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.[1,5] 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[6] and patients with coronary heart disease (CHD).[7] 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
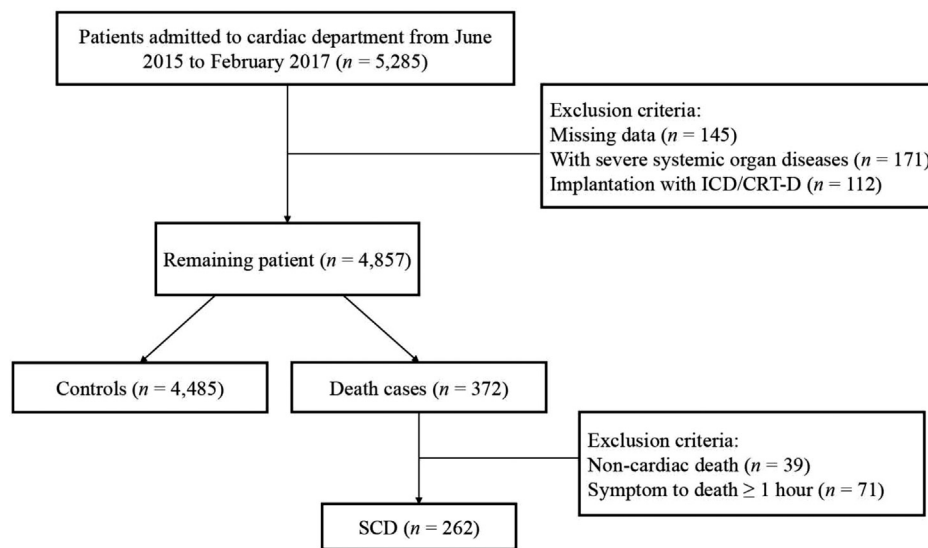
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Figure 1: Flow diagram describing the study population. CRT-D: Cardiac resynchronization therapy defibrillator; ICD: Implantable cardioverter defibrillator; SCD: Sudden cardiac death.

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 defibrillator (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-

tion. Ventricular premature beats (VPBs) and non-sustained ventricular tachycardia (NSVT) were measured using a 24-h Holter monitor. Left ventricular ejection fractions (LVEF) were determined by echocardiography. Alanine transferase (ALT), aspartate aminotransferase (AST), uric acid (UA), triglyceride (TG), total cholesterol (TC), low-density lipoprotein-cholesterol (LDL-C), high-density lipoprotein-cholesterol (HDL-C), hemoglobin, and creatinine levels were obtained from blood laboratory testing, and estimated glomerular filtration rate (eGFR) was calculated by the MDRD Study China equation.[8]

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 $\geq$ 140 mm Hg, a diastolic blood pressure of $\geq$ 90 mm Hg, or the use of antihypertensive drugs. DM was defined as a fasting glucose of $\geq$ 7.0 mmol/L (126 mg/dL), non-fasting glucose of $\geq$ 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 $\geq$3 consecutive ventricular beats at $\geq$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 $\pm$ 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. We used the point system developed by Sullivan et al[9] to assign a score for every risk factor. The performance and calibration of the risk score was assessed using the area under receiver operating characteristic (AUROC) curve and the Hosmer-Lemeshow goodness-of-fit test. Logistic regression analysis was used to analyze the risk of in-hospital SCD by risk score stratification. 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	16.528*	<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	171 (65.3)	3382 (75.4)		
Current	48 (18.3)	807 (18.0)		
Former	43 (16.4)	296 (6.6)		
Drinking, *n* (%)			3.056†	0.217
Never	218 (83.2)	3897 (86.9)		
Current	30 (11.5)	386 (8.6)		
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/m^2), 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 (P$_{25}$, P$_{75}$)	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	384.56 ± 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*	0.156
LDL-C (mmol/L), mean ± SD	2.94 ± 1.27	2.91 ± 1.04	0.448*	0.654
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
eGFR (mL/min per 1.73 m^2), mean ± SD	76.93 ± 24.38	91.48 ± 31.74	−7.295*	<0.001

* *t* values. † χ^2 values. ‡ Z 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 fractions; 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, accounting for 68.7%.[1] This finding might also explain why family history of SCD for the inherited arrhythmogenic diseases and cardiomyopathy, which were only present in a small proportion of the hospitalized popula-

Table 2: Value assignment of variables

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 (U/L)	<50 = 0, ≥50 = 1
eGFR (mL/min per 1.73 m^2)	≥40 = 0, <40 = 1

ALT: Alanine transferase; 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 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 1.73m^2	1 point

