Prediction for cardiovascular diseases based on laboratory data: An analysis of random forest model

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Abstract

Background: To establish a prediction model for cardiovascular diseases (CVD) in the general population based on random forests.

Methods: A retrospective study involving 498 subjects was conducted in Xi’an Medical University between 2011 and 2018. The random forest algorithm was used to screen out the variables that greatly affected the CVD prediction and to establish a prediction model. The important variables were included in the multifactorial logistic regression analysis. The area under the curve (AUC) was compared between logistic regression model and random forest model.

Results: The random forest model revealed the variables, including the age, body mass index (BMI), fasting blood glucose (FBG), diastolic blood pressure (DBP), triglyceride (TG), systolic blood pressure (SBP), total cholesterol (TC), waist circumference, and high-density lipoprotein-cholesterol (HDL-C), were more significant for CVD prediction; the AUC was 0.802 in CVD prediction. Multifactorial logistic regression analysis indicated that the risk factors for CVD included the age (odds ratio (OR): 1.14, 95% confidence intervals (CI): 1.10-1.17, \(P < .001\)), BMI (OR: 1.13, 95% CI: 1.06-1.20, \(P < .001\)), TG (OR: 1.11, 95% CI: 1.02-1.22, \(P = .023\)), and DBP (OR: 1.04, 95% CI: 1.02-1.06, \(P = .001\)); the AUC was 0.843 in CVD prediction. The established logistic regression prediction model was Logit \(P = \log\left(\frac{P}{1 - P}\right) = -11.47 + 0.13 \times \text{age} + 0.12 \times \text{BMI} + 0.11 \times \text{TG} + 0.04 \times \text{DBP}; P = 1/(1 + \exp(-\text{Logit} P))\). People were prone to develop CVD at the time of \(P > .51\).

Conclusions: A prediction model for CVD is developed in the general population based on random forests, which provides a simple tool for the early prediction of CVD.

Keywords
cardiovascular disease, prediction model, random forest, risk factors
1 | INTRODUCTION

Cardiovascular diseases (CVD) have been always the most common cause of morbidity and mortality worldwide. In 2013, 17.3 million people were estimated to die of CVD among 54 million total deaths, approximately accounting for 31.5% of all global deaths.1 With economic growth, the prevalence of CVD is increasing obviously in developing countries. In 2016, there were about 4.34 million deaths from CVD in China, including 2.01 million deaths due to stroke and 1.74 million deaths due to coronary heart disease (CHD).2,3 Early detection of CVD phenotypes and effective interventions is conductive to slowing the progression, during which CVD risk assessment and stratification play important roles.4

Cardiovascular diseases risk assessment is not only an essential method of screening high-risk CVD individuals, but also an important evidence of individualized therapeutic regimens formulated by clinicians. At present, several CVD risk prediction models have been developed, including Framingham risk model,5 SCORE project,6 QRISK model,7 and pooled cohort equation,8 which all belong to 10-year risk assessment models of major CVDs in European and American countries.3 According to the well-established risk factors, the standard CVD risk prediction models can predict the future risk of CVD and make an unambiguous assumption that each risk factor is associated with CVD outcomes in a linear manner.9 However, these models may oversimplify the complicated relationships involving a lot of risk factors without linear interactions.10 Additionally, multiple versions of Framingham models are reported to overpredict the incidence of CHD,11-13 and Framingham CVD model is poor in the calibration of the end point of major CVD.14 There are also evidences suggesting that QRISK2 based on ORISK model is continuously updated to include additional risk factors like extension of age range, type 2 diabetes mellitus, and type 1 diabetes mellitus.15,16 Therefore, it is necessary to investigate an approach that can integrate various risk factors better and confirm more accurate associations between outcomes and risk factors.

The random forest, a key data mining method in the field of machine-learning that depends on a computer to learn all the complicated and non-linear interactions among variables through minimization of errors between observed and predicted outcomes,17 can achieve a higher accuracy in the disease prediction by using bootstrap aggregation and randomization of predictors.18-21 Until now, there are few studies on the application of random forests to predict CVD risks in the general population. Therefore, this study was performed to establish a prediction model for CVD in the general population based on random forests.

2 | METHODS

2.1 | Study population

This study was conducted in Xi’an Medical University between 2011 and 2018, and a total of 498 subjects undergoing a physical examination were involved. All the subjects had complete physical examination data and could move by themselves; those staying in bed and those with CVD were excluded. The included subjects voluntarily participated in the study and were informed consent.

