Mass to voltage ratio index predicts mortality following TAVI

Alon Porat MD1 | Max Gordon MD1 | Gidon Perlman MD1 | David Planer MD1 | Haim Danenberg MD2 | Ronny Alcalai MD1 | David Leibowitz MD1

1Department of Cardiology, Hadassah Medical Center and Faculty of Medicine, Hebrew University of Jerusalem, Israel
2Department of Cardiology, Edith Wolfson Medical Center, Holon, Israel

Correspondence
David Leibowitz. Coronary Care Unit Hadassah-Hebrew University Medical Center Mt Scopus, Jerusalem 91240, Israel.
Email: OLEIBO@hadassah.org.il

Abstract
Transcatheter aortic valve implantation (TAVI) is commonly performed in elderly patients with aortic stenosis. Better methods of risk stratification are needed in this population with high morbidity. There is a relatively high incidence of cardiac amyloidosis in this population and high LV mass index (LVMI) to QRS voltage may help identify patients with worse prognosis following TAVI. This retrospective study enrolled consecutive patients who underwent TAVI in our institution between the years 2008–2019. Mass voltage ratio index (MVRi) was calculated as the ratio of LV mass index on echocardiogram to voltage using the Sokolow-Lyon criteria on 12 lead ECG performed within 3 months before the intervention. Two hundred and fifty-one patients (mean age 80.8 years, 49% men) were enrolled. One hundred and sixty-eight (67%) patients were alive at 3 years follow up. MVRi was a statistically significant predictor of 3 year mortality (p < 0.005). Patients were divided categorically into tertiles based on MVRi score; the "high" group had significantly higher 3-year mortality (p < 0.001). In the multivariate model only Euroscore (p < 0.009) and MVRi (p < 0.011; OR: 2.32; CI: 1.15–4.964) were statistically significant predictors of mortality. The "high" group had a significantly lower survival rate after 3 years follow up on Kaplan-Meier analysis (p < 0.001). Our findings suggest that MVRi is a strong, independent predictor of increased post-TAVI mortality. This may be a simple clinical tool to assist in the assessment of patients prior to before TAVI.

KEYWORDS
echocardiography, electrocardiography, transcatheter aortic valve replacement

1 | INTRODUCTION

Aortic stenosis (AS) is a common valvular heart condition whose incidence increases with age. In populations aged over 75 years the prevalence of severe AS can reach 3.4%. In recent years transcatheter aortic valve implantation (TAVI) has become standard of care in elderly populations. Although less invasive than surgical valve replacement, TAVI is associated with complications and long-term prognosis may be limited given the comorbidities in this population. Patients with low EF (<30%), moderate/severe aortic regurgitation, pulmonary hypertension, COPD and CRF had a worse prognosis after TAVI procedures. The predictive value of tools...
such as Euroscore/STS are limited and there remains a need for better risk stratification of this complex population.

Another common pathology in the elderly is transthyretin cardiac amyloidosis (CA). This disorder is characterized by extracellular deposits of fibrillar proteins in different organs including the heart which can result in LV dysfunction. Both CA and AS are common pathologies in the elderly population and previous studies have reported an incidence of CA of up to 15% in TAVI candidates. A combination of both diseases carries a poor prognosis regardless of left ventricular ejection fraction and aortic valve replacement procedures. Both AS and CA cause increased left ventricular mass (LVM) but for different reasons. In AS there is a true increase in LVM in response to pressure overload while in CA it is due to amyloid deposits in the myocardium. Because of this difference, the ECG of patients with AS often demonstrates high QRS amplitudes on ECG due to “true” left ventricular hypertrophy (LVH) whereas patients with CA may demonstrate low QRS amplitudes due to deposits of amyloid in the heart. The ratio between the LV mass index (LVMi) (obtained by echocardiogram) to QRS amplitude has been used as a diagnostic tool in CA. We postulated that this finding would be even more pronounced in a population of patients with significant AS and consequent LV hypertrophy.

