Association Between Healthy Lifestyle Index Score and Cardiovascular Mortality in Patients with Stable Coronary Artery Disease

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INTRODUCTION

Cardiovascular disease (CVD) remains one of the leading causes of death worldwide and continues to represent a major source of morbidity and mortality in the United States.[1] Among individuals diagnosed with stable coronary artery disease, long-term prognosis is influenced not only by clinical risk factors but also by modifiable lifestyle behaviors.[2] Preventive health behaviors such as maintaining a healthy diet, engaging in physical activity, avoiding smoking, and managing cardiometabolic risk factors have been shown to play an important role in reducing cardiovascular complications and mortality. As healthcare systems increasingly emphasize prevention and lifestyle modification, understanding the relationship between lifestyle behaviors and survival outcomes has become an important area of cardiovascular research. Healthy lifestyle indices have been increasingly used in epidemiologic and clinical research to summarize multiple behavioral factors into a single measure of overall health behavior.[3] These composite indices provide a more comprehensive understanding of lifestyle patterns than evaluating individual risk factors independently. Prior research suggests that healthier lifestyles are associated with improved cardiovascular outcomes;[4] however, the extent to which healthier baseline behaviors reduce the risk of cardiovascular mortality among low-risk patients with stable coronary artery disease remains an important clinical question.

The purpose of this study is to evaluate whether a healthier lifestyle, measured using the Healthy Lifestyle Index Score (HLIS), is associated with a reduced rate of death due to cardiovascular causes among low-risk patients with stable coronary artery disease. Specifically, this analysis examines differences in survival between patients with higher versus lower HLIS scores, while accounting for important demographic and clinical covariates, including age, systolic blood pressure, smoking status, diabetes status, and gender.

METHODS

All analyses were conducted using the pre-processed dataset *M215_Midterm_1.csv* provided for this assignment. The dataset included 4,475 low-risk patients with stable coronary artery disease. Variables included were time to cardiovascular death in months (t2cvdeath), cardiovascular death status (death; 1 = death, 0 = censored), Healthy Lifestyle Index Score (tx, 0 = HLIS < 4, 1 = HLIS ≥ 4), baseline age in years (age), systolic blood pressure (sbp), gender (0 = male, 1 = female), current smoking status (cursmk; 0 = no, 1 = yes), and current diabetes status (diab; 0 = no, 1 = yes). All analyses were conducted using R version 4.4.2. The primary exposure of interest was the Healthy Lifestyle Index Score (HLIS), dichotomized as high (≥ 4) and low (< 4). The outcome of interest was time to cardiovascular death. Continuous variables (age and systolic blood pressure) were summarized using means and standard deviations, and categorical variables (gender, smoking status, and diabetes status) were summarized using counts and percentages. Differences in these variables by HLIS were assessed using independent two-sample t-tests and chi-square tests, respectively.

To evaluate the association between HLIS and time to cardiovascular death, survival analyses were conducted. Survival probabilities by HLIS were visualized using Kaplan–Meier curves, and differences were assessed using the log-rank test. Two Cox proportional hazards models were fit: an unadjusted model and a multivariable model adjusting for age, systolic blood pressure, gender, smoking status, and diabetes status. Tied event times were handled using the Breslow method. The proportional hazards assumption was evaluated using Schoenfeld residuals. To assess potential effect modification by gender, an interaction term between HLIS and gender was later included in both the unadjusted and multivariable Cox models. All statistical tests were conducted using a significance level of $\alpha = 0.05$.

RESULTS

A. Baseline Characteristics by Healthy Lifestyle Index Score

Baseline characteristics of the cohort are summarized in Tables 1A and 1B, stratified by HLIS. Among 4,475 patients, 49.1% had HLIS < 4 and 50.9% had HLIS ≥ 4. Overall, demographic and clinical characteristics were well balanced between groups. Median age and systolic blood pressure were similar, with no statistically significant differences observed. Similarly, the distribution of categorical variables such as gender, smoking status, and diabetes status did not differ significantly between HLIS categories.

