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A randomized controlled trial of the short-term effect of rosuvastatin on the corrected QT interval

Lijun Zhu, Wenjun Shen, Wanying Hu, Haiwang Guan, Yanwei Wang, Conglin Wang, Jing Lin, Li Chen & Qingjun Jiang✉

Rosuvastatin can block human ether-a-go-go related gene (hERG) currents and prolong the corrected QT (QTc) interval, but this effect has not been confirmed in the population. This study compared the changes of QTc interval between populations receiving atorvastatin and rosuvastatin, explained the effect of rosuvastatin on QTc interval, and the correlation between rosuvastatin and QT prolongation. The QTc interval decreased by 0.83 ms from baseline in atorvastatin group and increased by 6.57 ms in rosuvastatin group. More patients in the rosuvastatin group had an increased QTc interval (62.7% vs. 46.6%, p< 0.001). Rosuvastatin increased the risk of newly emerged QT prolongation by 42% (95% CI 1.10–1.85, $p = 0.008$). But there was no correlation between rosuvastatin and severe QT prolongation (RR 1.23, 95% CI 0.74–2.06, $p = 0.426$). In conclusion, rosuvastatin exhibits a modest adverse effect on the QTc interval. Rosuvastatin monotherapy does not appear to increase the risk of arrhythmia. But this study did not assess the usage of rosuvastatin with other QT-prolonging drugs, the potential for an additive effect should be considered when combining rosuvastatin with other QT-prolonging drugs.

QT prolongation predisposes patients to the development of torsades de pointes (TdP), which can cause cardiac arrest and sudden cardiac death. It is considered to be a surrogate marker of proarrhythmia risk. In patients with congenital long QT syndrome (LQTS), the risk of TdP increases by 5–7% for every 10ms increase in the corrected QT (QTc) interval, and 2–3 fold when the QTc interval exceeds 500ms[1,2]. Patients with acquired LQTS induced by QT-prolonging drugs have a 2.5–2.7 fold increased risk of cardiac arrest and sudden cardiac death, and concurrent use of two or more QT-prolonging drugs increases the risk by over fourfold[3,4].

Drugs are among the most common causes of acquired LQTS. In light of the potentially fatal outcome of drug-induced QT prolongation, non-clinical (ICH S7B) and clinical (ICH E14) regulatory guidance from the International Conference on Harmonization have emphasized the importance of QT liability testing[5,6]. Prolonging the QTc interval is a major safety concern in the development of new drugs. QT liability and torsadogenic potential have become common reasons for the withdrawal of many drugs from the market[7]. Several organizations and institutions are working to provide up-to-date QT-prolonging drug lists[7,8]. Nevertheless, the risk of QT prolongation for many commonly prescribed medications remains uncertain[9].

Rosuvastatin is a fully synthetic-selective HMG-CoA reductase inhibitor. Among the existing statins, it has the largest reduction in low-density lipoprotein cholesterol (LDL-C) and has been proven to be the most effective agent for reducing cardiovascular risk[10]. Rosuvastatin is the third most used statin in the US, with approximately 29 million prescriptions filled annually[11]. In China, it is second only to atorvastatin, accounting for 24.6% of the market share. Although the safety and tolerability of rosuvastatin have been confirmed by previous studies[10–14], some animal and in vitro cell experiments in recent years have found that rosuvastatin can block human ether-a-go-go related gene (hERG) potassium channels and prolong the QTc interval, raising concerns about the safety of rosuvastatin[15,16].

A case-control study based on real-world data showed that the proportion of patients who use rosuvastatin in the QT prolongation group was higher than that in the control group (2.7% vs. 0.9%), and rosuvastatin was associated with QT prolongation (OR 1.30, 95% CI 1.21–1.39). However, this small sample, retrospective, cohort study did not find the association between rosuvastatin and QT prolongation to be statistically significant ($p = 0.065$)[9]. In this context, more research about rosuvastatin and QT prolongation is needed .

Department of Cardiology, Ningbo Medical Centre Lihuili Hospital, No.57 Xingning Road, Ningbo 315040, Zhejiang, China. ✉email: lhlyyxnk068@163.com

This study aimed to compare the changes of QTc interval between patients receiving atorvastatin and rosuvastatin, explore the effect of rosuvastatin on QTc interval and explain the correlation between rosuvastatin and QT prolongation.

