV should be evaluated with more sensitive tests.

Keywords: Acne, sympathetic skin response, dermatology

Introduction

Acne vulgaris (AV) is a common pilosebase unit disease characterized by peripheral inflammation. Several mechanisms have been proposed for the etiopathogenesis of AV. These include sebaceous gland hyperplasia, increased sebum production, follicular hyperkeratinization and p. acnes colonization [1].

In addition, psychoemotic stress may also affect the onset and/or exacerbation of AV. Because it is associated with the peripheral nervous system, the cranial nervous system and the autonomic nervous system, the skin is a neuroimmunoendocrine organ. In chronic stress, the hypothalamo-pituitary adrenal (HPA) axis activates and stimulates the release of corticotropin-releasing hormone (CRH), adrenocorticotropin hormone (ACTH), alpha-melanocyte stimulating hormone (α-MSH) and β-endorphin. These are excreted from the skin as well as the central nervous system. The increase in these causes an increase in the inflammatory response in the pilosebase, which stimulates keratinocyte proliferation and differentiation.

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Stress also activates epinephrine and norepinephrine secretion. These catecholamines are produced locally by the kerotinocytes as well as the sympathetic nervous system fibers. They increase cytokine release by lymphocyte proliferation and increase immunity and inflammation [2]. In a study conducted, it was observed that there was a marked decline in the acnes as a coincidence in those with sympathetic nerve blockade [3]. However, the relationship between changes in the sympathetic activity of the skin and AV is unknown.

Sympathetic skin response (SSR) is a commonly used, simple and reliable test to assess the function of the sympathetic nervous system. SSR is a temporary change in the electrical potential of the skin, both spontaneously and reflexively against various internal and external stimuli [4].

In this study, we aimed to evaluate the changes (if any) in sympathetic nervous system activity in AV patients.

Material and Methods

The study protocol was approved by the local Ethics Committee. All the subjects read and signed the informed consent forms before enrolling in the study.

Patient group and study protocol

Thirty one AV patients (mean age; 22.48 ± 2.35 years, 20 women, 11 men) and 29 healthy volunteers (mean age; 23.19 ± 1.27 years, 18 women, 11 men) between the ages of 18-30 who applied to the dermatology policlinic were taken into the study. All patient and control groups were questioned about detailed disease and medication intake history. Dermatologic examination was performed by a single experienced dermatologist, and global acne score (GAS) was calculated. Neurological examinations were performed by an experienced neurologist.

Patients with drug use that may cause acne or history of endocrinological pathology, known malignancy, systemic disease (such as diabetes mellitus, liver failure, renal insufficiency), inflammatory or infectious disease were excluded from the study.

Patients diagnosed with polyneuropathy that may affect SSR in neurological examination or having thyroid dysfunction that may increase polyneuropathy susceptibility, vitamin B12 deficiency, alcoholism, renal insufficiency, diabetes mellitus accompanied by autonomic neuropathy, and idiopathic Parkinson's disease were excluded from the study. Also, those with the use of medication (L-dopa, decongestants, bronchodilators, beta blockers, etc.) that could affect CNS functions were not included in the study.

SSR Measurement

The SSR was assessed by the same clinician (HKO), using a Neuropack MEB-2200 Nihon Kohden® (Tokyo, Japan) electromyography device, in a semi-dark, silent room, in supine position, between 12.00-16.00 to avoid being affected by circadian changes, and when patient awake. Silver chloride electrodes were used for the shots and the hand skin temperature was kept at about 32°C. The active electrode is placed on the palm of the hand, the reference electrode is placed on the back of the hand and the ground electrode is placed on the wrist. To avoid habituation, stimuli were given to both median neurons separately in irregular and unexpected times. The duration of the stimuli was 0.2 ms and the intensity was between 10-30 mA. The frequency filters is set to 0.5-2000 Hz and the analysis time is set to about 10 seconds. It was accepted that SSR could not be obtained at least 5 times as pathologically The SSR latencies were recorded in seconds (s) and the amplitudes were recorded in millivolts (mV).

Statistics

The Shapiro Wilk test was used to check the normal distribution of continuous variables. Student t test (age) was used for comparison of 2 independent groups of variables with normal distribution, and Mann Whitney U test was used for variables with no normal distribution. Comparisons of categorical data were made by chi-square test. The relationship between Latans (right and left) and amplitude (right and left) and GAS in AV patients was evaluated by Spearman correlation analysis. SPSS for Windows version 22.0 packet program was used for statistical analysis and P <0.05 was considered statistically significant.

Results

The mean age and sex of the groups were similar (p> 0,05, for each). There was no significant difference between the AV and healthy control groups in terms of SSR latencies and amplitudes (p> 0.05, for each; Table 1)

The mean GAS in AV patients was 15.61. There was no correlation between GAS and latency and amplitude values of both extremities in AV patients (p> 0.05).

Table 1. Latency and amplitude levels in all groups

	Acne Vulgaris	Healthy Control	P
Age (year)	22.48±2.35	23.19±1.27	0,099
Gender (F/M)	20/11	18/11	0,844
RLat (msec)	1.14±1.06	2.23±3.61	0,990
RA (mV)	3.49±3.72	3.18±4.91	0,738
Llat (msec)	1.37±1.00	0.83±2.0	0,254
LA (mV)	2.89±3.11	3.20±2.32	0,696

Rlat, Right latans; RA, right amplitude; Llat, Left latans; LA, Left amplitude

Discussion

In this study, it was determined that SSR did not show any significant change in AV patients compared to healthy patients.

