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Predicting mortality and non-fatal cardiovascular events in patients with stable coronary heart disease

Panakhova D.Z., Mammaev S.N.

Dagestan State Medical University of the Ministry of Health of the Russian Federation,
Makhachkala, Russia.

AUTHORS:

Diana Z. Panakhova*, PhD, MD, Assistant at the Department of Faculty therapy, Dagestan State Medical University of the Ministry of Health of the Russian Federation, Makhachkala, Russia. ORCID: 0000-0003-0719-8980
Suleiman N. Mammaev, PhD, MD, Professor, Department of Hospital Therapy, Dagestan State Medical University of the Ministry of Health of the Russian Federation, Makhachkala, Russia. ORCID: 0000-0001-8898-8831

Objective. The aim of the study was to identify the factors, associated with poor prognosis in patients with stable coronary heart disease (CHD), and to build a statistically validated model for predicting the risk of mortality and non-fatal cardiovascular events (CVEs) in patients with stable CHD.

Methods. This retrospective observational cohort study included 85 patients, admitted to the Dagestan Center of Cardiology and Cardiovascular Surgery for planned inpatient treatment from 01.01.2015 to 31.12.2017 and with a giagnosis of stable exertional angina. The data were collected from patients' medical records and their long-term outcomes were consequently verified. Simultaneously, a telephone contact was established with enrolled patients to ascertain vital status and to record cardiovascular events. The patients were invited for reassessment, which included clinical and anamnestic data, laboratory and instrumental diagnostics. For the prognostic model, binary logistic regression was used to evaluate the impact of certain factors on the probability of adverse outcomes development.

Results. Over the 4-year period of observation, 5.9% (5 people) of 85 patients died. In 84.7% (72 patients) of all cases, admission due to CHD worsening was registered. In 15.3% of patients (13 people), the primary composite endpoint, which included all-cause mortality and CVE development, was reached.

The prognostic model for evaluation of probability of reaching the primary endpoint, depending on the influence of variable factors was built. The most significant factors included: hematocrit, echocardiographic left atrial volume, and coronographic chronic occlusion of the left circumflex artery. The obtained model was proven statistically significant ($p<0.001$), and had high sensitivity (85.7%) and specificity (97.4%).

* Corresponding author. Tel. +7 988-631-96-25; e-mail: . E-mail: panaxova92@mail.ru

Conclusion. In this study, certain factors that contribute to the risk of death and non-fatal CVEs in patients with stable CHD were identified. This allowed for the development of a prognostic model to estimate these risks and facilitate the further implementation of secondary prevention measures in clinical practice.

Keywords: coronary heart disease, stable exertional angina, cardiovascular events, life expectancy, survival.

Conflict of interest: None declared.

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Introduction

According to the World Health Organization, cardiovascular diseases (CVDs) remain one of the leading causes of mortality worldwide, despite continuing advances in diagnosis and treatment [1]. Statistical data indicate that CVD mortality in the Republic of Dagestan (RD) rose from 203.1 in 2018 to 208.4 per 100,000 population in 2022 [2]. Mortality from coronary heart disease (CHD) accounted for 23.8% of all-cause deaths and, specifically, 54.2% of CVD deaths, placing it first among CVDs in Russia in 2022 [2]. Between 2019 and 2022, CHD mortality in Russia increased from 301.4 to 307.4 per 100,000 population, representing a growth rate of 2.0%. Furthermore, during the first two years of the COVID-19 pandemic, a sharp increase in mortality was observed (from 347.3 in 2020 to 348.1 per 100,000 population in 2021) compared with 2019 [3]. An analysis of age-standardized CHD mortality across 45 countries demonstrated that the Russian Federation belongs to the group of countries with high CHD mortality, and that the burden of CHD in Russia exceeds that of economically developed nations [4].

CVD mortality ranks first in the overall mortality structure in the RD, accounting for 40.6%[1] in 2024. Between 2016 and 2019[2], CVD mortality in the RD declined gradually by 5.3%, from 209.4 to 198.7 per 100,000 population. However, in 2020, against the backdrop of the COVID-19 pandemic, CVD mortality spiked sharply to 244.4 per 100,000 population, which may be attributed to constraints in the organizational and methodological work on cause-of-death analysis across nosological groups during the pandemic. Over the period from 2020 to 2024, CVD mortality in the RD declined from 244.4 to 191.8 per 100,000 population. The highest CVD mortality rates were recorded in the highland districts of the RD, where older age groups predominate. It should be noted that CHD has remained the primary driver of high CVD mortality over the past decade, accounting for 58.4% in 2024[1].

