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Elevated uric acid level: the chicken or the egg?

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The article entitled *Predictive value of serum uric acid on left atrial spontaneous echo contrast in non-valvular atrial fibrillation patients* by Liao, *et al*. has given important data regarding the association between uric acid level and spontaneous echo contrast in patients with non-valvular atrial fibrillation.[1] This relationship has become an attractive interest and hereafter more comprehensive studies should be conducted. We would like to contribute to the study for some methodological points.

Recent studies have shown that hyperuricemia may damage endothelial function and increases the cardiovascular event risk.[2] Moreover, there is increasing evidence that uric acid may play a role in coronary artery disease, cerebrovascular disease, chronic respiratory disease, peripheral artery disease etc., by atherosclerotic involvement. However uric acid level may be affected by several factors and its elimination is a challenging issue especially for retrospective studies. In this well designed study, the authors had compared groups for well-known ischemic risk factors such as hypertension, dyslipidemia, diabetes mellitus, etc. In addition, alcohol consumption or hypothyroidism are well known confounders for uric acid level so it would have been better if the authors had mentioned to these parameters too.[3,4]

In the last paragraph of the discussion part, the authors suggested that, serum uric acid level could be regarded as a therapeutic target for the prevention of thromboembolic events and may be an indicator for antithrombotic therapy decision. We think that, uric acid could be a consequence of systemic endothelial involvement, not the reason. Although conflicting results, some studies showed that the association between serum uric acid (SUA) elevations and cardiovascular disease was considered to be 'epiphenomenal' and not causal.[5] The effects of uric acid lowering therapy on total mortality, major cardiovascular events, hospitalization and adverse effects are not reliable for using in medical practice yet.[6]

In conclusion, this article enlightens the relationship between uric acid and ischemic conditions in non-valvular atrial fibrillation patients. However, new studies with more detailed risk factors assessment may contribute to our knowledge in this area.

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Authors' reply

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Thank you very much for the excellent comments from Dr. Dogan's group on our recently published article. In the article, we demonstrated the relationship between SUA and left atrial spontaneous echo contrast (LA-SEC) screened by transesophageal echocardiography (TEE) in a total of 1345 non-valvular atrial fibrillation (AF) patients.

In clinical practice, most of AF patients had taken amiodarone for long-term rhythm control. Although catheter ablation candidates discontinued at least five half-life periods prior to ablation, thyroid function had presented abnormal condition, which was even irrevesible in short-term. Therefore, it was difficult to accurately evaluate the real effect of thyroid function on SUA in AF patients. And there was the same situation for alchol consumption, due to the difficulty of quantifying the volume, frequency and category of alchol.

There were similar interpretations for the relationship mentioned in our study between SUA and LA-SEC: direct pathophysiological process, indirect or reversed causal relationship, innocent bystander association with other cardiac conditions or statistical bias.

Many evidences had presented uric acid promoted tissue inflammation and increased the likelihood of both AF and thrombogenesis.[2,3]

SUA might be an indicator of profibrillatory/prothrombotic course via a direct or indirect way. SUA was simply associated with confounder that promote LA-SEC, even thrombus, such as hypertension, diabetes, and vascular disease.[4,5] In the end, the observed link between SUA and LA-SEC was likely to lead to a statistical bias for the retrospective design. Actually, they were controversial based on present evidences; a prospective study would be the most valuable way to validate the independent contribution of SUA to LA-SEC.

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