report and drug prescription information transferred to the primary care physician very likely prolongs adequate patient management, keeping the patient within the "radar" of the health care system.

In conclusion, in a real-life, prospective, all-comers study that enrolled old, frail and vulnerable patients, an early short-term strategy aiming to reduce 30-day readmission rates remained beneficial up to 1-year, largely driven by HF-related readmissions.

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Clinical Characteristics and Prognosis of Very Elderly Patients With Acute Coronary Syndrome Treated With Ticagrelor: Insights From the LONGEVO-SCA Registry

Perfil clínico y pronóstico del paciente muy anciano con síndrome coronario agudo tratado con ticagrelor. Datos del registro LONGEVO-SCA

To the Editor,

Clinical practice guidelines recommend ticagrelor or prasugrel as first line drugs in non–ST-elevation acute coronary syndrome (NSTEMI), and clopidogrel has been relegated to patients with contraindications to these drugs (especially high risk of bleeding).[1] Elderly patients are under-represented in the clinical trials that support these recommendations. Possibly because of that, under-use of these drugs in everyday clinical practice has been described, especially in elderly patients with comorbidities.[2–4] There is very little information on antiplatelet treatment and its impact on geriatric assessment in elderly patients with NSTEMI.

The LONGEVO-SCA registry included patients aged ≥ 80 years with NSTEMI from 44 Spanish hospitals, where the patients underwent an in-hospital geriatric assessment and their 6-month prognosis was analyzed.[5] The primary endpoint of the study was total mortality and its causes at 6 months; secondary endpoints were the readmission, bleeding, and reinfarction rates and new revascularization procedures.

The aim of this analysis was to describe the clinical profile and outcomes in patients who survived to hospital admission, according to whether or not they were prescribed ticagrelor on discharge, excluding patients treated with oral anticoagulants (n = 86). The analysis included total mortality, readmissions, bleeding (BARC 2, 3, or 5) and ischemic events (cardiac mortality, reinfarction, or new revascularization procedures) at 6 months. Cox regression was used for the adjusted analysis, with the variables that showed an association ($P < 0.1$) with either exposure (ticagrelor) or effect: admitting unit, age, previous heart failure, atrial fibrillation, Killip class, hemoglobin, creatinine clearance, invasive management, left main trunk stenosis, revascularization during admission, GRACE, CRUSADE and PRECISE-DAPT scores, and Lawton-Brody, Charlson, nutritional risk, and frailty indexes.

The analysis included 413 patients, 63 of whom (15.2%) received ticagrelor on discharge. These patients were admitted more often to critical care units, were younger, and more often male (Table 1). They had a higher prevalence of atrial fibrillation and bleeding prior to admission. Furthermore, they had slightly lower GRACE scores, with a lower bleeding risk profile. They underwent coronary angiography more often and had a higher percentage of left main trunk stenosis and a higher frequency of percutaneous revascularization.

The patients in the ticagrelor group had a greater capacity for instrumental activities, lower degrees of comorbidity, and a lower prevalence of frailty and nutritional risk.

The incidence of bleeding was low in both groups, with no significant differences (3.2% vs 5.4%). The patients in the ticagrelor group had a slightly lower incidence of ischemic events and a lower incidence of death or readmission (Figure 1). After adjustment for confounding factors, the effect of treatment with ticagrelor was clearly not significant for either ischemic events (hazard ratio [HR] = 0.81; 95% confidence interval [95%CI], 0.33-4.21; $P = .807$) or mortality or readmission (HR = 0.79; 95%CI, 0.37-1.73; $P = .565$).

The findings of this study are in line with those of previous publications and show the low rate of ticagrelor use in elderly patients in our setting,[2] which is inversely proportional to the ischemic and bleeding risk.[3,4]

Some factors limit the robustness of these findings. This was an observational registry, with probable selection bias and unmeasured confounding factors. The small size of the ticagrelor group made it difficult to study the impact of treatment on outcomes. Finally, a longer follow-up would have allowed us to optimize the study of mid-term outcomes, although it is known that the highest risk of bleeding is concentrated in the first months after an event.