The aim of this study was to examine whether the ratio between LVMi to QRS amplitude before TAVI predicts mortality 3 years post-TAVI.

2 | MATERIALS AND METHODS

All patients who underwent TAVI at our institution (Hadassah-Hebrew University Medical Center Jerusalem) were eligible for inclusion in the study. Study approval was provided by the institutional ethics committee and the study was conducted according to the principles of the Helsinki Declaration. Patients with an echocardiogram and a 12 lead ECG performed up to 3 months before the procedure and who had body height and weight recorded were included in the study. Severe AS was defined as aortic valve area (AVA) < 1.0 cm². Low flow low gradient (LFLG) patients were defined as those with mean gradient less than 40 mmHg and AVA less than 1.0 cm².

Patients who did not have a known outcome for at least 3 years follow up after the procedure (i.e., were alive and did not yet complete 3 years follow up at the time the study ended) or who died within 24 h of the procedure were excluded (n = 73). Mortality data was taken from the Ministry of Health and Hadassah hospital registry. Additional relevant clinical, electrographic, and echocardiographic data was taken from review of patient hospital files. Euroscore II was calculated for all patients. Diabetes was defined as use of oral medical or insulin therapy.

2.1 | Calculation of ECG-A and LVMi

12-lead electrocardiograms were performed with standard instrument sensitivity of 10 mm = 1 mv. ECG amplitude was calculated using the Sokolow Lyon method (sum of S wave in lead I + R wave in V5 or V6).

Patients with pacemakers and left and right bundle branch blocks were included in the study.

LV mass was calculated on echocardiogram using the Penn-cube M-Mode method. 0.8 [(LVEDD + IVSd + PWd)³ – LVEDD³] + 0.6 where LVEDD = LV end-diastolic diameter, and PWd and IVSd posterior wall thickness in diastole.

2.2 | Calculation of the index

Mass voltage ratio index (MVRI) is calculated by dividing LVMi by the sum of ECG leads voltage as per the Sokolow Lyon method.

The study population was analyzed with MVRI as a continuous variable and categorically by dividing the population into tertiles. Tertiles were chosen as mortality could potentially be related to both high or low MVRI ratios in this population.

2.3 | Statistical analysis

The association between categorical variables was assessed using the Chi-square test. Comparison of a continuous variable between two independent groups was performed using the t test. The comparison of a continuous variable between three independent groups was carried out using the analysis of variance procedure with the Dunnett Post Hoc tests. Variables thought to be clinically relevant on the basis of previous literature underwent univariate analysis. The variables which were found to be significantly associated with mortality using the univariate approach, were entered into a multivariate logistic regression model with backwards selection for a dichotomous dependent variable (mortality yes/no). The Kaplan–Meier model was applied for assessing the effect of MVRI on survival, with the log rank test for the comparison of survival curves. All statistical tests applied were two-tailed, and a p value of 0.05 or less was considered statistically significant. All statistical analysis was done using SPSS software (version 26; IBM).

3 | RESULTS

Three hundred and twenty-four patients who had the relevant data recorded in their patient file were reviewed, 70 patients with less than 3-year follow-up and three who died within 24 h of the procedure were excluded leaving a study population of 251 (mean age 80.8 ± 6.7 years, 123 M/128 F, mean Euroscore 5.5 ± 5.3). Out of 251 patients with 3 years follow up 83 (33%) died within 3 years. The demographic and characteristics of the study population are shown in Table 1.

In a univariate model the following variables were found to be statistically significant predictors of mortality within 3 years: Euroscore (p = 0.003), aortic valve mean pressure gradient (AVMP)
As well as LFLG ($p = 0.05$), Diabetes mellitus ($p = 0.037$), RBBB ($p = 0.036$), and age ($p = 0.034$). MVRi score was compared between the 3 years survival and mortality groups as a continuous variable and was a statistically significant predictor of mortality with $p = 0.005$ (shown in Table 1). There was no significant correlation between LVMi and RBBB or AV mean pressure.

