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Since the test was positive, upgrade to dual-chamber ICD (5) was successfully implanted on the following days.

The patient was asymptomatic at follow-up, and presented isolated episodes of monomorphic ventricular tachycardia during ICD control, which reversed with ATP.

In these cases, programmed stimulation can be performed from the site where the ventricular lead is implanted, so a conventional EPS with stimulation from the right ventricular outflow tract has to be performed in cases of non-induced arrhythmia from the RV apex.

The timely use of resources available in our daily practice will help us provide adequate follow-up to our patients, with the safest and most appropriate diagnostic and therapeutic options, always by experienced operators.

In addition to its safety and reliability, inductive telemetry completely rules out any complication associated with the puncture site, and avoids radiation exposure received during any invasive method.

Induction of complex and sustained arrhythmias should be performed in the coronary care unit with all the necessary resources to manage possible complications.

In addition to induction of sustained arrhythmias, asynchronous and/or programmed stimulation from implantable devices can be very useful as diagnostic tool, even for the evaluation of the permanent programming mode. Through this means, we can:

- Determine the Wenckebach point in those patients with preferential atrial stimulation (MVP, AAIsafeR, etc.).
- Determine sinus node recovery time to assess sinus function, and consider (or not) the frequency response sensor activation.
- Measure antegrade conduction refractory periods.

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High-Sensitivity Troponin for Prediction of Myocardial Infarct Size in Patients with ST Segment Elevation

The assessment of myocardial lesion size after acute ST-segment elevation myocardial infarction (STEMI) has significant prognostic and therapeutic implications. (1) Different imaging and lab techniques can be used, echocardiography being the most widely employed. High-sensitivity troponin T (hs-Tn) has recently been available in clinical practice, and to date, the information regarding its correlation with infarct size has been discordant. (2, 3)

The purpose of this report is to correlate the level of echocardiographic involvement, measured with the wall motion score index (WMSI), and ejection fraction (EF), with hs-Tn values obtained during the first hours of STEMI.

For this purpose, a retrospective analysis was conducted, including 67 patients admitted for STEMI from May 2012 to January 2013. Patients with previous infarction that could alter WMSI were excluded. STEMI definition was taken from the clinical practice guidelines of the European Society of Cardiology, in its third universal definition of infarction. (4) Infarction was confirmed with hs-Tnl >14 ng measured by the Roche method with a 2010 Elecsys® analyzer. Blood was systematically withdrawn for lab tests on admission and between 6 and 12 hours. Spearman's test was used to correlate troponin with WMSI.

Mean age was 59 ± 10.1 years, and 91% were male patients (Table 1). The mean pain-to-balloon time was 221 (110-311) minutes and, on admission, 91% were in Killip class A or B.

The main culprit vessel was the anterior descending artery in 44.6% of cases, but significant lesions in two or more vessels were found in 71% of patients.

Median hs-Tn on admission was 241 ng/L (27.5-1,350) and 1,965 ng/L (655.2-6,770) at 6-12 hours, with a median EF of 50% (41.5-57.5). When the relationship with WMSI was analyzed, we found that hs-Tn at 6-12 hours had a moderate but significant correlation (r=0.54, p=0.005) (Figure 1). In turn, correlation between hs-Tn on admission and 6-12 h and EF was not significant (p=0.545 and p=0.253, respectively).

Lack of correlation with EF could be explained because several factors are involved in its assessment after STEMI: compensatory mechanism of normal tissue, affected myocardial mass, location, level SCIENTIFIC LETTERS 155

Table 1. Baseline population characteristics

Variable	n = 67
Age, years	59 ± 10.1
Male subjects, %	91
DM, %	11.9
SMK/EXSMK, %	73.1
HFH, %	14.9
HTN, %	59.7
Dyslipidemia, %	64.2
Physical inactivity, %	13.4
Obesity, %	16.4
Systolic blood pressure, mm Hg	127.8 ± 21.7
Diastolic blood pressure, mm Hg	72.7 ± 13.1
Pain-to-balloon time, min	221 (110-311)
Heart rate on admission, bpm	74.8 ± 16.5
Troponin on admission, ng/L	241 (27.5-1350)
Troponin 6-12 hours, ng/L	1965 (655.2-6770)
Creatinine on admission, mg/dl	1.1 ± 0.22
Killip & Kimball on admission, %	
А	68
В	23
С	0
D	9
VT/VF, %	6
Aspiration thrombectomy, %	32.8
IIb/IIIa inhibitors, %	29.9
Culprit vessel, %	
ADA	44.6
RCA	26.2
Cx	29.2
Number of vessels, %	
1	29
2	35.8
3	35.8
Emergency surgery, %	3
Ejection fraction, %	50 (41.5-57.5)
In-hospital mortality, %	3
Hospital stay, days	4 (3-5)

DM: Diabetes mellitus. SMK: Smoker. EXSMK: Ex-smoker. HFH: Hereditary family history. HTN: Hypertension. VT/VF: Ventricular tachycardia/ Ventricular fibrillation ADA: Anterior descending artery. RCA: Right coronary artery. Cx: Circumflex artery.

of involvement of myocardial tissue surrounding the necrotic area, previous ventricular hypertrophy, and associated multivessel disease, among other factors.

