

Assessment of Serum Uric Acid in Young Male Patients with Ankylosing Spondylitis

To the Editor:

We read with great interest the article entitled “Low levels of serum uric acid increase the risk of low bone mineral density in young male patients with ankylosing spondylitis¹”. Thanks to Kang and colleagues for their valuable investigation in which they concluded that lower serum uric acid (SUA) levels were associated with lower bone mineral density in young male patients with ankylosing spondylitis and that SUA might be a predictive biomarker or therapeutic target in these patients. However, we think that some issues have to be emphasized when assessing SUA levels.

Generating the study population is perhaps the most important step of a research study. In this respect, exclusion criteria carry a big importance. In their study, the authors defined several exclusion criteria that we consider insufficient. Several researchers have suggested that these diseases can affect SUA concentrations: metabolic syndrome, systemic inflammation, cardiovascular diseases, diabetes mellitus, nonalcoholic fatty liver disease, peripheral arterial disease, hypertension, multiple sclerosis, dyslipidemia, and Parkinson disease^{2,3}. In addition to these disorders, several kinds of drugs were suggested to alter SUA levels, such as hypoglycemic agents (e.g., metformin), weight-reducing drugs (e.g., orlistat), lipid-lowering drugs (e.g., fenofibrate, simvastatin, ezetimibe, atorvastatin), calcium channel blockers (e.g., amlodipine), angiotensin-converting enzyme inhibitors (e.g., captopril, enalapril, ramipril), angiotensin II receptor antagonists (e.g., losartan), estrogens, corticosteroids, and the dietary food supplements vitamin C, vitamin A, iron, flavonoids, omega 3 fatty acids, zinc, and β -carotene^{4,5}. The authors have to define whether the participants have any of these contributing factors to present a robust study population.

The authors did not state when they obtained blood samples. Fustin, *et al* showed that the purine synthesis is under the control of the hepatic clock, and consequently degradation products of purine nucleotides, UA, showed fluctuation⁶. It is essential to standardize the sampling time, which can cause bias and can lead to misinterpretation of the results.

Kang, *et al* contributed valuable data to the medical literature. But clarifying the above concerns will provide a clearer picture to the readers of *The Journal*.

TOLGA DOGAN, MD, Department of Internal Medicine, Gulhane Military Medical Academy, Ankara; MEHMET AGILLI, MD, Department of Biochemistry, Agri Military Hospital, Agri, Turkey. Address correspondence to Dr. T. Dogan, Department of Internal Medicine, Gulhane Military Medical Academy, Ankara, Turkey.
E-mail: dr_tolgadogan94@yahoo.com

REFERENCES

1. Kang KY, Hong YS, Park SH, Ju JH. Low levels of serum uric acid increase the risk of low bone mineral density in young male patients with ankylosing spondylitis. *J Rheumatol* 2015;42:968-74.
2. Katsiki N, Karagiannis A, Athyros VG, Mikhailidis DP. Hyperuricaemia: more than just a cause of gout? *J Cardiovasc Med* 2013;14:397-402.
3. Shankar A, Klein BE, Nieto FJ, Klein R. Association between serum uric acid level and peripheral arterial disease. *Atherosclerosis* 2008;196:749-55.
4. Nagao A, Seki M, Kobayashi H. Inhibition of xanthine oxidase by flavonoids. *Biosci Biotechnol Biochem* 1999;63:1787-90.
5. Schievink B, de Zeeuw D, Parving HH, Rossing P, Lambers Heerspink HJ. The renal protective effect of angiotensin receptor blockers depends on intra-individual response variation in multiple risk markers. *Br J Clin Pharmacol* 2015;80:678-86.
6. Fustin JM, Doi M, Yamada H, Komatsu R, Shimba S, Okamura H. Rhythmic nucleotide synthesis in the liver: temporal segregation of metabolites. *Cell Rep* 2012;1:341-9.

J Rheumatol 2016;43:3; doi:10.3899/jrheum.150577