Methods

Study design and setting

This study was a pilot parallel, single blind, randomized controlled trial, which was conducted in accordance with the ethical standards described in the Declaration of Helsinki and was approved by the Ethics Committee of Ningbo Medical Centre Lihuili Hospital (KY2023SL211). Informed consent was obtained from all participants and/or their legal guardians. This study was retrospectively registered in Chinese Clinical Trials Registry (ChiCTR2400092701) on November 21, 2024. We followed the Consolidated Standards of Reporting Trials (CONSORT) extension for the Pilot and Feasibility Trials Statement. We also followed the recommendations of trial protocol regulations of the World Health Organization.

Selection of participants

Patients with suspected coronary artery disease (CAD) hospitalized in the Department of Cardiology, Ningbo Medical Centre Lihuili Hospital from August 2023 to May 2024 were consecutively enrolled. Exclusion criteria were: (1) taking statins within 3 months prior to admission; (2) taking known QT-prolonging drugs within 3 months prior to admission; (3) history of TdP or sudden cardiac death; and (4) severe liver and kidney dysfunction, myopathy, pregnancy, breastfeeding, or statin allergy.

Patients with the following conditions were eliminated during follow-up: (1) discontinued statin or changed statin type; (2) failed to complete the ECG as required; (3) severe arrhythmias unrelated to QT prolongation, such as atrial flutter, atrial fibrillation with fast ventricular rate, atrial tachycardia, supraventricular tachycardia, grade II type 2 and III atrioventricular block, sinus arrest, and significant sinus bradycardia rate < 50 beats per minute (bpm); (4) pacemaker implantation or cardiac radiofrequency ablation; (5) acute heart failure; (6) acute myocardial infarction; (7) severe electrolyte disturbance (serum potassium < 3.0 mmol/L or > 5.5 mmol/L; serum calcium, sodium, and magnesium deviated from the normal range); and (8) incomplete data.

Methods of measurement and interventions

Patients were screened, enrolled, and received the intervention by well-trained cardiologists following standardized procedures. All patients underwent the necessary examinations and treatments in accordance with clinical norms and national guidelines[17–19].

Patients were grouped by random number table method with an allocation ratio of 1:1, using a random number table to generate 1000 random numbers. After the subjects were enrolled, they obtained the corresponding random number according to their serial number. Those with odd random numbers were included in the experimental group and those with even numbers were included in the control group. The experimental group received a prescribed dose of rosuvastatin (10 mg) and the control group received a prescribed dose of atorvastatin (20 mg) at 8 p.m. every day. To conceal randomization allocation, the random number was read just prior to the first statin administration.

All patients completed the standard 10-s 12-lead ECG on the day of admission. After receiving two consecutive doses of statins, ECGs were followed up again at 8–12 a.m. on the third day of admission. The QT interval was measured from the first wave of the QRS complex to the end of the T-wave which was determined by the tangent method. The QTc interval was automatically calculated using the NaLong aECG Acquisitor digital electrocardiograph system (Version 3.0) by the Bazett's formula, and verified by at least one ECG expert. Patients and doctors could recognize the type of statins patients received, but this information was hidden from ECG experts.

Demographic characteristics and clinical data, including age, sex, height, weight, smoking, drinking, and comorbidities (hypertension, diabetes, atrial fibrillation, and chronic heart failure) were collected by trained resident physicians. Blood samples were collected by nurses before 8 a.m. on the day after admission and sent to the biochemical laboratory for analysis. Echocardiography was performed at the ultrasound diagnostic center.

Outcome measures

The primary outcome was newly emerged QT prolongation. QT prolongation was defined as a QTc interval > 450 ms for males and 470 ms for females[20]. Patients with normal QTc interval at baseline but with QT prolongation after statin were regarded as newly emerged QT prolongation. The secondary outcomes were increased QTc interval and severe QT prolongation. Increased QTc interval was defined as the follow-up QTc interval being longer than the baseline QTc interval. Severe QT prolongation was defined as either a follow-up QTc interval exceeding 500 ms or an increase in QTc interval exceeding 60 ms from the baseline[21].

Sample size estimation

We based the sample size for statistical power on the primary outcome. A previous small sample study showed that rosuvastatin increased the risk of QT prolongation by 2.5 times (HR 2.54, 95% CI 0.94–6.80)[9]. We hypothesized that rosuvastatin increased the risk of newly emerged QT prolongation by 12.5% and atorvastatin by 5%. The total sample size required to detect this difference in event rates with a two-tailed alpha level of 0.05 and 80% power is 438[22]. We increased our sample size to 500 to take into account a projected 10% elimination during follow-up.