Stable exertional angina is the most frequently diagnosed form of CHD. It is characterized by a relatively predictable pattern of ischemic chest pain that occurs with physical exertion or emotional stress and is rapidly relieved by rest [5].

Patients with stable angina are at elevated risk of subsequent myocardial infarction or sudden cardiac death due to rupture of an unstable atherosclerotic plaque. The mean annual event rate is approximately 2% [5].

Although modern treatment strategies substantially improve the prognosis of patients with stable CHD, individualized risk stratification for adverse outcomes in this population remains an important clinical challenge. A key trend in contemporary medicine in general, and in cardiology in particular, is the shift toward personalized medicine, which requires precise risk stratification based on each patient's individual characteristics. However, existing prognostic scores are frequently limited either by a narrow set of parameters or by insufficient adaptation to real-world clinical practice.

In this context, predicting mortality and non-fatal cardiovascular events (CVEs) in patients with stable CHD using clinical, anamnestic, laboratory, and instrumental data routinely available in clinical prac-

[1] Resolution of the Government of the Republic of Dagestan On approval of the regional program of the Republic of Dagestan "Optimal medical rehabilitation for health restoration in the Republic of Dagestan" dated June 30, 2025 № 219.

[2] Resolution of the Government of the Republic of Dagestan On Amendments to the State Program of the Republic of Dagestan "Combating Cardiovascular Diseases" dated June 30, 2021 № 159.

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tice represents a task of both scientific and practical importance.

Objective

The aim of this study was to identify factors associated with poor prognosis in patients with stable CHD and to develop a statistically validated prognostic model for mortality and non-fatal CVE risk based on clinical and anamnestic data available at initial hospitalization.

Methods

This retrospective, observational, cohort study enrolled 85 patients who underwent planned inpatient treatment between 01.01.2015 and 31.12.2017 with a diagnosis of CHD with stable exertional angina at the Dagestan Center of Cardiology and Cardiovascular Surgery (DCCVS). Data were collected from medical records archived at the DCCVS for the above period, followed by verification of long-term outcomes. Telephone contact was established with enrolled patients to ascertain vital status and to record cardiovascular events. Patients were invited to the DCCVS for a follow-up assessment comprising clinical and anamnestic data collection and laboratory and instrumental investigations (complete blood count, biochemical blood panel, lipid profile, electrocardiography, echocardiography).

Inclusion criteria

Inclusion criteria: planned hospitalization at the DCCVS from 01.01.2015 to 31.12.2017 with a diagnosis of stable exertional angina; residence within the RD; coronary angiography (CAG) performed during the index hospitalization; availability of a medical record in the hospital archive containing clinical, anamnestic, and laboratory/instrumental data at the time of the index hospitalization.

Exclusion criteria

Exclusion criteria: refusal to participate in the study; diagnosis of acute myocardial infarction or unstable angina at admission or within one month prior to the index hospitalization; emergency CAG or percutaneous coronary intervention performed within the first 24 hours of admission; residence outside the RD; relocation outside the RD; absence of CAG data at the index hospitalization; absence of a medical record in the hospital archive; presence of an active malignancy at the time of index hospitalization.

The study identified factors associated with adverse outcomes in patients with stable CHD, assessed the incidence of the primary composite endpoint (PCE)—defined as all-cause death or non-fatal CVE (myocardial infarction, stroke, or transient ischemic attack)—and developed a statistically validated model for predicting PCE risk based on clinical and anamnestic data available at the index hospitalization.

PCE components observed in the study population were: 5 deaths, 2 myocardial infarctions, 3 strokes, and 3 transient ischemic attacks.

The study was conducted in accordance with Good Clinical Practice standards and the principles of the Declaration of Helsinki. The Ethics Committee of Dagestan State Medical University of the Ministry of Health of the Russian Federation approved the study protocol. Written informed consent was obtained from all participants prior to enrollment.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics, version 26. The null hypothesis served as the basis for significance testing, with a p-value of <0.05 considered statistically significant. A binary logistic regression model with stepwise backward elimination (Wald method) was used to identify independent predictors of PCE. Model quality was assessed, and sensitivity and specificity were calculated by constructing receiver operating characteristic (ROC) curves with determination of the area under the curve (AUC).

