High-sensitivity Troponin T Assay in Asymptomatic High Cardiovascular Risk Patients. The TUSARC Registry

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A B S T R A C T

Introduction and objectives: High-sensitivity troponin T assays (Hs-TnT) have been carried out in selected populations in clinical trials and in registries of the general population with low cardiovascular risk (CVR). The aim of this study was to determine the proportion of individuals with elevated Hs-TnT and the proportion of individuals with elevated Hs-TnT in a Spanish population of asymptomatic individuals with very high CVR, as well as the parameters associated with Hs-TnT elevation.

Methods: The study included 690 patients. Hs-TnT detection and Hs-TnT elevation (>99th percentile value), as well as the association of elevated Hs-TnT and clinical, analytical, and treatment data were analyzed.

Results: Hs-TnT was analyzed in 646 patients and was detected in 645. Elevated TnT was detected in 212 patients (32.9%). On multivariate analysis, elevated TnT was independently associated with male sex (OR, 2.81; 95%CI, 1.67-4.73; P < .001), older age (OR, 1.06; 95%CI, 1.04-1.09; P < .001), a higher body mass index (OR, 1.07; 95%CI, 1.02-1.12; P < .002), insulin therapy (OR, 1.99; 95%CI, 1.15-3.46; P < .01), history of heart failure (OR, 3.92; 95%CI, 1.24-12.39; P = .02), and estimated glomerular filtration rate calculated by CKD-EPI (OR, 0.96; 95%CI, 0.95-0.97; P < .001).

Conclusions: In a Spanish population of asymptomatic individuals at very high CVR, Hs-TnT was associated with older age, male sex, higher body mass index, insulin therapy, history of heart failure, and lower glomerular filtration rate.

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INTRODUCTION

Cardiac troponin is a component of the cardiomyocyte contrac-
tile apparatus that is released to the bloodstream after myocardial
injury. The high-sensitivity troponin T (hs-TnT) assay detects
smaller amounts of troponin T than conventional assays, permit-
ting earlier and more precise diagnosis and improved treatment.\(^1\)
However, the use of hs-TnT assays also increases the proportion of
emergency patients and outpatients with detectable levels of
cardiac troponin T. High-sensitivity troponin T is detected not only
in the acute phase of coronary events, but also in clinical trial
patient populations with ischemic heart disease,\(^2\) heart failure
(HF),\(^3\) and kidney disease; in healthy populations with no evident
heart disease,\(^4\)-\(^6\); and in emergency room patients with no
coronary event.\(^7\)

There is currently no information on the prevalence in Spain of
detectable hs-TnT and elevated hs-TnT (≥ the 99th percentile
value) in asymptomatic individuals at very high cardiovascular risk
(CVR) and receiving stable medical treatment. The aim of this study
was to determine the proportion of individuals with detectable Hs-
TnT and the proportion of individuals with elevated Hs-TnT in a
Spanish population of asymptomatic individuals with very high
CVR, as well as the parameters associated with Hs-TnT elevation.

METHODS

Study Population

The TUSARC registry (Troponina T UltraSensible en pacientes de
muy Alto Riesgo Cardiovascular [High-sensitivity troponin T in
patients at high cardiovascular risk]) compiles data from a
prospective cohort of asymptomatic individuals who meet the
criteria for very high CVR according to the previous European
Society of Cardiology definition: cardiovascular disease diagnosed
by invasive or noninvasive methods; previous myocardial infar-
cction, percutaneous or surgical coronary revascularization, or
stroke; peripheral artery disease; type 1 or 2 diabetes mellitus
associated with another CVR factor or target organ involvement;
SCORE risk ≥ 10%; or glomerular filtration rate (GFR) ≤ 60 mL/min/
1.73 m\(^2\).\(^8\) The registry was approved by the Burgos University
Hospital Ethics Committee (ref. CEIC 1008), and all participants
gave written informed consent.