Nonetheless, in light of these results, it seems justified to assert that, although the adjusted analysis did not show a clinical benefit, ticagrelor is reasonably safe for selected patients ≥ 80 years, despite their theoretical bleeding risk profile (more than 85% of the ticagrelor group had a PRECISE-DAPT score ≥ 25, considered high

Table
Baseline Characteristics, Treatment and Prognosis According to Ticagrelor Prescription at Discharge

	Ticagrelor at discharge (n = 63)	No ticagrelor at discharge (n = 350)	*P*
Admitting unit			.011
Intensive care	9 (14.3)	20 (5.7)	
Coronary care unit	17 (27)	73 (20.9)	
Cardiology ward	33 (52.4)	221 (63.1)	
Internal medicine	0	22 (6.3)	
Elderly care	0	5 (1.4)	
Other	4 (6.3)	9 (2.6)	
Age. y	82.7 ± 2.6	84.8 ± 4	.001
Male	49 (77.8)	206 (58.9)	.006
Body mass index	27.5 ± 4	26.6 ± 4	.084
Hypertension	53 (84.1)	297 (84.8)	.642
Diabetes mellitus	27 (42.9)	133 (38)	.531
Previous stroke	6 (9.5)	51 (14.3)	.515
Peripheral vascular disease	6 (9.5)	50 (14.3)	.288
Previous myocardial infarction	18 (28.6)	127 (36.3)	.203
Previous heart failure	4 (6.3)	57 (16.3)	.037
Previous atrial fibrillation	1 (1.6)	31 (8.9)	.027
Previous bleeding	1 (1.6)	23 (6.6)	.089
Previous neoplasm	9 (14.3)	58 (16.6)	.612
Killip class ≥ II on admission	12 (19.0)	126 (28.9)	.078
Baseline hemoglobin, g/dL	13.1 ± 2	12.6 ± 2	.081
Creatinine clearance	53 ± 20)	48 ± 20	.042
LVEF, %	56 ± 11	53 ± 12	.191
Invasive management	59 (93.7)	258 (73.7)	.001
Left main trunk stenosis	17 (28.8)	38 (14.7)	.001
Multivessel disease	38 (64.4)	137 (53.1)	.053
Revascularization			.001
No	8 (12.7)	177 (50.6)	
PCI	54 (85.7)	167 (47.7)	
Coronary surgery	1 (1.6)	6 (1.7)	
GRACE score	159 ± 22	166 ± 29	.090
CRUSADE score	36 ± 11	42 ± 13	.001
PRECISE-DAPT score	32.9 ± 10	39 ± 12	.001
Geriatric syndromes			
Disability (Barthel index)			.135
Independent	49 (77.8)	217 (62)	
Mild dependency	12 (19)	94 (26.9)	
Moderate dependency	1 (1.6)	19 (5.4)	
Severe dependency	1 (1.6)	11 (3.1)	
Completely dependent	0	9 (2.6)	
Instrumental activities (Lawton-Brody index)	6.3 ± 2	5.3 ± 3	.001
Comorbidity (Charlson index)	2 ± 1.7	2.5 ± 1.9	.040
Cognitive impairment (Pfeiffer test)			.149
None	49 (77.8)	227 (64.9)	
Moderate	13 (20.6)	112 (32)	
Severe	1 (1.6)	9 (2.6)	
Nutritional risk (MNA-SF)	24 (38.7)	189 (54)	.020
Frailty (FRAIL scale)			.007
No	29 (46)	110 (31.4)	
Pre-frail	27 (42.9)	140 (40)	
Frail	7 (11.1)	100 (22.6)	
Events at 6 months			
Bleeding	2 (3.2)	19 (5.4)	.420
Readmission due to bleeding	0	14 (4)	.087

Table *(Continued)*
Baseline Characteristics, Treatment and Prognosis According to Ticagrelor Prescription at Discharge

