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Editorial

Atrial fibrillation in acute coronary syndromes: Could white blood cell count be the link?

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The development of atrial fibrillation (AF) in acute coronary syndromes (ACS) is associated with increased morbidity and mortality. Therefore, being able to predict AF in this group of patients could lead to a change in the management strategy that could impact outcomes.

One common clinical dilemma is the patient presenting with their first episode of AF as a response to infection. Once the infection has been treated and the AF resolved, the conundrum is what their future risk of AF is and whether they need to be anticoagulated.

In a paper written by Tran et al. [1], the authors examined the association between white blood cell count (WBCC) in patients admitted for ACS, and changes in WBCC during hospitalization, with the development of new-onset AF. They reported that WBCC levels taken alone did not predict AF, but rather a greater increase in WBCC during hospitalization was associated with the risk of developing new onset AF.

Previous studies have shown that an elevated WBCC at hospital admission is an independent predictor of death at 6 months in patients with acute coronary syndromes [2]. In addition, elevations in WBCC were associated with impaired epicardial and myocardial perfusion and more extensive CAD [3]. Furthermore, WBCC has been independently associated with the incidence of AF. In community-based cohorts, an increased WBCC was associated with incidence of AF during a long period follow-up [4,5]. Such evidence suggests that the inflammatory component could potentially act as a link between AF and ACS.

In the study by Tran et al. [1] WBCC was proposed as a readily-available, and cost-effective tool to predict the occurrence of AF after an acute coronary event. The authors demonstrated that the magnitude of WBCC increase was associated with an increased incidence of AF. One strength of this study is that it included a diverse population from different socioeconomic classes and also a whole spectrum of patients presenting with ACS from those who were medically managed to those who underwent coronary artery bypass graft (CABG). Although the authors did not manage to collect information regarding to the type and the duration of AF, it is not clear how important this is, as the risk of different durations and persistence of AF remains a point of controversy and is not used in risk assessment tools.

Nevertheless, due to the nature of the study, there was a lack of data on the subtypes of WBCC as well as the timing of WBCC rise and its link to AF. This would help to further clarify the role of WBCC in AF prediction. Finally, the use of WBCC to predict AF was mainly in the acute setting. It is unclear if the patients who had a rise in their WBCC were sicker and had a longer hospital stay, hence allowing 'more time' for AF to be diagnosed while being an inpatient. What would complete the picture would be to see if the impact of WBCC increases on AF development in the longer term up to one year post index event.

This interesting paper holds promise for the use of a cost effective readily available marker for prediction and management of AF in patients with ACS. So far, a rise of WBCC during the in-patient setting could help clinicians, as it could prove a useful tool to guide management of those patients (e.g. dual versus triple therapy) if AF is timely diagnosed.

Since a significant proportion of patients with ACS develop AF and as most of these patients are asymptomatic [6], WBCC predictive role could potentially help to avoid invasive and expensive methods of monitoring.

To conclude, the authors should be congratulated for their study. Despite the observational nature and the methodological issues arising from confounding factors, they included a large sample of patients making the results valid. However, there is still need for more mechanistic studies to evaluate the AF-related role of WBCC in ACS.

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Conflict of interest

We declare no conflict of interest.

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