

Response to Letter Regarding Article, “Good Prognosis for Pericarditis With and Without Myocardial Involvement: Results From a Multicenter, Prospective Cohort Study”

We thank Drs Mewton and Bresson for their interest in our article.¹ The issues raised by the authors are essentially 3: the diagnosis being based on subjective criteria, the exclusion of patients with systemic inflammatory diseases, and the rationale for the definition of perimyocarditis and myopericarditis not being based on solid pathophysiological evidence.

First, in clinical practice, a spectrum of myopericardial syndromes can be encountered, ranging from pure pericarditis to increasing degrees of inflammatory myocardial involvement (myopericarditis and perimyocarditis) to pure myocarditis.^{2,3} Diagnostic criteria for acute pericarditis are well recognized and established.³⁻⁵ Although not supported by guidelines and consensus documents, myopericarditis and perimyocarditis definitions also have been proposed on the basis of clinical criteria.³ Myopericarditis is a primarily pericardial inflammatory syndrome occurring when clinical diagnostic criteria for pericarditis are satisfied and concurrent mild myocardial involvement is documented by elevation of biomarkers of myocardial damage (ie, increased troponins).^{1,3}

In our article,¹ a clinical diagnosis of myopericarditis was made in patients with a definite diagnosis of acute pericarditis and elevation of cardiac markers of injury (troponin I or T, creatine kinase-MB fraction) without new onset of focal or diffuse depressed left ventricular function by echocardiography or cardiac magnetic resonance. Perimyocarditis was diagnosed in patients with clinical criteria for acute pericarditis, elevation of cardiac markers of injury, and evidence of new onset of focal or diffuse depressed left ventricular function by echocardiography or cardiac magnetic resonance. The rationale for these diagnostic criteria is that pure pericardial or predominant pericardial inflammatory involvement is not characterized by significant impairment of myocardial function and that, on the contrary, focal or diffuse abnormalities of ventricular wall motion or function imply substantial myocardial inflammatory involvement.³ On this basis, we disagree and believe that reported clinical criteria have been published several times previously and are based on the best available clinical evidence. Obviously, there is a need for further research and especially a better understanding of the pathophysiology, but a proper classification with or without myocardial involvement and with or without ventricular dysfunction is clinically useful and may help guide different management strategies (similar for pericarditis and myopericarditis, ie, pericarditis with mild myocardial involvement and preserved ventricular function, and similar for perimyocarditis and myocarditis).

Second, the article included all consecutive patients with pericardial inflammatory syndromes (acute pericarditis, myopericarditis, and perimyocarditis) and thus there is no reason to exclude patients with systemic inflammatory syndromes who may be a part of the group.^{2,3}

Third, the pathophysiology of inflammatory myopericardial syndromes is still under investigation, and the adopted classification and diagnostic criteria are clinically based and useful in clinical practice. We would like also to clarify that troponin was measured serially in all patients.

In conclusion, we presented commonly adopted clinical criteria that are useful for the clinical management of patients with mixed forms of inflammatory myopericardial involvement. Further research is obviously needed to better understand the pathophysiology and to improve treatment, and study of the long-term outcomes is needed.

Disclosures

None.

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