Study participants were recruited during outpatient consulta-
tions in the cardiology and internal medicine departments at our
hospital. To be included, patients had to have at least 1 of the high
CVR factors listed above, had have no treatment modifications in
the 3 months preceding inclusion, and be asymptomatic at the
time of recruitment. Asymptomatic status was defined as follow:
\(a)\) absence in the 3 months preceding inclusion of any clinical
cardiovascular event (angina, acute myocardial infarction [AMI],
cerebrovascular ischemic accident, or clinical HF episode); \(b)\) a
minimum of 3 months since surgical coronary or peripheral
revascularization, and \(c)\) a minimum of 6 months since percuta-
neous coronary revascularization. The 6-month wait period after
percutaneous coronary revascularization was chosen because this is
the highest risk period for in-stent restenosis. At the time of
inclusion, none of the patients was in New York Heart Association
functional class III-IV, and none mentioned difficulties with day-
to-day activities. A history of clinical HF was identified from a
review of clinical event history showing objective evidence of
water retention in chest X-ray, a description of edema or
symptoms compatible with HF, or clinical improvement after
diuretic therapy.

Between March 2013 and March 2015, a total of 690 patients
gave informed consent to participate in the study. Of the patients
who initially gave consent, 44 did not attend the scheduled visit for
blood extraction and were excluded from the analysis. Thus, a total
of 646 patients were examined for Hs-TnT and were included in the
statistical analysis. For all participants, a specifically designed data
sheet was used to record anthropometric data, CVR factors, clinical
characteristics, relevant comorbidities, and medical treatment. In
addition to Hs-TnT determination, analytical biochemistry par-
ameters were recorded for all patients, including a study of kidney
function. Kidney function was assessed from measurements of
serum creatine, microalbumin, the albumin-creatinine index (deter-
mined in first-morning urine samples), and GFR estimated with the
Chronic Kidney Disease Epidemiology Collaboration equation
(CKD-EPI). Left ventricular hypertrophy (LVH) was diagnosed by
electrocardiogram traces according to the Cornell criteria. Periph-
eral vascular disease and carotid atheromatosis were diagnosed by
the presence of ≥ 50% stenosis.

Laboratory Parameters

Blood samples were collected in EDTA tubes and centrifuged
within 4 hours of collection, and the plasma samples obtained
were frozen at −21 °C. Batches of frozen plasma samples were sent
periodically by specialized messenger service for processing at the
central laboratory (Hospital Central de la Defensa Gómez Ulla,
Madrid, Spain). In this study, troponin T concentration was
measured using the Cobas 6000 platform for high-sensitivity
determination, with a minimum detection level of 3 ng/L and a
predetermined reference value of 14 ng/L for the 99th percentile in
the healthy population. Data quality was ensured by the use of
internal and external controls. The internal control was provided
by Roche Diagnostics (Preci Control Troponin). The control for low
concentrations (lot No. 175680000) had a calculated variation
coefficient of 2.4% and the control for high concentrations (lot No.
17405200) had a calculated variation coefficient of 3%. For the
external control, we used the Seironom Immunonassay Liq L-1/
Immunoprotein Liq system. Values were assigned according to the
stipulations of European directive 98/79/CE on in vitro diagnostic
medical devices and international standard ISO 17511. The lot
number was 1208448, with a variation coefficient of 5.1%.

Elevated Hs-TnT was defined as a concentration ≥ 14 ng/L.

Statistical Analysis

Statistical analysis was conducted with Stata 13.1 (StataCorp;
College Station, Texas, United States). Quantitative variables are
presented as mean ± standard deviation and qualitative variables as
numbers and frequencies. Quantitative variables were analyzed by
the Student t test and qualitative variables by the chi-square test.
The adjusted model for the multivariable analysis included all variables
found to be significant in the univariable analysis. We calculated the
odds ratio (OR), 95% confidence interval (95%CI) and its statistical
significance (P value), and the C statistic for the final predictive model.
The final predictive model was the equation with largest area under the curve of all possible equations that included the significant variables in the multivariable analysis.

