

**LETTER TO THE EDITOR**

# New-onset atrial fibrillation after atrial ischemia

To the editor:

We read with great interest “New-onset atrial fibrillation in patients with acute coronary syndrome may be associated with worse prognosis and future heart failure.” by Nagai M et al published in *Journal of Arrhythmia*.<sup>1</sup> The authors have reported that new-onset atrial fibrillation (AF) after acute coronary syndrome (ACS) may be associated with worse prognosis and future heart failure. We have further comments about the study.

First, selective atrial artery occlusion during elective percutaneous transluminal coronary angioplasty is associated with myocardial ischemic damage, atrial arrhythmias, and intra-atrial conduction delay.<sup>2</sup> AF occurs when structural and/or electrophysiological abnormalities change atrial tissue to facilitate abnormal impulse formation and/or propagation in addition to a combination of multiple factors, such as genetic components, heart failure, atrial stretch and ischemia, sympathovagal influences, inflammation, and fibrosis. In the setting of acute myocardial infarction (AMI), some reports have demonstrated that sinus node artery and atrial branches affect the development of AF.<sup>3,4</sup> However, these reports did not consider the relationship between coronary artery occlusion involving atrial branches and other factors of AF, including advanced age, heart failure symptoms, and depressed left ventricular function. The authors also have not touched on the impacts due to coronary artery occlusions involving atrial branches on the development of AF after ACS. Atrial ischemia may play a key role in the development of AF.

Second, the authors have reported that 67 of 645 patients had new-onset AF after ACS during the follow-up period ( $1.4 \pm 1.2$  years). AF is a common supraventricular arrhythmia after ACS and an indicator of worse prognosis in the short term and long term.

Clinical atrial infarction may manifest with supraventricular arrhythmias, atrial rupture, hemodynamic compromise from loss of atrial “kick,” and thromboembolic phenomena. Moreover, atrial ischemia and infarction may result in atrial electrophysiological changes and the propensity for AF, forming the dominant substrate for AF in myocardial infarction. In fact, atrial and supraventricular tachycardia were recorded more frequently after inferior wall myocardial infarction in patients with right ventricular involvement than in those without right ventricular involvement.<sup>5</sup> A strategy that protects atrial branches from the right coronary artery may be effective for preventing new-onset AF after percutaneous coronary intervention. However, it is difficult to objectively


evaluate these electrocardiographic changes in the clinical setting. Thus far, no modality can confirm atrial ischemic damage, including scintigraphy and echocardiography, after AMI. Further studies are necessary to evaluate the association between atrial ischemia and new-onset AF after ACS.

Lastly, the clinical implication of this study is that strict continuous follow-up of atrial arrhythmias is necessary for patients with new-onset AF in the acute phase after ACS for the purpose of preventing congestive heart failure requiring hospitalization. Furthermore, optimal medical therapy and catheter ablation therapy should be considered to improve the clinical outcomes in this population. However, there is a lack of data concerning optimal treatment modalities of AF and the timing of the start of AF treatment in this setting. Therefore, randomized trials evaluating effects of antithrombotic and/or antiarrhythmic management should be designed and executed.

We would be very interested in a commentary from the authors on the above. We thank the authors for their valuable data about new-onset AF after ACS.

## CONFLICT OF INTEREST

Authors declare no conflict of interests for this article.

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## REFERENCES

1. Nagai M, Itoh T, Ishida M, Fusazaki T, Komatsu T, Nakamura M, et al. New-onset atrial fibrillation in patients with acute coronary syndrome may be associated with worse prognosis and future heart failure. *J Arrhythm*. 2019;35(2):182–9. <https://doi.org/10.1002/joa3.12154>.

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2. Álvarez-García J, Vives-Borrás M, Gomis P, Ordoñez-Llanos J, Ferrero-Gregori A, Serra-Peñaranda A, et al. Electrophysiological effects of selective atrial coronary artery occlusion in humans. *Circulation*. 2016;133(23):2235–42.
3. Alasady M, Abhayaratna WP, Leong DP, Lim HS, Abed HS, Brooks AG, et al. Coronary artery disease affecting the atrial branches is an independent determinant of atrial fibrillation after myocardial infarction. *Heart Rhythm*. 2011;8(7):955–60.
4. Kyriakidis M, Barbetseas J, Antonopoulos A, Skouros C, Tentolouris C, Toutouzas P. Early atrial arrhythmias in acute myocardial infarction. Role of the sinus node artery. *Chest*. 1992;101(4):944–7.
5. Rechavia E, Strasberg B, Mager A, Zafrir N, Kusniec J, Sagie A, et al. The incidence of atrial arrhythmias during inferior wall myocardial infarction with and without right ventricular involvement. *Am Heart J*. 1992;124(2):387–91.