

Our study has 2 main limitations. The first is possible selection bias, since the IPF was determined in the first morning blood test following hospitalization, and this sample is collected in patients at highest risk. Hence, our results would be valid for this population. The second limitation is the small sample size and limited number of adverse events, which impedes precise adjustment for all the variables potentially related to mortality, and limits the robustness of the results.

Therefore, we conclude that in patients hospitalized for ACS, elevated IPF values determined in the first 24 h following admission are associated with a poorer in-hospital prognosis due to an increase in mortality, even among patients who are not considered at high-risk according to the GRACE score. These patients, who can be identified by standard blood testing, may benefit from more intensive medical treatment or use of a prompt revascularization strategy. Future studies should confirm the association between IPF and mortality and investigate the pathophysiology of these findings.

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Early Anticoagulation May Improve Preprocedural Patency of the Infarct-related Artery in Primary Percutaneous Coronary Intervention

La anticoagulación precoz podría mejorar la permeabilidad de la arteria causante del infarto antes de la angioplastia primaria

To the Editor,

Primary percutaneous coronary intervention (pPCI) is the preferred reperfusion strategy in most patients with ST-segment elevation myocardial infarction (STEMI). In these patients, adjuvant antithrombotic therapy (antiplatelet and anticoagulant) is essential to prevent thrombotic events mediated by platelet activation or the coagulation cascade.¹ In pPCI, various parenteral anticoagulants are used, such as unfractionated heparin (UFH), enoxaparin and bivalirudin. However, the optimal time at which to start anticoagulation therapy in patients with STEMI—whether at diagnosis or at the start of pPCI—is unknown and consequently there are no recommendations on this issue in the current clinical practice guidelines.^{2,3} The aim of this study was to evaluate the impact of early administration of anticoagulation therapy (at diagnosis) compared with its application in the cardiac catheterization laboratory at the start of the procedure on the initial patency of the infarct-related artery (IRA) in patients undergoing pPCI as a reperfusion strategy.

All consecutive patients (between October 2009 and November 2011) admitted to a tertiary center with a diagnosis of STEMI who underwent pPCI were prospectively registered. Patients diagnosed in the tertiary center and in other centers or by the emergency service in their homes and who were transported to the catheterization laboratory for pPCI were included. Patients who had received early enoxaparin therapy were excluded due to their small number and the heterogeneity of doses and routes of administration used. The patients were thus divided into 2 groups according to the treatment received at diagnosis (group 1: those

receiving early UFH; group 2: those not receiving anticoagulation therapy until their arrival at the catheterization laboratory). The glycoprotein IIb/IIIa inhibitors and the anticoagulant employed in the catheterization laboratory (in patients in group 2) were used according to the operator's criteria. Once in the catheterization laboratory, group 1 patients underwent ACT (activated clotting time) determination and could receive an extra dose of UFH to achieve an ACT of 250-350 s (in those receiving glycoprotein IIb/IIIa inhibitors, the ACT target was 200-250 s), while group 2 patients received intravenous UFH doses of 1 mg/kg, except for 8% of the patients, who received bivalirudin. The patency of the IRA was evaluated with the initial Thrombolysis in Myocardial Infarction (TIMI) flow grade dichotomized into 2 arbitrary categories: poor flow (TIMI 0-1) vs good flow (TIMI 2-3). This categorization was used because TIMI grade 2-3 flow allows complete visualization of the distal area of the lesion, facilitating the procedure. The statistical analysis was performed by using a logistic regression model (backward stepwise method), which included initial TIMI flow (dichotomized) as the dependent variable and anticoagulation therapy (groups 1-2) as the independent variable, adjusting by variables considered clinically relevant and baseline characteristics not evenly distributed between the 2 groups ($P < .20$).