RESULTS

Univariable Analysis

In total, Hs-TnT was detected in 645 of the 646 patients (99.85%), and was ≥ 14 ng/L (elevated Hs-TnT) in 212 patients (32.9%; 95%CI, 29.36%). Among patients without elevated Hs-TnT (n = 434), mean Hs-TnT was 7.92 ± 3.11. Among patients with elevated Hs-TnT, mean Hs-TnT was 27.7 ± 38.23. Demographic characteristics, analytical biochemistry variables, and cardiovascular and medical treatment history are listed in Table 1 according to whether patients had elevated or nonelevated Hs-TnT. Patients with elevated Hs-TnT tended to be older and more overweight as assessed by body mass index (BMI), and were predominantly male. In relation to CVR factors, patients with elevated Hs-TnT had higher blood pressure but showed no differences from the other patients in analytical biochemistry parameters except for those related to kidney function: serum creatine, microalbumin, albumin-creatinine index, and GFR calculated by CKD-EPI. The elevated Hs-TnT population had more history of clinical HF and higher rates of LVH, ischemic stroke, and peripheral arterial disease/peripheral revascularization. In addition, significantly more patients with elevated Hs-TnT had a GFR < 60 mL/min/1.73 m². The 2 patient groups showed no difference in any specifically coronary event (angina AMI, and percutaneous or surgical revascularization). Significant between-group differences were detected in the rates of treatment with acenocoumarol, the use of insulin to control diabetes mellitus, and the use of angiotensin II receptor blockers and calcium antagonists.

Multivariable Analysis

For all variables that were statistically significant in the univariable model (P < .05), a multivariable logistic regression analysis was conducted with elevated Hs-TnT (≥ 14 ng/L) as the dependent variable (Table 2). The only variables showing an independent association with elevated Hs-TnT were age, male sex, BMI, a history of clinical HF, and insulin therapy. Among kidney function parameters, only GFR estimated by CKD-EPI was associated with elevated Hs-TnT. In the resulting final model, the area under the curve was 0.8001 (95%CI, 0.76-0.84) (Figure).

DISCUSSION

The TUSARC registry evaluates the presence of nonelevated and elevated Hs-TnT in a population of asymptomatic patients at very high CVR and the association of Hs-TnT elevation with a range of recorded parameters. The main findings in this registry are as follows: a) Hs-TnT was detected in all but 1 of the patients included in the registry; b) elevated Hs-TnT levels were detected in a third of the patients (32.9%); c) Hs-TnT was associated with older age, high BMI, and male sex; d) the only clinical event associated with elevated Hs-TnT was a history of clinical HF; no correlation was observed between elevated Hs-TnT and a history of coronary events or coronary revascularization; e) elevated Hs-TnT was independent of blood sugar and cholesterol levels at the time of inclusion and was also independent of microalbumin and albumin-creatinine index, but was associated with lower CKD-EPI-estimated GFR, and f) insulin was the only treatment associated with elevated Hs-TnT.

Values for Hs-TnT have been recorded in several general population registries.4-6 Population registries provide real world data, without the selection bias inherent in clinical trials.

These general population registries included more patients than our population. However, comparisons are difficult because, unlike our registry, earlier registries had a low prevalence of CVR and previous cardiovascular disease.

Nonetheless, like our registry, general population registries show an association of Hs-TnT with sex and age, with higher Hs-TnT values recorded in men and older patients.7

The differences in Hs-TnT detection between registries appear to be explained by differences in CVR. For example, all patients included in the Cardiovascular Health Study were older than 65 years, and 18% of them had a history of cardiovascular disease.3 This registry thus had higher values for mean patient age and cardiovascular history than the other population registries,4,6 reflected in the detection of Hs-TnT in 66.2% of patients.