	Ticagrelor at discharge (n = 63)	No ticagrelor at discharge (n = 350)	*P*
Required transfusion	0	9 (2.5)	.211
Intervention due to bleeding	1 (1.6)	3 (0.9)	.496
Change in antiplatelet agent	1 (1.6)	13 (3.7)	.326
Fatal bleeding	0	1 (0.3)	.843
Cardiac death, reinfarction, or new revascularization	5 (7.9)	61 (17.4)	.057
Cardiac death	2 (3.2)	26 (7.4)	.168
Reinfarction	4 (6.3)	37 (10.6)	.299
New revascularization	1 (1.6)	20 (5.7)	.138
Total mortality	2 (3.2)	44 (12.6)	.029
Readmission	10 (15.9)	131 (30)	.018
Death or readmission	11 (17.5)	127 (36.3)	.004

LVEF, left ventricular ejection fraction; MNA-SF, Mini nutritional asssessment-Short Form; PCI, percutaneous coronary intervention.
Values are expressed as No. (%) or mean ± standard deviation.

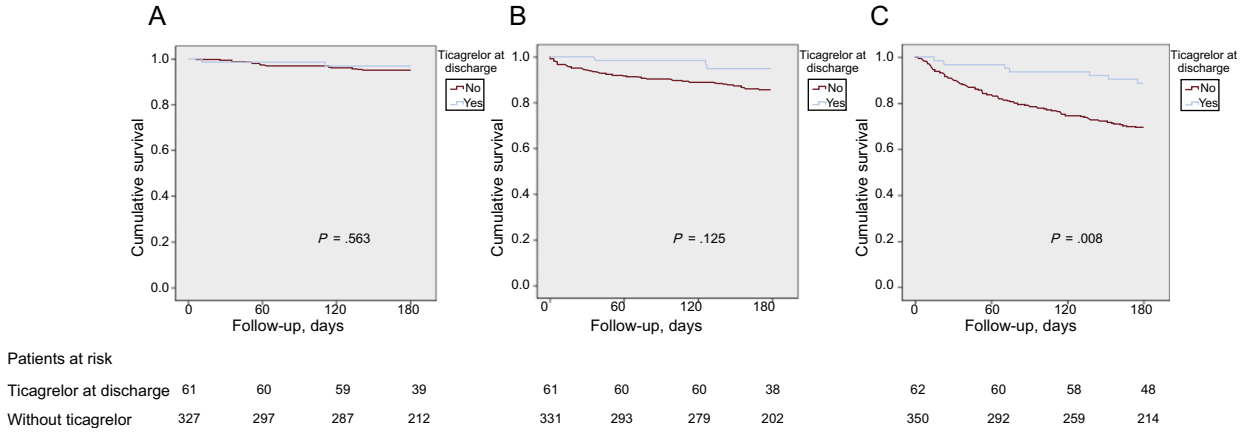

Figure. Cumulative survival free from bleeding events (A), ischemic events (B) and death or readmission (C), according to ticagrelor prescription at discharge.

bleeding risk in the recent guidelines[1]). This patient profile has scarcely been studied yet continues to grow in our everyday clinical practice.

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Mitral Repair as a Treatment of Outflow Tract Obstruction in Hypertrophic Cardiomyopathy: "Myectomy Without Myectomy"

Tratamiento de la obstrucción del tracto de salida en la miocardiopatía hipertrófica mediante reparación mitral: «miectomía sin miectomía»

To the Editor,

Hypertrophic cardiomyopathy is the most common type of cardiomyopathy, with a prevalence of 0.2% in the adult population. The diagnosis is based on finding an increased myocardial thickness of $\geq$ 15 mm that is unexplained by abnormal loading conditions.[1]

Dynamic left ventricular outflow tract obstruction (LVOTO), defined by a peak Doppler gradient $\geq$ 30 mmHg, is a common condition that is found at presentation in a third of patients and is provocable in another third. This phenomenon is produced due to the combined action of septal hypertrophy and anterior systolic motion (ASM) of the mitral valve, which usually has morphological abnormalities. LVOTO increases morbidity and mortality, as it is associated with heart failure, angina, syncope, and sudden death.[1,2]

