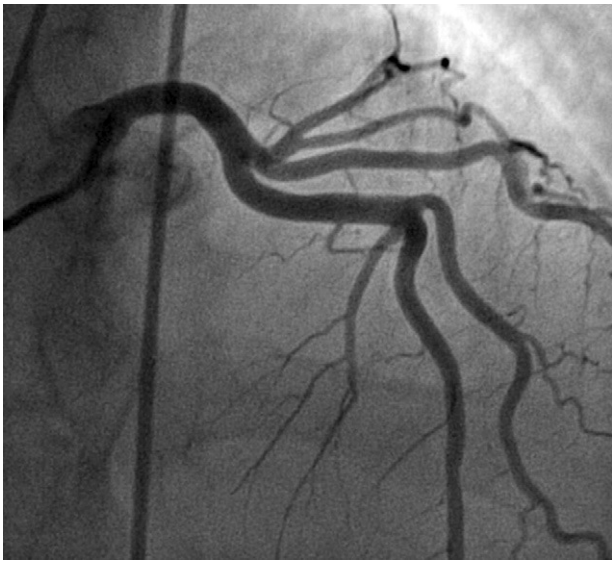

Figure 2. Follow-up coronary angiogram at 7 days. The intracoronary thrombus has disappeared.

venous or arterial thrombosis. The case was discussed with members of the Hematology department, who indicated starting anticoagulation therapy with acenocumarol. Seven days after the infarction, the coronary angiography was repeated and it was confirmed that the thrombus had disappeared (Fig. 2). Three months after the infarction, the patient remained asymptomatic and with improved ventricular function (LVEF, 50%) in the follow-up echocardiography but FVIII levels were still high (210%).

Although the main cause of myocardial infarction is atherosclerotic disease, other causes such as coronary artery thrombosis should be kept in mind, particularly in younger patients. Study of coronary artery disease in younger individuals includes patients younger than 45 years. Our patient was 45 years old, the cutoff age, with no cardiovascular risk factors, and he presented with acute anterior myocardial infarction caused by a thrombus in the left anterior descending artery, with no angiographic evidence of coronary artery disease.

Among coagulation system disorders traditionally associated with arterial thrombosis are hyperfibrinogenemia; protein C, protein S, and antithrombin factor III deficiencies; increased thrombin activity; and mutation of Leiden factor V.[1,2] For several years, FVIII elevation has been included in the list of prothrombotic factors. FVIII is a glycoprotein that acts as a cofactor for factor IX and is essential for thrombus formation. Several studies have shown that elevated FVIII is an independent risk factor for arterial thrombosis (myocardial infarction, ischemic stroke, and peripheral arterial thrombosis), venous thrombosis, and thrombotic recurrence.[3–5] Most patients included in these studies had their first event at a younger age than our patient. It is also known that patients with acute myocardial infarction with elevated FVIII have a worse prognosis. However, the underlying mechanism for this FVIII elevation is not known,[6] and there is little indication in the literature about management of patients who have suffered thrombotic events and how long anticoagulation therapy should be maintained in the absence of a recurrence. We followed the indications of colleagues from the Hematology department, who recommended oral anticoagulation for 6 months.

In summary, FVIII should be included in the study of hypercoagulation of a patient with myocardial infarction and no arteriosclerotic disease.

Verónica Hernández, Nuria Muñoz, M. Antonia Montero, Agustín Camacho, Fernando Lozano, and Vicente Hernández*

Servicio de Cardiología, Hospital General de Ciudad Real, Ciudad Real, Spain

*Corresponding author:
E-mail address: veronicahernandezz@hotmail.com (V. Hernández).

