Validation of the HCM Risk-SCD model in patients with hypertrophic cardiomyopathy following alcohol septal ablation

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Aims

The HCM Risk-SCD model for prediction of sudden cardiac death (SCD) in hypertrophic cardiomyopathy recommended by the 2014 European Society of Cardiology (ESC) guidelines has not been validated after septal reduction therapy. The aim of this study was to validate the HCM Risk-SCD model in patients undergoing alcohol septal ablation (ASA) and to compare its performance to previous models.

Methods and result

A total of 844 ASA patients without prior SCD event were included. The primary endpoint was a composite of SCD and appropriate implantable cardioverter defibrillator (ICD) therapy, identical to the HCM Risk-SCD endpoint. A distinction between periprocedural (<30 days) and long-term (>30 days) SCD was made to discern procedure-related adverse arrhythmic events caused by the ASA-induced myocardial infarction from long-term SCD risk. Twenty patients reached the SCD endpoint within the first 30 days. During a follow-up of 6.5 ± 4.2 years, another 46 patients reached the SCD endpoint. The predicted 5-year SCD risk according to the HCM Risk-SCD model was 5.1%, and the observed 5-year SCD risk was 4.0%. The C-statistics for the use of the HCM Risk-SCD model was 0.61 (P = 0.02), the C-statistics for the use of the 2003 American College of Cardiology/ESC guidelines was 0.59 (P = 0.051), and the C-statistic for the use of the 2011 American College of Cardiology Foundation/American Heart Association guidelines was 0.58 (P = 0.054). Maximal left ventricular wall thickness, syncope after ASA, and fulfilling the 2014 ESC recommendations for primary ICD implantation according to the HCM Risk-SCD model, respectively, predicted SCD during long-term follow-up.

Conclusion

The HCM Risk-SCD model can be used for SCD prediction in patients undergoing ASA.

Keywords

Hypertrophic cardiomyopathy • Alcohol septal ablation • Septal reduction therapy • Sudden cardiac death • Implantable cardioverter defibrillator

Introduction

Hypertrophic cardiomyopathy (HCM) is the most common inherited cardiac disease (prevalence 1:500) and carries a risk for sudden cardiac death (SCD) of 0.5 to 1% per year.1 Predicting which patients are at risk of SCD has proven difficult. The 2003 American College of Cardiology (ACC)/European Society of Cardiology (ESC) and 2011 American College of Cardiology Foundation (ACCF)/American

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Heart Association (AHA) guidelines use five conventional risk factors to estimate the risk for SCD: a family history of SCD, maximal left ventricular wall thickness (LVWT) \(>30\) mm, non-sustained ventricular tachycardia (NSVT), and abnormal blood pressure response to exercise.\(^2,3\) O’Mahony et al.\(^4\) demonstrated in 2013 that the models based on these conventional risk factors were limited in distinguishing high from low-risk patients. The following year, the HCM Outcomes Investigators presented a novel risk prediction model (HCM Risk-SCD), which provided a calculated 5-year SCD risk using four of the conventional (family history of SCD, NSVT, maximal LVWT, and unexplained syncope) and three additional risk factors: age, left atrial (LA) diameter, and left ventricular outflow tract (LVOT) gradient.\(^5\) This model was more accurate in predicting SCD risk compared with the previous models and was commended in the 2014 ESC guidelines. However, the HCM Risk-SCD model has not been validated in patients with obstructive HCM who have undergone surgical myectomy or alcohol septal ablation (ASA), and application of the model in these patients is therefore not recommended.\(^5\) Since its introduction over 20 years ago, a main concern regarding ASA has been its possible effect on SCD risk, due to the intracoronary injection of ethanol, creating a potentially arrhythmogenic ablation scar. On the other hand, LVOT obstruction has been independently associated with SCD,\(^6-10\) and was therefore incorporated in the HCM Risk-SCD model.\(^5\) The aims of this study were to (i) validate the HCM Risk-SCD model in patients undergoing ASA and to compare its performance to previous models; and (ii) identify predictors of SCD in HCM patients after ASA.