Variables related to the procedure are shown in Table 2. Correlation between these variables and 3 years survival was assessed using a univariate model. The only variable found to be a statistically significant predictor of mortality was vascular access—not femoral with $p = 0.01$.

The study population was then divided into three groups ($n = 83/84$ in each group) based on MVRi scores (shown in Table 3): “low”: MVRi < 4.19, “middle”: 4.19 < MVRi < 6.58, and “high”: MVRi > 6.58. The demographic characteristics and a univariate model comparing different variables are shown in Table 3. Three-year survival was compared between the groups based on the MVRi score in a univariate model. MVRi was found to be a statistically significant predictor of mortality after 3 years with $p = 0.001$.

All variables found to be statistically significant along with the MVRi score were then put in a multivariate model (Table 4). Euroscore, MVRi, vascular access—not femoral remained the only independent predictors of 3 years mortality. Euroscore had a $p = 0.01$, adjusted odds ratio (OR): 1.07, 95% confidence interval (CI: 1.102–1.1016). Vascular access—not femoral had a $p = 0.04$, adjusted OR: 0.261, 95% CI: 0.07–0.95. MVRi had a $p = 0.011$, adjusted OR: 2.32, 95% CI: 1.16 to 4.96 (shown in Table 4).

A Kaplan–Meier survival curve model (shown in Figure 1) was applied to the study population comparing 3-year mortality between different variables.

### Table 1: Demographic and characteristics of the study population

| Variable | Study population | Survival 3 y | Mortality 3 y | $p$ value |
|----------|------------------|--------------|---------------|-----------|
| N        | 251              | 168          | 83            |           |
| Men, n (%) | 123 (49.0)       | 87 (51.7)    | 36 (43.3)     | 0.210     |
| Age, years | 80.8 ± 6.67      | 81.4 ± 6.0   | 79.53 ± 7.7   | 0.034     |
| BSA, m²   | 1.79 ± 0.20      | 1.79 ± 0.2   | 1.78 ± 0.2    | 0.669     |
| COPD, n (%) | 49 (19.5)        | 29 (17.2)    | 20 (24.0)     | 0.199     |
| AF, n (%) | 28 (11.2)        | 15 (8.9)     | 13 (15.6)     | 0.111     |
| LBBB, n (%) | 20 (8.0)         | 11 (6.5)     | 9 (10.8)      | 0.237     |
| RBBB, n (%) | 30 (12.0)        | 15 (8.9)     | 15 (18.0)     | 0.036     |
| LAHB, n (%) | 42 (16.7)        | 27 (16.0)    | 15 (18.0)     | 0.690     |
| AVB, n (%) | 42 (16.7)        | 26 (15.4)    | 16 (19.2)     | 0.448     |
| Pacemaker (b), n (%) | 34 (13.5) | 20 (11.9) | 14 (16.8) | 0.280 |
| Pacemaker (a), n (%) | 60 (23.9) | 35 (20.8) | 25 (30.1) | 0.105 |
| CAD, n (%) | 133 (53.0)       | 88 (50.0)    | 49 (59.0)     | 0.177     |
| Prior CVA, n (%) | 30 (12.0) | 22 (13.0) | 8 (9.6) | 0.427 |
| Diabetes mellitus, n (%) | 98 (39.0) | 58 (34.5) | 40 (48.1) | 0.037 |
| HTN, n (%) | 209 (83.3)       | 140 (83.3)   | 68 (83.1)     | 0.968     |
| CRF, n (%) | 103 (41.0)       | 63 (37.5)    | 40 (48.1)     | 0.105     |
| EF, %     | 58.13 ± 13.1     | 59.1 ± 13.2  | 56 ± 12.7     | 0.097     |
| Euroscore | 5.49 ± 5.28      | 4.7 ± 4.4    | 7.1 ± 6.5     | 0.003     |
| AVMP, mmHg | 44.37 ± 14.48    | 45.8 ± 14.2  | 41.5 ± 14.7   | 0.028     |
| AVA, cm   | 0.67 ± 0.17      | 0.67 ± 0.16  | 0.66 ± 0.19   | 0.793     |
| LFLG      | 91 (36.2)        | 46 (27.3)    | 37 (44.5)     | 0.054     |
| MVRi (con) | 7.14 ± 5.64      | 6.36 ± 5.18  | 8.74 ± 6.57   | 0.005     |