Table 1A. Baseline Characteristics by Healthy Lifestyle Index Score (Categorical Variables)

Characteristic	**HLIS < 4, N = 2,200[1]**	**HLIS > 4, N = 2,275[1]**	**p-value[2]**
Gender			0.4
Female	384 (17%)	419 (18%)	
Male	1,816 (83%)	1,856 (82%)	
Current Smoking Status			0.4
No Smoking	1,845 (84%)	1,887 (83%)	
Yes Smoking	355 (16%)	388 (17%)	
Current Diabetes Status			0.2
No Diabetes	1,188 (54%)	1,273 (56%)	
Yes Diabetes	1,012 (46%)	1,002 (44%)	

[1]n (%), [2]Pearson's Chi-squared test

Table 1B. Baseline Characteristics by Healthy Lifestyle Index Score (Continuous Variables)

Characteristic	**HLIS < 4**, N = 2,200[1]	**HLIS > 4** , N = 2,275[1]	**p-value**[2]
Age	50 (44, 57)	51 (44, 57)	0.081
SBP	129 (118, 141)	129 (119, 140)	0.8

[1]Median (Q1, Q3), [2]Welch Two Sample t-test

B. Association Between Healthy Lifestyle Index Score and Cardiovascular Mortality

Kaplan–Meier survival curves were generated to evaluate the association between Healthy Lifestyle Index Score (HLIS) and cardiovascular mortality (Figure 1). Median survival was 84 months (95% CI: 82–86) among patients with a high HLIS and 80 months (95% CI: 79–82) among those with a low HLIS. At 36 months, estimated survival was similar between groups (High HLIS: 0.960 vs. Low HLIS: 0.961). By 60 months, survival was higher in the high HLIS group (0.838, 95% CI: 0.821–0.855) compared with the low HLIS group (0.792, 95% CI: 0.773–0.811). The difference in survival between groups was statistically significant based on the log-rank test ($p < 0.001$).

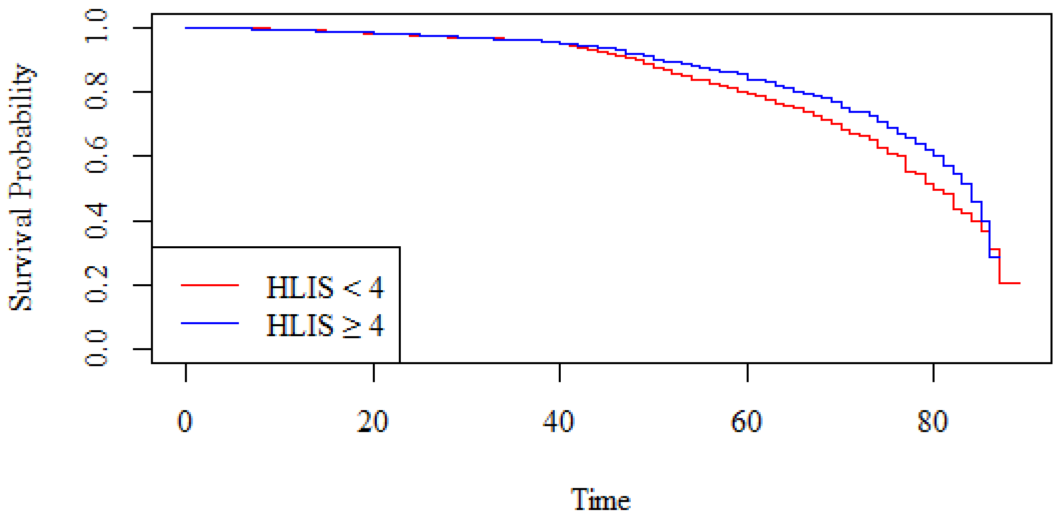

Figure 1. KM Plot for Low HLIS and High HLIS

Table 2 displays the unadjusted Cox proportional hazards regression, illustrating a higher HLIS was associated with a lower hazard of cardiovascular death. The hazard ratio (HR) for HLIS ≥ 4 compared with HLIS < 4 was 0.76 (95% CI: 0.67–0.86; $p < 0.001$). The multivariable Cox proportional hazards model estimates the association between HLIS and cardiovascular mortality while adjusting for age, systolic blood pressure, gender, smoking status, and diabetes status (Table 3). The results were similar as HLIS ≥ 4 was associated with a lower hazard (HR = 0.76; 95% CI: 0.67–0.86; $p < 0.001$). On the other hand, covariates associated with higher hazard included higher systolic blood pressure ($p = 0.049$) and current smoking ($p = 0.018$). Age, gender, and diabetes status were not statistically significant at the 0.05 level. The hazard ratio for HLIS was unchanged after multivariable adjustment, suggesting minimal confounding by age, systolic blood pressure, gender, smoking status, or diabetes.

