Comment on: Monocyte-to-HDL ratio as an inflammatory marker: does it reflect blood pressure patterns?

Sirs,

We read with great interest the article by Astan and Dayanan entitled *Monocyte-to-HDL ratio as an inflammatory marker: does it reflect blood pressure patterns*? published in the Journal of Medicine and Palliative Care (1). The authors are to be congratulated for investigating the potential relationship between the monocyte/HDL ratio (MHR) and circadian blood pressure (BP) patterns, an area of growing clinical interest given the interaction between inflammation and hypertension. While the study provides valuable data, several methodological issues need to be addressed. First, although patients with diabetes mellitus, chronic kidney disease, and overt cardiovascular disease were excluded, other confounding factors affecting dipping status, such as obesity, obstructive sleep apnea, shift work, endocrine diseases, and psychosocial stress, were not assessed (2, 3). These factors are well-documented determinants of nocturnal BP decline and may partially explain the observed lack of association between MHR and circadian BP profiles. Additionally, insufficiently described inclusion criteria and study period raise concerns regarding potential selection bias. The marked imbalance between hypertensive and normotensive groups further limits comparability; increasing the size of the normotensive cohort would strengthen the validity of between-group analyses. Moreover, antihypertensive medication classes and dosing schedules, which are known to influence circadian BP patterns, were not reported and may have acted as additional confounders. Second, the study divided patients into dipper and non-dipper groups. However, the reverse dipper and extreme dipper phenotypes have distinct prognostic implications, with reverse dippers exhibiting the highest cardiovascular risk (4). Inclusion of these categories could have provided a more comprehensive understanding of circadian BP variability. Third, reliance on a single 24-h ABPM also limits the interpretation of dipping status, as circadian patterns may vary over time depending on lifestyle and clinical conditions (5). Repeated or longitudinal ABPM measurements are necessary to strengthen the results. Furthermore, analysis of biochemical parameters raises additional concerns. MHR and related laboratory values (*e.g.* HDL, triglycerides, liver enzymes) can be significantly affected by factors such as dietary intake, alcohol consumption, smoking status, medication use (*e.g.* statins, fibrates, corticosteroids, oral contraceptives), acute infections, thyroid dysfunction, liver failure, obesity, metabolic syndrome, and rheumatologic diseases (6-8). These potential confounders were not systematically assessed in the present study. Without controlling for such influences, the observed MHR stability between dipper and non-dipper groups may reflect unmeasured variability rather than a true absence of association. Although the authors discussed MHR within an inflammatory framework, it should be noted that inflammation is not limited to rheumatologic conditions, and multiple systemic disorders may influence this biomarker. In conclusion, the study by Astan and Dayanan provides important insights but also highlights the complexity of associating inflammation with circadian BP variability. Future research should include diverse dipping phenotypes, utilise repeated ABPM, control for biochemical and lifestyle confounders, and incorporate multimodal inflammation profiling (*e.g.* cytokines such as IL-6 and TNF-α, oxidative stress markers, acute-phase reactants, monocyte/macrophage activation indices) to better clarify the role of MHR in hypertension and cardiovascular risk stratification.

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