

4. Sündermann SH, Grünenfelder J, Corti R, Rastanc AJ, Linke A, Lange R, et al. Feasibility of the Engager aortic transcatheter valve system using a flexible over-the-wire design. *Eur J Cardiothorac Surg*. 2012;42:e48-52.
5. Sündermann SH, Holzhey D, Bleiziffer S, Treede H, Falk V. Medtronic Engager bioprosthesis for transapical transcatheter aortic valve implantation. *EuroIntervention*. 2013;9:S97-100.
6. Sündermann SH, Grünenfelder J, Corti R, Rastanc AJ, Linke A, Lange R, et al. Outcome of patients treated with Engager transapical aortic valve implantation: One-year results of the feasibility study. *Innovations (Phila)*. 2013;8:332-6.

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SAME-TT₂R₂ Score Does Not Predict Time in Therapeutic Range in Atrial Fibrillation Patients After Hospitalization for Acute Decompensated Heart Failure



La puntuación SAME-TT₂R₂ no predice el tiempo en rango terapéutico tras un ingreso por insuficiencia cardíaca aguda en pacientes con fibrilación auricular

To the Editor,

The SAME-TT₂R₂ score has been proposed as a way to predict the acceptability of anticoagulation control in patients with nonvalvular atrial fibrillation.¹ This score has been shown to be useful in different cohorts,² but there have been no assessments of its usefulness in patients with recently decompensated heart failure (HF).

Given that atrial fibrillation and HF often occur concurrently and that HF is associated with poor anticoagulation control,¹ we believe that validation of the SAME-TT₂R₂ score in patients with HF could be a useful exercise for clinical practice. Thus, with the aim of extending the validity of this scale, we assessed its predictive and discriminative capacity in a cohort of patients with nonvalvular atrial fibrillation receiving anticoagulation therapy with vitamin K antagonists and with recently decompensated HF.

The study was conducted according to the tenets of the Declaration of Helsinki. The retrospective analysis used data collected for a prospective registry in the cardiology department of a tertiary hospital between January 2008 and September 2011. The study included all patients with nonvalvular atrial fibrillation who received anticoagulant therapy with vitamin K antagonists after discharge following an episode of acute decompensated heart failure. Patients with a contraindication for the new anticoagulants and those with fewer than 2 international normalized ratio (INR) measurements in the 6 months after hospital discharge were excluded (n = 19). The SAME-TT₂R₂ score was calculated at the time of inclusion for all patients. The time in therapeutic range (TTR) was estimated according to the Roosendaal method and poor anticoagulation control was defined as TTR < 65%. A binary logistic regression analysis was conducted to assess the association between SAME-TT₂R₂ score and INR control. Discriminatory capacity was analyzed by calculating the area under the receiver operating characteristic (ROC) curve. The model was calibrated using the Hosmer-Lemeshow goodness of fit test.

In total, 108 patients were included. The median number of INR measurements was 8 (interquartile range [IQR], 6.25-10; range, 3-16). The median SAME-TT₂R₂ score was 2 (IQR, 1-2). Overall, 70% (n = 76) of the patients had an SAME-TT₂R₂ score ≥ 2. The mean estimated TTR during follow-up was 48% ± 24% and 73% (n = 79) had a TTR < 65%. As shown in the Table, of the components of the score, no factor was significantly associated with poor INR control in this population.

The SAME-TT₂R₂ score was similar in patients with TTR ≥ 65% and in those with TTR < 65% (1.9 ± 0.8 vs 2.1 ± 1.0, respectively; P = .415). The percentage of patients with SAME-TT₂R₂ score ≥ 2 did not differ between those with TTR ≥ 65% and those with TTR < 65% (69% vs 75%, respectively; P = .504; Figure). The calibration of the

score was good (Hosmer-Lemeshow test, P = .75). However, in the discrimination analysis, the area under the ROC curve was 0.54 with a 95% confidence interval of 0.42-0.66 and so it was not possible to establish a cutoff to predict INR control.

In our cohort of patients with nonvalvular atrial fibrillation and recently decompensated HF, INR control was poor and the SAME-TT₂R₂ score was not useful for identifying patients with good or poor anticoagulation with vitamin K antagonists. These results contrast with those of a study in which prior HF was not significantly associated with worse INR control in patients with nonvalvular atrial fibrillation (TTR < 65%: 47.4% in patients with HF vs 52.6% in other patients; P = .189).³ However, we believe that it is not history of HF that is responsible for poor anticoagulation control but rather the severity of HF. In our study, all patients had recently decompensated HF, placing them in a high risk group. In view of our results, we believe that these patients should be more closely monitored, or they could be treated with direct oral anticoagulants given the poor anticoagulation control with vitamin K antagonists in most cases. In fact, in large randomized phase III trials with currently available direct anticoagulants, patients with HF were well represented (between 32.0% and 62.5%) and, in the subgroup analysis, there was no statistically significant heterogeneity with respect to efficacy or safety in the treatment of these patients.⁴⁻⁶ Our results show the need for additional validation studies for the SAME-TT₂R₂ score in these patients. One of the main limitations of the study is the small sample size and its retrospective design. The clinical profile of our patients (as in other series of acute HF), with SAME-TT₂R₂ scores > 2 in 70%, can be explained by the low discriminatory capacity in this context. Finally, it would be clinically useful to develop new scoring systems that could identify patients who will have poorly controlled anticoagulation with vitamin K antagonists in this scenario.

