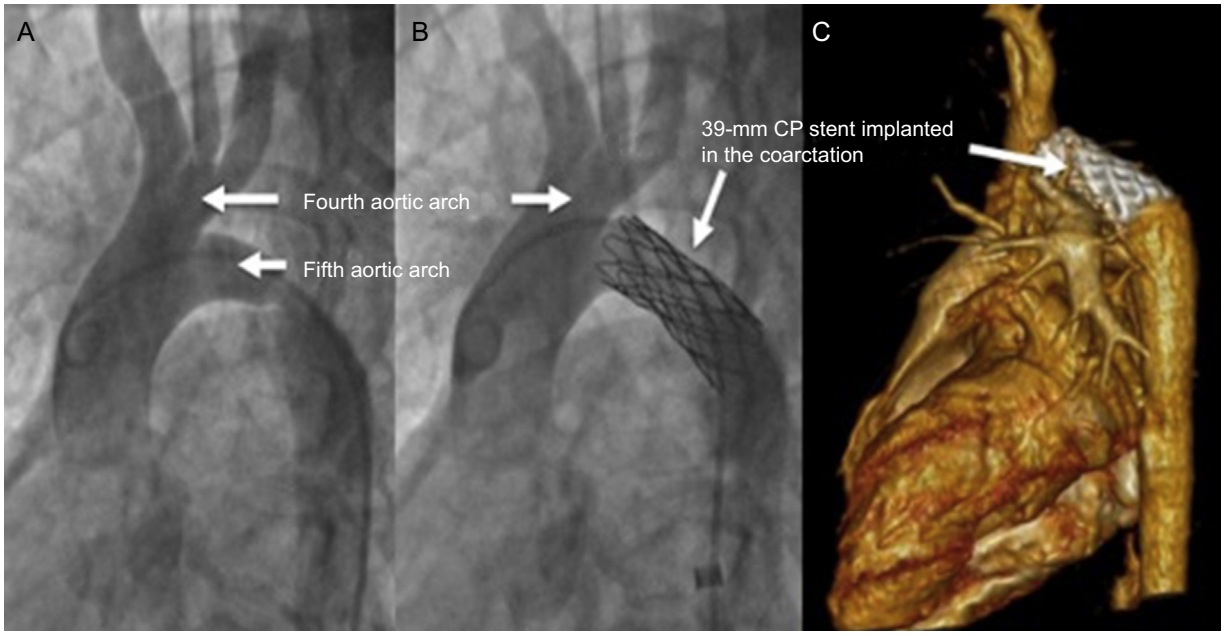

Figure 2. Catheter angiography of the ascending aorta before and after implantation of a 39-mm CP stent at the coarctation site (A y B). Post-intervention computed tomography with contrast (C).

with a 30-mmHg hemodynamic gradient (Figure 2A). The coarctation was repaired by placement of a 39-mm covered CP stent premounted on a 14 × 40 mm Maxi LD valvuloplasty balloon, with overdilation of the distal third of the stent with a 16 × 40 mm Maxi LD balloon. Postprocedure angiography showed a positive outcome, with a residual gradient of 2 mmHg (Figure 2B). The patient was kept under observation for 48 hours before discharge, without incident.

During follow-up at 3 months postimplantation, computed tomography with contrast showed a normally positioned 38-mm stent, beginning 5 mm distal to the single trunk of the ascending aorta that opens to the supra-aortic vessels (Figure 2C). No further intervention was required during 12 months of follow-up.

To our knowledge, this is the first case report of stent placement in a pediatric patient to treat coarctation of a type B persistent fifth aortic arch, associated with interruption of the fourth aortic arch. The patient required no reintervention during the first 12 months of follow-up. We believe that stent angioplasty is a safe and successful treatment for this type of anatomy and should be considered as an alternative to surgery.

Paulo Valderrama,[a,*] Teresa Álvarez,[b] Fernando Ballesteros,[b] Alejandro Rodríguez,[b] and José Luis Zunzunegui[b]

[a]*Servicio de Cardiología Pediátrica, Hospital Luis Calvo Mackenna, Clínica Santa María, Santiago, Chile*
[b]*Servicio de Hemodinámica Pediátrica, Hospital General Universitario Gregorio Marañón, Madrid, Spain*

* Corresponding author:
E-mail address: paulovalderrazo@gmail.com (P. Valderrama).