The influence of CVR would explain not only the detection of Hs-TnT in all but 1 patient in our population, but also the high percentage of patients with elevated Hs-TnT (32.9%). While avoiding the selection bias associated with clinical trials, our results are similar to those found in clinical trials in which the inclusion criteria include ischemic heart disease or HF, in which Hs-TnT is detected in almost all patients and is elevated in 11% to 50% of HF patients.4,8,9

One of the most notable findings in our study is the lack of association between Hs-TnT and a history of ischemic heart disease, manifested as angina, AMI, or revascularization, despite the high percentage of patients in our registry with a history of angina or AMI (54%). Although this finding could at first seem surprising, it is consistent with previously published series. For example, in the Dallas Heart Study,4 although the incidence of previous coronary disease was very low (3.3%), a large majority of those patients who did have coronary disease (83%) had Hs-TnT below 14 ng/L. Similarly, in clinical trials that used ischemic heart disease as an inclusion criterion, the fraction of patients with elevated Hs-TnT did not exceed 40%.2,10 This finding reinforces the idea that factors other than previous ischemic heart disease should be considered as possible causes of elevated Hs-TnT in asymptomatic patients at high CVR.

In this regard, another important finding in our study is the association between Hs-TnT and previous clinical HF events. Elevated Hs-TnT was found in almost all patients with HF associated with ventricular dysfunction, whether acute11 or chronic.3,12 In our population, 70% of patients with a clinical history of HF had elevated Hs-TnT. The many mechanisms involved in TnT release in HF patients are not limited to the presence of ischemic heart disease, and include injury mediated by inflammatory cytokines, oxidative stress, and various mechanisms triggered by wall stress, for example cellular apoptosis.13 Although Hs-TnT could be elevated by episodes of acute decompensation, our study population had no clinical events in the 3 months preceding inclusion, which should be sufficient time for TnT levels to return to baseline. The chronic Hs-TnT elevation in these patients might reflect changes to cardiac anatomy, muscular architecture, or function that are maintained beyond the release associated with acute events.

In this regard, elevated Hs-TnT has been linked to the presence of LVH measured by cardiac magnetic resonance in healthy populations, indicating troponin as a marker of structural heart disease.1 In our study, LVH showed no correlation with Hs-TnT in either the multivariable analysis or the univariable analysis. The apparent discrepancy with the cited findings may reflect the lower sensitivity of electrocardiography compared with cardiac magnetic resonance for the detection of LVH. In the low-risk ARIC registry population,5 left ventricular hypertrophy was measured according
**Table 1**
Demographic, Clinical, and Treatment Characteristics of Patients Classified According to the Presence or Absence of Elevated Hs-TnT (>99th Percentile Value)