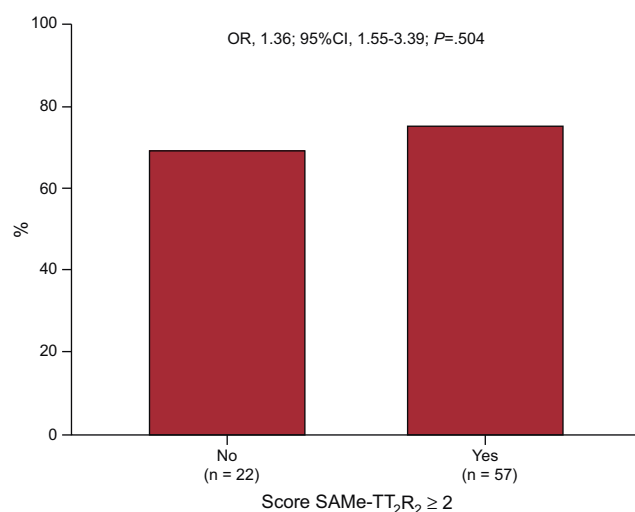


Figure. The percentage of patients with time in therapeutic range < 65% according to the SAME-TT₂R₂ score. CI, confidence interval; OR, odds ratio.

Table

Distribution of Different Variables According to Anticoagulation Quality During Follow-up

Variables	Overall population (n = 108)	TTR		P
		< 65% (n = 79)	≥ 65% (n = 29)	
Female	56 (52)	41 (52)	15 (52)	.987
Age < 60 y	9 (8)	8 (10)	1 (3)	.439
Comorbidity ^a	103 (95)	75 (95)	28 (97)	.723
Hypertension	92 (85)	68 (86)	24 (83)	.667
Diabetes mellitus	38 (35)	27 (34)	11 (38)	.717
Ischemic heart disease	36 (33)	25 (32)	11 (38)	.539
Peripheral artery disease	9 (8)	5 (6)	4 (14)	.246
Symptomatic heart failure	108 (100)	79 (100)	29 (100)	1.000
Left ventricular systolic function ≤ 45%	46 (43)	31 (39)	15 (52)	.245
Right heart failure	9 (8)	7 (9)	2 (7)	.856
Prior stroke or transient ischemic accident	20 (19)	14 (18)	6 (21)	.725
Chronic obstructive pulmonary disease	21 (19)	15 (19)	6 (21)	.843
Liver or kidney disease ^b	11 (10)	10 (13)	1 (3)	.282
Drugs with interactions ^c	25 (23)	19 (24)	6 (21)	.756
Current smoker	13 (12)	10 (13)	3 (10)	1.000
White	108 (100)	79 (100)	29 (100)	1.000
SAMe-TT ₂ R ₂ score, mean ± SD	2.0 ± 0.9	2.1 ± 1.0	1.9 ± 0.8	.415

TTR, time in therapeutic range.

Data are expressed as no. (%) or mean ± standard deviation.

^a History of 2 or more of the following: hypertension, diabetes mellitus, ischemic heart disease, peripheral artery disease, heart failure, stroke or transient ischemic accident, pulmonary disease, and liver or kidney disease.^b Kidney disease defined as creatinine > 2.6 mg/dL, kidney transplant, or hemodialysis. Liver disease defined as cirrhosis, elevated bilirubin twice limit of normal, or elevated transaminases 3 times the limit of normal.^c Medication for rhythm control.

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REFERENCES

1. Apostolakis S, Sullivan RM, Olshansky B, Lip GY. Factors affecting quality of anticoagulation control among patients with atrial fibrillation on warfarin: the SAMe-TT₂R₂ score. *Chest*. 2013;144:1555–63.
2. Esteve-Pastor MA, Roldán V, Valdés M, Lip GY, Marín F. The SAMe-TT₂R₂ score and decision making between a vitamin K antagonist or a non-vitamin K antagonist oral anticoagulant in patients with atrial fibrillation. *Expert Rev Cardiovasc Ther*. 2015;1–11 [Epub ahead of print].
3. Anguita Sánchez M, Bertomeu Martínez V, Cequier Fillat A. Insuficiencia cardiaca, fibrilación auricular no valvular y control de la anticoagulación con antagonistas de la vitamina K. *Rev Esp Cardiol*. 2016;69:75–6.
4. Connolly SJ, Ezekowitz MD, Yusuf S, Eikelboom J, Oldgren J, Parekh A, et al. Dabigatran versus warfarin in patients with atrial fibrillation. *N Engl J Med*. 2009;361:1139–51.
5. Patel MR, Mahaffey KW, Garg J, Pan G, Singer DE, Hacke W, et al. Rivaroxaban versus warfarin in nonvalvular atrial fibrillation. *N Engl J Med*. 2011;365:883–91.
6. Granger CB, Alexander JH, McMurray JJV, Lopes RD, Hylek EM, Hanna M, et al. Apixaban versus warfarin in patients with atrial fibrillation. *N Engl J Med*. 2011;365:981–92.

<http://dx.doi.org/10.1016/j.rec.2016.01.009>