Establishment of a predictive model for inpatient sudden cardiac death in a Chinese cardiac department population: a retrospective study

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Abstract

Background: Little is known about the risk factors for sudden cardiac death (SCD) in the overall hospitalized cardiac department population. This study was conducted to investigate the risk factors and develop a predictive model for SCD in a hospitalized cardiac department population.

Methods: We conducted a retrospective study of patients admitted to the cardiac department of the First Affiliated Hospital of Xinjiang Medical University from June 2015 to February 2017. We collected the clinical data from medical records. Multiple stepwise logistic regression analysis was carried out to confirm the risk factors for SCD and develop a predictive risk model. The risk score was assessed by the area under receiver operating characteristic (AUROC) curve and the Hosmer-Lemeshow goodness-of-fit test.

Results: A total of 262 patients with SCD and 4485 controls were enrolled in our study. Logistic regression modeling identified eight significant risk factors for in-hospital SCD: age, main admitting diagnosis, diabetes, corrected QT interval, QRS duration, ventricular premature beat burden, left ventricular ejection fraction, and estimated glomerular filtration rate. A predictive risk score including these variables showed an AUROC curve of 0.774 (95% confidence interval: 0.744–0.805). The Hosmer-Lemeshow goodness-of-fit test showed the chi-square value was 2.527 (P = 0.640). The incidence of in-hospital SCD was 1.3%, 4.1%, and 18.6% for scores of 0 to 2, 3 to 5 and ≥6, respectively (P < 0.001).

Conclusions: Age, main admitting diagnosis, diabetes, QTc interval, QRS duration, ventricular premature beat burden, left ventricular ejection fraction, and estimated glomerular filtration rate are factors related to in-hospital SCD in a hospitalized cardiac department population. We developed a predictive risk score including these factors that could identify patients who are predisposed to in-hospital SCD.

Keywords: Sudden cardiac death; inpatient; risk factors; predictive risk score

Introduction

Sudden cardiac death (SCD) is defined as an unexpected natural death attributable to cardiac reasons that usually takes place within 1 h of the onset of symptoms.[1] Today, SCD is a global public health problem that affects both developed countries and developing countries.[2] In the United States, the overall prevalence rate of SCD is 55 per 100,000 per year in the general population.[3] In China, this rate is 41.8 per 100,000 per year.[4] SCD leads to approximately 3.7 million deaths annually worldwide and accounts for 15% to 20% of all deaths.[2]

SCD continues to raise considerable concern worldwide. Most SCD cases occur in the general population without any prior warning.[3] Previous studies have also focused on the prevention of SCD in the general population. Many large population-based epidemiological studies have identified the risk factors for SCD in the general population and even developed predictive risk stratification models.[1,3] For predicting the risk of SCD in patients with diagnosed cardiovascular diseases, most studies have concentrated on specific populations, such as patients with hypertrophic cardiomyopathy[5] and patients with coronary heart disease (CHD).[6] However, the risk factors for SCD in the overall hospitalized cardiac department population are poorly understood, and thus far, there is no risk score available in Asia. The lack of identification and stratification of patients at high risk of SCD will lead to serious consequences, such as high medical costs and...
death. Therefore, a predictive risk score for in-hospital SCD that can be used clinically is necessary. Our aim was to determine the risk factors for in-hospital SCD in the cardiac department population and develop a predictive risk score using conventional and low-cost clinical information. Initial diagnosis and assignment of an early risk stratification score can help doctors to promptly identify patients who are likely to present with SCD and provide better treatment and care to reduce mortality.

Methods

Ethical approval

This study protocol was reviewed and approved by the Institutional Review Board of the First Affiliated Hospital of Xinjiang Medical University (Ethical Approval Number: 20150130-01) and conformed to the principles and guidelines of the Declaration of Helsinki. As a retrospective study, this study was exempt from the informed consent from patients.

Study design and population

We performed a retrospective, single center study over a 2-year period from June 2015 to February 2017. A total of 262 hospitalized patients died of SCD and 4485 control patients from the cardiology department of the First Affiliated Hospital of Xinjiang Medical University were eventually enrolled in our study [Figure 1]. The exclusion criteria were as follows: incomplete clinical data; implantation with implantable cardioverter debrillator (ICD) or cardiac resynchronization therapy-defibrillator; and severe systemic organ diseases such as infectious disease, malignant tumor, or other serious devastating diseases.