Note: Demographic and characteristics of the study population. Values are mean ± SD or %. Univariate model comparing survival after 3 years between patients based on a number of variables. Abbreviations: AF, atrial fibrillation; AHB, left anterior hemi-block; AVB, atrioventricular block; AVA, aortic valve area; AVMP, aortic valve mean pressure; BSA, body surface area; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; CRF, chronic renal failure; DM, diabetes mellitus; EF, ejection fraction; HTN, hypertension; LBBB, left bundle branch block; LFLG, low flow low gradient; MVRi, Mass voltage ratio index (continuous variable). Pacemaker, b—before TAVI procedure. a—up to 7 days after TAVI procedure; RBBB, right bundle branch block.
the three groups based on the MVRi score ("low,” “middle,” and “high”). In a 3-year follow-up the “high” score group showed a significantly higher mortality rate than the "low" and "middle" groups over 3 years follow-up. The difference was statistically significant in a Log Rank test with a \( p = 0.001 \).

4 | DISCUSSION

Our findings demonstrate that assessment of MVRi utilizing ECG and echocardiogram is an independent predictor of mortality following TAVI and may be a simple, widely available, and inexpensive tool for risk assessment before the procedure. High MVRi may be indicative of coexisting CA common in this elderly population with AS.

4.1 | Prognosis following TAVI

Many patients undergoing evaluation for TAVI are elderly and often have many co-morbidities, and it is important to correctly assess which patients can best benefit from the procedure.\(^{19-24}\) Previous studies have questioned whether surgical risk algorithms accurately predict mortality post-TAVI.\(^{25,26}\) Some, but not all previous studies have suggested the importance of cardiovascular factors such as reduced EF, pulmonary hypertension, and low aortic valve gradients secondary to low stroke volumes. In the study of Furer et al. from the PARTNER-2 trial, EF < 50% was associated with higher mortality however in the multivariate model reduced EF was no longer significantly associated with all-cause mortality, findings consistent with our study.\(^{27}\) In very elderly population, it is possible other factors such as low gradient AS more accurately reflect the effect of myocardial dysfunction than EF. In our study, low gradient AS was significantly lower in patients who died but only on univariate analysis.

4.2 | Amyloidosis in TAVI patients

Recent studies have shown a high prevalence of CA in patients referred for TAVI,\(^{28}\) a finding generally associated with worse prognosis after TAVI.\(^{7}\) Presumed mechanisms for the negative influence of CA on prognosis after TAVI include low output state leading to multisystem failure, an increased incidence of arrhythmias as well as involvement of amyloid in other organ systems. Several previous studies have examined the predictive value of an elevated MVRi for the presence of CA. Castano et al. prospectively screened candidates for TAVI for CA using PYP scintigraphy. They demonstrated a higher MVRi in the subgroup of patients with suspected CA on nuclear imaging.\(^{29}\) Scully et al. prospectively screened 200 patients pre TAVI using DPD scintigraphy and found evidence of CA in 13% of subjects. MVRi was higher in the CA group in this study as well but was not predictive of CA on multivariate analysis.\(^{15}\) A recent study by Rosenblum et al including 204 pre-TAVI patients from two academic centers did not find a significant difference in MVRi in patients with or without CA.\(^{30}\) Nitsche et al. performed a comprehensive diagnostic evaluation including myocardial biopsies for CA in 191 patients before TAVI. In this study using stricter diagnostic criteria the prevalence of CA was 8% and was significantly associated with elevated MVRi on multivariable analysis, findings potentially consistent with our study.\(^{31}\) However, the presence of CA did not affect prognosis after TAVI in this study. This may be due to selection bias as performance of TAVI in this population was not randomized.