### Methods

#### Patient population

An international multicentre observational cohort design was used. The study population consisted of 1146 consecutive HCM patients (mean age 58 ± 14 years; 47% female) who underwent ASA because of symptomatic LVOT obstruction despite optimal medical therapy. Procedures were performed in six European tertiary invasive centres (Germany (n = 513); Bad Oyenhausen; The Netherlands (n = 314): Nieuwegein and Rotterdam; Czech Republic (n = 161); Prague; Scandinavia (n = 158); Copenhagen and Oslo) between January 1996 and August 2015. Each patient had an established diagnosis of HCM (based on otherwise unexplained left ventricular hypertrophy of \(\geq 15\) mm) and a (provocable) LVOT gradient \(\geq 50\) mmHg. The choice of ASA instead of surgical myectomy was based on patient profile (age, coronary anatomy, comorbidities, etc.) and patient preference.

### Follow-up and endpoints

Follow-up started at the time of ASA. There were some differences in post-ASA follow-up regimens between the centres. In general, all patients had a first clinical follow-up 3–6 months after the procedure and annual routine check-ups thereafter. Adverse events were retrieved from national patient registries, from hospital patient records at the centre where follow-up was performed, and from information provided by patients themselves and/or their general practitioners. All implantable cardioverter defibrillator (ICD) shocks were evaluated by an experienced electrophysiologist, unaware and independent of the study purpose and endpoints.

The primary endpoint of SCD was equivalent to the endpoint used in the HCM Risk-SCD study.\(^5\) It was a composite endpoint consisting of (i) instantaneous and unexpected death within 1 h of a witnessed collapse in patients who were previously in a stable clinical condition or nocturnal death with no antecedent history of worsening symptoms, (ii) successful resuscitation after cardiac arrest, and (iii) appropriate ICD interventions for ventricular fibrillation (VF) or fast VT (\(>200\) beats per minute). Patients who reached the SCD endpoint were censored at the time of its occurrence. A distinction between peri-procedural (\(\leq 30\) days) and long-term (\(>30\) days) SCD was made to discern procedure-related adverse arrhythmic events caused by the ASA-induced myocardial infarction from long-term SCD risk.

### Risk stratification

Conventional risk factors for SCD and additional variables necessary to calculate the 5-year SCD risk according to the HCM Risk-SCD model were evaluated at baseline (before ASA). The following risk factors were identified: (i) a family history of SCD in \(\geq 1\) first-degree relatives aged \(<40\) years or in a first-degree relative with confirmed HCM at any age, (ii) maximal LVWT, (iii) history of unexplainable syncope, (iv) documented NSVT \(\geq 3\) beats at a rate of \(\geq 120\) beats per minute, (v) abnormal blood pressure response during exercise, (vi) (provocable) LVOT gradient, (vii) LA diameter measured in parasternal long axis, and (viii) age. Patients with \(>2\) missing risk factors, or with a history of cardiac arrest or sustained VT were excluded.

The 5-year SCD risk for individual patients was calculated using the HCM Risk-SCD formula:

\[
P_{\text{SCD at 5 years}} = 1 - 0.999^{\text{Risk Index}}
\]

where Prognostic Index = 0.15939858 \times \text{maximal LVWT} (mm) - 0.00294271 \times \text{maximal LVWT}^2 (mm^2) + 0.0259082 \times \text{LA diameter} (mm) + 0.000446131 \times \text{maximal LVOT gradient (mmHg)} + 0.04583082 \times \text{family history of SCD} + 0.82639195 \times \text{NSVT} + 0.71650361 \times \text{unexplained syncope} - 0.01799934 \times \text{age at evaluation (years)}.

According to the 2014 ESC guidelines, a calculated 5-year SCD risk of \(\geq 6%\) confers a recommendation (Class IIa) for ICD implantation for primary prevention of SCD.\(^1\)

For comparison, risk profiles based on the 2003 ACC/ESC and 2011 ACCF/AHA guidelines were calculated. In the 2003 ACC/ESC guidelines, \(\geq 2\) conventional risk factors (factors 1–5 above) conferred a recommendation for ICD implantation for primary prevention of SCD.\(^2\) Sudden cardiac death risk stratification according to the 2011 ACCF/AHA guidelines is similar, except that family history of SCD, maximal LVWT \(\geq 30\) mm, or unexplained syncope as a sole risk factor are also considered to confer an ICD recommendation (Class IIa).\(^3\)

### Statistical analysis

SPSS version 24 (IBM, Armonk, NY, USA), and Microsoft Excel 2010 (Microsoft Corporation, Redmond, WA, USA) were used for all statistical analyses. Categorical variables are summarized as percentages. Normally distributed continuous data are expressed as mean ± standard deviation and non-normally distributed data as median ± interquartile range (IQR). To compare continuous variables Student’s t-test or
Mann–Whitney U test were used, and to compare categorical variables the χ² test was used.

