

or can develop during hospitalization in non-ST-segment elevation ACS.⁴ In the study by Bardají et al,¹ there are no data on the time to onset of heart failure symptoms. The authors should comment on the classification of heart failure patients and its possible relationship with ACS.

In conclusion, patients with elevated troponin levels and without ACS may have poor prognosis due to concomitant systemic diseases. Patient classification as ACS or not may need further examination in the study by Bardají et al.¹

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Elevated Troponin Levels in Patients Without Acute Coronary Syndrome: What is the Real Diagnosis? Response



Valores de troponina elevados en pacientes sin síndrome coronario agudo: ¿cuál es el diagnóstico real? Respuesta

To the Editor,

We very much appreciate Dr. Eyuboglu's comments regarding our article on elevated troponin values¹ and the opportunity to respond and clarify some of the questions raised. The first comment refers to those patients with chest pain, without syncope and with other diagnoses who, despite their elevated troponin levels, were not diagnosed with acute coronary syndrome (ACS). Indeed, according to the most recent recommendations, the diagnosis of acute myocardial infarction is established within a clinical context compatible with myocardial ischemia when an increase in troponin levels is detected.² It is precisely this clinical criterion that enables us to classify the patients referred to by Dr. Eyuboglu as not belonging to the ACS group. We all agree that the simple presence of chest pain, syncope, or any other symptom, even when it coincides with an elevated troponin level, is not a sufficient basis for establishing a diagnosis of acute myocardial infarction and ACS. The reason for the increase in troponin levels in these patients may be multifactorial³ and, unfortunately, as occurs in other series, there is always a group of patients with elevated troponin levels of uncertain cause.⁴

With respect to the patients with bradyarrhythmias or tachyarrhythmias and increased troponin levels, the decision not to classify them as ACS patients is based on the recommendations for the diagnosis of type 1 and type 2 myocardial infarctions, proposed in the third universal definition of myocardial infarction² and, more specifically, in the diagnostic criteria established in the publication by Saaby et al.⁴ After reviewing all of these cases, we consider that most of these patients meet the criteria for type 2 acute myocardial infarction, as explained in the discussion section of our article, clarifying that those patients with arrhythmic events clearly secondary to myocardial ischemia were diagnosed

as having ACS and do not belong to the group with bradyarrhythmias or tachyarrhythmias. The term ACS, at the present time, refers basically to cases of type 1 myocardial infarction, the pathophysiological mechanism of which is supposedly the complication of an atheromatous plaque located in a coronary artery, and the patients have a well-established prognosis and treatment (anticoagulation therapy, antiplatelet therapy, statins, revascularization, etc.). This does not apply to most patients with bradyarrhythmias or tachyarrhythmias because, in reality, they have type 2 myocardial infarctions. In our opinion and that of other prominent authors,⁵ patients with type 2 acute myocardial infarction should not be reported as having ACS.

Finally, with respect to patients with a diagnosis of heart failure and elevated troponin levels, we agree that, in some cases, it may prove very difficult to determine whether or not they have an ACS as an underlying problem.² All the available clinical information in these cases has been reviewed by 2 experienced clinical cardiologists, and it has been possible to rule out, in a reasonable manner, the possibility of ACS. Again, we are convinced that some of these patients with heart failure and elevated troponin levels have a type 2 myocardial infarction, especially if they show the hemodynamic conditions associated with this diagnosis.

In conclusion, we consider that the relevance of our report lies in its demonstration that, in clinical practice, the definitive diagnosis of patients with increased troponin levels is often not as easy or evident as could be supposed from the simple application of a series of international recommendations.

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