The outcome in our study was that SCD is defined as death within 1 h of the onset of symptoms. The timing of symptoms before death was determined from the rescue records and death information in the medical records. Because in-patients with severe condition generally have ECG monitoring and more nursing care compared with out-of-hospital patients, the symptoms are easier to be mastered. The symptoms in our study were defined as sudden loss of consciousness, abrupt blood pressure drop less than 90/60 mmHg (1 mm Hg = 0.133 kPa), sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) shown by ECG monitoring, cardiac arrest shown by ECG monitoring, and escape rhythm shown by ECG monitoring. The cause of death for each case was verified by an experienced and professional team of cardiologists. Patients identified with a specific non-cardiac cause of death were excluded.

Data collection

The potential clinical risk markers chosen for predictive risk score development were from the published literature. To expand the applicability of the risk model, especially in areas with insufficient medical resources, potential risk markers were chosen from conventional and low-cost clinical examinations. All data were collected on admission. We collected clinical data from the medical records of the study population, including demographic characteristics, lifestyle, medical history, physical examination, 12-lead ECG, 24-h Holter, 2-dimensional echocardiography, and blood laboratory testing.

Age, gender, and ethnicity were recorded from the identification card of the patients. Smoking, drinking habits, family history of SCD, and recent hospitalization were determined by self-report. The history of hypertension and diabetes mellitus (DM) were identified by the combination of self-report and clinical information from previous and present hospitalization. Height and weight were measured using standard and calibrated instruments, and body mass index (BMI) was calculated as weight (in kilograms) divided by height (in meters) squared. The heart rate, corrected QT (QTc) interval, QRS duration, and J-point were confirmed by the first ECG after hospitaliza-
Diagnosis standard

Current smokers were defined as smoking at least one cigarette per day for more than 6 months. Former smokers were defined as having stopped smoking for more than 6 months. Current drinkers were defined as consuming alcohol at least once per week for more than 6 months. Former drinkers were defined as having stopped drinking alcohol for more than 6 months. Family history of SCD was defined as SCD occurrence among any family member. Hypertension was defined as a systolic blood pressure of ≥ 140 mm Hg, a diastolic blood pressure of ≥ 90 mm Hg, or the use of antihypertensive drugs. DM was defined as a fasting glucose of ≥ 7.0 mmol/L (126 mg/dL), non-fasting glucose of ≥ 11.1 mmol/L (200 mg/dL), or the use of hypoglycemic medications. Post myocardial infarction (MI) was defined as a diagnosis of MI more than 30 days previously or a remote MI shown in an ECG during hospitalization. VPB burden was calculated as the number of VPBs divided by the total heart beats on a 24-h Holter monitor. NSVT was defined as ≥3 consecutive ventricular beats at ≥120 beats/min and lasting <30 s.

Data analysis

SPSS version 22.0 software (SPSS Inc., Chicago, IL, USA) was used to analyze the data. Continuous data were presented as the means ± standard deviation (SD) or median and interquartile range (25th, 75th percentiles) and were compared by t-test analysis or Wilcoxon log-rank tests. Categorical data were presented as the frequencies and percentages of the total in each category and were compared with the Pearson chi-square test. Variables that reached statistical significance in analyses comparing SCD cases and controls were included in a multivariate Logistic regression analysis to identify the risk factors for in-hospital SCD. In the multivariate Logistic regression analysis, continuous variables were grouped into convenient categories. The risk factors were presented as odds ratios (ORs) and 95% confidence intervals (95% CIs). A forward procedure with the Wald test was used to determine the best model. The Hosmer-Lemeshow goodness-of-fit test was used to assess the model fit. All tests were two-sided, and a P value of <0.05 was considered as statistically significant.

Results

Comparison of SCD cases with controls

The cohort included 262 SCD cases and 4485 control cases. The clinical characteristics of the patients who developed SCD and those who did not develop SCD are delineated in Table 1. No differences in gender, drinking, family history of SCD, BMI, J-point, AST, UA, TG, TC, LDL-C, HDL-C or hemoglobin were observed between SCD cases and controls (all P > 0.05). SCD cases were older, had a greater Han ethnicity rate, smoking rate, recent hospitalization rate, hypertension rate, DM rate, heart rate, QTc interval, QRS duration, number of VPBs, NSVT, and ALT compared with controls. SCD cases had lower levels of LVEF and eGFR than controls. The main admitting diagnosis was different between the two groups.