A total of 1075 patients were included, of which 95 were excluded because they had received early enoxaparin therapy. Therefore, 980 patients were included in this analysis, divided into the following 2 groups: group 1, n=566 (intravenous UFH 0.75-1 mg/kg), and group 2, n=414. None of the patients received glycoprotein IIb/IIIa inhibitors before reaching the hospital. Among the baseline characteristics (Table), differences were found in the following variables (included in the model): prior acute myocardial infarction, prior PCI, Killip class on admission, clopidogrel loading dose, and the time between activation of the catheterization laboratory to the start of coronary angiography. In the multivariable analysis, the only predictor was the early use of UFH, which was associated with a significantly higher percentage of TIMI 2-3 flow in the IRA (27% vs 16.7%; odds ratio=1.84 [confidence interval,

Table
Characteristics Prior to Primary Percutaneous Coronary Intervention

	UFH (n=566)	Without early AC (n=414)	P
Age, years	61.6±12.4	62.2±13.8	.473
Weight, kg	77.6±13.1	78.2±14.0	.825
Males	450 (79.5)	324 (78.3)	.637
<i>Antecedents</i>			
Diabetes mellitus	133 (23.5)	102 (24.6)	.680
Hypertension	301 (53.2)	234 (56.5)	.299
Dislipidemia	310 (54.8)	230 (55.6)	.807
Active smoking	272 (48.0)	196 (47.3)	.842
Prior stroke	33 (5.8)	32 (7.7)	.221
Prior AMI	50 (8.8)	57 (13.8)	.014
Prior PCI	39 (6.9)	42 (10.1)	.110
Renal insufficiency	27 (4.8)	212 (4.6)	.895
Time (min) from diagnosis-PCI*	73.2±36.3	66.5±37.1	.006
<i>AMI Localization</i>			
Anterior	307 (54.2)	212 (51.2)	
Inferior	251 (44.3)	199 (48.1)	
Other	8 (1.4)	3 (0.7)	
Maximal ST elevation (mm)	3.5±2.1	3.4±1.9	.390
Number of leads with ST elevation	4.3±1.7	4.4±1.7	.699
Killip class on admission (II-IV)	86 (15.2)	89 (21.5)	.071
<i>Origin</i>			
Other center	75 (13.3)	64 (15.5)	
Tertiary hospital	491 (86.7)	350 (84.5)	
ASA	566 (100.0)	412 (99.5)	.098
<i>Clopidogrel loading dose</i>			
Without clopidogrel	5 (0.9)	13 (3.1)	
300 mg	23 (4.1)	64 (16.7)	
600 mg	538 (95.0)	337 (81.4)	

AC, anticoagulation; AMI, acute myocardial infarction; ASA, acetylsalicylic acid; PCI, percutaneous coronary intervention; UFH, unfractionated heparin.

* Time from diagnosis to PCI starts with the diagnosis of ST elevation AMI—when antiplatelet and anticoagulation therapy is started (in patients receiving early therapy) and the system of transferring the patient to the catheterization laboratory is activated—until the beginning of PCI.

1.33–2.55]; $P < .001$), in both cases compared with administration in the catheterization laboratory.

The results of this study show that, in patients with STEMI undergoing pPCI, early administration of parenteral anticoagulation therapy (at diagnosis and before transfer to the catheterization laboratory) was associated with greater patency of the IRA on starting the procedure than initiating anticoagulation in the catheterization laboratory. The most important and novel characteristic of the present study is that it directly compares 2 anticoagulant administration strategies (early administration and administration at the start of pPCI), while previous studies have focused on comparing distinct agents rather than the optimal timing of administration.^{4–6}

We acknowledge the limitations of this study due to its observational design; in addition, the possibility of a selection bias when administering early anticoagulation therapy cannot be excluded. To confirm our results, a randomized study should be performed. Such a study is warranted by the clinical implications of our findings.

CONFLICTS OF INTEREST

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Transfemoral Edwards-SAPIEN XT Aortic Valve Implantation Without Previous Valvuloplasty

Implantación de prótesis aórtica Edwards-SAPIEN XT transfemoral sin valvuloplastia previa

To the Editor,

An 88-year-old woman with diabetes, hypertension, chronic kidney failure (Cockcroft-Gault 42 mL/min), a logistic EuroSCORE of 8% and 2 of the Fried frailty criteria (reversible frailty) was considered unsuitable for surgery by her healthcare center and was referred for transcatheter implantation of an aortic prosthesis¹ for severe symptomatic aortic stenosis.^{2,3} Coronary angiography revealed the absence of lesions and computed tomography showed an iliofemoral axis suitable for transfemoral prosthesis implantation with maximum and minimum aortic annulus diameters of 23.9 mm and 16.5 mm. Transesophageal echocardiography showed substantial degenerative aortic stenosis, with transaortic gradient >80 mmHg, a moderately calcified valve, symmetrical opening and 17-mm annulus.