2.2 | Data collection

A medical examination questionnaire was designed to obtain the basic information of subjects, including the age, gender, history of various diseases, and smoking. The body height, body weight, blood pressure, and waist circumference were achieved by the physical examination. The body mass index (BMI) was calculated based on the formula of the body weight in kilograms/body height in meters squared. Laboratory indicators included fasting blood glucose (FBG), total cholesterol (TC), triglycerides (TG), low-density lipoprotein-cholesterol (LDL-C), and high-density lipoprotein-cholesterol (HDL-C). The levels of FBG and blood lipids were detected in the next morning after an overnight of at least 8 hours though blood sampling.

2.3 | Outcomes

The occurrence of a fatal or non-fatal cardiovascular event was considered as the primary outcome. The outcome was based on the follow-up results and was confirmed according to international classification of diseases-10 (ICD-10) diagnosis codes, especially I20-I25 for coronary (ischemic) heart conditions and I60-I69 for cerebrovascular conditions.22

2.4 | Statistical analysis

STATA 14.0 software (Stata Corporation) and R software (version 3.6.1) were used to analyze the data. The data with normal distribution were presented with the mean ± standard deviation (±s) by t test, whereas those with abnormal distribution were described with the median and quartile (M|Q1, Q3) using Mann-Whitney U rank-sum test. The enumeration data were manifested as n (%) by chi-square test or Fisher’s exact test. P < .05 was considered statistically significant.

The random forest was a collection of various decision tree models.23 Each tree was developed from bootstrap samples in the training dataset, and the randomly selected best subset of explanatory variables or features was used to split each node. In the forest, the class predictions produced by each tree were assembled and the model prediction was finally determined according to the majority vote.24 Random allocation was used to assess the random forest prediction model; namely, the samples were randomly assigned into the nonoverlapping training samples for establishing the prediction model and testing samples for calculating the sensitivity, specificity, accuracy, positive predictive value, and negative predictive value of prediction models. The importance of variables
### TABLE 1  The basic characteristics of samples in the training set and testing set ([x ± s]/[M(Q1, Q3)]

| Variables                          | Training set (n = 335) | Testing set (n = 163) | χ²/t/Z | P       |
|------------------------------------|------------------------|-----------------------|--------|---------|
| **CVD (n, %)**                     |                        |                       |        |         |
| Yes                                | 169 (50.40)            | 78 (47.90)            | 0.29   | .587    |
| No                                 | 166 (49.60)            | 85 (52.10)            |        |         |
| **Age (y)**                        | 42.44 ± 9.24           | 42.27 ± 9.54          | -1.89  | .850    |
| **Gender (male/female)**           | 235 (70.10)/100 (29.90)| 121 (74.20)/42 (25.80)| 0.90   | .244    |
| **BMI (kg/m²)**                    | 23.97 ± 3.58           | 24.40 ± 4.01          | 1.15   | .248    |
| **Waist circumference (cm)**       | 83.90 ± 14.82          | 85.25 ± 14.71         | 0.96   | .339    |
| **FBG (mmol/L)**                   | 5.02 (4.37, 5.67)      | 5.00 (4.43, 5.65)     | -0.02  | .986    |
| **Diastolic blood pressure (DBP, mm Hg)** | 80.84 ± 13.25          | 80.79 ± 11.76         | -0.04  | .967    |
| **Systolic blood pressure (SBP, mm Hg)** | 123.29 ± 17.69          | 123.70 ± 17.09        | 0.25   | .805    |
| **TC (mmol/L)**                    | 4.26 (3.70, 5.08)      | 4.48 (3.80, 5.20)     | -1.55  | .121    |
| **TG (mmol/L)**                    | 1.65 (1.10, 2.50)      | 1.62 (1.10, 2.69)     | -0.37  | .707    |
| **HDL-C (mmol/L)**                 | 1.47 (1.17, 2.26)      | 1.57 (1.18, 2.51)     | -1.13  | .260    |
| **LDL-C (mmol/L)**                 | 1.76 (1.23, 2.50)      | 1.69 (1.20, 2.40)     | 0.69   | .485    |
| **Activity level**                 |                        |                       |        |         |
| Low                                | 93 (27.80)             | 54 (33.10)            | 2.78   | .249    |
| Middle                             | 193 (57.60)            | 81 (49.70)            |        |         |
| High                               | 49 (14.60)             | 28 (17.20)            |        |         |
| **Smoking**                        |                        |                       |        |         |
| No                                 | 188 (56.10)            | 86 (52.80)            | 0.69   | .707    |
| Smoking cessation                  | 27 (8.10)              | 16 (9.80)             |        |         |
| Yes                                | 120 (35.80)            | 61 (37.40)            |        |         |
| **Stroke**                         |                        |                       |        |         |
| No                                 | 332 (99.10)            | 162 (98.80)           | 0.12   | .728    |
| Yes                                | 3 (0.90)               | 2 (1.20)              |        |         |
| **Pulmonary tuberculosis a**       |                        |                       |        |         |
| No                                 | 334 (99.70)            | 162 (99.40)           |        | .602    |
| Yes                                | 1 (0.30)               | 1 (0.60)              |        |         |
| **Chronic bronchitis a**           |                        |                       |        | .999    |
| No                                 | 331 (98.80)            | 162 (99.40)           |        | .999    |
| Yes                                | 4 (1.20)               | 1 (0.60)              |        |         |
| **Pneumonia a**                    |                        |                       |        | .600    |
| No                                 | 161 (98.80)            | 333 (99.40)           |        | .600    |
| Yes                                | 2 (1.20)               | 2 (0.60)              |        |         |
| **Lung cancer a**                  |                        |                       |        | .999    |
| No                                 | 335 (100.00)           | 163 (100.00)          |        | .999    |
| Yes                                | 0 (0.00)               | 0 (0.00)              |        |         |
| **Pulmonary emphysema a**          |                        |                       |        | .999    |
| No                                 | 334 (99.80)            | 163 (100.00)          |        | .999    |
| Yes                                | 1 (0.30)               | 0 (0.00)              |        |         |
| **Family history of hypertension** |                        |                       |        | .495    |
| No                                 | 222 (66.30)            | 113 (69.30)           | 0.46   | .495    |
| Yes                                | 113 (33.70)            | 50 (30.70)            |        |         |