Statistical analysis

Categorical variables were represented as numbers (%) and analyzed using the chi-square test or Fisher's exact test. Continuous variables were represented as mean ± S.D. and analyzed using Student's t-test or Mann - Whitney U test. Logbinomial regression was used to estimate the relative risk (RR) of rosuvastatin with end-point events. Logistic regression analysis was used to investigate the independent correlation between rosuvastatin and QT prolongation after correcting confounding factors. Data analysis was performed using IBM SPSS Statistics, Version 25.0 (Armonk, NY, USA), with a p value of < 0.05 considered significant.

Results

A total of 522 patients fulfilled the research criteria and were classified as research subjects. 466 patients completed follow-up and were included in the analysis, 228 in the rosuvastatin group and 238 in the atorvastatin group. The CONSORT Flow-diagram (Fig. 1) describes the flow of patients through the trial.
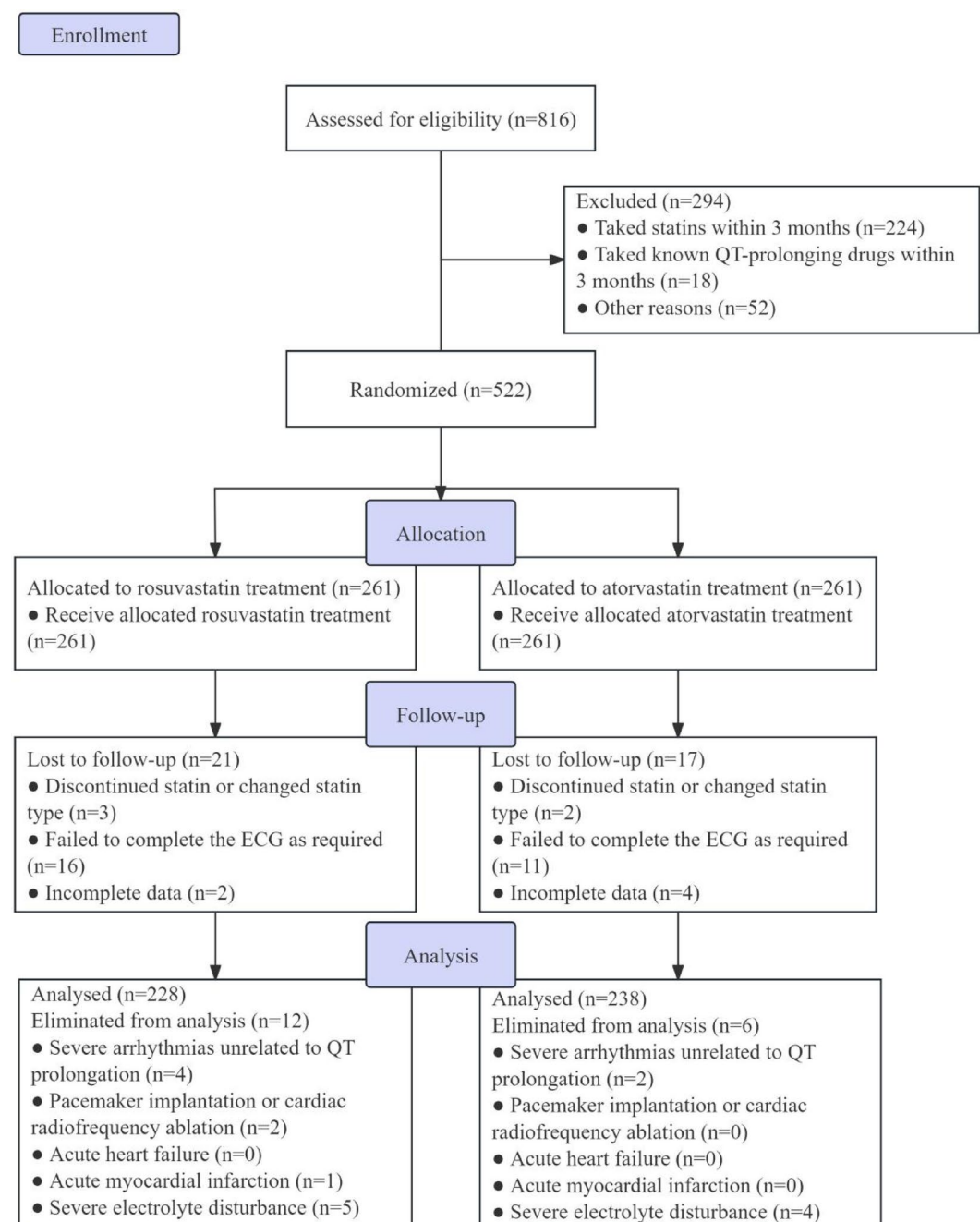

Fig. 1. CONSORT flow diagram.