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Diagnostic Implication of the Percentage Change in Troponin I in Normal Range in Patients With Suspected Unstable Angina

Implicación diagnóstica de la variación porcentual de troponina I en el rango de normalidad en pacientes con sospecha de angina inestable

To the Editor,

Unstable angina (UA) is a frequent cause of hospital admission. Risk stratification should be the first step in managing diagnosis and treatment.[1] Nevertheless, despite the existing diagnostic resources, a substantial percentage of the patients that are at high risk according to the most widely used prognostic scoring systems (PSS) still show no evidence of coronary artery disease (CAD) in the angiographic study performed during the hospital stay.[2]

With the aim of providing information to complement electrocardiography (ECG) and PSS in the management of these patients in the scenario described here, we studied whether variations within normal range (NR) in the levels of troponin I (TnI), measured serially in the emergency service using a conventional (as opposed to ultrasensitive) method, predicted the detection of CAD in the hemodynamic study.

For this purpose, we evaluated all the patients admitted to our service with UA in 2010. We included prospectively all the patients with no history of ischemic heart disease and a negative result in serial testing for enzymes indicative of myocardial damage, defined as levels below the 99th percentile of the upper reference limit (URL), associated with intermediate or high risk according to the Thrombolysis in Myocardial Infarction (TIMI) and Global Registry of Acute Coronary Events (GRACE) risk scores, who had undergone coronary angiography during the hospital stay.

The final sample included 78 patients, with a mean age of 64.5 years. The study received the approval of the Ethics and Clinical Research Committee and all the patients signed the informed consent form.

At admission, the patient history and cardiovascular risk factors (CVRF) were recorded, as were the curves of the enzymes TnI and creatine kinase (CK), and the creatine kinase MB isoenzyme (CK-MB). The levels of these markers were determined by immunoassay using the UniCel® DxI 800 analyzer with the Access AccuTnI®, Synchron LX20® and Access CK-MB® kits, respectively (Beckman Coulter, Inc). An ECG finding of ST segment depression greater than 0.5 mm and/or the presence of T wave inversion greater than or equal to 2 mm in 2 or more contiguous leads was considered to be indicative of disease.[3] The sample was divided based on the presence or absence of significant CAD in the hemodynamic study, defined as stenosis greater than 50% in left main coronary artery or greater than 70% in the remaining coronary arteries.

The patients with CAD presented higher peak TnI levels (0.098 ng/mL versus 0.012 ng/mL; $P<.001$; 99th percentile URL, 0.30 ng/mL); the distribution of the CVRF was homogeneous in both groups (Table). The analysis of the diagnostic yield of the percent increase in the TnI level within NR (between minimum and maximum values) for the detection of CAD had an optimal cut-off point of 20%. Thus, the highest percentages in the serial determination had a specificity of 95% and a positive predictive value of 96% in the diagnosis of CAD, with an area under the receiver operator characteristic (ROC) curve of 0.77 (95% confidence interval, 0.67-0.87; $P<.001$) (Fig.) and an overall test accuracy of 70.6%. The positive likelihood ratio was 14.2.

Although to a great extent the ECG changes were able to distinguish the patients with CAD, the additional information provided by analysis of the TnI level significantly improved diagnostic specificity. We should point out that the PSS employed at the time of admission did not identify the presence of significant CAD.

We can conclude that, in the initial evaluation of the patient who is admitted for UA, analysis of the changes in TnI within the NR provides the clinician with supplementary information when establishing early invasive management due to its ability to predict angiographically significant CAD.

Diagnosis of UA is based on the combination of chest pain assessment and ECG changes and the determination of biomarkers. In the presence of the typical signs, with an estimate of intermediate or high risk according to the standard PSS, hospital admission is recommended for the purpose of evaluating the presence of CAD. In centers in which ultrasensitive TnI testing is not available, levels below the 99th percentile URL are considered to be a negative result, without taking into account in clinical practice changes in the concentrations that remain within this range. Previous studies have demonstrated the prognostic value of minor increases in TnI and the potential benefits of an early invasive strategy in patients with UA.[4]

Table
Characteristics of the Sample According to the Presence of Coronary Artery Disease and After Application of the Optimal Cut-off Point of 20%.