(Continues)
### TABLE 1 (Continued)

| Variables                              | Training set (n = 335) | Testing set (n = 163) | $\chi^2/t/Z$ | $P$ |
|----------------------------------------|------------------------|-----------------------|--------------|-----|
| Family history of CHD                  |                        |                       |              |     |
| No                                     | 288 (86.00)            | 137 (84.00)           | 0.32         | .570|
| Yes                                    | 47 (14.00)             | 26 (16.00)            |              |     |
| Family history of diabetes mellitus    |                        |                       |              |     |
| No                                     | 283 (84.50)            | 140 (85.90)           | 0.17         | .670|
| Yes                                    | 52 (15.50)             | 23 (14.10)            |              |     |
| Family history of stroke               |                        |                       |              |     |
| No                                     | 305 (91.00)            | 153 (93.90)           | 1.18         | .277|
| Yes                                    | 30 (9.00)              | 10 (6.10)             |              |     |
| Family history of lung cancer<sup>a</sup> |                        |                       |              |     |
| No                                     | 159 (97.50)            | 328 (97.90)           | –            | .755|
| Yes                                    | 4 (2.50)               | 7 (2.10)              |              |     |

<sup>a</sup>Represented the data were analyzed by Fisher’s exact test.

### TABLE 2  The basic characteristics of subjects in case group and control group ($\bar{x} \pm s$)/[M(Q1, Q3)]

| Variables                        | Case group (n = 247) | Control group (n = 251) | $\chi^2/t/Z$ | $P$  |
|----------------------------------|----------------------|-------------------------|--------------|------|
| Age (y)                          | 47.04 ± 7.87         | 37.82 ± 8.36            | 12.66        | <.001|
| Gender (male/female)             | 202 (81.78)/45 (18.20) | 154 (61.35)/97 (38.60)  | 25.48        | <.001|
| BMI (kg/m<sup>2</sup>)           | 25.25 ± 3.55         | 23.01 ± 3.57            | 7.02         | <.001|
| Waist circumference (cm)         | 87.74 ± 14.19        | 81.00 ± 14.61           | 5.22         | <.001|
| FBG (mmol/L)                     | 5.20 (4.64, 6.15)    | 4.81 (4.26, 5.33)       | 5.12         | <.001|
| DBP (mm Hg)                      | 84.84 ± 13.87        | 76.87 ± 10.16           | 7.30         | <.001|
| SBP (mm Hg)                      | 128.66 ± 19.36       | 118.28 ± 13.61          | 6.91         | <.001|
| TC (mmol/L)                       | 4.41 (3.84, 5.25)    | 4.20 (3.60, 4.93)       | 2.59         | .010 |
| TG (mmol/L)                       | 1.93 (1.32, 3.00)    | 1.40 (0.95, 2.13)       | 5.48         | <.001|
| HDL-C (mmol/L)                    | 1.48 (1.15, 2.43)    | 1.50 (1.19, 2.26)       | −0.14        | .886 |
| LDL-C (mmol/L)                    | 1.74 (1.18, 2.54)    | 1.70 (1.25, 2.40)       | −0.014       | .989 |
| Activity level                    |                      |                         |              |      |
| Low                               | 74 (30.00)           | 73 (29.10)              | 9.55         | .008 |
| Middle                            | 147 (59.50)          | 127 (50.60)             |              |     |
| High                              | 26 (10.50)           | 51 (20.30)              |              |     |
| Smoking                           |                      |                         |              |      |
| No                                | 110 (44.50)          | 164 (65.30)             | 22.11        | <.001|
| Smoking cessation                 | 28 (11.30)           | 15 (6.00)               |              |     |
| Yes                               | 109 (44.10)          | 72 (28.70)              |              |     |
| Stroke<sup>a</sup>                |                      |                         |              |      |
| No                                | 243 (98.40)          | 250 (99.60)             | –            | .213 |
| Yes                               | 4 (1.60)             | 1 (0.40)                |              |     |
| Pulmonary tuberculosis            |                      |                         |              |      |
| No                                | 246 (99.60)          | 250 (99.60)             | 0.00         | .991 |
| Yes                               | 1 (0.40)             | 1 (0.40)                |              |     |