The performance of the novel risk model, and the models based on conventional risk factors for SCD, was determined by the C-statistic, which in the current context indicates how well the model discriminates between high and low risk of SCD in post-ASA patients. A C-statistic of 0.5 indicates no predictive value and 1.0 indicates perfect discrimination. Univariate Cox regression analysis was performed to identify predictors of outcome. Kaplan–Meier graphs were used to show SCD rates and differences in SCD rate were assessed by the log-rank test. All tests were two-sided, and P < 0.05 was considered statistically significant. To handle missing data, we used a similar approach outlined in the HCM Risk-SCD study where missing data were identified and imputed using multiple imputation techniques based on chained equations. A total of 25 imputed data sets were generated and pooled.

### Results

#### Clinical characteristics

After exclusion of patients with a history of aborted SCD (n = 27) or with >2 missing SCD risk factors, 844 patients (mean age 56 ± 14 years; 46% female) were available for further analyses. Table 1 lists the baseline characteristics of these patients. An ICD had been implanted for primary prevention in 18 (2%) patients before ASA (Table 2). Risk stratification was complete in 536 (63%) patients; exercise testing was lacking in 212 (25%) patients, Holter monitoring in 202 (24%) patients, and LA diameter in 21 (2%) patients. The remaining five risk factors were missing in <0.5% of patients.

#### Periprocedural sudden cardiac death

Twenty patients (mean age 60 ± 15 years; 55% female) reached the SCD endpoint within the first 30 days post-ASA and were therefore excluded from model validation. One patient died of VF, and 19 patients received successful electrical cardioversion for VT/VF. Patients with a periprocedural SCD endpoint had larger infarcts following ASA [maximum creatine kinase (CK)-MB 168 (IQR 468) vs. 87 (IQR 107) IU/L, P < 0.01]. A maximum CK-MB >240 IU/L was found to be the only significant predictor of periprocedural SCD [hazard ratio (HR) 7.51, 95% confidence interval (CI) 2.29–24.61, P < 0.01]. The 240 IU/L cut-off was based on a previous analysis showing an increased risk of adverse arrhythmic events during follow-up above this level. Volume of injected alcohol was not associated with peri-procedural SCD.

#### Predicted vs. observed sudden cardiac death endpoints

In the 824 patients who did not reach the SCD endpoint within the first 30 days, the follow-up duration was 6.5 ± 4.2 years. A total of 29 (4%) patients had an ICD implanted after ASA (Table 2). During long-term follow-up, 46 patients (mean age 54 ± 16 years; 50% female) reached the SCD endpoint, which translated to 0.8 events per 100 patient-years; 30 patients died suddenly (mean age 57 ± 15 years; 63% female; mean follow-up 4.4 ± 2.7 years), 4 patients were successfully resuscitated (mean age 40 ± 22 years; 25% female; mean follow-up 3.9 ± 3.9 years), and 12 patients received an appropriate ICD shock (mean age 50 ± 15 years; 25% female; mean follow-up 3.7 ± 3.5 years).