SSR is considered to be a simple way of measuring sudomotor activity. Thus, sympathetic activity is assessed as rapid and painless. SSR has been evaluated in a variety of diseases such as migraine, multiple sclerosis, fibromyalgia, and lumbosacral radiculopathy [5-8]. In addition, SSR has been evaluated in skin diseases such as leprosy, palmoplantar hyperhidrosis, vitiligo and psoriasis [9-12]. However, it has not been evaluated so far in AV.

It has also been observed that people who have been treated with sympathectomy for hyperhidrosis treatment have concurrent acne recovery. This suggests that there may be an increase in sympathetic activity in AV patients. The authors suggested that this may be due to changes in the epithelial melanocyte system as a responsible mechanism [3]. Apart from α-MSH melanogenesis, pilosebase is involved in the inflammatory and immune response of the unit [13]. Increased α-MSH and MC-1R expression in sebaceous glands has been shown in patients with acne in the case of systemic or cutaneous stress. The α-MSH / MC1R interaction has been suggested to be closely related to keratinocyte proliferation and differentiation [14]. In this study, it was observed that SRS amplitude and latency values of AV patients were not different from healthy controls. Many studies have found different normal values for the latencies and amplitudes of SRS [15-17].

Because of SRS latency and amplitude normal values vary and are affected by factors such as skin thickness and body temperature, sensitivity of this method is considered to be relatively weak in assessing autonomic dysfunction [18]. For this reason, we think that the studies using different electrophysiological and biochemical methods are needed to clarify the role of the sympathetic system in AV pathophysiology.

The limitation of this study is that the number of cases is limited and a parameter that measures the currently valid sympathetic response is not studied.

As a result, it was found that SSR did not differ from normal

people in AV cases. Changes in the autonomic nervous system in AV should be evaluated with more sensitive tests. Detecting a possible relationship can create a different perspective for AV pathogenesis and treatment.

Conclusion

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Refrences

1. Antiga E, Verdelli A, Bonciani D, Bonciolini V, Caproni M, Fabbri P. Acne: a new model of immune-mediated chronic inflammatory skin disease. G Ital Dermatol Venereol. 2015;150(2):247-54.

2. Honeyman JF. Psychoneuroimmunology and the skin. Acta Derm Venereol. 2016;217:38–46.

3. Kaplan T, Gunduz O, Oznur B, Han S. Could thoracoscopic sympathicotomy for hyperhidrosis also improve acne vulgaris?. Kardiochir Torakochirurgia Pol. 2014;11(3):264–7.

4. Uçar MK, Bozkurt MR, Bozkurt F. A Survey of SSR. Mechanism and application. Int J Comput Appl. 2013;73:875–87.

5. Ozkul Y, Ay H. Habituation of sympathetic skin response in migraine and tension type headache. Auton Neurosci. 2007;134(1-2):81-4.

6. Thomas-Pohl M, Weglinski L, Salga M, Blum C, Manceau P, Amarenco G. Sympathetic skin response and multiple sclerosis: descriptive and prospective study and association with neuroperineal disorders. Prog Urol. 2014;24(11):691-6.

7. Ozkan O, Yildiz M, Arslan E, Yildiz S, Bilgin S, Akkus S, Koyuncuoglu HR, Koklukaya E. A study on the effects of sympathetic skin response parameters in diagnosis of fibromyalgia using artificial neural networks. J Med Syst. 2016;40(3):54.

8. Erdem Tilki H, Coşkun M, Unal Akdemir N, Incesu L. Axon count and sympathetic skin responses in lumbosacral radiculopathy. J Clin Neurol. 2014;10(1):10-6.

9. Bir LS, Aktan S. Sympathetic skin response in psoriasis and vitiligo. J Auton Nerv Syst. 1999:77:68-71.

10. Dogramacı AC, Okuyucu EE. Sympathetic Skin Response in Patients with Vitiligo. Turkderm. 2009;43:58-60

11. Wilder-Smith A, Wilder-Smith E. Electrophysiological evaluation of peripheral autonomic function in leprosy patients, leprosy contacts and controls. Int J Lepr Other Mycobact Dis .1996;64:433-40.

12. Chu EC, Chu NS. Patterns of sympathetic skin response in palmar hyperhidrosis. Clin Auton Res. 1997;7:1-4.

13. Madva EN, Granstein RD. Nerve- derived transmitters including peptides influence cutaneous immunology. Brain, behaviour and Immunity. 34(2013):1-10.

14. Ganceviciene R, Böhm M, Fimmel S, Zouboulis CC. The role of neuropeptides in the multifactorial pathogenesis of acne vulgaris. Dermato- Endocrinology. 2009:1:3,170-6.

15. Goizueta-San Martín G, Gutiérrez-Gutiérrez G, Godoy-Tundidor H, Mingorance-Goizueta B, Mingorance-Goizueta C, Vega-Piris L, Gutiérrez-Rivas E. Sympathetic skin response: reference data for 100 normal subjects. Rev Neurol. 2013;56(6):321-6.

16. Uncini A, Pullman SL, Lovelace RE, Gambi D. The sympathetic skin response: normal values, elucidation of afferent components and application limits. Journal of Neurological Sciences. 1988;87(2-3):299-306.

17. Baba M, Watahiki Y. Sympathetic skin responce in healthy man. Electromyogr. Clin. Neurophysiol. 1988;28(5):277-83.

18. Mimori Y, Tanaka H. Sympathetic skin response(SSR). Nihon Rinsho. 1992;50:753–8.