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Validation of a New Risk Score for Predicting Post-discharge Cardiovascular Events in Patients With Acute Coronary Syndrome

Validación de una nueva escala de riesgo para predecir eventos cardiovasculares poshospitalización en pacientes con síndrome coronario agudo

To the Editor,

In recent years, several studies have shown a high incidence of adverse clinical events following acute coronary syndrome (ACS).[1–4] Abu-Assi et al[1] suggested using a new risk scoring system to predict post-discharge cardiovascular events in patients with ACS. However, as the authors acknowledge, one of the main limitations of their excellent work was the lack of an external validation process. Therefore, to improve the validity of this new scoring system, we assessed its predictive power and discriminatory power in a contemporary cohort of patients with ACS.

We carried out a retrospective study in accordance with the principles of the Declaration of Helsinki. The study was based on the data from a prospective registry of all patients with ACS admitted to a tertiary hospital in Spain. The inclusion period was from January 2012 to September 2014 (n = 1039). The study excluded patients who died in hospital (n = 55), those lost to follow-up (n = 7), and those who had incomplete data to calculate the score (n = 95; 98%

Table 1
Baseline Population Characteristics According to Adverse Events at 1 Year

	Combined events		
	No (n = 805)	Yes (n = 77)	*P*
Demographic data			
Age, y	65 ± 12	71 ± 12	<.001
Sex (women)	180 (22)	21 (27)	.326
Medical history			
Active smoker	284 (35)	15 (20)	.005
Hypertension	566 (70)	64 (83)	.017
Diabetes mellitus	354 (44)	46 (60)	.008
Dyslipidemia	599 (74)	60 (78)	.498
Peripheral vascular disease	50 (6)	15 (20)	< .001
Ischemic stroke	77 (7)	15 (20)	< .001
Ischemic heart disease[a]	224 (28)	25 (33)	.387
Heart failure	26 (3)	6 (8)	.041
Atrial fibrillation/flutter	96 (12)	14 (18)	.112
COPD	62 (8)	10 (13)	.106
Cancer	38 (5)	9 (12)	.016
In-hospital data			
Type of ACS			
NSTEACS	480 (60)	44 (57)	.397
STEACS	282 (35)	26 (34)	
Indeterminate ACS	43 (5)	7 (9)	
Killip > 1 on admission	134 (17)	25 (33)	< .001
Initial heart rate	77 ± 20	83 ± 21	.012
Initial systolic blood pressure	136 ± 27	139 ± 27	.336
Creatinine, mg/dL	1.06 ± 0.47	1.23 ± 0.88	.007
CKD-EPI, mL/min/1.73 m²	63 ± 21	56 ± 23	.009
Hemoglobin, mg/dL	14.1 ± 1.9	13.3 ± 1.9	< .001
LVEF, %	55 ± 12	50 ± 13	< .001
LVEF < 50%	200 (25)	35 (46)	< .001
Coronary angiography	797 (99)	75 (97)	.215
Multivessel disease	385 (48)	48 (62)	.015
Reperfusion strategy			
PCI	626 (77.8)	56 (72.7)	.560
Surgery	50 (6.2)	4 (5.2)	
Mixed	1 (0.1)	0 (0)	
Isolated thrombolysis	3 (0.4)	1 (1.3)	
None	125 (15.5)	16 (20.8)	
Treatment on discharge			
ASA	788 (98)	71 (93)	.019
Clopidogrel	570 (71)	59 (78)	.225
Prasugrel	127 (16)	6 (8)	.065
Ticagrelor	66 (8)	3 (4)	.185
Beta-blockers	737 (92)	69 (91)	.737
Statins	789 (99)	75 (99)	.900
ACEI/ARB	715 (89)	70 (92)	.424
Aldosterone antagonists	133 (17)	21 (28)	.016

ACEI, angiotensin-converting enzyme inhibitors; ACS, acute coronary syndrome; ARB, angiotensin receptor blockers; ASA, acetylsalicylic acid; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration; COPD, chronic obstructive pulmonary disease; LVEF, left ventricular ejection fraction; NSTEACS, non-ST-segment elevation acute coronary syndrome; PCI, percutaneous coronary intervention; STEACS, ST-segment elevation acute coronary syndrome.