| Chronic bronchitis                |                      |                         |              |      |
| No                                | 234 (94.70)          | 248 (98.80)             | 6.63         | .011 |
| Yes                               | 13 (5.30)            | 3 (1.20)                |              |     |

(Continues)
could be reflected by the mean decreased Gini (MDG) index in random forest output results. The greater the MDG index, the more significant the variable. In this study, the random forest analysis was performed using R software, and the random forest algorithm was used to screen out the variables that greatly affected the CVD prediction and to establish a prediction model. Then, the screened important variables were included in the multifactorial logistic regression analysis. Stepwise method was used to screen the variables. The receiver operating characteristic (ROC) curve was drawn using CVD probability in the subjects of testing data-set and their physical examination results, and the area under the curve (AUC) of logistic regression model was compared with that of random forest model using Z test.

### TABLE 2 (Continued)

| Variables                                             | Case group (n = 247) | Control group (n = 251) | $\chi^2/t/Z$ | P     |
|-------------------------------------------------------|----------------------|-------------------------|--------------|-------|
| Pneumonia<sup>a</sup>                                 |                      |                         |              |       |
| No                                                     | 246 (99.60)          | 248 (98.80)             | –            | .624  |
| Yes                                                    | 1 (0.40)             | 3 (1.20)                |              |       |
| Lung cancer<sup>a</sup>                               |                      |                         |              |       |
| No                                                     | 247 (100.00)         | 251 (100.00)            | –            | .999  |
| Yes                                                    | 0 (0.00)             | 0 (0.00)                |              |       |
| Pulmonary emphysema<sup>a</sup>                        |                      |                         |              |       |
| No                                                     | 247 (100.00)         | 250 (99.60)             | –            | .330  |
| Yes                                                    | 0 (0.00)             | 1 (0.40)                |              |       |
| Family history of hypertension                         |                      |                         |              |       |
| No                                                     | 150 (60.70)          | 185 (73.70)             | 9.52         | .002  |
| Yes                                                    | 97 (39.30)           | 66 (26.30)              |              |       |
| Family history of CHD                                 |                      |                         |              |       |
| No                                                     | 208 (84.20)          | 217 (86.50)             | 0.50         | .479  |
| Yes                                                    | 39 (15.80)           | 34 (13.50)              |              |       |
| Family history of diabetes mellitus                    |                      |                         |              |       |
| No                                                     | 207 (83.80)          | 216 (86.10)             | 0.49         | .483  |
| Yes                                                    | 40 (16.20)           | 35 (13.90)              |              |       |
| Family history of stroke                               |                      |                         |              |       |
| No                                                     | 220 (89.10)          | 238 (94.80)             | 5.58         | .018  |
| Yes                                                    | 27 (10.90)           | 13 (5.20)               |              |       |
| Family history of lung cancer<sup>a</sup>              |                      |                         |              |       |
| No                                                     | 244 (98.80)          | 247 (98.40)             | –            | .999  |
| Yes                                                    | 3 (1.20)             | 4 (1.60)                |              |       |

<sup>a</sup>Represented the data were analyzed by Fisher’s exact test.

