Systemic inflammation and arrhythmogenesis in the context of major cardiac surgeries: A glimpse into further implications

Kenan Yalta[1], Ugur Ozkan[1], Tulin Yalta[2], Ertan Yetkın[3]

[1]Department of Cardiology, Trakya University, Edirne, Turkey
[2]Department of Pathology, Trakya University, Edirne, Turkey
[3]Department of Cardiology, Türkiye Hospital, Istanbul, Turkey

Correspondence to:
Kenan Yalta, MD,
Cardiology Department,
Trakya University,
Balkan Yerleşkesi, 22030, Edirne,
Turkey,
phone: +90 505 657 98 56,
e-mail: kyalta@gmail.com,
akenanyalta@trakya.edu.tr

DOI: 10.33963/v.phj.101059

Received:
June 5, 2024

Accepted:
June 6, 2024

Early publication date:
June 7, 2024

In recent decades, systemic inflammation has been implicated the development and prognosis of various cardiovascular conditions including acute coronary syndromes and cardiac arrhythmias [1–4]. In particular, multi-inflammatory index (MII) is defined as a group of indices based on blood counts of certain cells (including neutrophils, platelets, lymphocytes) and levels of C-reactive protein, and has clinical implications in a variety of cardiovascular disorders [1]. On the other hand, postoperative atrial fibrillation (AF) has been a challenging condition potentially associated with adverse outcomes in the setting of coronary artery bypass grafting (CABG) [2]. The recent study by Yuksel et al. [1] has demonstrated the usefulness of preoperative MII in predicting post-operative AF in patients undergoing off-pump CABG. We highly appreciate the novel findings of the study [1]. However, we would also like to highlight a variety of points as well.

First, it seems highly likely that further increases in systemic inflammation parameters and associated blood cells (as demonstrated by MII) following CABG may have served as the primary trigger for post-operative AF evolution in the study population [1]. Previously, it has been highlighted that systemic inflammation could potentially trigger cardiac arrhythmias through a variety of direct and indirect mechanisms, including the impact of cytokines on cardiac ion channels, induction of the coagulation cascade and myocardial dysfunction [4]. Notably, an existing structural heart disease may significantly lower the threshold for arrhythmogenesis due to increased systemic inflammation [4], which may also occur in the setting of cardiac surgeries [1]. Accordingly, advanced age was previously suggested as an important predictor of post-operative AF [1] possibly due to its potential association with microstructural changes involving the atrial myocardium. Therefore, subtle changes involving the atrial as well as ventricular myocardium (including fibrosis and impaired myocardial strain, etc.) might have significantly facilitated the evolution of postoperative AF occurence in the study [1]. Did the authors plan further follow-up tests including cardiac magnetic resonance imaging and advanced echocardiographic evaluation in those with postoperative AF in an attempt to detect subclinical cardiac disease?

Second, malignant arrhythmias, including ventricular tachycardia and ventricular fibrillation, may also have an important relationship with systemic inflammation, and may significantly contribute to adverse outcomes following CABG [4]. We wonder about the authors' comment regarding the clinical value of MII in predicting ventricular arrhythmias after major cardiac surgeries. Did the patients with AF have a higher incidence of ventricular arrhythmias?

Finally, systemic inflammation may likely persist, to variable degrees, following the postoperative recovery period in a portion of the study population, particularly in those with hyperinflammation at baseline (1) unless specific anti-inflammatory strategies are implemented [4]. Of note, eradication of the underlying inflammatory trigger (infections,

rheumatological diseases, etc.) appears to be the mainstay of therapy in the setting of systemic inflammation [4]. However, some of patients with inflammation-induced arrhythmias may not have an obvious underlying trigger of systemic inflammation [4]. Importantly, the initiation of certain agents including colchicine and interleukin blockers (including anakinra and canakinumab) may significantly suppress arrhythmic episodes in this group of patients [4]. From a therapeutic perspective, adjunctive anti-inflammatory strategies in these patients may potentially reduce the need for more sophisticated antiarrhythmic strategies [4] not only in the short term (for instance; perioperative period) but also in the long term. We wonder about the authors' long-term anti-inflammatory strategies in those with post-operative AF who have no identifiable trigger of systemic inflammation.

In summary, the study by Yuksel et al. [1] has expanded the clinical utility of MII in the setting of cardiovascular disease. However, further implications of MII in the setting of cardiac arrhythmias remain to be established.

Article information

Conflict of interest: None declared.

Funding: None.

REFERENCES

1. Yuksel A, Velioglu Y, Atasoy MS, et al. Multi-inflammatory index as a novel predictor of new-onset atrial fibrillation after off pump coronary artery bypass grafting. Pol Heart J. 2024, doi: 10.33963/v.phj.100847, indexed in Pubmed: 38845422.
2. Engin M. Postoperative atrial fibrillation after coronary bypass surgery: Where should a cardiac surgeon stand? Pol Heart J. 2024; 82(6): 683–684, doi: 10.33963/v.phj.100978, indexed in Pubmed: 38845440.
3. Özkan U, Gürdoğan M, Öztürk C, et al. Systemic immune-inflammation index: A novel predictor of coronary thrombus burden in patients with non-ST acute coronary syndrome. Medicina (Kaunas). 2022; 58(2): 143, doi: 10.3390/medicina58020143, indexed in Pubmed: 35208466.
4. Yalta T, Yalta K. Systemic inflammation and arrhythmogenesis: A review of mechanistic and clinical perspectives. Angiology. 2018; 69(4): 288–296, doi: 10.1177/0003319717709380, indexed in Pubmed: 28539056.

Response

The authors of the article cited were invited to reply. They do not respond to it.