The general characteristics of the study population are summarized in Table 1. Compared with the rosuvastatin group, more patients in the atorvastatin group received beta-blockers (17.6% vs. 11.0%, $p = 0.04$). There were no significant differences in the other characteristics between the two groups ($p > 0.05$).

The paired Student's t-test revealed no significant change in the QTc interval for the atorvastatin group post-therapy (430.34 ms vs. 429.51 ms, $p = 0.563$), whereas the rosuvastatin group exhibited an increase in the QTc interval (425.85 ms vs. 432.42 ms, $p < 0.001$).

ALT, alanine transaminase; AST, aspartate transaminase; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin-receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; SGLT-2i, sodium-dependent glucose transporter 2; LVDs, left ventricular end systolic diameter; LVDd, left ventricular end diastolic diameter; IVST, interventricular septal thickness; LVPW, left ventricular posterior wall.

The changes in electrocardiographic parameters after therapy are shown in Table 2. The heart rate decreased by 4.73 ± 10.32 bpm from baseline in the atorvastatin group and 3.70 ± 9.50 bpm in the rosuvastatin group ($p = 0.261$). The QTc interval of the atorvastatin group was decreased by 0.83 ± 22.07 ms, while that of the rosuvastatin group increased by 6.57 ± 20.32 ms, with statistically significant ($p < 0.001$). More patients in the rosuvastatin group had an increased QTc interval (62.7% vs. 46.6%, $p < 0.001$). Patients with newly emerged QT prolongation in rosuvastatin group were significantly more than those in atorvastatin group (9.2% vs. 4.2%, $p = 0.030$). However, there was no significant difference in the incidence of severe QT prolongation between the two groups (2.6% vs. 1.7%, $p = 0.537$). During follow-up, none of the patients in either group developed TdP and severe statin adverse reactions.

As shown in Fig. 2, the correlation between statins and end-point events was analyzed by log-binomial regression. Compared with atorvastatin, rosuvastatin increased the risk of a longer QTc interval by 35% ($p = 0.001$), and the risk of newly emerged QT prolongation by 42% ($p = 0.008$), but there was no correlation between rosuvastatin and severe QT prolongation ($p = 0.426$).

Univariate logistic regression analysis was used to analyze the correlation between patient characteristics and QT prolongation of baseline ECG, and the results are shown in Fig. 3. Table 3 lists the factors related to QT prolongation and the results of multivariate analysis. Age was associated with QT prolongation independently, the risk of QT prolongation increased by 45% (95% CI 1.11–1.91, $p = 0.007$) for each additional 10 years of age. Atrial fibrillation (OR 2.89, 95% CI 1.24–6.74, $p < 0.001$) and heart failure (OR 3.85, 95% CI 1.83–8.12, $p < 0.001$) were independent risk factors closely related to QT prolongation. Serum calcium was negatively correlated with QT prolongation, an increase in serum calcium of 0.1 mmol/L was associated with a 28% reduction in the risk of QT prolongation (95% CI 0.53–0.99, $p = 0.042$).

The variables serum calcium and magnesium were statistically transformed to make their values 10 times those of the original, and the values of age and creatinine were statistically transformed to 0.1 times of the original. LVEF and LVD are causally related to chronic heart failure, so they were not included in the multivariate regression model.

In order to eliminate the influence of confounding factors on QT prolongation induced by rosuvastatin, logistic regression analysis was used again to anlyze the correlation between rosuvastatin and newly emerged QT prolongation. Univariate analysis showed that they were closely related (OR 2.31, 95% CI 1.06–5.03, $p = 0.034$). After adjusting for age, heart failure, atrial fibrillation and drug use, it showed that rosuvastatin was an independent risk factor for newly emerged QT prolongation (OR 2.57, 95% CI 1.12–5.90, $p = 0.025$).

Anticoagulants were associated with newly emerged QT prolongation (OR 2.91, 95% CI 1.03–8.17, $p = 0.043$), but the association disappeared after adjusting for atrial fibrillation. Beta-blockers (OR 1.82, 95% CI 0.75–4.42, $p = 0.184$) and any other drugs were not associated with newly emerged QT prolongation ($p > 0.05$).