3 | RESULTS

3.1 | Selection and basic characteristics of study population

Totally, 1809 people underwent a physical examination, in which 307 cases of CVD were excluded. Among the rest 1502 people, 247 cases of CVD found by the physical examination were set as the case group, and then, 20% (n = 251) of 1255 people without CVD were randomly selected as the control group. A total of 498 subjects were finally included in this study. They were 22-60 years old, with the mean age of (42.39 ± 9.33) years; their BMI was 16.50-43.00 kg/m<sup>2</sup>, with the mean BMI of (24.12 ± 3.73) kg/m<sup>2</sup>. These subjects were randomly divided into the training set (n = 335) and testing set (n = 163). As shown in Table 1, no significant differences were shown between the training set and testing set regarding the basic characteristics of samples (P > .05). Additionally, the basic characteristics of subjects in case group and control group were compared in Table 2, and the results indicated that the differences were pronounced between two groups in the age, gender, BMI, waist circumference, FBG, SBP, DBP, TC, TG, smoking, activity level, chronic bronchitis, family history of stroke, and hypertension (P < .05).

3.2 | Random forest model

Totally 335 training samples were used to establish the random forest model. As indicated in Figure 1, 24 variables were ordered according to the MDG index. It was found that the variables, such as
age, BMI, FBG, DBP, TG, SBP, TC, waist circumference, and HDL-C, were more significant for CVD prediction. The random forest could better distinguish the people who would develop CVD from those who would not (Figure 2). The minimum error was 21.49% when the number of decision tree was 258 (Figure 3).

A total of 163 testing samples were adopted to assess the efficacy of prediction model, and the results revealed that the accuracy, sensitivity, specificity, positive predictive value, and negative predictive value of prediction model were 72.89%, 78.82%, 79.23%, 73.62%, and 75.00%, respectively.

3.3 | Logistic regression model

Nine variables more significant for CVD prediction in Figure 1 were involved in the multifactorial logistic regression analysis. As listed in Table 3, the risk factors for CVD included the age [odds ratio (OR): 1.14, 95% confidence intervals (CI): 1.10-1.17, \( P < .001 \)], BMI (OR: 1.13, 95% CI: 1.06-1.20, \( P < .001 \)), TG (OR: 1.11, 95% CI: 1.02-1.22, \( P = .023 \)), and DBP (OR: 1.04, 95% CI: 1.02-1.06, \( P = .001 \)).

The efficacy of prediction model was assessed using testing samples. It was found that the accuracy, sensitivity, specificity, positive predictive value, and negative predictive value of prediction model were 77.91%, 78.50%, 78.50%, 78.19%, and 77.65%, respectively.

3.4 | Comparison of two prediction models

The AUCs of two prediction models were presented in Figure 4. In cardiovascular risk prediction, the ROC-AUCs were 0.802 (95% CI: 0.735-0.870, \( P < .001 \)) for random forest model and 0.843 (95% CI: 0.808-0.877, \( P < .001 \)) for logistic regression model, and no statistical significance was shown (\( P > .05 \)). In this study, however, the
logistic regression model was chosen as a prediction model due to its supply of estimated relative risk (RR) for influencing factors. With Logit $P$ as a dependent variable, logistic regression prediction model was established: $\text{Logit } P = \log\left[p/(1-p)\right] = -11.47 + 0.13 \times \text{age} + 0.12 \times \text{BMI} + 0.11 \times \text{TG} + 0.04 \times \text{DBP}; P = 1/[1 + \exp(-\text{Logit } P)]$. People were prone to develop CVD when the value of $P$ was more than .51.

### DISCUSSION

In this study, we depended on random forests to establish a prediction model for CVD, and the results indicated that the random forest algorithm was effective in distinguishing the individuals who would develop CVD from those who would not. Unlike other established risk assessment methods, the random forest not confined to a small number of risk factors could incorporate all available risk factors. Additionally, the logistic regression prediction model based on the random forest was established; namely, the risk of developing CVD could be predicted according to the age, BMI, TG, and DBP, and the CVD might occur in people with the value of $P > .51$.