The predicted 5-year SCD risk according to the HCM Risk-SCD model was 5.1 ± 4.5%, and the observed 5-year SCD risk was 4.0% (Figure 1). The C-statistic for use of the HCM Risk-SCD model was 0.61 (95% CI 0.52–0.69, P = 0.02). The C-statistic for risk stratification with use of the 2003 ACC/ESC guidelines was 0.59 (95% CI 0.50–0.67, P = 0.051), and the C-statistic for the 2011 ACCF/AHA guidelines was 0.58 (95% CI 0.50–0.67, P = 0.054). The performance of the different SCD risk prediction models is summarized in Table 3.
Predictors of sudden cardiac death

Predictors of SCD during long-term follow-up were maximal LVWT $\geq 30$ mm (HR 3.48, 95% CI 1.62–7.46, $P < 0.01$), syncope after ASA (HR 2.88, 95% CI 1.37–6.06, $P < 0.01$), and a 2014 ESC recommendation for primary prophylactic ICD implantation according to the HCM Risk-SCD model (HR 1.96, 95% CI 1.09–3.54, $P = 0.02$; Table 4). An indication for primary prophylactic ICD implantation according to the 2003 ACC/ESC and 2011 ACCF/AHA guidelines was not predictive of SCD (HR 1.74, 95% CI 0.89–3.39, $P = 0.10$, and HR 1.63, 95% CI 0.91–2.90, $P = 0.10$, respectively). Kaplan–Meier estimates for risk of SCD are shown in Figure 2. Compared with the different SCD risk prediction models, the risk factors LVWT $\geq 30$ mm and syncope after ASA retained a high specificity but lower sensitivity for predicting SCD (Table 3).

### Discussion

This is the first study to validate the HCM Risk-SCD model in patients undergoing ASA. The principal findings of this study were that (i) the HCM Risk-SCD model discriminated better between patients with high or low risk of SCD following ASA, as compared with the models proposed by the 2003 ACC/ESC and 2011 ACCF/AHA guidelines; and (ii) maximal LVWT, syncope after ASA, and fulfilling the 2014 ESC recommendations for primary prophylactic ICD implantation according to the HCM Risk-SCD model, respectively, predicted SCD during long-term follow-up.

From 2011, the American and European HCM guidelines differentiated in their approach to SCD risk stratification. The 2011 ACCF/AHA guidelines continued the path of the 2003 ACC/ESC guidelines in using the conventional risk factors for SCD, with the modification that the individual risk factors family history of SCD, maximal LVWT $\geq 30$ mm, and unexplained syncope alone were also considered sufficient for a primary ICD recommendation. However, O’Mahony et al. demonstrated in 2013 that the models based on these conventional risk factors were limited in distinguishing high-risk from low-risk patients. The HCM Outcomes Investigators introduced the novel HCM Risk-SCD model based on an initial systematic review, identifying risk factors independently associated with SCD in at least one published multivariable analysis. A subsequent univariate Cox regression analysis on a population of 3675 consecutive HCM patients found seven out of the eight pre-specified risk factors to be associated with SCD at a 15% significance level. Finally, these predictors and their respective statistical weights were used to build an equation that calculated the risk of SCD in 5 years for an individual HCM patient. The model was internally validated using bootstrapping and incorporated in the 2014 ESC guidelines. Subsequently, an external validation of the asqHCM Risk-ICD model in patients undergoing alcohol septal ablation was performed using a database of 844 HCM patients from The Netherlands, and Kaplan–Meier estimates were compared with the different SCD risk prediction models. The discrimination of the HCM Risk-SCD model was significantly better than the 2014 ESC recommendations for primary prophylactic ICD implantation, with the risk factors LVWT $\geq 30$ mm and syncope after ASA retaining high specificity but lower sensitivity for predicting SCD.

### Table 2
Clinical characteristics of 844 HCM patients before ASA and (for patients without periprocedural SCD) at last follow-up before censoring

<table>
<thead>
<tr>
<th></th>
<th>Before ASA (n = 844)</th>
<th>After ASA (n = 824)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>56 ± 14</td>
<td>63 ± 14</td>
</tr>
<tr>
<td>LA diameter (mm)</td>
<td>47 ± 7</td>
<td>46 ± 7</td>
</tr>
<tr>
<td>LVOT gradient (mmHg)</td>
<td>95 ± 48</td>
<td>19 ± 27</td>
</tr>
<tr>
<td>Syncope</td>
<td>185 (22%)</td>
<td>71 (9%)</td>
</tr>
<tr>
<td>ICD</td>
<td>18 (2%)</td>
<td>29 (4%)</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation or n (%).

ICD, internal cardioverter defibrillator; LA, left atrium; LVOT, left ventricular outflow tract; ASA, alcohol septal ablation; SCD, sudden cardiac death.