Our study greatly expands previous work by looking directly at the effect of MVRi on intermediate-term mortality following TAVI from a single institution in a larger number of patients. We utilized indexed LV mass to normalize for body size and discriminate between physiologic changes related to body habitus and changes related to pathologic stimuli such as CA.\(^{32}\)

It is important to note that we did not systematically screen our patient population for the presence of CA, and it is possible that the influence of MVRi is due to additional pathologies. However, other clinical findings that may influence ECG voltage such as BMI, a history of COPD

---

**Table 2** Procedural characteristics

|                          | Study population | Survival 3y | Mortality 3y | p value |
|--------------------------|------------------|-------------|--------------|---------|
| N                        | 251              | 168         | 83           |         |
| Valve pathology—combined stenosis and regurgitation, n (%) | 6 (2.3) | 2 (1.2) | 4 (4.8) | 0.077 |
| Valve-in-valve procedure, n (%) | 5 (2) | 3 (1.8) | 2 (2.4) | 0.74   |
| Vascular access—not femoral artery, n (%) | 14 (5.4) | 5 (3) | 9 (10.8) | 0.011 |
| Valve type—Edwards, n (%) | 72 (28.7) | 51 (30.4) | 21 (25.3) | 0.4    |

Note: Demographic and characteristics of the procedure for the study population. Values are N (%). Univariate model comparing survival after 3 years between patients based on a number of variables related to the procedure. Vascular access, patients who went through the procedure with either direct aortic or axillary artery access compared to femoral artery access. Valve pathology, patients with combined aortic stenosis and regurgitation, compared to patients with aortic stenosis alone. Valve type, patients who had an Edwards valve implanted compared to Medtronic valve.
and ejection fraction were not significantly associated with mortality making the presence of undiagnosed CA a potential explanation of our findings.

### 4.3 Study limitations

The limitations of our study include its retrospective nature although the use of the hard endpoint of mortality limits bias. The number of patients included in the study is relatively small although similar to other studies on the topic. The possibility of mortality related to operator experience and a learning curve for the procedure cannot be excluded with certainty. We examined all-cause mortality as specific causes of death are not available from the centralized database. As mentioned, we did not perform a specific clinical assessment for the presence of CA. Our study examined an elderly population and may not be applicable to younger, more low-risk patients undergoing TAVI.

Our study did not include significant numbers of patients with either advanced renal disease or oxygen dependency and frailty was not systematically assessed in our population and these variables could not be assessed in our population.
CONCLUSION

Our study shows that elevated MVRi in patients undergoing TAVI predicts 3-year mortality, possibly due to coexisting CA. Further prospective trials with larger number of patients are necessary to examine whether using MVRi as a simple screening tool will improve prognosis of patients undergoing TAVI.

TABLE 4

| Variable                  | Univariate model | Multivariate model |
|---------------------------|------------------|-------------------|
| MVRI                      | 0.001            | 0.013             |
| RBBB                      | 0.021            | 0.337             |
| LFLG                      | 0.054            | 0.398             |
| Vascular access-not femoral | 0.011           | 0.041             |
| DM                        | 0.037            | 0.816             |
| Age, years                | 0.034            | 0.078             |
| Euroscore                 | 0.003            | 0.018             |
| AVMP, mmHg                | 0.028            | 0.953             |

Note: Multivariate model for variables found to be statistically significant predictors of mortality in univariate model.

Abbreviations: CI, confidence interval, lower limit; CI,H, higher limit.

ACKNOWLEDGMENTS

This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

FIGURE 1

Kaplan–Meier curve, comparing 3 years survival between groups based on MVRI score. Kaplan–Meier curve comparing survival rates between groups with different MVRI scores. Population study was divided into tertiles based on MVRI score (“low”: MVRI < 4.19, “middle”: 4.19 < MVRI < 6.58, and “high”: MVRI > 6.58). Survival between the different groups was compared using Log Rank test. The “high” score group showed a significantly greater mortality rate than the “low” and “middle” groups over 3 years follow-up. The difference was statistically significant with a p = 0.001. These results demonstrate the association of MVRI score and 3 years prognosis after TAVI. MVRI, Mass voltage ratio index; TAVI, transcatheter aortic valve replacement [Color figure can be viewed at wileyonlinelibrary.com]
DATA AVAILABILITY STATEMENT
The data that support the findings of this study are available from the corresponding author upon reasonable request.