Risk factors for in-hospital SCD

Table 2 shows the value assignment of variables that reached statistical significance in the analyses comparing SCD cases and controls. Table 3 shows the multiple logistic regression analyses of in-hospital SCD. Multiple logistic regression analysis showed that age, main admitting diagnosis, DM, QTc interval, QRS duration, VPB burden, LVEF, and eGFR were independent significant predictive factors related to in-hospital SCD.

Predictive risk score building and verification

According to the results of the multiple Logistic regression analysis, we established a clinical risk score for predicting inpatient SCD, as shown in Table 4. Each risk factor was assigned a value ranging from 1 to 3 points, and the maximum total score for one patient was 11 points. The incidence of in-hospital SCD showed an upward trend with an increasing risk score [Figure 2]. The AUROC curve value was 0.774 (95% CI: 0.744–0.805) [Figure 3]. The Hosmer-Lemeshow goodness-of-fit test showed a chi-square value of 2.527 and a P value of 0.640.

Risk score stratification

Patients were stratified into three risk groups: low (0–2 points), intermediate (3–5 points), and high (6 or more points). As shown in Table 5, the incidence of in-hospital SCD was 1.3%, 4.1%, and 18.6% in the low-, intermediate-, and high-risk group, respectively (P < 0.001). Logistic regression analysis showed that the intermediate-risk group was associated with an OR of 3.125 (95% CI: 1.942–5.028, P < 0.001), and in the high-risk group, the risk of SCD was increased to OR 16.866 (95% CI: 10.569–26.914, P < 0.001).

Discussion

This study investigated the risk factors and established a predictive risk score of SCD in a hospitalized Chinese cardiac department population. Our predictive risk score provides a practical, conventional, non-invasive and low-cost method to help doctors identify those hospitalized
Table 1: The clinical characteristics of controls and SCD cases

| Characteristics                        | SCD cases (n = 262) | Controls (n = 4485) | Statistics | P   |
|----------------------------------------|---------------------|---------------------|------------|-----|
| Age (years), mean ± SD                 | 67.79 ± 12.68       | 56.69 ± 10.43       | 16.328†    | <0.001 |
| Gender (male), n (%)                   | 163 (62.2)          | 2601 (58.0)         | 1.813†     | 0.178  |
| Ethnicity, n (%)                       |                     |                     | 12.455†    | <0.001 |
| Han                                    | 209 (79.8)          | 3117 (69.5)         |            |      |
| Other                                  | 53 (20.2)           | 1368 (30.5)         |            |      |
| Main admitting diagnosis, n (%)        |                     |                     | 138.442†   | <0.001 |
| Heart failure exacerbation             | 70 (26.7)           | 871 (19.4)          |            |      |
| Post MI or unstable angina             | 39 (14.9)           | 1421 (31.7)         |            |      |
| AMI within 30 days                     | 141 (53.8)          | 1159 (25.8)         |            |      |
| Other                                  | 12 (4.6)            | 1034 (23.1)         |            |      |
| Smoking, n (%)                         |                     |                     | 36.790†    | <0.001 |
| Never                                  | 218 (83.2)          | 3897 (86.9)         |            |      |
| Current                                | 30 (11.5)           | 807 (18.0)          |            |      |
| Former                                 | 14 (5.3)            | 202 (4.5)           |            |      |
| Family history of SCD, n (%)           | 4 (1.5)             | 51 (1.1)            | 0.328†     | 0.567  |
| Hospitalization within 1 month, n (%)  | 39 (14.9)           | 364 (8.1)           | 14.602†    | <0.001 |
| Hypertension, n (%)                    | 199 (76.0)          | 2868 (63.9)         | 15.609†    | <0.001 |
| DM, n (%)                              | 103 (39.3)          | 1359 (30.3)         | 9.433†     | 0.002  |
| BMI (kg/m²), mean ± SD                 | 25.04 ± 4.10        | 25.42 ± 3.11        | -1.885‡    | 0.060  |
| Heart rate (beats/min), mean ± SD      | 89.98 ± 21.21       | 83.06 ± 18.29       | 5.897*     | <0.001 |
| QTc interval (ms), mean ± SD           | 452.85 ± 67.24      | 436.18 ± 57.78      | 4.496*     | <0.001 |
| QRS duration (ms), mean ± SD           | 117.58 ± 30.26      | 106.29 ± 24.25      | 7.215*     | <0.001 |
| J-point, n (%)                         |                     |                     | 3.975†     | 0.137  |
| Normal                                 | 237 (90.5)          | 4157 (92.7)         |            |      |
| Elevation>1 mm                         | 23 (8.8)            | 318 (7.1)           |            |      |
| Decline≥1 mm                           | 2 (0.8)             | 10 (0.2)            |            |      |
| Number of VPBs, median (P25, P75)      | 3259 (803, 8472)    | 415 (98, 1024)      | 14.119†    | <0.001 |
| NSVT, n (%)                            | 50 (19.1)           | 188 (4.2)           | 115.278†   | <0.001 |
| LVEF (%), mean ± SD                    | 46.29 ± 12.33       | 56.18 ± 10.65       | -14.476*   | <0.001 |
| ALT (U/L), mean ± SD                   | 32.15 ± 11.79       | 25.32 ± 6.23        | 16.141†    | <0.001 |
| AST (U/L), mean ± SD                   | 34.40 ± 10.83       | 33.95 ± 8.92        | 0.784      | 0.433  |
| UA (μmol/L), mean ± SD                 | 384.36 ± 69.30      | 383.23 ± 50.18      | 0.407      | 0.684  |
| TG (mmol/L), mean ± SD                 | 1.35 ± 0.40         | 1.39 ± 0.33         | -1.883*    | 0.060  |
| TC (mmol/L), mean ± SD                 | 4.01 ± 1.13         | 3.92 ± 0.99         | 1.419*     | 0.136  |
| LDL-C (mmol/L), mean ± SD              | 2.94 ± 1.27         | 2.91 ± 1.04         | 0.448*     | 0.634  |
| HDL-C (mmol/L), mean ± SD              | 0.95 ± 0.38         | 0.98 ± 0.29         | -1.596*    | 0.110  |
| Haemoglobin (g/L), mean ± SD           | 118.29 ± 29.46      | 119.86 ± 20.77      | -1.158*    | 0.247  |
| cGFR (ml/min per 1.73 m²), mean ± SD   | 76.93 ± 24.38       | 91.48 ± 31.74       | -7.295*    | <0.001 |

