# Editorial board comments on the article «The frequency of atrial infarction in patients with supraventricular arrhythmias»

(authors: Bocharov A.V., Popov L.V. DOI: 10.18087/cardio.2022.3.n1648)

Atrial myocardial infarction (Atrial infarction AI) is a very rare disorder. AI can be caused by occlusion of the supplying artery (atherosclerosis, clot). It can also result from closure of the ostium of the atrial branch by a proximal right coronary artery or circumflex artery stent [1].

The publication of the paper of interest aims to draw the attention of readers and authors to studying left atrial (LA) performance and the diagnosis of LA disorders. The objective of the article is to assess the incidence of AI in patients with supraventricular arrhythmia after suppression of arrhythmia with verapamil. AI was diagnosed based on electrocardiographic (ECG) criteria, positive troponin test, and findings of selective coronary angiography. In our opinion, the article contains some contradictory theses and inconsistencies.

1. MI was diagnosed based on a positive qualitative response to cardiac troponins. This approach is not currently recommended for the diagnosis of MI. Small thickness of the necrotic atrial wall does not cause a significant increase in the levels of cardiac troponins. Moreover, a positive troponin test requires ruling out some other diseases, including myocarditis.
2. Clinical manifestations of acute atrial failure were not described in the article.
3. The authors used slow flow in the left atrial branch of the sinoatrial nodal artery as a diagnostic criterion for atrial MI. It is noteworthy that slow flow is not a diagnostic criterion for MI in general.
4. Parenteral administration of verapamil can cause coronary slow flow because of the coronary artery dilation (vasodilating effect of calcium channel blockers).
5. Selective coronary angiography showed no coronary lesions, but the authors state 82% of patients had coronary artery disease.
6. The presence of atrial MI usually results in atrial dysfunction followed by dilatation [2]. The article says nothing about it.
7. The pathophysiology of the sequence of events in this study is unclear: sinus rhythm recovered, after which atrial MI developed. The real-life sequence of events is different: first, atrial MI develops, after which, because of atrial wall necrosis, the generation and conduction of electrical impulses is disrupted, followed by the development of atrial fibrillation (AF).
8. The article does not report the incidence of the combination of atrial MI and infarctions of other sites. In most cases, the atria are involved in MI of larger areas (most often the inferior left ventricular (LV) wall).
9. Atrial dysfunction and atrial MI is verified by magnetic resonance imaging of the heart [2], which was not used in this study.

Taking into consideration the data presented by the authors, it can be assumed that, after the suppression of supraventricular arrhythmia and the restoration of sinus rhythm, patients experienced atrial tissue hibernation (?) combined with slow flow in the sinoatrial nodal artery due to the use of verapamil.

In our opinion, the involvement of the atria in the infarction process should be confirmed (or disconfirmed) in the presence of larger MI and atrial dilatation and dysfunction and/or sudden onset of AF. No effort, cost, or time should be spent to detect isolated atrial MI in the absence of any objective reasons.

However, the editorial board decided to publish this paper to draw attention to studying LA, its function, role in LV filling, diagnostic and prognostic possibilities, everything that has recently been defined as left atrial cardiomyopathy. It is necessary to develop up-to-date diagnostic approaches using contrast-enhanced MRI of the heart, stress-strain EchoCG, and other techniques. We look forward to further studies of LA lesions in ischemia and heart rhythm disorders, chemotherapy cardiotoxicity and, of course, in myocarditis and cardiomyopathies, including COVID-19 cases.

## REFERENCES

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