For patients with significant obstruction and limiting symptoms despite pharmacological treatment, invasive treatment, either surgical or alcohol septal ablation, is the therapeutic option of choice. The classic surgical approach is transaortic myectomy, or Morrow technique, whose results in terms of gradient resolution and symptomatic improvement have been proven extensively. However, the technique is not free from complications, mainly atrioventricular block, ventricular septal defects, and the onset of aortic regurgitation.[2]

Recently, new surgical techniques have been developed that combine myectomy with mitral interventions. Dulguerov et al.[3] described good outcomes using a combined intervention that included transaortic and transmitral myectomy, elongation of the anterior mitral leaflet using a pericardial patch, partial resection of the posterior mitral leaflet, and annuloplasty. Other groups such as that of Ferrazzi et al.[4] reported that performing shallow

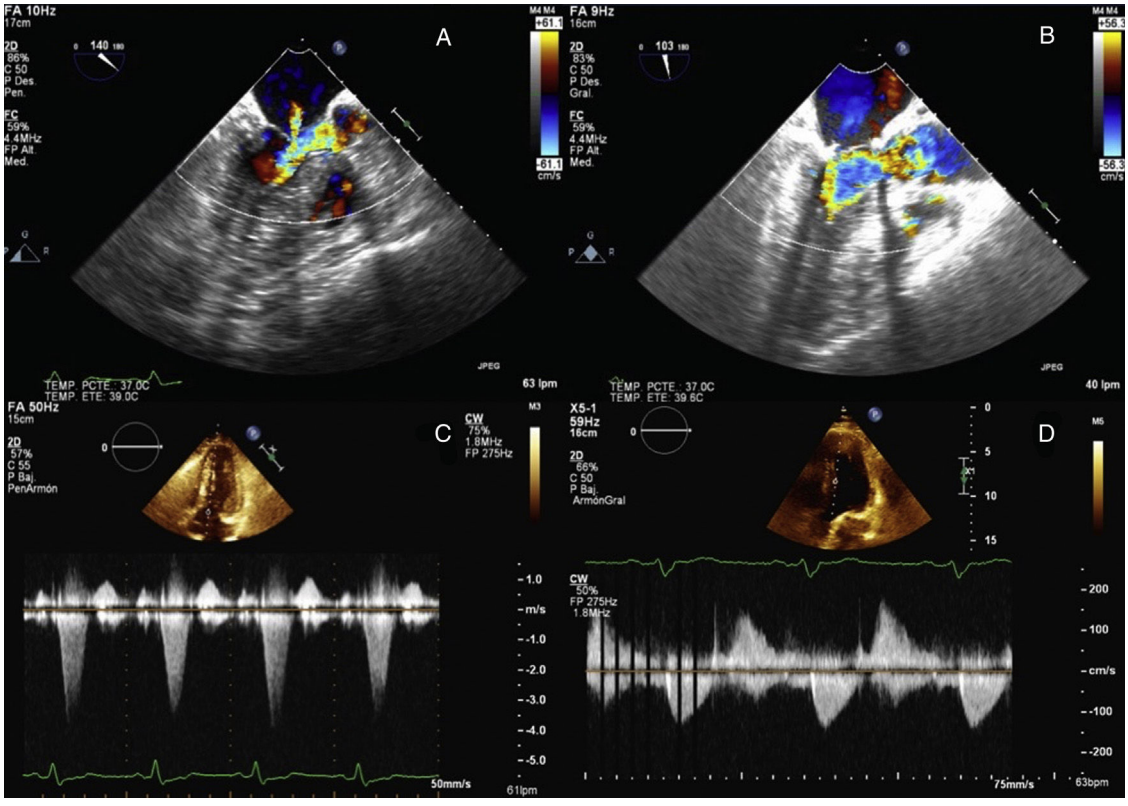

Figure 1. A: transesophageal echocardiogram before surgery; color Doppler of the outflow tract showing obstruction; the arrow indicates mitral regurgitation. B: transesophageal echocardiogram after surgery; color Doppler of the outflow tract showing resolution of the mitral regurgitation and of the obstruction. C: stress echocardiogram before surgery; continuous Doppler of the outflow tract showing a significant gradient. D: stress echocardiogram after surgery; continuous Doppler of the outflow tract showing resolution of the gradient.