Results

The study included 85 patients: 62 men (72.9%) and 23 women (27.1%). Mean age at index hospitalization was 58.55 ± 8.5 years (95% CI: 56.39–60.71) in men and 61.96 ± 8.6 years (95% CI: 58.23–65.69) in women. At the time of follow-up assessment, mean age was 62.84 ± 8.6 years (95% CI: 60.58–65.11) in men and 66.36 ± 8.9 years (95% CI: 62.38–70.35) in women.

Patients were followed for 4 years. During this period, all-cause mortality was 5.9% (5 patients): 1 woman (1.7%) and 4 men (6.7%). The mean age at death among men was 63 ± 7.26 years (95% CI: 51.45–74.55); the female decedent was 66 years old.

The Kaplan-Meier survival curve (Fig. 1) depicts the timing of deaths in the study population. Survival analysis yielded a mean survival time of 55.9 ± 0.69 months (95% CI: 54.54–57.26 months). The median

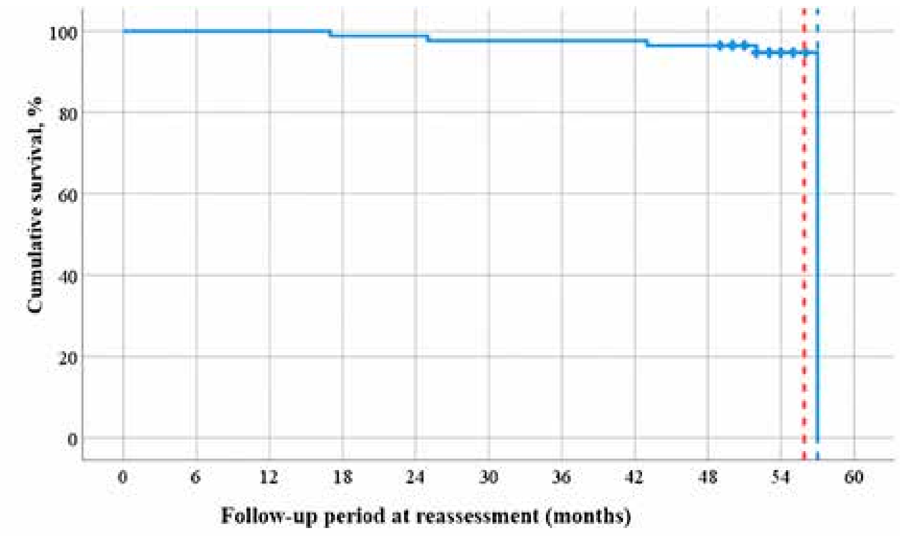

Fig. 1. Kaplan-Meier curve, characterizing the survival of the patients with stable CHD (months)

survival time, corresponding to the estimated time to death in at least 50% of patients, was 57 months.

Among the causes of death, 2 cases were classified as sudden cardiac death (40% of decedents), 2 deaths were attributable to concomitant malignancies, and the cause of death could not be established in 1 patient (20% of decedents).

The incidence of deaths and CVEs as a proportion of the total study population (n=85) and per 1,000 patient-years over the follow-up period is presented in Table 1.

Table 1. **Incidence of deaths and CVEs recorded during 4 years of follow-up in the study population, (n=85)**

Event	Number of recorded events		Event frequency to 1000 patient-years
	n	%	
Death	5	5.9 %	21.4
Myocardial infarction	2	2.4 %	6.1
Stroke	3	3.5 %	9.3
Transient ischemic attack	3	3.5 %	9.1

During the 4-year follow-up, 84.7% of patients (72) required repeated hospitalizations due to CHD progression.

The PCE was reached in 15.3% of patients (n=13).

The Kaplan-Meier curve (Fig. 2) shows the time to PCE. Event-free survival analysis yielded a mean time to PCE of 52.62 ± 1.42 months (95% CI: 49.83–55.40 months). The median time to PCE, corresponding to the estimated time at which at least 50% of patients would have reached the endpoint, was 57 months.

Univariate analysis identified echocardiographic ventricular dilatation, chronic occlusion of the left circumflex artery (LCx) on CAG, and stenosis of the posterior descending artery (PDA) on CAG as factors with the greatest statistically significant impact on the odds of PCE (Table 2).