Discussion

In this study, we observed that compared with the atorvastatin group, more patients in the rosuvastatin group had an increased QTc interval after therapy, and more patients with normal QTc interval at baseline developed QT prolongation. Rosuvastatin can increase the risk of QT prolongation.

Statins are the first-line treatment for lowering LDL-C, consistently recommended by national guidelines, and are widely used for primary and secondary prevention of atherosclerotic cardiovascular disease[17–19]. It is estimated that approximately 145.8 million people (2.6%) in the world are taking statins[23]. Previous studies have shown that atorvastatin and simvastatin can shorten the QTc interval and QTc dispersion[24,25], and the mechanism may be related to the improvement of blood cholesterol level[26–28]. However, this effect was not observed in the study of rosuvastatin. In contrast, rosuvastatin can potentially disrupt the transport of immature hERG potassium channels to the membrane and increase the degradation of mature hERG potassium channels, thereby blocking the delayed rectifier potassium current (IKr) at the late phase of rapid repolarization of the myocardial action potential, resulting in increased QTc interval[15–16]. Koo et al. analyzed 1,040,752 ECG results from a database and found that patients with QT prolongation had a higher rate of rosuvastatin use than those with a normal QTc interval. They speculated that rosuvastatin may be associated with QT prolongation, which provided new evidence for the effect of rosuvastatin on QTc interval[9].

However, that case-control study could not infer whether exposure factor "rosuvastatin" was a cause or a result of QT prolongation, and the small sample cohort study did not find a statistically significant correlation between rosuvastatin and QT prolongation. To this end, we conducted this randomized controlled trial that was strictly controlled for potential interfering factors. Our study demonstrated that rosuvastatin can induce a longer QTc interval and increase the risk of QT prolongation. Compared with atorvastatin, rosuvastatin increased the risk of newly emerged QT prolongation by 42%, and we found that rosuvastatin alone rarely directly leads to severe QT prolongation. This explains why Koo et al. found in their investigation of the National Health Insurance Service (NHIS) database in South Korea that the long-term use of rosuvastatin does not increase mortality.

Variables	Rosuvastatin ($n = 228$)	Atorvastatin ($n = 238$)	p-Value
Age (years)	63.71 ± 10.97	64.96 ± 11.32	0.229
Male	129 (56.6%)	145 (60.9%)	0.341
Female	99 (43.4%)	93 (39.1%)	
Height (cm)	163.11 ± 8.35	163.26 ± 8.00	0.847
Weight (kg)	66.61 ± 11.84	66.45 ± 11.53	0.884
Smoking	76 (33.3%)	80 (33.6%)	0.949
Drinking	54 (23.7%)	42 (17.6%)	0.107
Hypertension	128 (56.1%)	140 (58.8%)	0.558
Diabetes	47 (20.6%)	48 (20.2%)	0.905
Atrial fibrillation	17 (7.5%)	13 (5.5%)	0.381
Chronic heart failure	18 (7.9%)	22 (9.2%)	0.603
Biochemical indicators			
Creatinine (μmoI/L)	69.40 ± 18.25	74.90 ± 42.79	0.074
AST (U/L)	24.49 ± 8.24	24.63 ± 11.17	0.879
ALT (U/L)	22.84 ± 17.15	21.87 ± 14.80	0.512
Serum potassium (mmoI/L)	3.89 ± 0.32	3.94 ± 0.35	0.090
Serum calcium (mmoI/L)	2.26 ± 0.10	2.27 ± 0.10	0.151
Serum magnesium (mmoI/L)	0.86 ± 0.07	0.85 ± 0.06	0.600
Serum sodium (mmoI/L)	140.25 ± 1.86	140.27 ± 1.90	0.914
Total cholesterol (mmol/L)	4.78 ± 1.06	4.88 ± 1.18	0.351
LDL-C (mmol/L)	2.73 ± 0.64	2.81 ± 0.73	0.245
HDL-C (mmol/L)	1.20 ± 0.26	1.19 ± 0.26	0.816
Triglycerides (mmol/L)	1.58 ± 1.13	1.62 ± 1.34	0.779
Lipoprotein (a) mg/dL	20.11 ± 22.38	17.30 ± 17.50	0.130
Inpatient medication			
Aspirin	186 (81.6%)	200 (84.0%)	0.482
Clopidogrel	182 (79.8%)	199 (83.6%)	0.246
Pantoprazole	185 (81.1%)	203 (85.3%)	0.230
Isosorbide mononitrate	35 (15.4%)	36 (15.1%)	0.946
Beta-blocker	25 (11.0%)	42 (17.6%)	0.040
ACEI/ARB/ARNI	68 (29.8%)	81 (34.0%)	0.330
Calcium channel blocker	58 (25.4%)	57 (23.9%)	0.709
Loop diuretic	18 (7.89%)	12 (5.04%)	0.210
Insulin	1 (0.4%)	2 (0.8%)	0.588
Metformin	15 (6.6%)	19 (8.0%)	0.552
SGLT-2i	9 (3.9%)	17 (7.1%)	0.133
α-glucosidase inhibitor	8 (3.5%)	5 (2.1%)	0.356
Sulphonylurea	17 (7.5%)	9 (3.9%)	0.084
Thiazolidinedione	3 (1.3%)	2 (0.8%)	0.618
Anticoagulant	17 (7.5%)	15 (6.3%)	0.623
Estazolam	2 (0.9%)	3 (1.3%)	0.688
Ultrasonic cardiogram			
LVEF (%)	64.36 ± 7.15	63.80 ± 7.55	0.411
LVDd (mm)	47.56 ± 4.68	47.65 ± 5.28	0.854
IVST (mm)	10.88 ± 1.56	10.83 ± 1.62	0.718
LVPW (mm)	9.86 ± 1.32	9.93 ± 1.28	0.592
Baseline ECG			
Heart rate (bpm)	71.83 ± 12.20	73.77 ± 12.51	0.091
QT interval (ms)	391.89 ± 38.51	392.43 ± 34.83	0.874
QTc interval (ms)	425.85 ± 26.54	430.34 ± 29.32	0.084
Followed - up ECG			
Heart rate (bpm)	68.14 ± 10.99	69.04 ± 10.85	0.371
QT interval (ms)	408.96 ± 34.21	404.05 ± 35.93	0.131
QTc interval (ms)	432.42 ± 28.46	429.51 ± 29.04	0.276