Characteristics	Sample	Without CAD	With CAD	*P*
Patients	78 (100)	24 (30.8)	54 (69.2)	—
Age, years	64.5±12.6	60.2±12.3	66.4±12.9	.810
Sex (men/women)	44 (56)/34 (44)	7 (34)/17 (66)	37 (68)/17 (32)	.600
Cardiovascular risk factors				
Hypertension	56 (71)	14 (58)	42 (77)	.070
Diabetes mellitus	30 (38)	8 (33)	22 (40)	.530
Dyslipidemia	51 (65)	17 (70)	34 (63)	.500
Smoking habit	44 (56)	13 (54)	31 (57)	.790
TIMI score	3.19±1.16	3.00±1.06	3.28±1.20	.190
GRACE score	115.21±29.39	107.71±25.72	118.60±30.51	.300
Hemoglobin, mg/dL	13.3±1.7	13.1±1.3	13.4±1.6	.770
Creatinine, mg/dL	1.1±0.8	0.9±0.2	1.1±0.3	.890
Disease-related electrocardiographic changes				
ST-T wave change	34 (44)	5 (21)	29 (54)	.060
Markers of myocardial necrosis				
Peak total CK, U/L	118.1±86.1	11.1±70.7	129.4±114.6	.650
Peak CK-MB, ng/mL	15±9.3	14.8±7.6	15.3±12.3	.110
%↑ CK-MB	11.2±41.2	2.2±31.9	16.3±71.8	.090
Peak TnI, ng/mL	0.071±0.090	0.012±0.005	0.098±0.009	.001
%↑ TnI	94.2±218.4	2.1±10.3	139.4±323.2	.001

Characteristics	Sample	%↑ TnI <20%	%↑ TnI >20%	*P*
TIMI score	2.24±1.20	2.21±1.20	2.27±1.20	.830
GRACE score	115.8±29.4	115.5±26.6	116.1±33.7	.940
ST-T wave change	34 (43)	18 (36)	16 (55)	.110
%↑ CK-MB	12±41.2	8.9±66.5	17.2±56.2	.560
%↑ TnI	94.2±48.4	1.9±0.8	258±78.5	.001
CAD on coronary angiography	54 (69)	26 (53)	28 (96)	.001

CAD, angiographically significant coronary artery disease; CK, creatine kinase; CK-MB, creatine kinase MB isoenzyme; GRACE, Global Registry of Acute Coronary Events; TIMI, Thrombolysis in Myocardial Infarction; TnI, troponin I measured by a conventional (as opposed to ultrasensitive) method; %↑, percent increase in serial measurements performed in the emergency service.
Data expressed as n (%) or mean ± standard deviation.

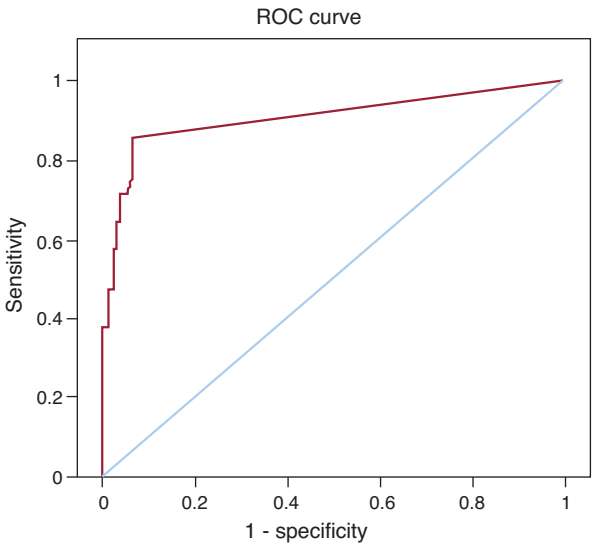

Figure. Receiver operator characteristic (ROC) curve corresponding to the diagnosis of coronary artery disease based on the percent increase in troponin I levels, within normal range, in serial measurements performed in the emergency service (optimal cut-off point, 20%).