PCE rates were significantly higher in patients with echocardiographic ventricular dilatation versus those without (p=0.036), in patients with chronic LCx occlusion on CAG versus those without (p=0.045), and in patients with PDA stenosis on CAG versus those without (p=0.022). The odds of PCE were 4.36-fold higher with ventricular dilatation (95% CI: 1.15–16.45), 6.8-fold higher with chronic LCx occlusion (95% CI: 1.20–38.45), and 6.89-fold higher with PDA stenosis (95% CI: 1.46–32.53). The associations between PCE and

Table 2. **Comparison of PCE rates by presence of echocardiographic ventricular dilatation, chronic LCx occlusion, and PDA stenosis on CAG at index hospitalization**

Risk factor	PCE rate				OR	95 % CI	p
	Factor present		Factor absent				
	n	%	n	%			
Echocardiographic ventricular dilatation	5	33.3	7	10.3	4.36	1.15–16.45	0.036*
Chronic LCx occlusion on CAG	3	50	10	12.8	6.8	1.2–38.45	0.045*
PDA stenosis on CAG	4	50	9	12.7	6.89	1.46–32.53	0.022*

Note *—statistically significant difference (p<0.05).

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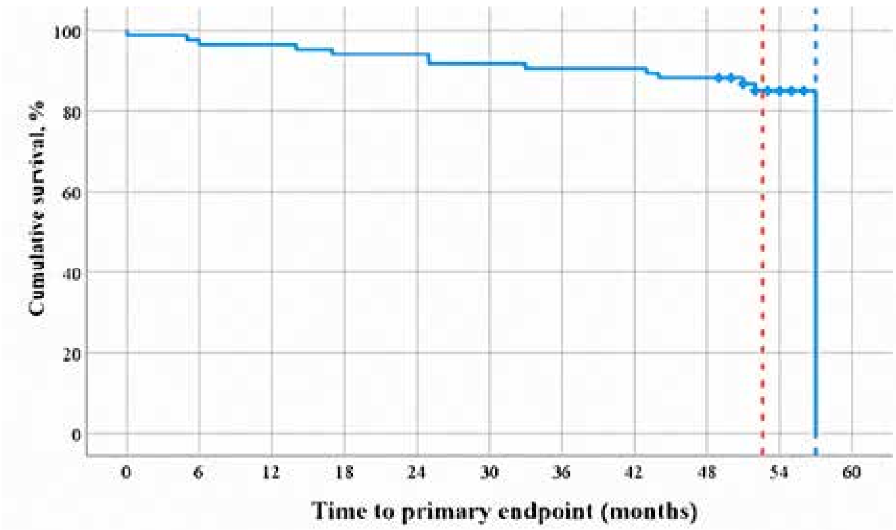

Fig. 2. Kaplan-Meier curve, characterizing reaching of the PCE over the period of observation (months)

each of these three factors were moderate (Cramér's V=0.252, 0.265, and 0.304, respectively).

However, in multivariate analysis, chronic LCx occlusion on CAG emerged as the sole statistically significant independent predictor, associated with a marked increase in the odds of PCE (OR ≈ 67.8; p=0.034) (Table 3).

Table 3. **Association between model predictors and PCE probability**

Factors	Change in odds with factor present		p
	OR	95 % CI	
Hematocrit level on CBC (%)	1.502	0.979-2.305	0.063
LA volume on Echo (mL)	0.854	0.728-1.002	0.053
Chronic LCx occlusion on CAG	67.791	1.387-3313.552	0.034*

Note *—statistically significant impact of a factor (p<0.05)

Using binary logistic regression, we developed a prognostic model for the probability of PCE in patients with stable CHD based on clinical and anamnestic data available at the index hospitalization. The relationship is described by equation (1):

$$P = 1 / (1 + e^{-z}) \times 100\ \%$$

$$z = -11.016 + 0.407 \times X_{HEMAT} - 0.158 \times X_{LAV} + 4.216 \times X_{OCCL.LCx} \quad (1),$$

where P—probability of PCE (%); X_{HEMAT} = hematocrit level on CBC (%); X_{LAV} = left atrial volume on Echo (mL); $X_{OCCL.LCx}$= chronic LCx occlusion on CAG (0 = absent, 1 = present).

The regression model was statistically significant (p<0.001). Based on the Nagelkerke R^2, model (1) accounted for 60.2% of the factors determining the probability of PCE.