Table 1. General characteristics of the study population.

Variables	Rosuvastatin ($n = 228$)	Atorvastatin ($n = 238$)	p-Value
Change of heart rate (bpm)	− 3.70 ± 9.50	− 4.73 ± 10.32	0.261
Change of QTc (ms)	6.57 ± 20.32	− 0.83 ± 22.07	< 0.001
Increased QTc interval	143 (62.7%)	111 (46.6%)	< 0.001
QT prolongation (baseline)	26 (11.4%)	38 (16.0%)	0.153
QT prolongation (follow - up)	40 (17.5%)	33 (13.9%)	0.275
Newly emerged QT prolongation	21 (9.2%)	10 (4.2%)	0.030
Severe QT prolongation	6 (2.6%)	4 (1.7%)	0.537

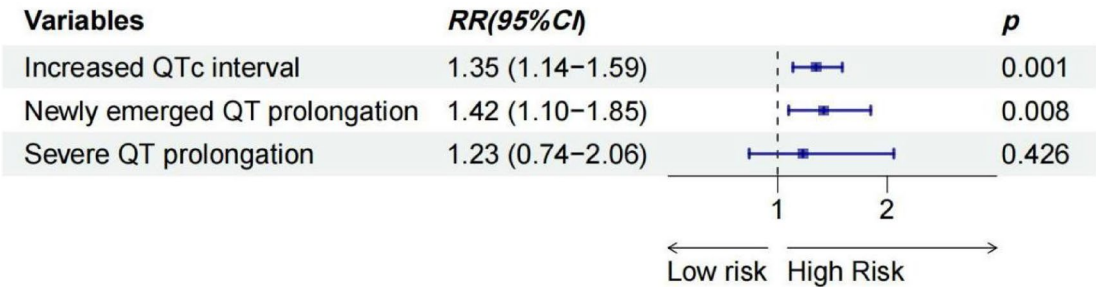**Table 2**. Changes of electrocardiographic parameters after Statin therapy.

Fig. 2. Relative risk of rosuvastatin with endpoint events.