While the consideration of TnI levels below the 99th percentile URL increases sensitivity in the diagnosis of CAD, it inherently involves a decrease in the specificity, although this can be improved by the combined analysis of temporal changes in TnI levels[5] and the ECG findings. In our study, the percent change in these levels within NR was found to be an excellent tool for predicting CAD in the hemodynamic study, and it could orient clinicians to the invasive management of these patients.

The limitations of this study lie in the small sample size, the selection of patients who underwent coronary angiography during their hospital stay, and the need for a validation cohort that confirms the hypothesis we propose.

Óscar Fabregat-Andrés,[a,*] Alfonso Valle-Muñoz,[b] Miguel Corbí-Pascual,[b] Mónica Ferrando-Beltrán,[a] Elena Lucas-Inarejos,[a] and Francisco Ridocci-Soriano[a]

[a]*Servicio de Cardiología, Consorcio Hospital General Universitario de Valencia, Valencia, Spain*
[b]*Servicio de Cardiología, Complejo Hospitalario Universitario de Albacete, Albacete, Spain*

[*] Corresponding author:
E-mail address: osfan@comv.es (Ó. Fabregat-Andrés).

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Aortic Stenosis and Porcelain Aorta: Could Percutaneous Valve Implantation Be a Valid Therapeutic Option?

Estenosis aórtica y aorta de porcelana: ¿el implante valvular percutáneo podría ser una opción terapéutica válida?

To the Editor,

In recent years, transcatheter aortic valve implantation (TAVI) has been established as a safe and effective alternative for the treatment of severe symptomatic aortic stenosis in high surgical risk patients.[1,2] The selection of candidates is based on the use of risk scores (the EuroSCORE being that most widely utilized) that assess the associated comorbidities of each patient and quantify the surgical risk on an individual basis.[3,4]

In the population we attend to, certain patients are considered to be inoperable due to comorbidities secondary to the aortic anatomy, such as the existence of porcelain aorta, that are not included among the EuroSCORE variables. There are unconventional surgical alternatives (for example, the apicoaortic conduit), but they have yet to be employed in series of patients with an adequate follow-up period.[2,5,6]

Porcelain aorta is a structural disease of the aortic wall defined as the extensive, circumferential calcification of the thoracic aorta, detected by means of computed tomography (CT) or fluoroscopy.[3] The incidence of porcelain aorta in different series of patients with aortic stenosis treated with TAVI varies, and can be as high as 18%.[2,3] This circumstance and the poor vascular access are the two main reasons for considering transapical access in treatment with TAVI.[3]

Little data has been published on the course of patients with porcelain aorta treated by means of TAVI with the Edwards Sapien

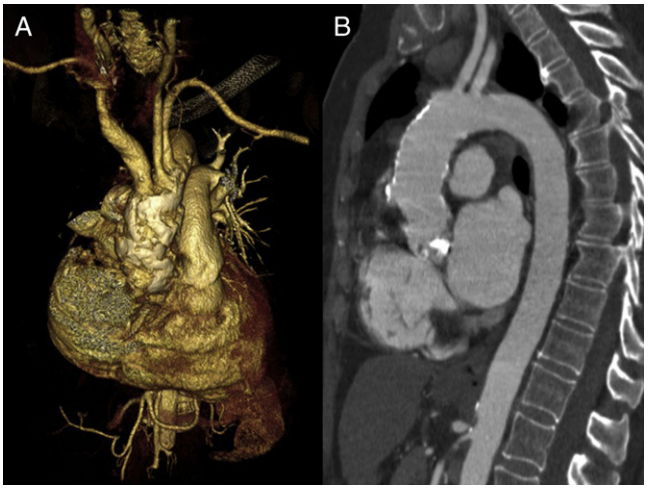

Figure 1. Three-dimensional reconstruction (A) and chest computed tomography (B) showing severe, diffuse, circumferential calcification of porcelain aorta (first case).