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Predictive role of Selvester QRS score in patients undergoing transcatheter aortic valve replacement

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Abstract

Introduction: Few data exist regarding the late clinical impact of the Selvester score prediction of myocardial fibrosis after transcatheter aortic valve replacement (TAVR). This study evaluated the predictive power of the Selvester score on survival in patients with aortic stenosis (AS) undergoing TAVR.

METHODS AND RESULTS: Patients with severe AS who had preoperative electrocardiograms were included. Clinical follow-up was obtained retrospectively. The primary endpoint was all-cause mortality. Secondary endpoints were cardiovascular death and major adverse cardiac events (MACEs). Two-hundred twenty-eight patients were included (mean age, 81.5 ± 7.4 years; women, 58.3%). Deceased patients had a higher mean score (4.6 ± 3.2 vs. 1.4 ± 1.3; $p < .001$). At a mean follow-up of 36.2 ± 21.2 months, the Selvester score was independently associated with all-cause mortality (hazard ratio [HR], 1.65; 95% confidence interval [CI], 1.48–1.84; $p < .001$), cardiovascular death (HR, 1.59; 95% CI, 1.38–1.74; $p < .001$), and MACE (HR, 1.55; 95% CI, 1.30–1.68; $p < .001$). After 5 years, the mortality risk was incrementally related to the Selvester score. The involvement of the inferior wall of the left ventricle was a lower mortality risk factor (HR, 0.42; 95% CI, 0.18–0.98; $p = .046$). For a Selvester score of 3, the area under the curve showed 0.92, 0.94, and 0.86 ($p < .001$), respectively, for 1, 2, and 3 years.

CONCLUSIONS: Elevated Selvester scores increase the risk of poor outcomes in patients with AS undergoing TAVR. The involvement of the anterior or lateral wall presents worse prognosis.

KEYWORDS

aortic stenosis, mortality, myocardial fibrosis, Selvester QRS score, transaortic valve replacement

1 | INTRODUCTION

Aortic stenosis (AS) occurs in up to 4.5% of the population over 75 years of age, and senile degeneration is the most frequently acquired etiology. Because of an aging population, the incidence of AS is expected to increase in the coming decades.[1] Transcatheter aortic valve replacement (TAVR) is often utilized in elderly patients with high surgical risk. Although well established with numerous proven benefits, the mortality rates of patients undergoing this procedure cannot be neglected. Thus, efforts have been made to identify the patient population profile with a better prognosis after this intervention, and those for whom treatment would be futile, that is, those cases in which the life expectancy is shorter than 1 year.[2-6]

Previous studies have established a correlation between myocardial fibrosis and prognosis in AS.[7-10] Imbalanced neuro-autonomic control, ventricular hypertrophy, and the presence of fibrosis were strong predictors of both all-cause and cardiovascular mortality in a group of elderly patients who underwent TAVR.[11]

In the 1970s, Selvester et al[12] correlated the findings of myocardial biopsies with the standard 12-lead electrocardiogram (ECG) and showed that changes in electrical activity can locate and estimate the sizes of fibrotic areas in patients with ischemic cardiomyopathy. Later studies have compared the correlation of the Selvester QRS score with the late enhancement in cardiac resonance[13,14] and other studies showed that this QRS score had an important prognostic value.[15-18]

In 2015, a small retrospective study reported a correlation between the Selvester QRS score and the final diastolic volume at 6 and 12 months after TAVR in patients who developed left bundle branch block (LBBB) postoperatively. Analysis of this correlation showed that those with high scores had worse left ventricular function at follow-up.[19] However, there is no data on the correlation between the Selvester score and mortality in patients undergoing TAVR. Therefore, we hypothesize that a widespread, noninvasive, and almost inexpensive clinical tool such as the ECG could identify a subgroup of patients with a higher percentage of myocardial fibrosis and would help in risk stratification, improving indications for TAVR procedures.

The current study investigated a possible relationship between the Selvester QRS score in patients who underwent TAVR and the cardiovascular outcomes. The clinical and echocardiographic evolution of these data was analyzed within 96 months of device implantation.