Vascular access was via the right femoral artery with a 16 Fr delivery catheter. Pre-implantation angiography showed mild-moderate aortic regurgitation and good calcium alignment (Fig. 1). Valve placement was guided by transesophageal echocardiography. A number 23 Edwards-SAPIEN XT prosthesis was advanced, without major difficulty, through the non-predilatated valvular orifice and implantation was performed with 200 bpm overstimulation. Perioperative echocardiography showed optimal prosthesis deployment, without regurgitation, and good placement (Fig. 2). The patient progressed satisfactorily. Follow-up echocardiography showed normal prosthetic function with a maximum gradient of 16 mmHg, without regurgitation.

With a EuroSCORE of 8% and reversible frailty, this patient was considered unsuitable for conventional surgery but suitable for transcatheter implantation,⁴ even though the transesophageal echocardiography-measured annulus diameter was 17 mm (Edwards Lifesciences recommend ≥ 18 mm annulus diameter for Edwards-SAPIEN XT valve implantation). Prior experience with a substantial number of transfemoral prosthesis implantation procedures has shown us that annulus diameter is simply one

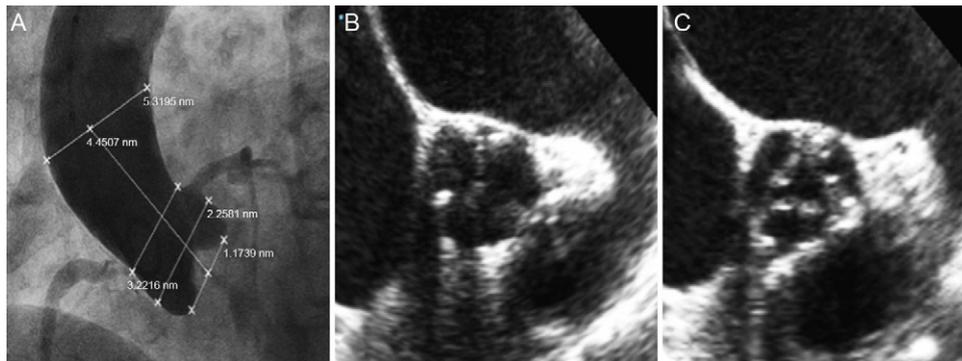


Figure 1. Images prior to percutaneous aortic valve implantation. A: aortogram. B and C: short-axis transesophageal echocardiography at the level of the aortic valve plane that allows visualization of the aortic valve, which is circular with moderate calcification and symmetrical distribution in systole and diastole.

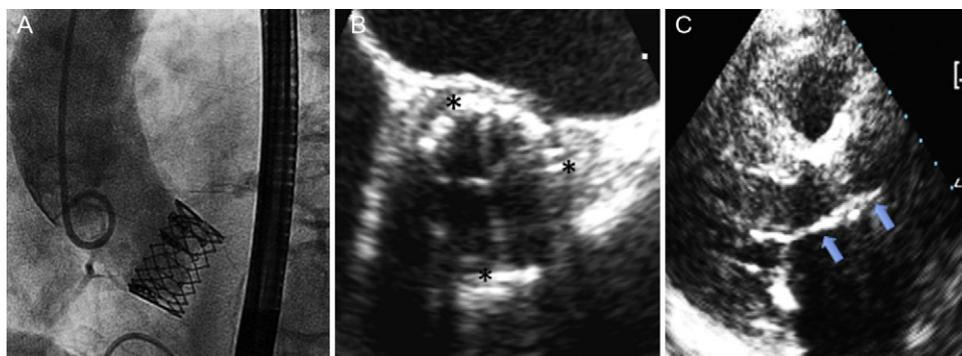


Figure 2. Images following implantation. A: aortogram, with adequate deployment and without aortic regurgitation. B: short-axis perioperative transesophageal echocardiography at the level of the aortic valve plane, with adequate prosthesis expansion (asterisks indicate the valve outline). C: long-axis parasternal follow-up transthoracic echocardiography, with adequate prosthesis implantation (arrows indicate the site of valve implantation).