EDITORIAL

Discharged from the emergency department with elevated troponin levels?

¿Alta desde urgencias con troponina elevada?

María Rubini Giménez, Christian Mueller

The clinical introduction of high-sensitivity troponins has taught us that myocardial damage due to non-coronary causes is more frequent than previously thought[1-13]. The exact mechanism leading to this myocardial damage is currently unknown and there is uncertainty as to whether the damage is always irreversible [as in acute myocardial infarction (AMI)] which inevitably leads to myocardial necrosis, or whether some entities may cause reversible damage. Similarly, in patients with acute non-coronary pathologies such as septic shock, there is usually disagreement as to whether troponin elevation is produced by a mechanism of ischemia due to increased demand, direct myocardial damage or even plaque rupture that is undetected. Therefore, secondary prevention measures (such as antiplatelets or statins) in patients with troponin elevation without acute coronary syndrome (ACS) can only be considered in later stages.

But then, why are troponins measured in these patients? Basically, for three reasons. First: because it is the standard of care accepted for patients with suspected AMI. Second, because in patients in whom coronary disease has been excluded, they help us to predict the risk (for example, in pulmonary embolism)[14]. And, third, because in patients with other cardiac pathologies such as heart failure or arrhythmias, the concentrations should help us to detect AMI as a possible trigger[15].

The clinical consequences of detecting this myocardial damage vary considerably. If the risk of future adverse events (death due to cardiovascular causes or AMI) is due, at least in part, to acute heart damage caused by the current episode (e.g. sepsis), then cardiac therapy aimed at minimizing myocardial damage may decrease the chance of a cardiovascular event. Conversely, if the risk of AMI or death from cardiovascular causes is attributable to the precipitating condition (and elevation of troponins is a marker of the severity of the current process, without the heart being responsible), cardiac therapy would not mean a decrease of adverse events in the future, since these would be due to damage in other vital organs, like the lungs or the kidneys.

Given all the above, the work published by Cediel *et al.*[10] in this issue of the journal is of great importance and represents a step forward in the case of patients discharged from Spanish emergency departments with high troponin levels and without ACS. The main message is clear: those patients with elevated troponins are at high risk of cardiovascular events and comorbidity and, especially important, mortality after one year is high. Similar results were observed in the study by Chew *et al.*[1], which included 38,161 patients with similar characteristics to the study by Cediel et al., showing a significant and direct relationship between troponin concentration and mortality in those patients with a diagnosis of non-coronary causes.

However, it is not possible to affirm that elevated troponin levels in patients with non-coronary causes are associated with features that predict an adverse event independently. In addition, it is unclear how many patients with elevated troponin levels and without ACS have chronic coronary artery disease, so the association of elevated troponins in these patients and cardiovascular adverse events in the future does not have to be a causal relationship. Therefore, the recommendation to develop new therapeutic strategies or to admit these patients is elusive, as already mentioned in the article. Even so, the results of the study by Cediel *et al.*[10] cannot be ignored: they report a high rate of cardiovascular adverse events in patients discharged from our emergency departments, and indicate that there is a possible association of myocardial damage with future adverse events.

Therefore, we encourage further research in this field, especially the possible relationship between myocardial damage and non-coronary or non-cardiac diseases, as well as to follow up these discharged patients with elevated troponins. The purpose is to determine whether the high risk during the first year is a result of the cardiac damage produced by the event or, conversely, a simple measure of the morbidity of these patients.

Author affiliation: Servicio Cardiología, Hospital Universitario de Basilea, Switzerlanz.
Authors' contributions: Both authors have confirmed their authorship in the author's responsibilities document, publication agreement and assignment of rights to EMERGENCIAS.
Corresponding author: María Rubini. Servicio de Cardiología. Hospital Universitario de Basilea. Spitalstrasse 21. 4056 Basel, Switzerlanz.
Email: Maria.Rubini@usb.ch
Information about the article: Received: 22-8-2016. Accepted: 5-9-2016. Online: 8-9-2016.
Editor in charge: Òscar Miró, MD, PhD.