Based on regression coefficients, hematocrit level and chronic LCx occlusion on CAG were positively associated with PCE probability, whereas echocardiographic left atrial (LA) volume was inversely associated. A 1% increase in hematocrit increased the odds of PCE 1.502-fold (95% CI: 0.979–2.305); chronic LCx occlusion on CAG increased the odds 67.791-fold (95% CI: 1.387–3313.552); a 1 mL increase in LA volume, conversely, decreased the odds 1.171-fold (OR=0.854; 95% CI: 0.728–1.002) (Table 3).

Although the contributions of hematocrit and LA volume did not reach strict statistical significance, both variables were retained in the prognostic model as clinically relevant parameters.

Figure 3 presents the OR values with 95% CI for the factors included in model (1).

The threshold value of logistic function P was determined by ROC curve analysis. The resulting curve is presented in Figure 4.

The area under the ROC curve for the relationship between predicted PCE probability in stable CHD patients and the logistic regression function values was 0.92 ± 0.07 (95% CI: 0.78–1.00). The model was statistically significant (p<0.001).

The cut-off value of function P (1) was 50.9%. Values of P >50.9% indicated high risk of PCE; values of P <50.9% indicated low risk. At this threshold, model sensitivity was 85.7% and specificity was 97.4%.

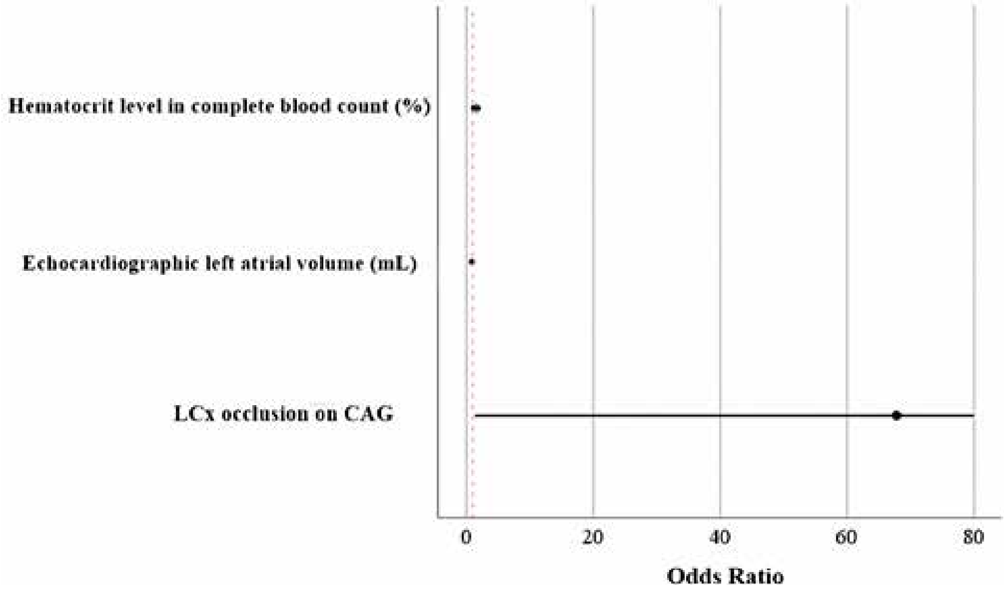

Fig. 3. Values of OR with 95 % CI for the studied factors, contributing to the reaching the PCE in patients with stable CHD

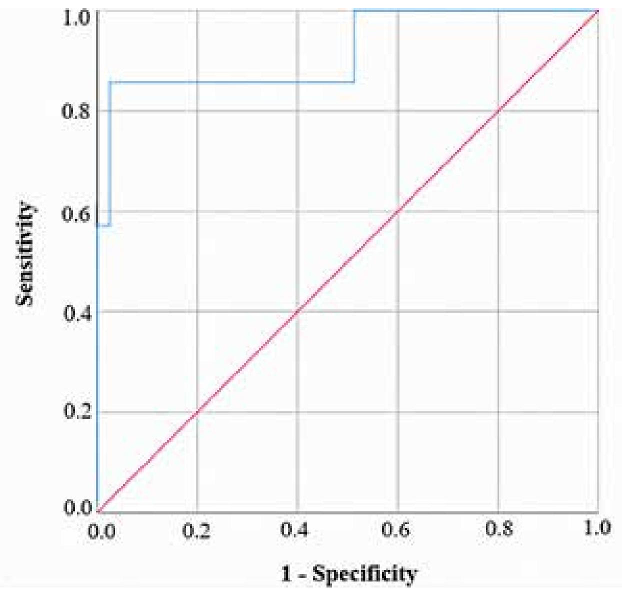

Fig. 4. ROC curve showing the performance of the prognostic model (1) in predicting the probability of the PCE in patients with stable CHD

Discussion

The overall mortality of 5.9%, corresponding to 21.4 deaths per 1,000 patient-years, is broadly consistent with mortality rates reported in comparable studies, such as the PROGNOZ IBS study [6], which enrolled patients with similar clinical characteristics.