2 | METHODS

2.1 | Patient population

This observational and retrospective study evaluated 228 consecutive patients with severe symptomatic AS who underwent TAVR from January 2009 to May 2016. The institutional heart team determined the indications for TAVR, valve type, access and deployment technique, according to each case characteristics. Procedures were performed as previously described.[20] Clinical outcomes were defined according to the Valve Academic Research Consortium (VARC)-2 definitions.[21] All patients provided written informed consent before undergoing TAVR. The following clinical data were recorded and tabulated: age, gender, functional class (New York Heart Association) and associated comorbidities, data from complete clinical examinations and tests such as ECGs, chest X-rays, laboratory tests, transthoracic echocardiography with aortic complex measurements, computed tomography (CT) of the heart, and aorta and coronary angiography. Transesophageal echocardiography was performed intraoperatively in most patients. This study was approved by the local institutional review board (Ethical Committee approval number 4668/2018) and follow the principles outlined in the Declaration of Helsinki.

2.2 | Electrocardiogram

A baseline ECG was recorded at the hospital prior to the procedure. Patients were assessed using the Selvester QRS score according to the recommendations reported in the most recent publication on the topic.[22] Initially, we categorized QRS complex morphology into six types: LBBB, right bundle branch block (RBBB), left anterior fascicular block (LAFB), LAFB with RBBB, LV hypertrophy, and no confounders. LV hypertrophy was defined as increased voltage according to Sokolow–Lyon or Cornell criteria and not meeting other classifications (see Appendix S1). Next, the amplitude, duration, amplitude ratio, and notch of the Q, R, and S waves were evaluated in each lead. The Leads III or aVR were excluded of scoring. Only the column of the selected morphology type is analyzed. Following each lead has its own scoring criteria (see Appendix S2). A single experienced board-certified cardiologist calculated a score between 0 and 27 for each ECG and the percentage of fibrosis was calculated by multiplying the score by 3, therefore estimating the myocardial fibrotic load. This investigator was blinded to patient outcomes. Three other board-certified cardiologists were selected to review the ECGs, being blinded to patient outcomes.

2.3 | Echocardiography and CT

A standardized comprehensive transthoracic echocardiographic examination was independently performed preoperatively according to established guidelines.[23] Intraoperatively, a transesophageal examination was performed. Images were reanalyzed offline in the workstations.

Images of the heart and aorta were obtained by CT scan to evaluate the annulus dimension and vascular accesses.

2.4 | Coronary angiography

All patients underwent coronary angiography preoperatively. When present, significant coronary artery disease (CAD) was treated with angioplasty at least 1 month before TAVR.

TABLE 1 Baseline clinical characteristics and echocardiography findings of study population

	Total (*n* = 228)	Selvester <3 (*n* = 153)	Selvester ≥3 (*n* = 75)	*p*
Females	133 (58.3)	93 (60.8)	40 (53.3)	.283
Body mass index, kg/m^2	26.5 ± 4.4	26.6 ± 4.4	26.5 ± 4.4	.822
Age, years	81.5 ± 7.4	81.6 ± 7.2	81.3 ± 7.8	.784
Death	57 (25.0)	16 (10.5)	41 (54.7)	<.001
NYHA functional class				
I	4 (1.8)	3 (1.9)	1 (1.3)	.734
II	38 (16.7)	30 (19.7)	8 (10.7)	.088
III	167 (73.2)	109 (71.2)	58 (77.3)	.726
IV	19 (8.3)	11 (7.2)	8 (10.7)	.372
Syncope	53 (23.2)	36 (23.5)	17 (22.6)	.884
Previous MI	49 (21.5)	26 (17.0)	23 (30.6)	.018
Carotid obstruction >50%	55 (24.1)	33 (21.5)	22 (29.3)	.197
Dyslipidemia	169 (74.1)	109 (71.2)	60 (80.0)	.155
Smoker	51 (22.4)	37 (24.1)	14 (18.6)	.347
Hypertension	198 (86.8)	127 (83.0)	71 (94.6)	.014
sPAP >55 mmHg	75 (32.9)	40 (26.1)	35 (46.6)	.001
eGFR, ml/min	44.48 ± 19.27	45.40 ± 18.73	42.47 ± 20.36	.291
eGFR <50 ml/min	122 (53.5)	78 (51.0)	44 (58.6)	.274
CAD >50%	113 (49.6)	70 (45.7)	43 (57.3)	.100
Previous stroke	23 (10.1)	16 (10.4)	7 (9.3)	.791
Atrial fibrillation	43 (18.9)	25 (16.3)	18 (24.0)	.164
Diabetes	89 (39)	54 (35.3)	35 (46.6)	.098
Insulin use	24 (10.5)	16 (10.4)	8 (10.7)	.961
COPD	34 (14.9)	23 (15.0)	11 (14.6)	.941
Porcelain aorta	16 (7)	10 (6.5)	6 (8.0)	.684
CABG	44 (19.3)	28 (18.3)	16 (21.3)	.585
Previous PCI	68 (29.8)	42 (27.4)	26 (34.6)	.263
Previous ABV	22 (9.6)	12 (7.8)	10 (13.3)	.187
EuroSCORE II	7.4 ± 5.8	6.9 ± 5.5	8.3 ± 6.4	.095
STS mortality	6.5 ± 6.2	6.1 ± 4.2	7.4 ± 9.0	.128
Follow-up, month	36.1 ± 21.2	39.4 ± 19.9	29.4 ± 22.2	<.001
Echocardiography findings				
Lad, mm	43.7 ± 5.5	43.2 ± 5.4	44.7 ± 5.6	.054
LV diameter, mm	49.6 ± 7.0	49.3 ± 6.5	50.2 ± 8.1	.345
LVEF, %	59.8 ± 11.4	61.0 ± 10.8	57.4 ± 12.1	.030
LVEF <50%, *n*	40 (17.5)	20 (13.1)	20 (26.6)	.011
LVEF <30%, *n*	5 (2.2)	2 (1.3)	3 (4.0)	.192
sPAP, mmHg	41.6 ± 14.8	40.2 ± 13.9	44.5 ± 16.2	.037
Ao VA pre, cm^2	0.7 ± 0.2	0.7 ± 0.2	0.7 ± 0.2	.961
Maximum SG, mmHg	65.4 ± 28.7	67.1 ± 30.1	61.8 ± 25.4	.149
Medium SG, mmHg	52.9 ± 15.8	53.8 ± 16.2	51.1 ± 15.2	.211
MR +3 and +4, *n*	30 (13.1)	19 (12.4)	11 (14.6)	.637