* t values, † Z values, ‡ p values. ALT: Alanine transferase; AMI: Acute myocardial infarction; AST: Aspartate aminotransferase; BMI: Body mass index; DM: Diabetes mellitus; eGFR: Estimated glomerular filtration rate; HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; LVEF: Left ventricular ejection fraction; MI: Myocardial infarction; NSVT: Non-sustained ventricular tachycardia; SCD: Sudden cardiac death; TC: Total cholesterol; TG: Triglyceride; UA: Uric acid; VPBs: Ventricular premature beats.

Patients most likely to have SCD. Data for the eight independent clinical predictive factors are not difficult to collect from primary medical institutions. Clinicians can apply this predictive tool to improve decision-making and provide the best treatment for patients. Moreover, this predictive risk score is a supplement to current ICD guidelines. Even if ICD therapy is not indicated for a patient, but the patient was confirmed as high risk based on our risk score stratification, the doctor should pay more attention since he/she had a higher risk of SCD than other patients. Multivariate analysis identified eight independent factors predictive of SCD among hospitalized patients, and our findings highlight the importance of history of CHD, cardiac systolic dysfunction, and abnormal cardiac electrical activity. CHD was the most common disease contributing to SCD, and in our study, a history of CHD was associated with SCD in the majority of cases, contributing to SCD.


| Variables | Value assignment |
|-----------|-----------------|
| Age (years) | <45 = 0, 45–64 = 1, ≥65 = 2 |
| Ethnicity | Han = 0, Other = 1 |
| Main admitting diagnosis | Other = 1, Heart failure exacerbation = 2, Post MI or unstable angina = 3, AMI within 30 days = 4 |
| Smoking | Never = 0, Current = 1, Former = 2 |
| Hospitalization within 1 month | No = 0, Yes = 1 |
| Hypertension | No = 0, Yes = 1 |
| DM | No = 0, Yes = 1 |
| Heart rate (beats/min) | <100 = 0, ≥100 = 1 |
| QTc interval (ms) | ≤450/460 (men/women) = 0, >450/460 (men/women) = 1 |
| QRS duration (ms) | ≤150 = 0, >150 = 1 |
| VPB burden | ≤20% = 0, >20% = 1 |
| NSVT | No = 0, Yes = 1 |
| LVEF (%) | ≥40 = 0, 25–39 = 2, <25 = 1 |
| ALT (UL) | ≤50 = 0, ≥50 = 1 |
| eGFR (mL/min per 1.73 m²) | ≥40 = 0, <40 = 1 |

ALT: Alanine transaminase; AMI: Acute myocardial infarction; DM: Diabetes mellitus; eGFR: Estimated glomerular filtration rate; LVEF: Left ventricular ejection fractions; MI: Myocardial infarction; NSVT: Non-sustained ventricular tachycardia; VPB: Ventricular premature beat.