Electrolyte abnormalities are an important cause of acquired LQTS and TdP. Previous studies have shown that hypokalemia and hypocalcemia can cause QT prolongation. Hypomagnesia does not produce specific ECG changes, but the injection of magnesium sulfate inhibits the onset of TdP in patients with QT prolongation[29]. This study excluded patients with severe electrolyte disorders and observed that serum potassium and magnesium levels were not independently associated with QT prolongation wtinin a relatively stable concentration range, whereas serum calcium was still associated with QT prolongation. Age is strongly associated with QT prolongation. Data analyses based on several large studies have shown that QTc interval increases with age. The Swedish pharmacovigilance database survey found that the most common risk factor for drug-induced TdP was age > 65 years, occurring in 72% of cases[30]. In this study, the risk of QT prolongation increased by 45% for each additional 10 years of patient age group. In addition, atrial fibrillation and heart failure were independent risk factors for QT prolongation, consistent with the results observed in others[31,32]. Interestingly, QT prolongation indicates a higher risk of future occurrence of atrial fibrillation and heart failure[33–35].

This study have some limitations. First, we followed the ECG at a specific time after the patient's medication, but continuous ECG monitoring was not performed. Secondly, the follow-up period of this study was short, so it was not possible to determine whether the QTc interval would further increase with prolonged medication duration. Finally, to prevent QT-prolonging drugs and acute cardiac events from interfering with the observation of the effect of rosuvastatin on QTc interval, we excluded patients with acute heart failure, acute myocardial infarction, severe non-ventricular arrhythmias, and those on QT-prolonging drugs. Therefore, the safety of rosuvastatin in these patients could not be confirmed.

Conclusion

Rosuvastatin can induce a longer QTc interval and increase the risk of QT prolongation. But rosuvastatin alone rarely directly leads to severe QT prolongation in patients. It is unnecessary to overly worry about the arrhythmogenic effect of rosuvastatin, but for patients who take rosuvastatin and other QT-prolonging drugs at the same time, the detection of QTc interval should be strengthened.
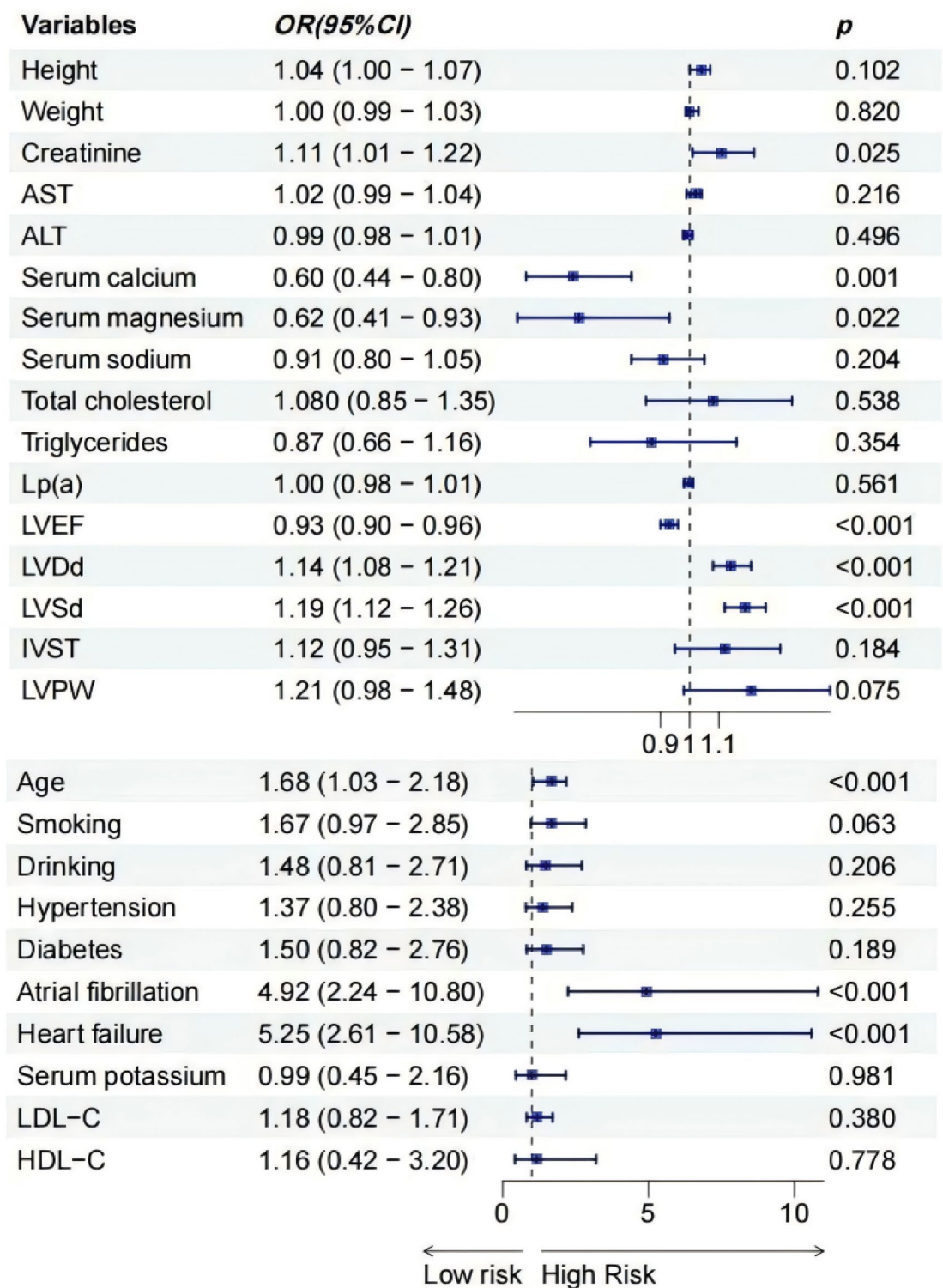
Variables	OR(95%CI)		p
Height	1.04 (1.00 − 1.07)		0.102
Weight	1.00 (0.99 − 1.03)		0.820
Creatinine	1.11 (1.01 − 1.22)		0.025
AST	1.02 (0.99 − 1.04)		0.216
ALT	0.99 (0.98 − 1.01)		0.496
Serum calcium	0.60 (0.44 − 0.80)		0.001
Serum magnesium	0.62 (0.41 − 0.93)		0.022
Serum sodium	0.91 (0.80 − 1.05)		0.204
Total cholesterol	1.080 (0.85 − 1.35)		0.538
Triglycerides	0.87 (0.66 − 1.16)		0.354
Lp(a)	1.00 (0.98 − 1.01)		0.561
LVEF	0.93 (0.90 − 0.96)		<0.001
LVDd	1.14 (1.08 − 1.21)		<0.001
LVSd	1.19 (1.12 − 1.26)		<0.001
IVST	1.12 (0.95 − 1.31)		0.184
LVPW	1.21 (0.98 − 1.48)		0.075