Note: Values are expressed as the mean ± *SD*, *n* (%) for categorical data.

Abbreviations: ABV, aortic balloon valvuloplasty; Ao VA pre, aortic valve area previous; CAD, coronary artery disease; CABG, coronary artery bypass grafting; COPD, chronic obstructive pulmonary disease; eGFR, glomerular filtration rate; EuroSCORE, European System for Cardiac Operative Risk Evaluation; Lad, left atrial diameter; LV, left ventricle; LVEF, LV ejection fraction; MI, myocardial infarction; MR +3 and +4, mitral regurgitation moderate and severe; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; SG, systolic gradient; sPAP, systolic pulmonary artery pressure; STS, Society of Thoracic Surgeons.

TABLE 2 Selvester QRS score data

	Total (*n* = 228)	Death (*n* = 57)	Survivor (*n* = 171)	*p*
Selvester score, *n* (%)	2.2 ± 2.4	4.6 ± 3.2	1.4 ± 1.3	<.001
Selvester score, %	6.6 ± 7.3	13.9 ± 9.7	4.2 ± 3.9	<.001
Anterior wall, *n* (%)	129 (56.6)	43 (75.4)	86 (50.3)	<.001
Lateral wall, *n* (%)	25 (11)	11 (19.2)	14 (8.1)	.020
Inferior wall, *n* (%)	36 (15.8)	13 (22.8)	23 (13.4)	.093
Conduction, *n* (%)				
LVH	106 (46.5)	21 (36.8)	85 (33.9)	.091
No confounder	64 (28.1)	23 (40.3)	41 (24.0)	.017
LBBB	37 (16.2)	8 (14.0)	29 (17.0)	.604
RBBB	15 (6.6)	3 (5.2)	12 (7.0)	.643
LAFB	4 (1.8)	1 (1.7)	3 (1.7)	1.0
RBBB + LAFB	2 (0.9)	1 (1.7)	1 (0.5)	.412

Note: Values are expressed as the mean ± *SD*, *n* (%) for categorical data.
Abbreviations: LAFB, left anterior fascicular block; LBBB, left bundle branch block; LVH, left ventricle hypertrophy; RBBB, right bundle branch block.

