THE ANATOLIAN JOURNAL OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

The Role of Inflammation in Atrial Fibrillation

LETTER TO THE EDITOR

To the Editor,

With great interest, I read the recent paper published in your journal entitled "Comparison of the Relationship Between Inflammatory Markers and Atrial Fibrillation Burden."[1] Atrial fibrillation (AF) is the most prevalent cardiac arrhythmia and poses a significant burden to patients and health-care systems worldwide. Atrial fibrillation has several underlying substrates. Electrophysiological and structural abnormalities result in atrial remodeling that promotes the development and persistence of AF. Experimental and clinical data indicate that inflammation plays an important role in the development and persistence of AF.[2] Knowing the pathophysiology of AF in detail is important for the treatment and prevention of AF. The authors have investigated sample inflammatory markers such as systemic immune inflammation index (SII), neutrophil–to-lymphocyte ratio, and platelet–to-lymphocyte ratio in the context of AF patterns.[1]

The study was well structured, and a sufficient number of patients were presented. However, there were some noteworthy points. First, the authors stated in the "Statistical Analysis" section that "if the data were parametric (data obtained with range, ratio scale, and normal distribution), the Kruskal–Wallis H test was used." The Kruskal–Wallis H test is a rank-based non-parametric test that can be used to determine if there are statistically significant differences between 2 or more groups of an independent variable on a continuous variable. In other words, it provides information about non-parametric (non-normally distributed) data. In the case of parametric data, it would be more appropriate to use one-way analysis of variance and the related post hoc test. Second, Table 2 indicates that there are statistically significant differences between the groups regarding echocardiographic "ascending aortic diameter (AAD), ejection fraction (EF), and left atrium (LA)" data but does not say exactly where they are. As far as i think, these data are non-parametric, as they are presented as median and range. Therefore, it would be more appropriate to use Kruskal–Wallis and the corresponding post hoc test. Third, in the "Conclusion" section, the authors stated that inflammation is associated with AF burden, and the SII is successful in reflecting this. However, no correlation or regression was applied between the AF pattern and inflammatory markers. Since the AF pattern (permanent and paroxysmal AF) is categorical data, it would be more reasonable to perform logistic regression analysis and obtain information about whether inflammatory markers are independently related to AF.[3]

In conclusion, appropriate statistical tests and presenting the results in an appropriate way contribute to the clarity of the data.

REFERENCES

1. Ömür SE, Zorlu Ç, Yılmaz M. Comparison of the relationship between inflammatory markers and atrial fibrillation burden. *Anatol J Cardiol*. 2023;27(8):486-493. [CrossRef]
2. Hindricks G, Potpara T, Dagres N, et al. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS): The Task Force for the diagnosis and management of atrial fibrillation of the European Society of Cardiology (ESC) Developed with the special contribution of the European Heart Rhythm Association (EHRA) of the ESC.. *Eur Heart J*. 2021;42(5):373-498. [CrossRef]
3. Tanboğa IH, Kurt M, Işik T, et al. Assessment of multivariate logistic regression analysis in articles published in Turkish cardiology journals. *Turk Kardiyol Dern Ars*. 2012;40(2):129-134. [CrossRef]

Abdulrahman Naser

Department of Cardiology, Kırklareli Training and Research Hospital, Kırklareli, Türkiye

Corresponding author:
Abdulrahman Naser
✉ abdulrahman_naser@hotmail.com

Cite this article as: Naser A. The role of inflammation in atrial fibrillation. *Anatol J Cardiol*. 2023;27(12):737.

DOI:10.14744/AnatolJCardiol.2023.3780