

## Detection of High-sensitivity Troponin T in Patients With Cardiovascular Risk. Response



Isabel Álvarez Nozal,<sup>a</sup> Héctor García Pardo,<sup>b</sup> and Diego Martín Raymondi<sup>b,\*</sup>

### Detección de troponina T ultrasensible en pacientes con riesgo cardiovascular. Respuesta

#### To the Editor,

We have read with interest the Letter to the Editor by Velilla Moliner et al.<sup>1</sup> concerning our article and appreciate their qualifications.

In contrast to diagnostic methods quantifying high-sensitivity troponin I, the methods used for high-sensitivity troponin T (hs-TnT) allow its detection in about 35% of the healthy population.<sup>2</sup> Without delving into the definition of "healthy" and if the 99th percentile should vary according to the characteristics of the population being studied,<sup>3</sup> this aspect has not undermined the usefulness of hs-TnT in both the diagnosis of acute coronary events and their exclusion (given its high negative predictive value<sup>4</sup>). In addition, as correctly highlighted by the authors, hs-TnT has shown prognostic value not only in heart disease populations, but also healthy populations.

The ideal biomarker would be useful for diagnosis and prognosis, as well as treatment-related aspects. Regarding the latter, hs-TnT is a marker of the effectiveness of the recommended treatment<sup>5</sup> for heart failure.

The findings of the TUSARC (*Troponina T UltraSensible en pacientes de muy Alto Riesgo Cardiovascular* [High-sensitivity troponin T inpatients at high cardiovascular risk]) registry and others oblige clinicians to investigate other causes of hs-TnT elevation beyond ischemia. Thus, the association of an hs-TnT elevation with heart failure and myocardial fibrosis is important<sup>6</sup> because it guides the role of elevated hs-TnT as a marker of both reversible and irreversible structural damage.

#### FUNDING

Roche Diagnostics provided the kits for the troponin determination, as well as both the internal and external controls.

## Diagnosis of Cardiac Amyloidosis: Is Imaging Enough?



2. A generic diagnosis of CA is insufficient. The substance deposited needs to be identified because prognosis and treatment vary considerably according to the type of CA.<sup>3</sup> This requires immunohistochemical characterization of the amyloid material found in the biopsy, as well as demonstration of circulating amyloid protein in serum (amyloid light-chain amyloidosis [AL]) or a causative genetic mutation (transthyretin familial amyloidosis).

Physicians can only recommend radical therapeutic options such as transplantation or chemotherapy after identification of the specific type of amyloidosis.<sup>3</sup> The patient studied by García-González et al.<sup>1</sup> does indeed have a high probability of having myeloma-associated AL amyloidosis but, due to his age and sex, might actually have senile CA (due to deposition of wild-type transthyretin), which would involve a different prognosis and therapeutic approach.<sup>4</sup> Only when senile CA is suspected (due to its more benign behavior and the absence of a specific treatment) is it suggested that <sup>99m</sup>technetium scintigraphy could obviate the need for endomyocardial biopsy.<sup>5</sup> Nonetheless, the appropriate course of action remains unclear.

In conclusion, we believe that the emergence of <sup>18</sup>F-florbetapir PET/CT for the diagnosis of CA is excellent news, especially if it is shown to be more sensitive than the other imaging techniques currently used for this purpose (ultrasound and magnetic resonance),<sup>6</sup> but biopsy of the affected organ is still required. In

1. A diagnosis of CA requires histological evidence of amyloid deposits, either in the heart itself or in biopsies from other affected organs.<sup>2</sup> If the biopsy is obtained from an organ other than the heart, the typical signs of CA need to be seen in cardiac imaging tests (echocardiography). False positives are possible in any imaging test, and CA diagnosis frequently has serious prognostic and therapeutic implications.

<sup>a</sup>Medicina Familiar y Comunitaria, Hospital Santos Reyes, Aranda de Duero, Burgos, Spain

<sup>b</sup>Servicio de Cardiología, Hospital Santos Reyes, Aranda de Duero, Burgos, Spain

\* Corresponding author:

E-mail address: dmartinr@saludcastillayleon.es  
(D. Martín Raymondi).

Available online 6 May 2017

## REFERENCES

1. Álvarez I, Hernández L, García H, et al. Troponina T ultrasensible en pacientes asintomáticos de muy alto riesgo cardiovascular. Registro TUSARC. Rev Esp Cardiol. 2017;70:261–266.
2. Saenger AK, Beyrau R, Braun S, et al. Multicenter analytical evaluation of a high-sensitivity troponin T assay. Clin Chim Acta. 2011;412:748–754.
3. Gore MO, Seliger SL, de Filippi CR, et al. Age- and sex-dependent upper reference limits for the high sensitivity cardiac troponin assay. J Am Coll Cardiol. 2014;63:1441–1448.
4. Body R, Burrows G, Carley S, et al. High Sensitive Cardiac Troponin T as an Early Biochemical Signature for Clinical and Subclinical Heart Failure: The Multi-Ethnic Study of Atherosclerosis. Clin Chem. 2015;61:983–989.
5. McMurray J, Packer M, Desai AS, et al. Angiotensin–Neprilysin Inhibition versus Enalapril in Heart Failure. N Engl J Med. 2014;371:993–1004.
6. Seliger SL, Hong SN, Christenson RH, et al. High Sensitive Cardiac Troponin T as an Early Biochemical Signature for Clinical and Subclinical Heart Failure: The Multi-Ethnic Study of Atherosclerosis. Circulation. 2017. <http://dx.doi.org/10.1161/CIRCULATIONAHA.116.025505>.

#### SEE RELATED CONTENT:

<http://dx.doi.org/10.1016/j.rec.2017.01.030>

<http://dx.doi.org/10.1016/j.rec.2017.04.004>

1885-5857/

© 2017 Sociedad Española de Cardiología. Published by Elsevier España, S.L.U. All rights reserved.