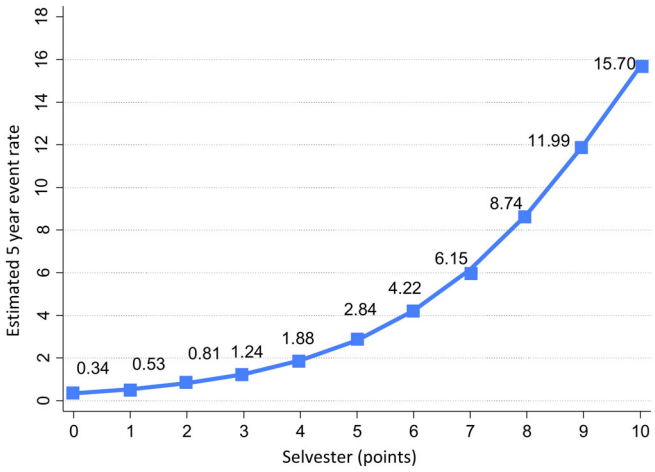

FIGURE 1 The predicted 5-year event rate relative to the Selvester QRS score points to the risk of all-cause mortality [Color figure can be viewed at wileyonlinelibrary.com]

2.5 | Follow-up

The same medical team followed all patients at prespecified time points: 1-month visit and then annually thereafter. The cause of death was categorized as cardiac or noncardiac. The primary endpoint was all-cause mortality; the secondary endpoint was cardiovascular death and major adverse cardiac events (MACEs; per VARC-2 criteria). Patients who died within 30 days postprocedure were excluded.

2.6 | Statistical analysis

Statistical analyses were performed using SPSS version 21 (IBM SPSS Statistics, IBM Corporation, Armonk, NY) and Stata release 14 (StataCorp, College Station, TX). Continuous variables were expressed as the mean ± *SD*, and categorical variables as counts and percentages. The Mann–Whitney and student's *t* tests for independent variables were used to compare continuous variables, and Fisher's exact or chi-square test were used for comparison of categorical variables. Survival rates were summarized using Kaplan–Meier estimates, and log-rank tests were used to compare groups. The index date was the date of the procedure. Student's *t* test was used to evaluate the difference on the Selvester QRS score mean regarding different outcomes. All clinical parameters were proposed for inclusion in a univariate Cox proportional hazards model, and all significant ($p < .10$) univariate correlates of survival were entered into a forward stepwise multivariate Cox model (cumulative outcomes). Receiver operating characteristic (ROC) curves were used to determine the sensitivity and specificity of the Selvester QRS score (empirical–nonparametric–ROC curve) as a predictor of all-cause mortality, and the area under the curve (AUC) was calculated to compare diagnostic efficiency with 95% confidence intervals (CIs). The incremental value of the percentage of fibrosis estimated by the Selvester QRS score for predicting the 5-year mortality risk was obtained through the AUC, integrated discrimination improvement, and net reclassification improvement. The CI was calculated and internally validated. The interobserver reproducibility of the Selvester QRS score was evaluated using an intraclass correlation reproducibility and 30% of the ECGs were randomly selected for this analysis. The interobserver agreement was evaluated by the *k* test.

3 | RESULTS

3.1 | Patients

Table **1** shows the main clinical characteristics and the echocardiographic findings of the study population. The mean age of the study

TABLE 3 Univariate and multivariate predictors of all-cause death, cardiovascular death and MACE after transcatheter aortic valve replacement