Table 2: Value assignment of variables

Table 4: The risk score of each factor for predicting inpatient SCD

| Risk factors | Integer coefficient |
|-------------|---------------------|
| Age ≥ 65 years | 1 point |
| Main admitting diagnosis | |
| Heart failure exacerbation | 2 points |
| Post MI or unstable angina | 1 point |
| AMI within 30 days | 3 points |
| DM | 1 point |
| QRS duration >150 ms | 1 point |
| QTc interval >450/460 ms (men/women) | 1 point |
| LVEF | |
| 25–39% | 1 point |
| <25% | 2 points |
| VPB burden >20% | 1 point |
| eGFR <40 mL/min per 1.73 m² | 1 point |

AMI: Acute myocardial infarction; DM: Diabetes mellitus; eGFR: Estimated glomerular filtration rate; LVEF: Left ventricular ejection fractions; MI: Myocardial infarction; OR: Odds ratio; SCD: Sudden cardiac death; VPB: Ventricular premature beat.

It had been the best-known risk factor for overall mortality and SCD due to progressive heart failure and ventricular arrhythmias. In addition, our study showed that the risk of in-hospital SCD increased with a decrease in LVEF, which was consistent with previous studies showing that the severity of heart failure and the degree of left ventricular systolic dysfunction were predictors of SCD. VT and VF had been proven to be the most common causes of out-of-hospital SCD. In our study, three abnormal cardiac electrical activity risk factors in ECG and 24-h Holter, prolonged QTc interval, QRS duration, and high rate of VPBs suggested a higher possibility of in-hospital SCD. The QT interval reflected the summed ventricular action potential durations.

Table 3: The risk factors for inpatient SCD

| Variables | β | SE | Wald | P | OR | 95% CI |
|-----------|---|----|------|---|----|-------|
| Age (years) | | | | | | |
| <45 | 0.080 | 0.141 | 0.320 | 0.571 | 1.083 | 0.82–1.43 |
| 45–64 | 0.716 | 0.144 | 24.794 | <0.001 | 2.047 | 1.54–2.71 |
| ≥65 | | | | | | |
| Main admitting diagnosis | | | | | | |
| Other | | | | | | |
| Heart failure exacerbation | 1.114 | 0.295 | 14.295 | <0.001 | 3.045 | 1.71–5.43 |
| Post MI or unstable angina | 0.601 | 0.227 | 7.013 | 0.008 | 1.824 | 1.17–2.85 |
| AMI within 30 days | 1.758 | 0.266 | 43.830 | <0.001 | 5.802 | 3.45–9.76 |
| DM | 0.547 | 0.155 | 12.503 | <0.001 | 3.439 | 2.82–4.23 |
| QTc interval | 0.775 | 0.136 | 32.543 | <0.001 | 2.170 | 1.66–2.83 |
| QRS duration | 0.721 | 0.143 | 23.533 | <0.001 | 2.057 | 1.56–2.72 |
| VPB burden | 0.536 | 0.242 | 4.931 | 0.026 | 1.710 | 1.07–2.75 |
| LVEF (%) | | | | | | |
| ≥40 | 0.594 | 0.288 | 4.424 | 0.039 | 1.811 | 1.03–3.19 |
| 25–39 | 1.263 | 0.236 | 28.578 | <0.001 | 3.536 | 2.23–5.62 |
| <25 | 0.622 | 0.297 | 4.380 | 0.036 | 1.863 | 1.04–3.34 |

AMI: Acute myocardial infarction; CI: Confidence interval; DM: Diabetes mellitus; eGFR: Estimated glomerular filtration rate; LVEF: Left ventricular ejection fractions; MI: Myocardial infarction; OR: Odds ratio; SCD: Sudden cardiac death; VPB: Ventricular premature beat.
Although the QT interval could be affected by LVEF, severity of CHD, and ventricular arrhythmia in hospitalized patients, it was still significant in our results.\textsuperscript{[14]} Prolonged QRS duration was a reflection of intraventricular conduction delay and abnormal repolarization, thus resulting in a decline in cardiac function and facilitation of re-entrant tachyarrhythmias.\textsuperscript{[15]} VPBs and NSVT were reflections of ventricular automaticity enhancement. Our study showed that NSVT was associated with SCD risk in the unadjusted comparison analysis but was not significant in the multiple adjusted analysis, and a high rate of VPBs (>20\%) eventually entered our risk score.