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

tion, did not enter our risk score. A previous study[10] and our study also showed that the risk of SCD was highest in the first 30 days after MI. The earlier period after MI had the greatest absolute risk, and the risk declined significantly over time, achieving a steady state until approximately 1 year.[10] A low LVEF was a clear sign of pump failure, and it had been the best-known risk factor for overall mortality and SCD due to progressive heart failure and ventricular arrhythmias.[11] 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.[12] VT and VF had been proven to be the most common causes of out-of-hospital SCD.[13] 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					1	
45–64	0.080	0.141	0.320	0.571	1.083	0.82–1.43
≥65	0.716	0.144	24.794	<0.001	2.047	1.54–2.71
Main admitting diagnosis						
Other					1	
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	1.729	1.28–2.34
QTc interval	0.775	0.136	32.543	<0.001	2.170	1.66–2.83
QRS duration	0.721	0.143	25.553	<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					1	
25–39	0.594	0.288	4.242	0.039	1.811	1.03–3.19
<25	1.263	0.236	28.578	<0.001	3.536	2.23–5.62
eGFR	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.
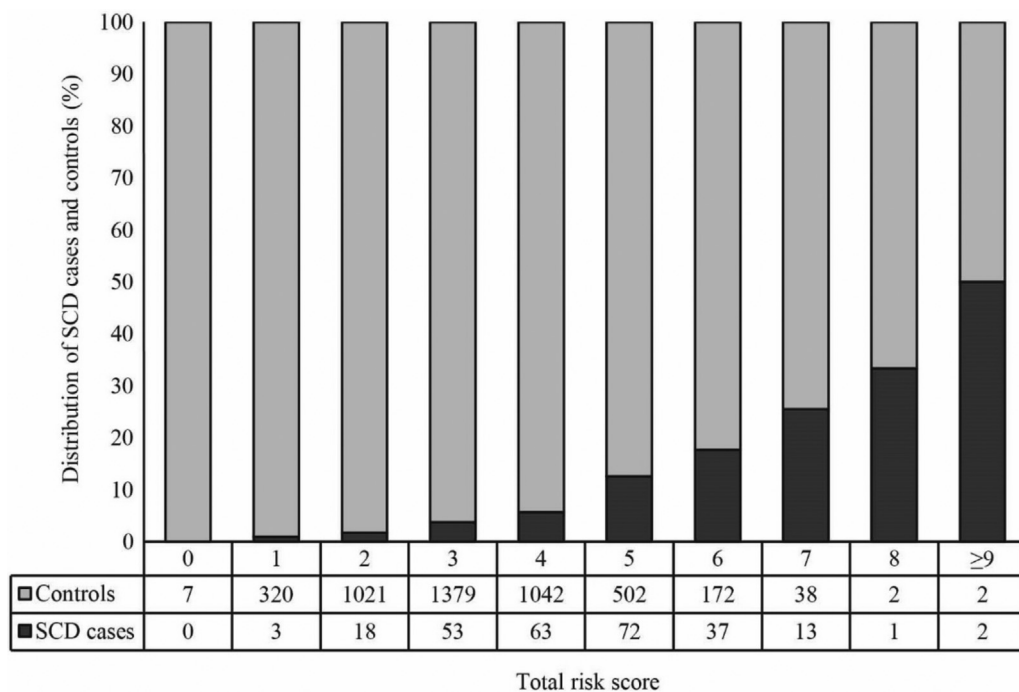
Figure 2: The numbers and distribution of in-hospital SCD cases and controls for each integer of the risk score. SCD: Sudden cardiac death.

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.[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.[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.[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.[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.[5]
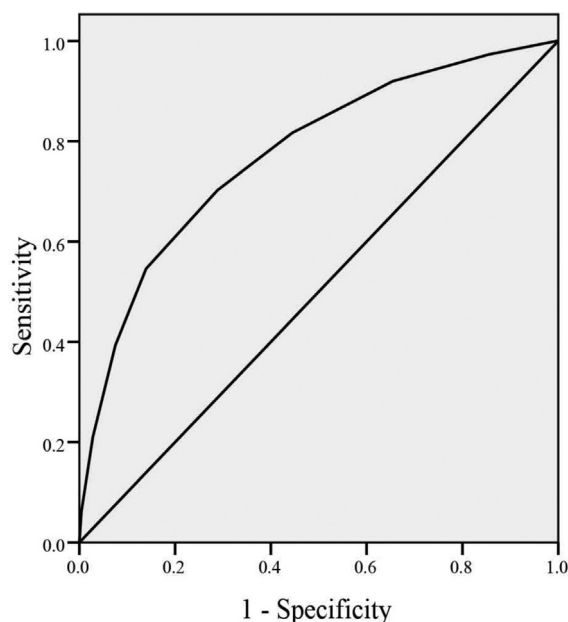
There are several differences between our results and those of previous prediction model and risk scores for the general population.[5,17,18] We believe that two reasons might explain these differences. First, the physical condition and underlying illnesses of the hospitalized population were different 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 *et al*'s paper[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

Figure 3: Receiver operating characteristic curve analysis of the risk score in predicting in-hospital SCD. SCD: Sudden cardiac death.

Table 5: Distribution and logistic regression analysis of inpatient SCD by risk score stratification

SCD risk score	Patients, *n*	SCD cases, *n* (%)	Controls, *n* (%)	*OR* (95% CI)
0–2 points	1569	21 (1.3)	1548 (98.7)	1
3–5 points	2410	98 (4.1)	2312 (95.9)	3.125 (1.942–5.028)
≥6 points	768	143 (18.6)	625 (81.4)	16.866 (10.569–26.914)

CI: Confidence interval; OR: Odds ratio; SCD: Sudden cardiac death.

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.

Conflicts of interest

There are no conflicts of interest.

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