### Table 3
Performance of SCD risk prediction models according to the 2014 ESC, 2003 ACC/ESC, and 2011 ACCF/AHA guidelines, and performance of the risk factors LVWT $\geq 30$ mm and syncope after ASA

<table>
<thead>
<tr>
<th>Model</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2014 ESC recommendation for primary ICD implantation</td>
<td>41</td>
<td>76</td>
<td>9</td>
<td>96</td>
</tr>
<tr>
<td>2003 ACC/ESC recommendation for primary ICD implantation</td>
<td>28</td>
<td>84</td>
<td>9</td>
<td>95</td>
</tr>
<tr>
<td>2011 ACCF/AHA recommendation for primary ICD implantation</td>
<td>50</td>
<td>65</td>
<td>8</td>
<td>96</td>
</tr>
<tr>
<td>LVWT $\geq 30$ mm</td>
<td>17</td>
<td>95</td>
<td>17</td>
<td>95</td>
</tr>
<tr>
<td>Syncope after ASA</td>
<td>23</td>
<td>91</td>
<td>13</td>
<td>95</td>
</tr>
</tbody>
</table>

ICD, internal cardioverter-defibrillator; NPV, negative predictive value; PPV, positive predictive value; ASA, alcohol septal ablation; LVWT, left ventricular wall thickness; ESC, European Society of Cardiology; ACC, American College of Cardiology; ACCF, American College of Cardiology Foundation; AHA, American Heart Association.
were found to have a high predicted risk (>6%/5 years) consistent with an ICD recommendation. In the latter report, however, no direct comparisons with SCD risk stratification according to the 2003 ACC/ESC and 2011 ACCF/AHA guidelines were made. Ruiz-Salas et al.\(^\text{14}\) conducted a study of 48 HCM patients who had received an ICD for primary SCD prevention, and found the HCM Risk-SCD model to be the only independent predictor of appropriate ICD therapy during a mean follow-up of 4 years. Also, none of the 11 patients with estimated low risk using the HCM Risk-SCD model experienced appropriate ICD therapy. Very recently, Fernández et al.\(^\text{15}\) conducted a fourth HCM Risk-SCD validation study in a South-American HCM population of 502 patients, and found the model to be an excellent predictor of SCD with a C-statistic of 0.93, compared with 0.76 and 0.71 when using the 2003 and 2011 models, respectively. These findings are in line with the present study which showed the HCM Risk-SCD model to be more predictive of SCD following ASA when compared with risk models used in previous guidelines. However, the performance of the HCM Risk-SCD model is far from perfect, and considering ICD implantation for primary prevention of SCD in patients with HCM obviously does not stop at a low calculated 5-year SCD risk. Therefore the model should be used, as also stated by the model developers, to complement clinical reasoning by providing objective individualized prognostic information.\(^\text{5}\) Moreover, the C-statistic of the HCM Risk-SCD model in the present study was lower compared with the original HCM Risk-SCD study (0.61 vs. 0.70, respectively). This might be explained by the fact that ASA can alter some of the risk factors in the HCM Risk-SCD model. Additional studies will be necessary to investigate if the HCM Risk-SCD model performs better when using parameters post-ASA.

**Limitations**

This study had several limitations. Patients who reached the SCD endpoint were censored at the time of its occurrence, and a distinction between periprocedural (<30 days) and long-term (>30 days) SCD was made to discern procedure-related adverse arrhythmic events caused by the ASA-induced myocardial infarction from long-term SCD risk. Consequently, 20 patients who reached the SCD endpoint within the first 30 days post-ASA were excluded from model validation. When the long-term follow-up data of these patients were included, however, a 2014 ESC recommendation for primary prophylactic ICD implantation according to the HCM Risk-SCD model remained a predictor for SCD (HR 2.00, 95% CI 1.13–3.56, \(P=0.02\)), and an indication for primary prophylactic ICD implantation according to the 2003 ACC/ESC and 2011 ACCF/AHA guidelines remained not predictive of SCD. The comparison of the different risk models was limited by the small number of SCD events. Finally, risk stratification was not completed in all patients, with one or two risk factors missing in 309 (37%) patients. However, since abnormal blood pressure response during exercise is not incorporated in the HCM Risk-SCD model, risk stratification for calculating the 5-year SCD risk according to the 2014 ESC guidelines was incomplete in only 222 (26%) patients. Consequently, more missing data were imputed using multiple imputations for calculating the C-statistics of the 2003 ACC/ESC and 2011 ACCF/AHA guidelines, compared with the HCM-Risk SCD model. The same approach to missing data was used in the HCM Risk-SCD study, in which risk stratification was incomplete in 22% of patients.\(^\text{5}\)