Table 2. Summary of Unadjusted & Adjusted Cox-PH Models

Unadjusted Model: Surv(t2cvdeath, death) ~ tx				
Variable	Hazard Ratio	Lower 95% CI	Upper 95% CI	p-value
HLIS (tx)	0.76	0.672	0.86	0.0001
Adjusted Model: Surv(t2cvdeath, death) ~ tx + age + sbp + gender + cursmk + diab				
Variable	Hazard Ratio	Lower 95% CI	Upper 95% CI	p-value
HLIS (tx)	0.758	0.670	0.858	0.0004
Age (age)	1.007	0.999	1.015	0.088
Systolic Blood Pressure (sbp)	1.004	1.000	1.008	0.049
Gender (gender)	0.868	0.736	1.024	0.093
Smoking Status (cursmk)	1.210	1.034	1.415	0.018
Diabetes (diab)	1.105	0.972	1.256	0.127

To explore whether the effect of HLIS on cardiovascular mortality differed by gender, an interaction term between HLIS and gender was included in both Cox models (Table 3). In both the unadjusted model and adjusted model, the interaction term (HLIS × gender) was not statistically significant ($p = 0.32$; $p = 0.28$), indicating no evidence of effect modification by gender. However, the main effect of HLIS in both models remained protective (HR = 0.78), suggesting a consistent association across males and females.

Table 3. Summary of Unadjusted & Adjusted Cox-PH Models with Gender Interaction

Unadjusted Model: Surv(t2cvdeath, death) ~ tx * gender				
Variable	Hazard Ratio	Lower 95% CI	Upper 95% CI	p-value
HLIS (tx)	0.784	0.684	0.897	0.00042
Gender (gender)	0.978	0.785	1.218	0.842
HLIS x Gender (tx:gender)	0.847	0.610	1.177	0.323
Adjusted Model: Surv(t2cvdeath, death) ~ tx * gender + age + sbp + cursmk + diab				
Variable	Hazard Ratio	Lower 95% CI	Upper 95% CI	p-value
HLIS (tx)	0.782	0.683	0.896	0.00002
Gender (gender)	0.944	0.758	1.177	0.610
Age (age)	1.007	0.999	1.015	0.083
Systolic Blood Pressure (sbp)	1.004	1.000	1.008	0.048
Smoking Status (cursmk)	1.211	1.035	1.417	0.017
Diabetes Status (diab)	1.105	0.972	1.257	0.126
HLIS x Gender (tx:gender)	0.833	0.599	1.157	0.275

DISCUSSION

In this cohort, a higher Healthy Lifestyle Index Score was associated with a lower risk of cardiovascular mortality. The similarity between unadjusted and adjusted hazard ratios suggests that the association is not substantially confounded by age, blood pressure, gender, smoking, or diabetes, indicating that HLIS may reflect aspects of cardiovascular risk beyond these traditional factors. Kaplan–Meier curves also demonstrated longer survival among individuals with higher HLIS. The protective association appears to reflect the combined effects of multiple healthy behaviors, which may improve vascular function, reduce inflammation, and support overall health. The absence of a significant interaction by gender indicates that the relationship between HLIS and mortality appears consistent in both males and females. Smoking and higher systolic blood pressure remained independently associated with increased mortality, consistent with established cardiovascular risk factors and existing research.

Several limitations should be considered when interpreting these results. First, the Healthy Lifestyle Index was dichotomized for analysis, which may obscure more nuanced relationships between lifestyle and survival. Additionally, all lifestyle data were assessed at baseline, and changes over time were not captured, potentially underestimating the true association. Finally, the cohort represents a specific low-risk population with stable coronary artery disease, which may limit generalizability to other populations with varying illnesses or higher-risk patients.

Despite these limitations, the study provides evidence that a healthier lifestyle is associated with improved survival, even after accounting for age, gender, blood pressure, smoking status and diabetes status. These results underscore the relevance of lifestyle interventions in healthcare and support continued efforts to promote healthy behaviors among patients with coronary artery disease. Future research should test additional variables such as BMI, other pre-existing health conditions, and mental health factors as well as include more demographic information such as race/ethnicity.

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