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Reply to the Letter to the Editor: "Uric Acid/ Albumin Ratio: Beyond Risk Stratification to Therapeutic Guidance in Hypertension"

Editöre Mektuba Yanıt: "Ürik Asit/Albümin Oranı: Hipertansiyonda Risk Stratifikasyonunun Ötesinde Terapötik Rehberlik"

To the Editor,

We thank the authors¹ for their interest and comments regarding our study, which demonstrated the relationship between the serum uric acid/albumin ratio (UAR) and the circadian rhythm of blood pressure (BP).²

In their initial comments, the authors brought attention to the potential role of serial UAR measurements as a complementary tool in assessing therapeutic response in patients with hypertension. Recent studies have demonstrated associations between UAR and established cardiovascular risk markers such as carotid intima-media thickness and peripheral artery disease severity in hypertensive patients.^{3,4} However, both our study and the existing literature are predominantly cross-sectional in design. As such, there is a lack of longitudinal data demonstrating the dynamics of UAR in response to pharmacological or lifestyle interventions. Until such longitudinal evidence is available, we propose that UAR be regarded as a complementary biomarker rather than a definitive tool for assessing therapeutic response in hypertensive patients.

In their second comment, the authors raised an important question regarding whether a high UAR should prompt more aggressive antihypertensive treatment, particularly in patients with a non-dipper blood pressure profile. Non-dipper hypertensive patients are known to have a higher risk of cardiovascular events and target organ damage.⁵ An elevated UAR reflects increased oxidative stress and inflammation, both of which contribute to vascular dysfunction and hypertension severity.⁶ In this context, a high UAR may help identify patients at elevated risk—particularly those with a non-dipping profile—who might benefit from closer follow-up or adjunctive therapies aimed at reducing oxidative stress and inflammation. However, current evidence does not support intensifying antihypertensive treatment based solely on UAR values. At this stage, UAR should be considered a complementary marker in risk stratification, while treatment decisions should be based on comprehensive clinical assessment, including ambulatory blood pressure monitoring and assessment of end-organ damage.

In their third comment, the authors posed a thought-provoking question regarding whether patients with elevated UAR might derive additional clinical benefit from uratelowering or anti-inflammatory therapies, particularly in the context of cardiovascular risk modulation. Current evidence indicates that UAR is not only associated with hypertension but also correlates with markers of atherosclerotic burden, endothelial dysfunction, and systemic inflammation.²⁻⁴ Urate-lowering therapies, such as allopurinol and febuxostat, have been shown to reduce inflammation and improve endothelial function, potentially mitigating cardiovascular risk.⁷ Similarly, anti-inflammatory agents like colchicine have demonstrated secondary cardiovascular protective effects in recent studies.⁸ However, specific prospective data assessing the efficacy of these therapies in patient subgroups characterized by elevated UAR remain limited. Therefore, while these therapies may offer promise, robust prospective clinical trials are needed to determine their true benefit in patients with elevated UAR.



LETTER TO THE EDITOR REPLY

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Available online at archivestsc.com. Content of this journal is licensed under a Creative Commons Attribution – NonCommercial-NoDerivatives 4.0 International License. We appreciate the authors' insightful question regarding the integration of UAR into chronotherapeutic models for hypertension management. Chronotherapy—administering antihypertensive medications at specific times to align with the body's circadian rhythms—has shown promise in improving BP control and reducing organ damage.⁹ For instance, evening dosing of angiotensin receptor blockers like valsartan has been associated with better nocturnal BP control and a more pronounced nocturnal BP dip. Incorporating UAR into chronotherapeutic models could offer a personalized approach to hypertension treatment. By identifying patients with elevated UAR and non-dipping BP patterns, clinicians might tailor medication timing to optimize therapeutic outcomes. However, prospective studies are needed to validate this approach and establish UAR as a reliable biomarker for guiding chronotherapy in hypertensive patients.

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