Inflammatory and non-inflammatory triggers of acute coronary syndromes

Inflammation plays an important role in the pathogenesis of acute coronary syndromes (ACS).¹ However, ACS include different clinical conditions that can present as an acute cardiac event, and it may also be seen in various clinical settings independent of underlying cardiovascular status and traditional risk factors.² Recently, Westerlind *et al*³ reported that siblings of patients with rheumatoid arthritis (RA) are at increased risk of ACS that could not be explained by traditional cardiovascular risk factors. This article provides excellent information regarding importance of inflammation and its hereditary role in the pathophysiology of ACS. Nevertheless, because of many clinical conditions may be the underlying cause of ACS and myocardial infarction (MI), there are some important points to be discussed to clarify the findings of the present article.

ACS and MI may also occur as an acute cardiac event in the absence of significant coronary artery disease (CAD) and inflammation. This type of MI is known as type 2 MI, and may occur in the context of a mismatch between myocardial oxygen supply and demand due to various diseases.⁴ Therefore, ACS is not only triggered by atherosclerotic plaque disruption, it may occur due to non-cardiovascular reasons, and this situation should be taken into consideration while defining ACS. Additionally, inflammation also seems to play a role in the mechanism of ACS secondary to non-atherosclerotic spontaneous coronary artery dissection.⁵ Hence, it would be worth to know the CAD severity and the underlying mechanism of ACS of the study groups in the study by Westerlind *et al*,³ to comment on the relationship between index event and patients' clinical conditions.

In conclusion, rheumatoid diseases and inflammation have important roles in the pathophysiology of CAD and ACS. However, ACS and MI do not occur only due to atherosclerotic plaque disruption. Non-cardiovascular and non-atherosclerotic reasons also may trigger ACS and MI in various clinical conditions. Hence, the underlying mechanism of ACS should be clearly stated to claim a relationship between siblings of patients with RA and an increased risk of ACS.

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