[a] Past history of acute myocardial infarction or coronary revascularization, either percutaneous or surgical.

Table 2
Discrimination and Calibration of Scoring in the Total Population and in the Different Subgroups

Population	C-statistic (95%CI)	Hosmer-Lemeshow, *P*
Total population (n = 882)	0.66 (0.63 to 0.69)	.279
NSTEACS (n = 524)	0.67 (0.63 to 0.71)	.135
STEACS (n = 308)	0.66 (0.61 to 0.72)	.784

95%CI, 95% confidence interval; NSTEACS, non-ST segment elevation acute coronary syndrome; STEACS, ST-segment elevation acute coronary syndrome.

due to unknown coronary anatomy). Thus, the final cohort comprised 882 patients. Follow-up at 1 year was carried out by a team of cardiologists and nursing staff, via telephone calls and review of clinical notes. Study events (cardiovascular death, acute myocardial infarction, and stroke) were defined according to the definitions used in the original study by Abu-Assi et al.[1] Causes of death were determined from information obtained from telephone calls to patients' relatives, review of clinical notes, and death certificates. In cases of uncertainty, or if the hospital notes were ambiguous or unavailable, we consulted the death register. Discriminatory power was analyzed by calculating the value of the area under the ROC curve. Calibration of the model was evaluated using the Hosmer-Lemeshow goodness of fit test. Calibration and discriminatory power were calculated for the total population and by subgroup according to type of ACS.

During the first year post-discharge, of 882 patients, 77 (8.7%) had a combined study event: 48 (5.4%) died of cardiovascular causes, 44 (5.0%) had a re-infarct, and 10 (1.1%) had a stroke. Table 1 compares patients with and without adverse clinical events: patients with adverse clinical events were older, had a higher rate of comorbidities (hypertension, diabetes mellitus, peripheral vascular disease, cerebrovascular disease, previous heart failure, and cancer) and had worse Killip class and renal function. They also had lower left ventricular ejection fraction and hemoglobin, and greater extent of coronary disease.

On the new scoring system, individuals with adverse events had a higher score than those without events: 8.01 ± 4.18 points versus 5.64 ± 4.51 points. Furthermore, analysis by category showed that as the risk score increased, the percentage of events also increased progressively (< 3 points, 3.3%; 3-7 points, 7.3%; and > 7 points, 13.5%; $P < .001$). The calibration of this scoring system was good, but the discriminatory analyses showed an acceptable (not excellent) predictive power for cardiovascular events during the first year post-discharge (C-statistics < 0.7) (Table 2).

In this study, we validated for the first time a recently proposed new scoring system for prediction of cardiovascular events during the first year post-discharge from hospital in patients with ACS. In our study, this new scoring system had an acceptable discriminatory power and calibration, in both the total population and in the 2 subgroups analyzed, which indicates its potential clinical usefulness as a stratification tool for patients with ACS in our environment.

Sergio Manzano-Fernández,[a,b,*] Pedro J. Flores-Blanco,[a] Miriam Gómez-Molina,[a] Ángel A. López-Cuenca,[c] María J. Sánchez-Galian,[a] and Mariano Valdés[a,b]

[a]*Servicio de Cardiología, Hospital Clínico Universitario Virgen de la Arrixaca, Instituto Murciano de Investigación en Biomedicina (IMIB), Murcia, Spain*

[b]*Departamento de Medicina Interna, Universidad de Murcia, Murcia, Spain*

[c]Servicio de Medicina Interna, Hospital de la Vega Lorenzo Guirao, Cieza, Murcia, Spain

* Corresponding author:
E-mail address: sergiosmf13@hotmail.com
(S. Manzano-Fernández).