The remaining three components of the risk score were age, DM, and reduced eGFR. Epidemiological investigation indicated that the incidence of SCD increased with age regardless of gender or race.\textsuperscript{[1]} The elderly population had degeneration of both reserve capacity and ability to withstand stress, which might lead to the high incidence of SCD. DM could contribute to cardiac ischemia, myocardial damage and scar formation and heterogeneity in atrial and ventricular repolarization, thus increasing the risk of SCD.\textsuperscript{[16]} Reduced eGFR was a reflection of impaired kidney function, which had been proven to be associated with a significantly elevated risk of SCD in the general population.\textsuperscript{[5]}

There are several differences between our results and those of previous prediction model and risk scores for the general population.\textsuperscript{[5,17,18]} We believe that two reasons might explain these differences. First, the physical condition and underlying illnesses of the hospitalized population were different from the general population, and the risk factors were varied. For example, most SCD victims in the general population did not have a pre-existing history of heart disease. A low LVEF was present in only 1\% of participants in Rajat Deo \textit{et al}’s paper\textsuperscript{[5]} and did not enhance SCD prediction in the general population, which limits the sensitivity of this technique. However, nearly 16\% of control cases and 80\% of SCD cases were diagnosed with decreased LVEF (<50\%), and a low LVEF was an important risk factor in our model. Second, for the general population, routine screening with...
echocardiography and 24-h Holter was difficult and expensive. However, in hospitalized patients, echocardiography and 24-h Holter were essential and routine. This might explain why the high occurrence of VPBs in the 24-h Holter monitor was a significant risk factor in our model.

**Study limitations**

This study had several limitations. First, it was a retrospective study with limitations inherent in this type of design, and the predictive ability of the risk score was not as accurate as a prospective study. In addition, monitoring these clinical data long-term was superior to obtaining just a single measurement because these risk factors change over time. Second, none of the deceased patients underwent autopsy due to our national conditions. Third, the modeling cohort was not from multiple centers and might not be representative of hospitalized patients in China. Further validation in a different population was required before the predictive risk score could be applied in clinical practice. Fourth, the study population was restricted to those with complete information available, which might lead to some bias in patient selection. Fifth, some risk factors that had been verified to be related to SCD in previous studies were not included in the database for this study, which might lead to some bias in the risk model.

**Conclusions**

In conclusion, we established a predictive risk score for in-hospital SCD in a hospitalized population, including age, main admitting diagnosis, DM, QTc interval, QRS duration, VPB burden, LVEF, and eGFR. These findings might help doctors in primary medical institutions to identify hospitalized patients who are expected to develop SCD.

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**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Deo R, Albert CM. Epidemiology and genetics of sudden cardiac death. *Circulation* 2012;125:620–637. doi: 10.1161/circulationaha.111.023838.

2. Mehrer R. Global public health problem of sudden cardiac death. *J Electrocardiol* 2007;40:S118–S122. doi: 10.1016/j.jelectrocard.2007.06.023.

3. Chugh SS, Jui J, Gunson K, Stecker EC, John BT, Thompson B, et al. Current burden of sudden cardiac death: multiple source surveillance versus retrospective death certificate-based review in a large U.S. community. *J Am Coll Cardiol* 2004;44:1268–1275. doi: 10.1016/j.jacc.2004.06.029.

4. Hua W, Zhang LF, Wu YF, Liu XQ, Guo DS, Zhou HL, et al. Incidence of sudden cardiac death in China: analysis of 4 regional populations. *J Am Coll Cardiol* 2009;54:1110–1118. doi: 10.1016/j.jacc.2009.09.016.