ORCID
Alon Porat http://orcid.org/0000-0002-8254-484X

REFERENCES
1. Eveborn GW, Schirmer H, Heggelund G, Lunde P, Rasmussen K. The evolving epidemiology of valvular aortic stenosis. The Tromsø Study. Heart. 2013;99(6):396-400.
2. Osnabrugge RLJ, Mylotte D, Head SJ, et al. Aortic stenosis in the elderly: disease prevalence and number of candidates for transcatheter aortic valve replacement: a meta-analysis and modeling study. J Am Coll Cardiol. 2013;62(11):1002-1012.
3. Siontis GCM, Praz F, Pilgrim T, et al. Transcatheter aortic valve implantation vs. surgical aortic valve replacement for treatment of severe aortic stenosis: a meta-analysis of randomized trials. Eur Heart J. 2016;37(47):3503-3512.
4. Rodes-Cabau J, Webb JG, Cheung A, et al. Long-term outcomes after transcatheter aortic valve implantation: insights on prognostic factors and valve durability from the Canadian Multicenter Experience. J Am Coll Cardiol. 2012;60(19):1864-1875.
5. Moat NE, Ludman P, De Belder MA, et al. Long-term outcomes after transcatheter aortic valve implantation in high-risk patients with severe aortic stenosis: The U.K. TAVI (United Kingdom transcatheter aortic valve implantation) registry. J Am Coll Cardiol [Internet]. 2011;58(20):2130-2138. doi:10.1016/j.jacc.2011.08.050
6. Carroll JD, Mack MJ, Vemulapalli S, et al. STS ACC TVT registry of transcatheter aortic valve replacement. J Am Coll Cardiol. 2020;76(21):2492-2516.
7. Treibei TA, Fontana M, Gilbertson JA, et al. Occult transthyretin cardiac amyloid in severe calcific aortic stenosis. Circ Cardiovasc Imaging. 2016;9(8):1-10.
8. Galat A,Guellich A, Bodez D, et al. Aortic stenosis and transthyretin cardiac myopathy: the chicken or the egg? Eur Heart J. 2016;37(47):3525-3531.
9. Ricci F, Ceriello L, Khanji MY, et al. Prognostic significance of cardiac amyloidosis in patients with aortic stenosis. JACC Cardiovasc Imaging. 2020;13(9):1801-1809.
10. Frank S,Johnson A,Ross J. Natural history of valvular aortic stenosis. Br Heart J. 1973;35(1):41-46.
11. Vogelsberg H, Mahrohldt H, Deluigi CC, et al. Cardiovascular magnetic resonance in clinically suspected cardiac amyloidosis: non-invasive imaging compared to endomyocardial biopsy. J Am Coll Cardiol. 2008;51(10):1022-1030.
12. Rapezzi C,Quarta CC,Riva L, et al. Transthyretin-related amyloidoses and the heart: a clinical overview. Nat Rev Cardiol. 2010;7(7):398-408.
13. Rahman JE,Helou EF, Gelzer-Bell R, et al. Noninvasive diagnosis of biopsy-proven cardiac amyloidosis. J Am Coll Cardiol. 2004;43(3):410-415. doi:10.1016/j.jacc.2003.08.043
14. Carroll JD,Gaasch WH, McAdam KPWJ. Amyloid cardiomyopathy: characterization by a distinctive voltage/mass relation. Am J Cardiol. 1982;49(1):9-13.
15. Scully PR,Patel KP,Treibel TA, et al. Prevalence and outcome of dual aortic stenosis and cardiac amyloid pathology in patients referred for transcatheter aortic valve implantation. Eur Heart J. 2020;41(29):2757-2767.
16. Sokolow M,Lyon TP. The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. Am J Heart. 1949;37(2):161-186.
17. Cokkinos DV, Demopoulos JN,Heimonas ET, Mallios C, Papazoglou N, Vorides EM. Electrocardiographic criteria of left ventricular hypertrophy in left bundle branch block. British Heart J. 1978;40:320-324.