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Percutaneous Transcatheter Treatment for Massive Pulmonary Embolism

Tratamiento percutáneo de la tromboembolia pulmonar aguda masiva

To the Editor,

Massive pulmonary thromboembolism (PTE) is characterized by sustained hypotension or cardiogenic shock, or both, and has high in-hospital mortality. In addition to hemodynamic and respiratory support, treatment includes anticoagulation and systemic fibrinolysis. Thrombolysis is contraindicated in between one third and one half of patients, mainly due to recent major surgery or trauma, etc., and is unsuccessful in approximately 8% of cases.[1] In these situations, the treatment options are surgical embolectomy, in select centers, or alternatively, percutaneous treatment.

In 2013, a protocol was implemented in our hospital for percutaneous intervention in patients with massive PTE and contraindication for thrombolysis. Since then, 24 such patients have been admitted and 5 of them (20%) received percutaneous intervention, performed by the interventional cardiologist (on-call available 24 hours). Prior to 2013, intervention had been performed, sporadically, in 3 patients. Thus a total of 8 patients have received attempted percutaneous treatment. Six patients (75%) had cardiorespiratory arrest with pulseless electrical activity. In 3 patients, the initial suspected diagnosis was cardiogenic shock secondary to acute coronary syndrome, with definitive diagnosis of PTE in the catheterization laboratory; in 2 patients, diagnosis was established by transesophageal echocardiography in the operating room; and in the remaining patients, diagnosis was confirmed on CT angiography. The angiographic and catheterization findings are described in the Table. Six patients had thrombotic occlusion of at least 1 pulmonary branch, and the mean pulmonary systolic pressure was 56 mmHg (standard deviation, 16 mmHg); in 2 patients, the pressures were not recorded due to hemodynamic instability. Five patients received variable doses of thrombolytic, administered as in situ intra-arterial boluses, divided between both pulmonary arteries according to the thrombus size.

Table
Clinical Data, Angiographic and Catheterization Details, Type of Percutaneous Intervention, In Situ Thrombolysis Dose, and Clinical Outcome

	Age	Sex	Diagnostic technique	Thrombolysis contra-indication	Angiographic findings	Preintervention PAP	In situ TL, dose	Transcatheter treatment	Postintervention PAP	Outcome
1	79	Female	CT-angio: bilateral PTE	HI	Bilateral segmental artery thrombus	35/18 (24)	No	No		Asymptomatic (15 months)
2	67	Female	Catheterization suspected ACS	HI	Complete occlusion RPA	Not recorded	Alteplase 25 mg RPA	Balloon fragmentation	Not recorded	In-hospital death due to ICH
3	44	Male	CT-angio: bilateral PTE	Knee surgery	Complete occlusion RPA	70/30 (45)	No	14 F aspiration	40/20 (26)	In-hospital death
4	33	Female	TEE: dilatation/ dysfunction RV	Surgery, hand replant	Occlusion RPA and inferior lobar branches LPA	60/20 (34)	Alteplase 10 mg RPA and 5 mg LPA	8 F aspiration	35/15 (16)	Asymptomatic (6 months)
5	42	Female	TEE: dilatation/ dysfunction RV and RPA thrombus	Surgery, skin graft	Occlusion RPA	51/21 (31)	Alteplase 20 mg RPA	Pigtail fragmentation 8 F aspiration	31/13 (19)	Asymptomatic (2 years)
6	71	Male	Catheterization suspected ACS	No	Bilateral occlusion of RPA and LPA	Not recorded	Alteplase 50 mg PT	8 F aspiration	Not recorded	Died in catheterization laboratory
7	68	Male	CT-angio: bilateral PTE	Hip surgery	Occlusion superior lobar artery	43/18 (26)	No	8 F aspiration	35/15 (22)	Asymptomatic (7 years)
8	70	Male	Catheterization suspected ACS	Hip surgery	Complete occlusion RPA	80/40 (53)	Alteplase 20 mg + 20 mg RPA	Balloon fragmentation	45/25 (31)	Asymptomatic (8 years)

ACS, acute coronary syndrome; CT-angio, computed tomography angiography; HI, head injury; ICH, intracranial hemorrhage; LPA, left pulmonary artery; postinterv. PAP, pulmonary artery pressure; PT, pulmonary trunk, PTE, pulmonary thromboembolism; RPA, right pulmonary artery; RV, right ventricle; TEE, transesophageal echocardiography; TL, thrombolytic.