5. Deo R, Norby FL, Katz R, Sotoodehnia N, Adabag S, DeFilippi CR, et al. Development and validation of a sudden cardiac death prediction model for the general population. *Circulation* 2016;134:806–816. doi: 10.1161/circulationaha.116.023042.

6. O’Mahony C, Jichi F, Pavlou M, Monserrat L, Anastasakis A, R apezzi C, et al. A novel clinical risk prediction model for sudden cardiac death in hypertrophic cardiomyopathy (HCM risk-SCD). *Eur Heart J* 2014;35:2010–2020. doi: 10.1093/eurheartj/het439.

7. El-Sherif N, Boutjdir M, Turitto G. Sudden cardiac death in ischemic heart disease: pathophysiology and risk stratification. *Cardio Electrophysiol Clin* 2017;9:681–691. doi: 10.1016/j.ccep.2017.08.003.

8. Ma YC, Zuo L, Chen JH, Luo Q, Yu XQ, Li Y, et al. Modified glomerular filtration rate estimating equation for Chinese patients with chronic kidney disease. *J Am Soc Nephrol* 2006;17:2937–2944. doi: 10.1681/asn.2006043638.

9. Sullivan LM, Massaro JM, D’Agostino RB Sr. Presentation of multivariate data for clinical use: The Framingham Study risk score functions. *Stat Med* 2004;23:1631–1660. doi: 10.1002/sim.1742.

10. Solomon SD, Zelenko夫ski S, McMurray JJ, Finn PV, Velazquez E, Ersl G, et al. Sudden death in patients with myocardial infarction and left ventricular dysfunction, heart failure, or both. *N Engl J Med* 2005;352:2581–2588. doi: 10.1056/NEJMoa043938.

11. Goldberger JJ, Can ME, Mohlerou SH, Kadish AH, Knight BP, Lauer MS, et al. American Heart Association/American College of Cardiology Foundation/Heart Rhythm Society Scientific Statement on Noninvasive Risk Stratification Techniques for Identifying Patients at Risk for Sudden Cardiac Death. A scientific statement from the American Heart Association Council on Clinical Cardiology Committee on Electrocardiography and Arrhythmias and Council on Epidemiology and Prevention. *J Am Coll Cardiol* 2008;52:1179–1199. doi: 10.1016/j.jacc.2008.05.003.

12. Epstein AE, DeMarco JP, Ellenbogen KA, Estes NA 3rd, Freedman RA, Gettes LS, et al. 2012 ACCF/AHA/HRS focused update incorporated into the ACCF/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol* 2013;61:e6–75. doi: 10.1016/j.jacc.2012.11.007.

13. John RM, Tedrow UB, Koplan BA, Albert CM, Epstein LM, Sweeney MO, et al. Ventricular arrhythmias and sudden cardiac death. *Lancet* 2012;380:1520–1529. doi: 10.1016/s0140-6736(12)64143-5.

14. Williams ES, Thomas KL, Broderick S, Shaw LK, Velazquez EJ, Al-Khatib SM, et al. Race and gender variation in the QT interval and its association with mortality in patients with coronary artery disease: results from the Duke Databank for Cardiovascular Disease.
Teodorescu C, Reinier K, Uy-Evanado A, Navarro J, Mariani R, Gunson K, et al. Prolonged QRS duration on the resting ECG is associated with sudden death risk in coronary disease, independent of prolonged ventricular repolarization. Heart Rhythm 2011;8:1562–1567. doi: 10.1016/j.hrthm.2011.06.011.

Vasiliadis I, Kolovou G, Mavrogeni S, Nair DR, Mikhailidis DP. Sudden cardiac death and diabetes mellitus. J Diabetes Complications 2014;28:573–579. doi: 10.1016/j.jdiacomp.2014.02.003.

Aro AL, Reinier K, Rusinaru C, Uy-Evanado A, Darouian N, Phan D, et al. Electrical risk score beyond the left ventricular ejection fraction: prediction of sudden cardiac death in the Oregon Sudden Unexpected Death Study and the Atherosclerosis Risk in Communities Study. Eur Heart J 2017;38:3017–3025. doi: 10.1093/eurheartj/ehx331.

Fishman GI, Chugh SS, Dumarco JP, Albert CM, Anderson ME, Bonow RO, et al. Sudden cardiac death prediction and prevention: report from a National Heart, Lung, and Blood Institute and Heart Rhythm Society Workshop. Circulation 2010;122:2335–2348. doi: 10.1161/circulationaha.110.976092.

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