18. Devereux RB,Alonso DR,Lutas EM, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol. 1986;57(6):450-458. doi:10.1016/0002-9149(86)90771-X
19. Nishimura RA, Otto CM,Bonow RO, et al. AHA/ACC focused update of the 2014 AHA/ACC guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2017;135:1195.
20. Lytvyn L,Guyatt GH,Manja V, et al. Patient values and preferences on transcatheter or surgical aortic valve replacement therapy for aortic stenosis: a systematic review. BMJ Open. 2016;6(9):e014327.
21. Makkar RR,Fontana GP,Jilaihawi H, et al. Transcatheter aortic valve replacement for inoperable severe aortic stenosis. N Engl J Med. 2012;366(18):1696-704.
22. Appel CF,Hultkist H,Nylander E, et al. Transcatheter versus surgical treatment for aortic stenosis: patient selection and early outcome1. Scand Cardiovasc J. 2012;46(5):301-307. doi:10.3109/14017431.2012.699636
23. Geneurex P,Head SJ,Wood DA, et al. Transcatheter aortic valve implantation: 10-year anniversary part II: clinical implications. Eur Heart J. 2012;33(19):2399-2402.
24. Puri R, Jung B, Cohen DJ, Rodes-Cabau J. TAVI or Not TAVI: Identifying patients unlikely to benefit from transcatheter aortic valve implantation. Eur Heart J. 2016;37(28):2217-2225.
25. Arangafage D,Cinadevilla C, Alkhoader S, et al. Agreement between the new EuroSCORE II, the Logistic EuroSCORE and the Society of Thoracic Surgeons score: implications for transcatheter aortic valve implantation. Arch Cardiovasc Dis. 2014;107(6–7):353-360.
26. Silaschi M,Conradi L,Selfert M, et al. Predicting risk in transcatheter aortic valve implantation: comparative analysis of EuroSCORE II and established risk stratification tools. Thorac Cardiovasc Surg. 2015;63(6):472-478.
27. Furer A,Chen S,Redfors B, et al. Effect of baseline left ventricular ejection fraction on 2-year outcomes after transcatheter aortic valve replacement. Circ Heart Fail. 2019;12:e005809.
28. González-López E,Gallego-Delgado M,Guzzo-Merello G, et al. Wild-type transthyretin amyloidosis as a cause of heart failure with preserved ejection fraction. Eur Heart J. 2015;36(38):2585-2594.
29. González-Lopez E,Gallego-Delgado M,Guzzo-Merello G, et al. Unveiling transthyretin cardiac amyloidosis and its predictors among elderly patients with severe aortic stenosis undergoing transcatheter aortic valve replacement. Eur Heart J. 2017;38(38):2879-2887.
30. Rosenblum H,Masi A,Narotsky DL. Unveiling transthyretin cardiac amyloidosis and its predictors among elderly patients with severe aortic stenosis undergoing transcatheter aortic valve replacement. Eur Heart J. 2017;38(38):2879-2887.
31. Nitsche C,Aschauer S,Kammerlander AA. Light chain and transthyretin cardiac amyloidosis in severe aortic stenosis: prevalence, screening possibilities, and outcome. Eur J Heart Fail. 2020;22(10):1852-1862.
32. Cuspidi C,Fachetti R,Bombelli M,Sala C,Grassi G,Mancia G.Differential value of left ventricular mass index and wall thickness in predicting cardiovascular prognosis: data from the PAMELA population. Am J Hypert. 2014;27(8):1079-1086.

How to cite this article: Porat A, Gordon M,Perlman G, et al. Mass to voltage ratio index predicts mortality following TAVI. Catheter Cardiovasc Interv. 2022;99:1918-1924. doi:10.1002/ccd.30117