Conflicting interests

Dr. Rubini has received conference fees from Abbot and grants from the Swiss Heart Society. Prof. Mueller has received grants from the Swiss Heart Society, Swiss National Founds, Abbott, Alere, Beckmann Coulter, BRAHMS, Critical Diagnostic, Nanosphere, Roche, Siemens, Singulex, 8sense and the Department of Internal Medicine (University Hospital Basel), as well as conference fees from Abbott, Alere, Biomerieux, Brahms, Cardiorentis, Novartis, Roche, Siemens, Singulex

Financing

The authors declare no external financing of this article.

Article commissioned and with internal revision by the Editorial Committee

References

1 Chew DP, Briffa TG, Alhammad NJ, Horsfall M, Zhou J, Lou PW, et al. High sensitivity-troponin elevation secondary to non-coronary diagnoses and death and recurrent myocardial infarction: An examination against criteria of causality. Eur Heart Acute Cardiovasr Care. 2015;4:419-28.
2 Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, et al. Third universal definition of myocardial infarction. Eur Heart J. 2012;33:2551-67.
3 Gualandro DM, Puelacher C, Mueller C. High-sensitivity cardiac troponin in acute conditions. Curr Opin Crit Care. 2014;20:472-7.
4 Twerenbold R, Jaffe A, Reichlin T, Reiter M, Mueller C. High-sensitive troponin T measurements: what do we gain and what are the challenges? Eur Heart J. 2012;33:579-86.
5 Thygesen K, Mair J, Katus H, Huber K, Weber M, Plebani M, et al. Recommendations for the use of cardiac troponin measurement in acute cardiac care. Eur Heart J. 2010;31:2197-204.
6 Haaf P, Drexler B, Reichlin T, Twerenbold R, Reiter M, Meissner J, et al. High-sensitivity cardiac troponin in the distinction of acute myocardial infarction from acute cardiac noncoronary artery disease. Circulation. 2012;126:31-40.
7 Thygesen K, Mair J, Giannitsis E, Mueller C, Lindahl B, Blankenberg S, et al. How to use high-sensitivity cardiac troponins in acute cardiac care. Eur Heart J. 2012;33:2252-7.
8 Bardaji A, Cediel G, Carrasquer A, de Castro R, Sanchez R, Boque C. Troponin elevation in patients without acute coronary syndrome. Rev Esp Cardiol. 2015;68:469-76.
9 Salinas M. Variabilidad en la oferta y en la solicitud de determinaciones de laboratorio en pacientes de servicios de urgencias hospitalarios. Emergencias. 2014;26:450-8.
10 Cediel G, Carrasquer A, Sánchez R, Boqué C, González-del-Hoyo M, Bardaji A. Pronóstico a un año de la elevación de Troponina I en los pacientes dados de alta directamente desde un servicio de urgencias hospitalario. Emergencias. 2016;28:298-304.
11 Rubini M. Biomarcadores en el paciente con dolor torácico: pasado, presente y futuro. Emergencias. 2014;26:221-6.
12 Domínguez-Rodríguez A. Biomarcadores inflamatorios en patología isquémica coronaria aguda. Emergencias. 2015;27:319-24.
13 Bueno H. Continuidad en la investigación cardiovascular: una llamada a incrementar la colaboración en investigación entre Cardiología y la Medicina de Urgencias y Emergencias. Emergencias. 2015;27:396-8.
14 Konstantinides SV. 2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2014;35:3145-6.
15 Januzzi JL, Jr., Filippatos G, Nieminen M, Gheorghiade M. Troponin elevation in patients with heart failure: on behalf of the third Universal Definition of Myocardial Infarction Global Task Force: Heart Failure Section. Eur Heart J. 2012;33:2265-71.