It is possible that as a result of this factors Chia Et al. found poor correlation between fourth generation troponin T at 12 hours and EF measured by SPECT five days after the event (r=0.39). (5) In turn, Steen et al. evaluated the correlation of AMI size with magnetic resonance imaging and fourth-generation troponin

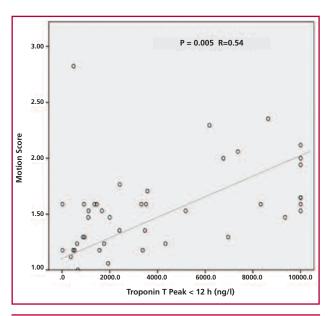


Fig. 1. Correlation between troponin T within 12 hours and echocardiography motion score.

T, measured 96 hours since symptom onset, and found an excellent association for STEMI (r=0.91). (6) Apparently, late troponin values improve correlation with infarct size, since baseline values are influenced not only by the affected myocardium but also by the level of reperfusion and time of ischemia.

The wall motion score index is simple to measure and is less dependent of the injured territory because the amounts of points for each segment is equal regardless of the arterial territory. In this case, hs-Tn improved its predictive ability, supporting the hypothesis that its concentration is associated with the affected myocardial tissue.

Ours is a retrospective study with the risk of bias involved in data collection. The echocardiographic assessment of infarct size was performed with portable equipment at the patient bedside, by operators who were not blind to the patient's condition.

Based on these results, we can conclude that hs-Tn values within 12 hours in patients with STEMI and no history of previous AMI present a regular predictive ability of infarct size assessed by WMSI.

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Simultaneous Percutaneous Closure of Paravalvular Aortic Leak and Aorto-Atrial Fistula Guided by Two-Dimensional Transesophageal Echocardiography

Paravalvular prosthetic leak is a complication of valve surgery caused by degeneration of annular tissue, affecting about 6% to 15% of surgically implanted prosthetic valves and annuloplasty rings. (1)

We report the case of a 75-year-old male patient, who underwent aortic valve replacement with a bioprosthesis (Biocor 27; St Jude®) and myocardial revascularization surgery (mammary artery bypass graft to the anterior descending artery and venous bypass graft to the first diagonal artery). The patient was discharged on the sixth day after uneventful postoperative course. Two months after the procedure, a control transthoracic echocardiography revealed a moderate anterior paravalvular leak (PVL), 5-6 hour of surgical view, and a fistula between the aorta and the right atrium (AO-RA), 8-9 hour of surgical view (Figure 1). There was no clinical, analytic, or echocardiographic evidence of endocarditis. During the 2-year follow-up, the patient had chronic hemolytic anemia and progressive heart failure despite optimal medical treatment; therefore, percutaneous leak closure was suggested.

Under two-dimensional transesophageal echocar-diography (TEE) and angiographic guidance, simultaneous paravalvular leak closure was performed via retrograde aorta with 8 mm Amplatzer Vascular Plug II (St Jude®) device, with immediate minimal residual shunt, and AO-RA fistula was closed with 6 mm Amplatzer Vascular Plug III (St Jude®) (Figure 2).

Today, three years after the percutaneous treat-

ment, the patient is asymptomatic, with no evidence of hemolysis. Control echocardiography showed mild paravalvular leak, with mild increase in transvalvular aortic gradient (mean gradient 24 mm Hg) and preserved left ventricular ejection fraction.

Paravalvular leak after cardiac valve replacement involves abnormal flow through the native tissue and prosthetic valve, due to incomplete apposition of the sewing ring to the native tissue. This is generally a consequence of suture dehiscence. It may develop more commonly in patients with heavy annular calcification, localized infection, or due to technical considerations. (2)

Paravalvular leaks are usually small and asymptomatic, and have a benign course. Larger PVLs -with greater clinical involvement occur in about 1-5% of the patients with prosthetic valves. (3) Biological valves are more commonly involved than mechanical valves.

Patients with significant PVLs present symptoms because blood flow through the valve and subsequent volume overload are associated with reduction of effective cardiac output and congestive heart failure, resulting in decreased exercise tolerance and dyspnea. Symptoms can also be associated with hemolytic anemia, which is caused by red cell fragmentation in the elevated shear stress of the regurgitant jet. Infectious endocarditis can be cause or consequence of PVLs.

Diagnosis is made based on clinical and echocardiographic findings, the latter being very difficult at times. Three-dimensional TEE allows for the visualization of the entire prosthetic valve, increasing the definition and characterization of PVLs. (4)

Reoperation is the treatment of choice in PVLs, either repairing the defect or, most commonly, replacing the valve. It is generally performed in very symptomatic patients due to severe anemia or progressive heart failure, and is associated with elevated morbidity and mortality. It also has the risk of recurrent paravalvular failure. (5)

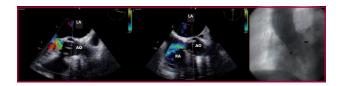


Fig. 1. Aortic (AO) paravalvular leak (left) and fistula to the right atrium (RA) (middle) in transesophageal echocardiography. Paravalvular leak (**) and fistula (*) in angiography (right).



Fig. 2. Post-procedural transesophageal echocardiography and angiography. Paravalvular residual shunt (left).