<table>
<thead>
<tr>
<th></th>
<th>TnT-us &lt; 14 ng/L (n = 434)</th>
<th>TnT-us &gt; 14 ng/L (n = 212)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td>.016</td>
</tr>
<tr>
<td>Age, y</td>
<td>285 (71.97)</td>
<td>148 (81.32)</td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>66.53 ± 14.59</td>
<td>75.11 ± 11.55</td>
<td>&lt; .0001</td>
</tr>
<tr>
<td><strong>CV risk factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>62 (15.7)</td>
<td>17 (9.34)</td>
<td>.063</td>
</tr>
<tr>
<td>DM</td>
<td>246 (62.12)</td>
<td>117 (64.29)</td>
<td>NS</td>
</tr>
<tr>
<td>HT</td>
<td>295 (74.24)</td>
<td>162 (89.01)</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Dyslipemia</td>
<td>335 (84.81)</td>
<td>157 (86.26)</td>
<td>NS</td>
</tr>
<tr>
<td>Family history of HF</td>
<td>59 (14.9)</td>
<td>16 (8.79)</td>
<td>.042</td>
</tr>
<tr>
<td><strong>Analytical biochemistry parameters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose, mg/dL</td>
<td>126.81 ± 36.97</td>
<td>125.53 ± 40.88</td>
<td>NS</td>
</tr>
<tr>
<td>HbA1c, %</td>
<td>6.8 ± 4.52</td>
<td>7.30 ± 6.94</td>
<td>NS</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td>169 ± 34.78</td>
<td>167.87 ± 36.59</td>
<td>NS</td>
</tr>
<tr>
<td>HDL, mg/dL</td>
<td>53.04 ± 13.76</td>
<td>51.66 ± 15.76</td>
<td>NS</td>
</tr>
<tr>
<td>LDL, mg/dL</td>
<td>89.97 ± 27.58</td>
<td>90.01 ± 31.06</td>
<td>NS</td>
</tr>
<tr>
<td>Triglycerides, mg/dL</td>
<td>131.33 ± 92.88</td>
<td>131.60 ± 98.44</td>
<td>NS</td>
</tr>
<tr>
<td>Creatinine, ml/min</td>
<td>0.94 ± 0.28</td>
<td>1.23 ± 0.47</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Microalbumin, mg/L</td>
<td>29.83 ± 195.11</td>
<td>118.49 ± 493.39</td>
<td>.001</td>
</tr>
<tr>
<td>Albumin-creatinine index, mg/gCr</td>
<td>31.12 ± 161.41</td>
<td>157.19 ± 616.42</td>
<td>.001</td>
</tr>
<tr>
<td>CKD-EPI eGFR, ml/min/1.73 m²</td>
<td>81.18 ± 4.23</td>
<td>62.24 ± 20.85</td>
<td>&lt; .001</td>
</tr>
<tr>
<td><strong>Cardiovascular history</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angina</td>
<td>97 (24.49)</td>
<td>43 (23.63)</td>
<td>NS</td>
</tr>
<tr>
<td>Infarction</td>
<td>137 (34.68)</td>
<td>57 (31.32)</td>
<td>NS</td>
</tr>
<tr>
<td>LVH</td>
<td>82 (20.71)</td>
<td>59 (32.42)</td>
<td>.002</td>
</tr>
<tr>
<td>Ischemic stroke</td>
<td>39 (9.85)</td>
<td>32 (17.58)</td>
<td>.009</td>
</tr>
<tr>
<td>PAD/Peripheral revascularization</td>
<td>27 (6.82)</td>
<td>26 (14.29)</td>
<td>.004</td>
</tr>
<tr>
<td>Coronary revascularization (percutaneous or surgical)</td>
<td>203 (51.26)</td>
<td>85 (46.7)</td>
<td>NS</td>
</tr>
<tr>
<td>Creatine clearance ≤ 60 ml/min</td>
<td>49 (12.37)</td>
<td>77 (42.31)</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>44 (11.11)</td>
<td>30 (16.48)</td>
<td>.073</td>
</tr>
<tr>
<td>Carotid plaque</td>
<td>24 (6.06)</td>
<td>12 (6.59)</td>
<td>NS</td>
</tr>
<tr>
<td>HF</td>
<td>6 (1.52)</td>
<td>14 (7.69)</td>
<td>.001</td>
</tr>
<tr>
<td><strong>Medical treatment</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>238 (60.1)</td>
<td>115 (63.19)</td>
<td>NS</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>39 (9.85)</td>
<td>22 (12.09)</td>
<td>NS</td>
</tr>
<tr>
<td>Acenocoumarol</td>
<td>51 (12.88)</td>
<td>43 (23.63)</td>
<td>.001</td>
</tr>
<tr>
<td>Aspirin + clopidogrel</td>
<td>18 (4.55)</td>
<td>13 (7.14)</td>
<td>NS</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>115 (29.04)</td>
<td>53 (29.12)</td>
<td>NS</td>
</tr>
<tr>
<td>ARB</td>
<td>131 (33.08)</td>
<td>79 (41.41)</td>
<td>.017</td>
</tr>
<tr>
<td>CA</td>
<td>100 (25.25)</td>
<td>64 (36.16)</td>
<td>.014</td>
</tr>
<tr>
<td>NSAID</td>
<td>18 (4.55)</td>
<td>11 (6.04)</td>
<td>NS</td>
</tr>
<tr>
<td>Insulin</td>
<td>49 (12.37)</td>
<td>46 (25.27)</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>142 (35.86)</td>
<td>73 (40.11)</td>
<td>NS</td>
</tr>
<tr>
<td>OAD</td>
<td>221 (55.81)</td>
<td>98 (53.85)</td>
<td>NS</td>
</tr>
<tr>
<td>Statins</td>
<td>316 (72.8)</td>
<td>149 (70.2)</td>
<td>NS</td>
</tr>
</tbody>
</table>