	All-case death				Cardiovascular death				MACE			
	Univariate HR		Multivariate HR		Univariate HR		Multivariate HR		Univariate HR		Multivariate HR	
	(95% CI)	*p*	Adjusted HR (95% CI)	*p*	(95% CI)	*p*	Adjusted HR (95% CI)	*p*	(95% CI)	*p*	Adjusted HR (95% CI)	*p*
Age, years	1.04 (1.00–1.09)	.056	1.02 (1.00–1.06)	.052	1.031 (1.003–1.098)	.033	1.052 (1.003–1.104)	.039	1.02 (1.01–1.05)	.049	1.026 (1.01–1.09)	.042
Male	1.58 (0.93–2.70)	.093	1.44 (0.98–1.99)	.108	1.01 (0.18–4.18)	.996	1.00 (0.44–2.25)	1.000	1.26 (0.95–1.78)	.126	1.04 (0.53–3.66)	.896
Dyslipidemia	2.36 (1.11–5.01)	.025	2.21 (1.06–5.19)	.032	2.18 (1.10–4.92)	.028	2.69 (1.24–5.87)	.013	2.01 (1.06–5.02)	.036	2.03 (1.18–4.99)	.031
STS mortality	1.02 (1.00–1.05)	.044	1.02 (1.01–1.06)	.049	1.00 (0.96–1.05)	.615	0.99 (0.95–1.04)	.813	1.08 (1.01–1.16)	.059	1.01 (0.92–1.12)	.112
Clinical success	0.33 (0.13–0.83)	.018	0.86 (0.53–0.96)	.048	0.92 (0.66–1.03)	.051	0.23 (0.09–0.59)	.002	0.80 (0.50–0.92)	.041	0.91 (0.66–0.98)	.048
Ao VA pre	0.21 (0.04–1.20)	.078	0.19 (0.04–1.12)	.073	1.12 (0.12–12.01)	.732	1.19 (0.09–15.22)	.891	0.38 (0.12–1.21)	.086	1.16 (0.18–11.12)	.689
sPAP	1.02 (1.00–1.03)	.057	1.01 (1.00–1.03)	.054	0.98 (0.95–1.03)	.499	0.99 (0.96–1.02)	.433	1.06 (1.01–1.13)	.058	0.96 (0.91–1.09)	.387
Atrial fibrillation	2.12 (1.18–3.82)	.012	2.69 (1.26–3.39)	.023	1.36 (0.68–4.12)	.860	1.28 (0.50–3.29)	.610	1.89 (1.12–3.01)	.038	1.66 (0.88–6.88)	.433
eGFR	0.98 (0.96–1.00)	.038	0.98 (0.96–1.00)	.038	1.02 (0.97–1.05)	.501	1.01 (0.98–1.05)	.541	0.95 (0.86–1.02)	.062	1.05 (0.95–1.12)	.667
Selvester (*n*)	1.54 (1.42–1.68)	<.001	1.49 (1.39–1.65)	<.001	1.59 (1.38–1.74)	<.001	1.65 (1.48–1.84)	<.001	1.46 (1.38–1.60)	<.001	1.55 (1.30–1.68)	<.001
Anterior wall	2.63 (1.44–4.83)	.002	2.53 (1.30–4.93)	.006	1.41 (0.50–4.00)	.516	0.96 (0.42–2.18)	.916	2.54 (1.31–4.94)	.007	1.45 (0.59–3.18)	.345
Lateral wall	2.53 (1.30–4.93)	.006	2.63 (1.44–4.83)	.002	0.96 (0.42–2.18)	.916	1.41 (0.50–4.00)	.516	2.60 (1.40–4.86)	.009	0.93 (0.39–1.88)	.826
Inferior wall	2.01 (1.08–3.76)	.028	1.89 (1.06–2.99)	.024	0.66 (0.28–0.97)	.041	0.42 (0.18–0.98)	.046	1.88 (1.12–2.77)	.022	0.60 (0.44–0.99)	.048
Conduction (ref. = LVH)						.914		.962				.820
No confounder	2.54 (1.39–4.66)	.002	2.11 (1.12–3.89)	.013	1.12 (0.55–2.55)	.799	1.08 (0.44–2.66)	.862	2.10 (1.10–3.80)	.026	1.26 (0.82–2.89)	.701

Abbreviations: Ao VA pre, aortic valve area previous; CI, confidence interval, eGFR, glomerular filtration rate; HR, hazard ratio; LVH, left ventricle hypertrophy; sPAP, systolic pulmonary artery pressure; STS, Society of Thoracic Surgeons.