Age is perhaps the most important risk factor for the short-term assessment of CVD.\(^{25}\) With advancing age, the structure and function of vasculature change owing to increased oxidative stress, premature cellular senescence, and damage in synthesis and/or secretion of endothelium-derived vasoactive molecules. These changes can result in the stiffening and thickening of vascular walls and other vascular complications.\(^{26-28}\) It was estimated that 4.0% of people aged 18-44 years had heart disease, and this prevalence increased to 11.9% in those aged 45-64 years and 23.1% in those aged 65-74 years, even up to 35.0% in those aged over 75 years.\(^{29}\) In this study, it was found that the risk of developing CVD in the general population would increase 0.14-folds at each addition of 1 year old.

BMI is used to classify the normal weight, overweight, and obesity, and is associated with the risk of developing CVD.\(^{30,31}\) The age-adjusted RR for CVD was higher in overweight people (males, RR: 1.21; females, RR: 1.20) and obese people (males, RR: 1.46; females, RR: 1.64).\(^{32}\) When compared with normal-weight adults, the overweight and obese adults had a higher level for each CVD biomarker, including C-reactive protein, TC, TG, and LDL-C.\(^{33}\) Our results suggested that the cardiovascular risk would increase 0.13-folds in the general population when 1 kg/m\(^2\) of BMI was added. It was reported that obesity was associated with a prothrombotic state, a proinflammatory state, atherogenic dyslipidemia, and insulin resistance,\(^{34,35}\) which could increase the overall cardiac workload to satisfy the metabolic needs of the expanded adipose tissue by elevating the cardiac output.\(^{36}\)
Atherogenic dyslipidemia is associated with poor cardiovascular outcomes, which is characterized by a combination of increased TG and LDL-C levels, lowered HDL-C level and a dominance of small-dense LDL-C particles. Dyslipidemia can increase the risk of developing CVD and determine the total risk of CVD in individuals through interaction with multiple risk factors. At present, lowering LDL-C level is considered as the primary therapeutic target in the management of dyslipidemia. In this study, TG was presented to be an independent risk factor for CVD and played a crucial role in cardiovascular prediction, which were supported by several studies. Additionally, in the population with LDL-C level reaching the target value who received lipid-lowering therapies, an association was present between TG and cardiovascular risk. TG is transported in plasma by specific triglyceride-rich lipoproteins (TRLs) which are thought to involve in the progression of atherosclerosis and CVD. Studies show that the variants in several key genes involved in the metabolism of TRLs have a strong correlation with CVD risk, and the strength of a variant’s impact on TG level is closely associated with the degree of its effect on CVD. And meanwhile, TRLs can promote the intimal cholesterol deposition and participate in activating and reinforcing some proapoptotic, proinflammatory, and procoagulant pathways.

A lower DBP (<70-80 mm Hg) had a correlation with an increased risk of all-cause death and myocardial infarction probably because of compromised coronary artery perfusion, consequently leading to cardiac ischemia. In the first asymptomatic carotid surgery trial, DBP was confirmed to be the single independent risk factor related to peri-procedural stroke or death. The combined results of 9 major prospective studies demonstrated that there were continuous, positive, and independent associations of DBP with stroke and with coronary heart disease. In this study, DBP was found to be a risk factor for CVD, and the risk of developing CVD would increase 0.04-folds when 1 mm Hg of DBP was augmented.

The strengths of this study were the use of random forests to predict the risk of developing CVD in the general population. The explanatory power of predictive analytics was based on the factors included. All the available risk factors were incorporated in this study via random forests, and then, important variables were screened out to establish the prediction model, which may make the predictive ability of the model more accurate. Moreover, the data required in the established prediction model could be easily obtained in clinic, which was conductive to the clinicians to promptly assess the individuals’ cardiovascular risk and implement personalized interventions for high-risk individuals. However, the study population mainly came from the city. Inadequate population diversity may cause a poor efficacy of the established model when used in other populations.

5 | CONCLUSIONS

A prediction model for CVD is developed in the general population based on random forests, which provides a simple tool for the early prediction of CVD. The risk of developing CVD can be predicted according to the individuals’ age, BMI, TG, and DBP.

AUTHOR CONTRIBUTIONS

XS and LS designed the study. XS, YYX, and ZJY drafted and wrote the manuscript. XW and PY contributed to data collection. YS and YYJ analyzed and interpreted the data. SJQ contributed to literature search. LS critically reviewed, edited, and approved the manuscript. All authors read and approved the final manuscript.

ETHICAL APPROVAL

The data in this article are from different provinces and different hospitals. It is finally compiled by a group company, so there is no ethics number when using it.
INFORMED CONSENT

Informed consent was obtained from all individual participants included in the study.

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