0.9 1 1.1

Age	1.68 (1.03 − 2.18)		<0.001
Smoking	1.67 (0.97 − 2.85)		0.063
Drinking	1.48 (0.81 − 2.71)		0.206
Hypertension	1.37 (0.80 − 2.38)		0.255
Diabetes	1.50 (0.82 − 2.76)		0.189
Atrial fibrillation	4.92 (2.24 − 10.80)		<0.001
Heart failure	5.25 (2.61 − 10.58)		<0.001
Serum potassium	0.99 (0.45 − 2.16)		0.981
LDL−C	1.18 (0.82 − 1.71)		0.380
HDL−C	1.16 (0.42 − 3.20)		0.778

0   5   10

Low risk High Risk

Fig. 3. Correlation between patient characteristics and QT prolongation of baseline ECG.

Variables	OR (95%CI)	p	OR (95%CI)	p
	Univariate analysis		Multivariate analysis	
Age	1.68 (1.30–2.18)	< 0.001	1.45 (1.11–1.91)	0.007
Atrial fibrillation	4.92 (2.24–10.80)	< 0.001	2.89 (1.24–6.74)	0.014
Heart failure	5.25 (2.61–10.58)	< 0.001	3.85 (1.83–8.12)	< 0.001
Creatinine	1.11 (1.01–1.22)	0.003	--	--
Serum calcium	0.60 (0.44–0.80)	< 0.001	0.72 (0.53–0.99)	0.042
Serum magnesium	0.62 (0.41–0.93)	0.032	--	--
LVEF	0.93 (0.90–0.96)	< 0.001	--	--
LVDs	1.14 (1.08–1.21)	< 0.001	--	--
LVDd	1.19 (1.12–1.26)	< 0.001	--	--

Table 3. Multivariate logistic regression analysis of the factors related to QT prolongation.

Data availability

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

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Author contributions

LJZ and QJJ designed this study. YWW and CLW led to recruitment and data acquisition. LJZ and JL completed the follow-up. LC and WJS verified ECG data. LJZ and HWG performed statistical analyses and drafted the manuscript. WYH and QJJ critically revised the manuscript. All authors revised and approved the final version of the manuscript.

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Declarations

Competing interests

The authors declare no competing interests.

Additional information

Correspondence and requests for materials should be addressed to Q.J.

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