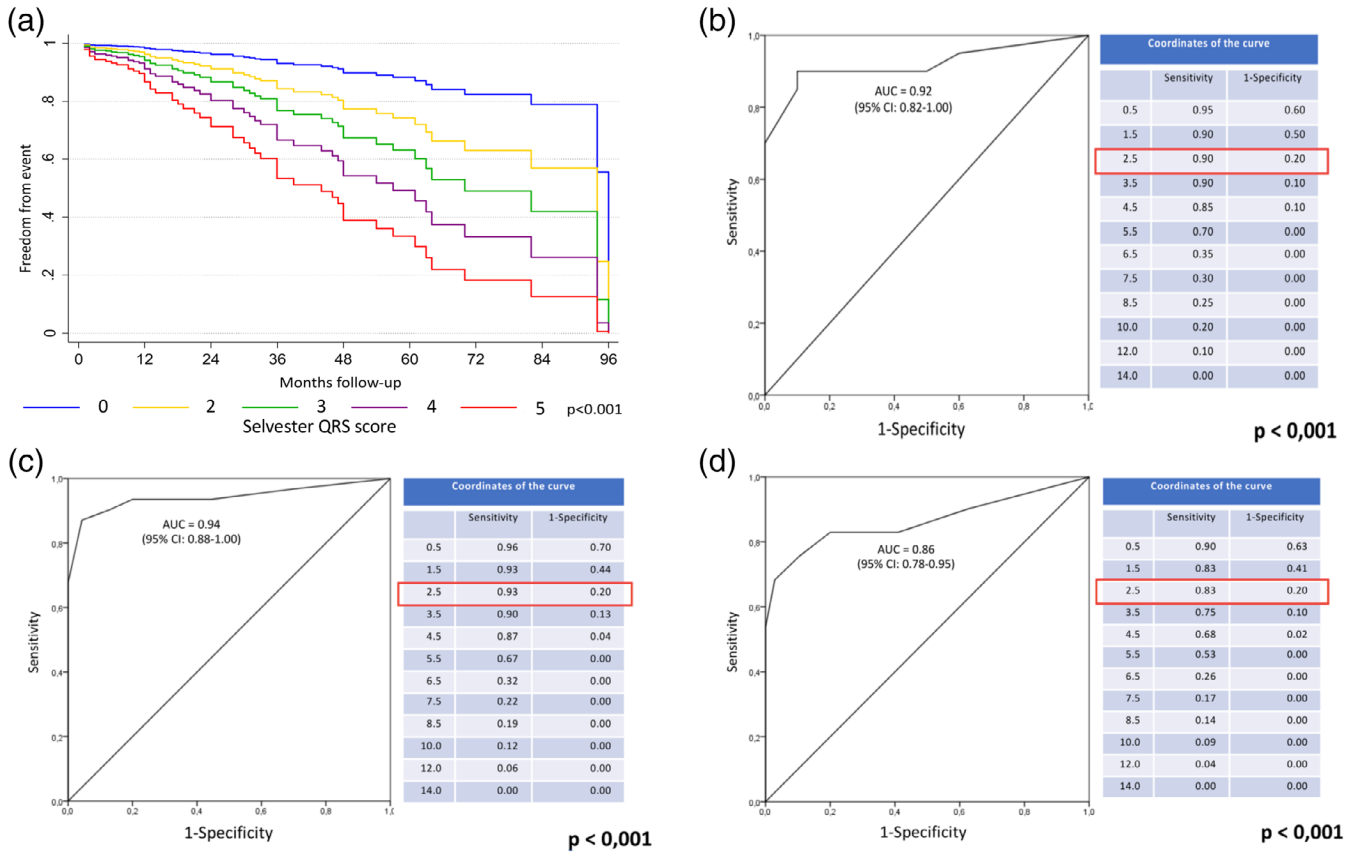

FIGURE 2 Relationship between the Selvester QRS score for fibrosis and all-cause mortality in 228 patients (a). The hazard plot is based on multivariable Cox regression analysis. The receiver operating characteristic curves for all-cause mortality show that the value with the best accuracy is 2.5 for 1, 2, or 3 years. Because the Selvester QRS scores are not fractional, Score 3 is the one that represents this point in the curve. The area under the curve shows 0.92 (b), 0.94 (c), and 0.86 (d) ($p < .001$), 1 year (b), 2 years (c) and 3 years (d) mortality [Color figure can be viewed at wileyonlinelibrary.com]

population was 81.5 ± 7.4 years, 58.3% were woman, and 75 (32.9%) patients had a Selvester QRS score of 3 or higher. This group with elevated Selvester QRS scores had a higher mortality rate (54.7% vs. 10.5%; $p < .001$), higher prevalence of previous myocardial infarction, hypertension, and systolic pulmonary arterial pressure (sPAP) over 55 mmHg. The echocardiographic findings indicated that this group had a lower mean ejection fraction, higher mean sPAP, and higher prevalence of left ventricle (LV) systolic dysfunction.

At a mean follow-up of 36.2 ± 21.2 months that extended to 96 complete months, 57 (25%) patients had died.

3.2 | Selvester QRS score and mortality

Table 2 shows the Selvester QRS score data of the study population. The mean score was 2.2 ± 2.4; among the patients who died, the mean was significantly higher (4.6 ± 3.2 vs. 1.4 ± 1.3; $p < .001$). The anterior wall was the most affected, considering the population as a whole. Among patients who died, the anterior and lateral walls were the most affected. According to the definition of intraventricular conduction of the Selvester QRS score, in our study population, the highest prevalence was LV hypertrophy followed by no confounder (i.e., patients without intraventricular conduction block as well as LV hypertrophy). Among the patients who died, the group of patients considered no confounder by Selvester QRS score was more prevalent (40.3 vs. 24.0%; $p = .017$).

