

Factors Contributing to the Low Rate of Surgical Coronary Revascularization in Spain. Are We Following the Recommendations? Response



Factores que contribuyen a la reducida indicación de la cirugía de revascularización coronaria en España. ¿Seguimos las recomendaciones? Respuesta

To the Editor,

We appreciate the comments and interest from Gualis Cardona et al and Lozano et al regarding our study.¹ Their comments support our impression that the results from our study can be extrapolated to most Spanish centers. They explained their opinions on the causes that have led to the infrequent use of coronary revascularization surgery in Spain. We would add to these causes the progressive increase in experience and confidence of the interventional cardiologists when it comes to treating lesions of the common trunk or multivessel disease, the advances in technology that have led to a low rate of restenosis and thrombosis, and patient preferences, particularly for patients of advanced age.

There is also a lack of published data on surgical results from hospitals in our environment and a low rate of procedures by some cardiac surgeons, owing to a low rate of referral. We think this could contribute to the perception some cardiologists have that the results of surgery in centers where practice is based on guideline recommendations are not comparable to results obtained nationwide.

However, our intention was not to perform an exhaustive analysis of the factors that have led to the situation we described, but to stress the need for their analysis.

During recent years it has been common to see studies and registers published in our environment that analyze rates of compliance with guidelines for such prevalent and relevant conditions as atrial fibrillation,² acute coronary syndrome,³ hypertension,⁴ or hyperlipidemia.⁵ However, we are not aware of any studies that have analyzed compliance with clinical guideline recommendations on coronary revascularization.

The lack of economic interest in promoting initiatives or support for this type of study should not be an impediment; the

scientific societies and administrative bodies can and must encourage the conduction of such studies.

Finally, we would like to acknowledge the comments from Gualis Cardona et al. regarding our study opening the door to an essential debate in our country. Should this happen, we will consider the main objective of our study¹ achieved.

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



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

To the Editor,

I read with great interest the article by Bardají et al.¹ In their study, the authors reported that patients with elevated troponin levels and no diagnosis of acute coronary syndrome (ACS) had higher mortality than patients with negative troponin without ACS, and had a similar prognosis to patients with ACS. In this article, I would like to emphasize some confusing factors about diagnosis in patients with elevated troponin levels and without ACS in that study. Firstly, in the study by Bardají et al,¹ the authors reported 9 patients with chest pain, 6 patients with syncope, and 8 patients with other diagnoses in the group of patients with

elevated troponin levels and without ACS. To our knowledge and according to current guidelines,² these 3 diseases are not possible non-ACS causes of troponin elevation. Therefore, the authors should comment on the mechanism of troponin elevation in these patient subgroups and the reasons for potentially false-positive troponin elevation. Secondly, the cause of troponin elevation was tachycardia in 25 patients and bradyarrhythmia in 6 patients in the group of patients with elevated troponin levels and without ACS. Tachycardia and bradyarrhythmia are possible causes of non-ACS troponin elevation. However, it is known that they can occur during ACS and can be the only sign of ACS. In particular, ventricular tachycardia and atrioventricular blocks can be associated with ACS. Therefore, the authors should comment on the types of tachycardia and bradycardia and their possible relationship with ACS to eliminate misdiagnosis. Finally, the cause of troponin elevation was defined as heart failure in 55 patients in the non-ACS group. Ischemic heart disease may present as ACS in the acute phase and heart failure in the chronic phase.³ In addition, it is known that acute heart failure may be the presentation of ACS

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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