The Framingham Heart Study reported 2-year rates of acute myocardial infarction and cardiovascular mortality of 6.2% and 3.8%, respectively, in women and 14.3% and 5.5% in men among patients with stable angina [7].

Well-established adverse prognostic factors in stable CHD include advanced age, severe angina, significant myocardial ischemia, extensive coronary atherosclerosis, proximal plaque localization, multivessel disease, severe chronic heart failure, tachycardia, severe depression, poor socioeconomic conditions, comorbidities such as chronic kidney disease, pulmonary disease, malignancy, and peripheral or cerebrovascular disease, as well as traditional cardiovascular risk factors (hypertension, dyslipidemia, diabetes mellitus [DM], smoking, obesity, etc.) [8, 9]. A relationship between survival in stable CHD patients and the extent of coronary atherosclerosis, degree of luminal narrowing, and anatomical distribution of stenoses has also been established [9]. According to the SCORE risk charts, the 10-year risk of cardiovascular mortality is approximately 4-fold higher in CHD patients aged 50 to 65 years, independent of other risk factors [10, 11].

The CLARICOR trial, which evaluated the predictive value of standard clinical parameters routinely available during outpatient visits for stable CHD patients presenting without new cardiac complaints, found that in univariate analysis smoking, DM, prior myocardial infarction, and use of calcium channel blockers, ACE inhibitors, long-acting nitrates, diuretics, cardiac glycosides, statins, high-sensitivity C-reactive protein, and reduced GFR were all significant predictors of the composite outcome comprising acute myocardial infarction, unstable angina, cerebrovascular disease, and all-cause mortality ($p<0.05$). In multivariate analysis, independent predictors of the composite outcome in stable CHD patients were smoking, DM, GFR, and use of long-acting nitrates and cardiac glycosides [12].

Comorbidity is a critical determinant of both the clinical course of stable CHD and long-term patient survival. Among CHD patients with comorbid conditions, those with concomitant chronic obstructive pulmonary disease (COPD) constitute a particularly important subgroup. Population-based data indicate that CHD patients with COPD face a 2- to 3-fold increase in the risk of cardiovascular death. Several studies have demonstrated that the leading cause of mortality in COPD patients is not respiratory failure but complications of coronary insufficiency [13].

Notably, several comorbidities—including COPD, bronchial asthma, and chronic kidney disease stages 3–5—that were associated with significantly increased PCE risk in prior studies such as PROGNOZ-IBS [8] did not demonstrate a statistically significant effect on PCE in our analysis, which may reflect the limited sample size and the low prevalence of these conditions in our cohort.

Our findings confirm the central role of coronary anatomy in predicting mortality and non-fatal CVEs in patients with stable CHD. Specifically, chronic LCx occlusion emerged as one of the most powerful independent predictors of adverse outcomes in our cohort. These results are consistent with established understanding of the prognostic significance of the location and severity of coronary atherosclerotic lesions in stable CHD [6].

The inclusion of laboratory and echocardiographic parameters (hematocrit level, echocardiographic LA volume) in our prognostic model reflects the multifactorial nature of outcome prediction and underscores the need for comprehensive risk assessment of mortality and CVEs in patients with stable CHD.

Conclusion

This study identified key factors associated with increased risk of death and CVEs in patients with stable CHD in the RD and enabled the development of a prognostic model based on clinical and instrumental parameters. The resulting model demonstrated high diagnostic accuracy and predictive precision.

The study results allow identification of patients at high risk of death and CVEs among those with stable CHD, thereby facilitating personalized treatment decisions and selection of patients who would benefit most from advanced therapeutic interventions. Furthermore, the findings provide a basis for optimizing secondary prevention of stable CHD in the RD healthcare system, enabling more rational and targeted allocation of public health resources.

Limitations of the study

The study was conducted with a limited number of patients (n=85) with a diagnosis of stable exertional angina. An additional limitation is the single-center study design.

Conflict of interest: none declared.

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