3.3 | Cumulative survival analysis and Selvester QRS score

Analysis of the outcomes showed that the mean survival time estimated by Kaplan–Meier was about 6 years (70.4 months, 95% CI, 64.8–76.0). Patients with higher Selvester scores had a higher incidence of mortality (1.40 ± 1.33 vs. 2.20 ± 2.40; $p < .001$), cardiovascular death (1.93 ± 2.04 vs. 5.26 ± 3.89; $p < .001$), and MACE (1.75 ± 1.91 vs. 2.97 ± 2.97; $p < .001$).

Univariate Cox analysis (Table 3) showed that high Selvester QRS score and no confounder conduction, as well as dyslipidemia, STS mortality, and presence of atrial fibrillation, increased the risk of all-

cause mortality in these patients. Clinical success and elevated creatinine clearance decreased the risk. In the presence of fibrosis, the involvement of the anterior wall was associated with higher risk, whereas involvement of the inferior wall was associated with a lower risk. Predictors of cardiovascular death and MACE showed similar results to all-cause mortality, except for clinical success.

The adjusted risk of all-cause death increased continuously and directly in relation to the Selvester QRS score, with each additional point associated with a 65% increase in risk. Over 5 years, the risks of death ranged from 0.53 to 2.84% in the Selvester score ≤5–2.84% to 15.70% of the Selvester score >5, and the absence of the Selvester score (0 score) was associated with a risk of 0.34% death in 5 years (Figure 1).

Clinical success (per VARC-2 criteria) reduced the event risk by 77% and when the other variables were the same, the occurrence of fibrosis only in the inferior wall reduced the risk by 58%. The survival Kaplan–Meier model is shown in Figure 2 at the 96-month follow-up time point for different Selvester QRS scores. Additionally, we performed a subanalysis evaluating the impact of Selvester QRS score according to the STS risk score and also according to gender, as previous studies suggest an increased risk in women[9,24] (please see Appendix S3).

The ROC curves for mortality showed that the value with the best accuracy to differentiate those patients who will and will not die, that is, the best combination of sensitivity and specificity, was 2.5 for 1, 2, or 3 years. Because the Selvester QRS score does not show fractional results, Score 3 represents that point in the curve. The AUC shows 0.92, 0.94, and 0.86 ($p < .001$), respectively, for 1, 2, and 3 years (Figure 2).

3.4 | Reproducibility

The interobserver and intraobserver agreement values (30% of ECG's were randomly selected for this correlation analysis) were high for the Selvester QRS score (interobserver, $R = .97$, $p < .01$, and coefficient of variation, 6.6%; and intraobserver, $R = .98$, $p < .01$, and coefficient of variation, 6.3%).

4 | DISCUSSION

The main finding of this is study is that the Selvester score, an easy and inexpensive ECG-based score related to myocardial fibrosis, could help in risk stratification of AS patients undergoing TAVR.

Although TAVR has proven benefits even in patients with low surgical risk,[25,26] an overall assessment is important. Prognostic factors have been investigated in patients who undergo percutaneous aortic prosthesis implantation.[1,7-10] However, because much of the data reflect laboratory abnormalities or comorbidities already used in prognostic surgical risk scores, they add little value for TAVR patients. The current study evaluated the estimation of fibrosis using the Selvester QRS score in patients with severe AS who underwent TAVR. We analyzed mortality and combined outcomes in the 228 cohort patients, with a long-term follow-up period up to 96 months.

The mean age of the study population was 81.5 years, similar to the main international cohorts.[27] The prevalence of comorbidities such as diabetes mellitus, CAD, carotid disease, stroke, and chronic obstructive pulmonary disease (COPD) was also similar to those studies and did not differ in the current sample among those with low scores (<3 points) and those with higher scores. The rates of prior myocardial infarction, hypertension, and sPAP exceeding 55 mmHg were similar to those reported in the literature, but differed significantly between the group with scores below three points and that with three points or higher.[28,29] In our study population, only dyslipidemia, age, the occurrence of clinical success according to VARC statement and the Selvester score were independent prognostic factors in the multivariate analysis. The surgical risk scores in the current study were somewhat lower than those found in the large samples but could still be considered high.[27,30] Female sex was associated with higher rates of major bleeding when undergoing TAVR, but data on gender-related mortality are conflicting.[9,24] In our population study, gender was not a predictor of all-cause mortality, cardiovascular mortality or MACE.

