Wien Klin Wochenschr (2016) 128:155
DOI 10.1007/s00508-015-0734-6

Wiener klinische Wochenschrift
The Central European Journal of Medicine

High proline-related inhibition of serum prolidase enzyme activity in scleroderma

Huseyin Kayadibi · Erdim Sertoglu · Metin Uyanik

Received: 29 May 2014 / Accepted: 19 January 2015 / Published online: 6 May 2015

Dear Editor

We read with great interest the recently published article by Savas et al. [1]. In this study, it was aimed to determine whether an association exists between serum prolidase enzyme activity (SPEA) and oxidative stress markers, as well as how collagen metabolism is affected in systemic sclerosis (SSc). In conclusion, SPEA was found to be significantly lower in SSc patients than in controls, and there was a negative weak correlation between prolidase and oxidative stress index (OSI). However, we would like to share our thoughts and contributions to this study.

First, patients who were pregnant, were older than 65 years or younger than 18 years, had previous psychiatric disorders, had a body mass index (BMI) more than 30 kg/m^2, with a diagnosis of myocardial infarction before the initiation of SSc, and with arterial hypertension, diabetes mellitus, renal dysfunction, and additional chronic diseases other than autoimmune disease were excluded from the study. Using these comprehensive exclusion criteria provided prevention of possible bias that may be derived from subjects who were included in the original study. We therefore can trust that the SPEA results are highly specific to SSc. This is an important factor that increases the value of the original study.

Second, collagen turnover (synthesis and degradation) works in accordance with the collagen accumulation properly in healthy individuals. However, SSc is characterized by excessive deposition of collagen and tissue fibrosis due to the reduced collagen degradation as a result of decreased SPEA. Moreover, at early stages of SSc, high amount of proline may be liberated from the breakage of proline-containing iminodipeptides or hydroxyproline due to the increased collagen turnover [2]. As we know, high proline has an inhibitory effect on SPEA [3]. Therefore, high proline levels may actually aggravate the severity of SSc by this inhibition, which can be determined by evaluating SPEA and urine/serum proline levels on the follow-up of scleroderma at regular time intervals.

In conclusion, lowering the serum proline concentration to the lower limit of normal may be helpful in the treatment of SSc. Prospective studies performed in SSc patients showing the inhibition of SPEA will provide more detailed information for the therapy of SSc.

Conflict of interest
The authors state that there is no conflict of interests regarding the publication of this article.

H. Kayadibi, MD, MSc (✉)
Biochemistry Laboratory, Adana Military Hospital,
Adana, Turkey
e-mail: mdkayadibi@yahoo.com

E. Sertoglu, MD
Biochemistry Laboratory, Anittepe Dispensary,
Ankara Mevki Military Hospital,
Ankara, Turkey

M. Uyanik, MD
Department of Medical Biochemistry,
Gulhane School of Medicine,
Ankara, Turkey

References

1. Savas E, Aksoy N, Pehlivan Y, Sayiner ZA, Oztürk ZA, Tabur S, et al. Evaluation of oxidant and antioxidant status and relation with prolidase in systemic sclerosis. Wien Klin Wochenschr. 2014;126(11–12):341–6.
2. Jayson MIV. Collagen changes in the pathogenesis of systemic sclerosis. Ann Rheum Dis. 1977;36(Suppl.):26.
3. Mock WL, Green PC. Mechanism and inhibition of prolidase. J Biol Chem. 1990;265:19606–10.