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**Figure 2** Kaplan–Meier graphs of sudden cardiac death (SCD) risk in 824 HCM patients following ASA, based on the 2014 European Society of Cardiology (ESC), the 2003 American College of Cardiology (ACC)/ESC, and the 2011 American College of cardiology Foundation (ACCF)/American Heart Association (AHA) guidelines. ICD, implantable cardioverter defibrillator.
Table 4  Univariate Cox regression model of predictors for SCD in 824 HCM patients

<table>
<thead>
<tr>
<th>Predictor</th>
<th>HR</th>
<th>95% CI</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>0.99</td>
<td>0.97–1.01</td>
<td>0.46</td>
</tr>
<tr>
<td>Female</td>
<td>1.20</td>
<td>0.68–2.15</td>
<td>0.53</td>
</tr>
<tr>
<td>LA diameter (mm)</td>
<td>1.04</td>
<td>1.00–1.08</td>
<td>0.07</td>
</tr>
<tr>
<td>LVWT &gt;30 mm</td>
<td>3.48</td>
<td>1.62–7.46</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Family history of SCD</td>
<td>0.91</td>
<td>0.41–2.04</td>
<td>0.82</td>
</tr>
<tr>
<td>NSVT</td>
<td>1.71</td>
<td>0.79–3.69</td>
<td>0.17</td>
</tr>
<tr>
<td>ABPR</td>
<td>1.17</td>
<td>0.45–3.01</td>
<td>0.75</td>
</tr>
<tr>
<td>Alcohol (mL)</td>
<td>0.92</td>
<td>0.71–1.19</td>
<td>0.51</td>
</tr>
<tr>
<td>CK-MB &gt;240 IU/L</td>
<td>1.32</td>
<td>0.52–3.39</td>
<td>0.56</td>
</tr>
<tr>
<td>LVOT gradient before ASA (mmHg)</td>
<td>1.00</td>
<td>1.00–1.01</td>
<td>0.60</td>
</tr>
<tr>
<td>LVOT gradient after ASA (mmHg)</td>
<td>1.00</td>
<td>0.99–1.01</td>
<td>0.94</td>
</tr>
<tr>
<td>Syncope before ASA</td>
<td>1.16</td>
<td>0.60–2.23</td>
<td>0.67</td>
</tr>
<tr>
<td>Syncope after ASA</td>
<td>2.88</td>
<td>1.37–6.06</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>2014 ESC recommendation for primary ICD implantation</td>
<td>1.96</td>
<td>1.09–3.54</td>
<td>0.02</td>
</tr>
<tr>
<td>2003 ACC/ESC recommendation for primary ICD implantation</td>
<td>1.74</td>
<td>0.89–3.39</td>
<td>0.10</td>
</tr>
<tr>
<td>2011 ACCF/AHA recommendation for primary ICD implantation</td>
<td>1.63</td>
<td>0.91–2.90</td>
<td>0.10</td>
</tr>
</tbody>
</table>

ABPR, abnormal blood pressure response to exercise; ASA, alcohol septal ablation; CI, confidence interval; CK, creatine kinase; HR, hazard ratio; LA, left atrium; LVOT, left ventricular outflow tract; LVWT, left ventricular wall thickness; NSVT, non-sustained ventricular tachycardia; SCD, sudden cardiac death; ICD, implantable cardioverter defibrillator; ESC, European Society of Cardiology; ACC, American College of Cardiology; ACCF, American College of Cardiology Foundation; AHA, American Heart Association.

Conclusion

The HCM Risk-SCD model can be used for SCD prediction in patients undergoing ASA.

Conflict of interest: none declared.

References