The analysis of the surgical scores has some prognostic accuracy, as shown in the PARTNER study cohort.[3,30] In the current study, higher STS scores were correlated with higher Selvester QRS scores; however, when adjusted by multivariate analysis, they did not prove to be an independent predictor of events. It should also be considered that these scores are not specific for valvular heart disease, and they use general data from patients with various comorbidities, which directly or indirectly affect surgical outcomes. Additionally, clinical peculiarities are not always contemplated in these scores.

The amount of myocardial fibrosis is not a component of traditional surgical risk scores, and in the literature some studies have reported a correlation between the amount of fibrosis and mortality, both in other heart diseases[17,18,30] and also in AS.[31] Other publications have shown that the Selvester QRS score was well correlated with the estimation of fibrosis and cardiac imaging studies.[32-36] Other publications have shown that use of the Selvester QRS score should be used with caution in patients with low LV ejection fraction and LBBB.[37] In patients with low pretest probability of myocardial scar, Selvester QRS score requires greater clinical evidence.[38] In the current study population, the calculation of the Selvester QRS score, which estimates the area of myocardial fibrosis, was correlated with increased mortality and was higher in patients who died. For each score point, there was a 65% increase in the patient's risk of death. When cardiovascular death was assessed, the patients who died had higher Selvester scores, and the same was true for MACE.

Echocardiographic variables showed that the group with scores of ≥3 had lower mean left ventricular ejection fraction and higher mean sPAP. The percentage of patients with LV systolic dysfunction was also higher in the group with a score of 3 or higher. Other studies have also shown a correlation between the percentage of fibrosis and systolic dysfunction.[17,18]

After adjustment for multivariate analysis, no clinical factors were correlated with outcomes, except for dyslipidemia, which may suggest that this comorbidity is related both to AS acceleration and its severity, as reported by others.[39-41] Thus, in our population, factors that have been classically correlated with mortality such as chronic kidney disease and COPD did not maintain significance after adjustments.[3,4,30,42,43] Sinning et al[7] reported that a severe increase in sPAP measured preoperatively was associated with an increased risk of death (hazard ratio [HR], 3.3, $p = .003$) and a higher incidence of comorbidities such as peripheral vascular arteriopathy, CAD, chronic kidney disease, and atrial fibrillation. These correlations were not found in the current study.

Piccirillo et al[11] analyzed some electrocardiographic criteria to predict events and, among them, LV hypertrophy was correlated with general mortality (AUC, 0.76, $p = .015$). In our study, patients without hypertrophy, considered no confounders, had a higher mortality rate, despites this finding may look controversial, we believe this may be because these patients did not have an adaptive alteration at high ventricular postload and, therefore, had higher mortality.

Patients with the same Selvester QRS scores and the same comorbidities had a different risk of death based on the location of the fibrosis. In ischemic patients, infarct areas that are located in the inferior wall also had a better evolutionary prognosis, probably because the involvement of this ventricular topography may cause less damage to the systole dynamics and is less related to falls in the LV ejection fraction.[44,45]

4.1 | Study limitations

The present analysis was a relatively small retrospective study, but it certainly generates an interesting hypothesis. This study is important in the context of TAVR in the world scenario, since this is the first analysis that correlated an electrocardiographic score of myocardial fibrosis with mortality. Based on the simplicity of this examination and that fact that ECG is innocuous to patients, the strong prediction of mortality is an important finding. The current analysis focused only on survival, whereas functional status and improvement were not considered. Additional prospective studies with more patients should be conducted to confirm the associations observed in the present study and to verify if they correspond to independent associations and therefore, identify those patients with higher mortality after TAVR.

5 | CONCLUSION

In the current study population, an association was found between the Selvester QRS score and an increased risk of death and MACE. A score of 3 had the best sensitivity and specificity. The involvement of the anterior or/and lateral LV walls was associated with a higher risk than involvement of the inferior wall.

IMPACT ON DAILY PRACTICE

Myocardial fibrosis is associated with adverse outcomes in AS and the Selvester QRS score is a simple and innocuous examination that estimates fibrosis and has a strong predictive power of mortality. Identifying patients with low survival may prevent futile treatment of AS. This study supports further investigations in larger populations to warrant to confirm the predictive power of the Selvester QRS score in patients with AS undergoing TAVR.

CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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