ACE, angiotensin converting enzyme; ARB, angiotensin receptor blockers; BMI, body mass index; CA, calcium antagonist; carotid plaques were defined by the presence of plaques causing > 50% stenosis; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration equation; DM, diabetes mellitus (antidiabetic medication or analytical variables at the time of inclusion); dyslipemia was defined as medication with lipid-lowering agents or LDL > 160 mg/dL; eGFR, estimated glomerular filtration rate; HF, clinical heart failure episode; Hs-TnT, high-sensitivity troponin T; HT, hypertension (medication with antihypertensive drugs or mean blood pressure > 140/90 mmHg in 3 consultations); LDL, low-density lipoprotein; LVH, left ventricular hypertrophy detected electrographically according to the Cornell criteria; NSAID, nonsteroidal anti-inflammatory drugs; OAD, oral antidiabetics, PAD, peripheral artery disease (claudication symptoms). Microalbumin and the albumin-creatinine index were determined in first-morning urine samples. Values are expressed as no. (%) or mean ± standard deviation.
Table 2
Final Model of Predictive Variables for High-sensitivity Troponin T

<table>
<thead>
<tr>
<th></th>
<th>OR (95%CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.06 (1.04-1.09)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Men</td>
<td>2.81 (1.67-4.73)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>BMI</td>
<td>1.07 (1.02-1.12)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>CKD-EPI eGFR (mL/min/1.73 m²)</td>
<td>0.96 (0.95-0.97)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>HF</td>
<td>3.92 (1.24-12.39)</td>
<td>.02</td>
</tr>
<tr>
<td>Insulin therapy</td>
<td>1.99 (1.15-3.46)</td>
<td>.01</td>
</tr>
</tbody>
</table>

95%CI, 95% confidence interval; BMI, body mass index; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration equation; eGFR, estimated glomerular filtration rate; HF, heart failure; OR, odds ratio.

Figure. Area below the curve ROC = 0.8001

CONCLUSIONS

High-sensitivity troponin T was detected in almost all high CVR patients receiving stable treatment in the 3 months preceding inclusion. A clinical history of HF was significantly associated with elevated Hs-TnT. High-sensitivity troponin T showed no association with a history of AMI, angina, or coronary revascularization. Patients with elevated Hs-TnT had a significantly diminished CKD-EPI-estimated GFR, although more than half of them had a GFR > 60 mL/min/1.73 m². Among the pharmacological treatments examined, only insulin therapy showed an association with Hs-TnT elevation.
FUNDING

This study was funded by Roche Diagnostics.

CONFLICTS OF INTEREST

None declared.

WHAT IS KNOWN ABOUT THE TOPIC?

– The level of Hs-TnT and the prevalence of Hs-TnT elevation have been studied in “healthy” populations (with few cardiovascular risk factors and little prior cardiovascular disease) and in clinical trial populations recruited according to strict inclusion criteria.

– There is a lack of population registries including patients at very high cardiovascular risk, especially in Spain.

– The results of previous registries and trials cannot be extrapolated to a high-risk population, leaving uncertainty about what proportion of high-risk patients have elevated Hs-TnT and the factors associated with this elevation.

WHAT DOES THIS STUDY ADD?

– The present study evaluated Hs-TnT and the prevalence of elevated Hs-TnT in an asymptomatic Spanish population at very high cardiovascular risk. Elevated Hs-TnT was associated with a history of heart failure and insulin therapy, but not with a history of infarction, angina, or coronary revascularization.

– The results suggest several possibilities related to Hs-TnT: a) It may be an early marker of cardiorenal syndrome. b) It may be a marker of myocardial injury in the asymptomatic population with diabetes. c) It may be a marker of structural heart disease. d) It might be associated with a worse prognosis during follow-up.

REFERENCES


