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Employment status as a predictor of adverse outcomes in patients with coronary artery disease

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ABSTRACT

Background: Lack of employment is associated with a poorer prognosis when compared to employment in the general population. Whether this association is present in patients with coronary artery disease (CAD) and similarly extends to adverse cardiovascular outcomes in those with CAD remains unknown. Herein, we analyzed the association of employment status and adverse outcomes in patients with CAD.

Methods: Non-retired participants enrolled in the Emory Cardiovascular Biobank during the years 2003–2023 were divided into employed and non-employed cohorts and analyzed for the primary composite outcome of cardiovascular death or non-fatal myocardial infarction (MI) by competing-risk regression, while accounting for non-cardiovascular death. The secondary outcome of all-cause death was assessed by Cox regression. Sensitivity analyses were performed to explore for heterogeneity of effect.

Results: The study sample consisted of 2809 participants with a mean age of 57 years, 35 % women, and 27 % Black. During a median follow-up of 5.1 years, 406 (14 %) cardiovascular death or non-fatal MI events and 537 (19 %) all-cause death events occurred. After adjustment for demographics, educational attainment level (EAL), and traditional cardiovascular risk factors, non-employment was found to be a significant predictor of cardiovascular death or non-fatal MI (sHR 1.78, 95 % CI 1.41–2.19, $P < 0.001$) and all-cause death (HR 2.46, 95 % CI 2.02–2.99, $P < 0.001$), when compared to employed participants.

Conclusions: Non-employment is associated with adverse outcomes in non-retired individuals with CAD, independent of demographics, EAL, and traditional cardiovascular risk factors. Non-employment due to disability was associated with the highest risk of adverse cardiovascular outcomes, highlighting a particularly vulnerable subgroup.

1. Introduction

Coronary artery disease (CAD) is a primary contributor to cardiovascular disease (CVD), the leading cause of mortality in the United States (US) [1]. Despite the identification of several risk factors traditionally associated with the development of CAD, individuals of lower socioeconomic status (SES) remain disproportionately affected [1,2]. The important role that SES plays in the prevalence and progression of CAD has become increasingly recognized and may in part explain the excess burden of poor outcomes in patients with CVD [3,4].

Employment status (ES) is a commonly utilized surrogate for SES and has been associated with both the development of incident CVD and

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related adverse outcomes in the general population [5–7]. Non-employment in the general population has been shown to portend a worse prognosis when compared to employed individuals, and even when examined in higher SES strata, the relationship between non-employment and adverse outcomes persists [8].

This association between non-employment and adverse outcomes has yet to be thoroughly examined in patients with CAD. Herein, we analyzed the association between employment status and adverse outcomes in working-age patients with CAD, with the hypothesis that non-employment would be significantly associated with incident cardiovascular and all-cause mortality in patients with CAD, independent of demographics, other indices of SES, and traditional cardiovascular risk factors, when compared to employment. We further examined the impact of self-reported race on the relationship between employment status and adverse outcome risk.

2. Material and methods

2.1. Study population

This study included participants enrolled in the Emory Cardiovascular Biobank (EmCAB). EmCAB is an ongoing, prospective registry of adults undergoing left heart catheterization at three Emory Healthcare-affiliated hospitals in Atlanta, Georgia, as detailed previously [9]. All participants enrolled between May of 2003 and November of 2023 were eligible for inclusion in the current study. EmCAB participants with a history of heart transplantation, severe congenital heart disease, severe valvular heart disease, severe anemia, recent blood transfusion, active inflammatory disease, active cancer, or dementia at the time of recruitment were excluded from EmCAB. This study was approved by the institutional review board of Emory University (Atlanta, GA) and was conducted and reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cohort studies. All participants provided written informed consent at time of enrollment into EmCAB.

2.2. Employment status

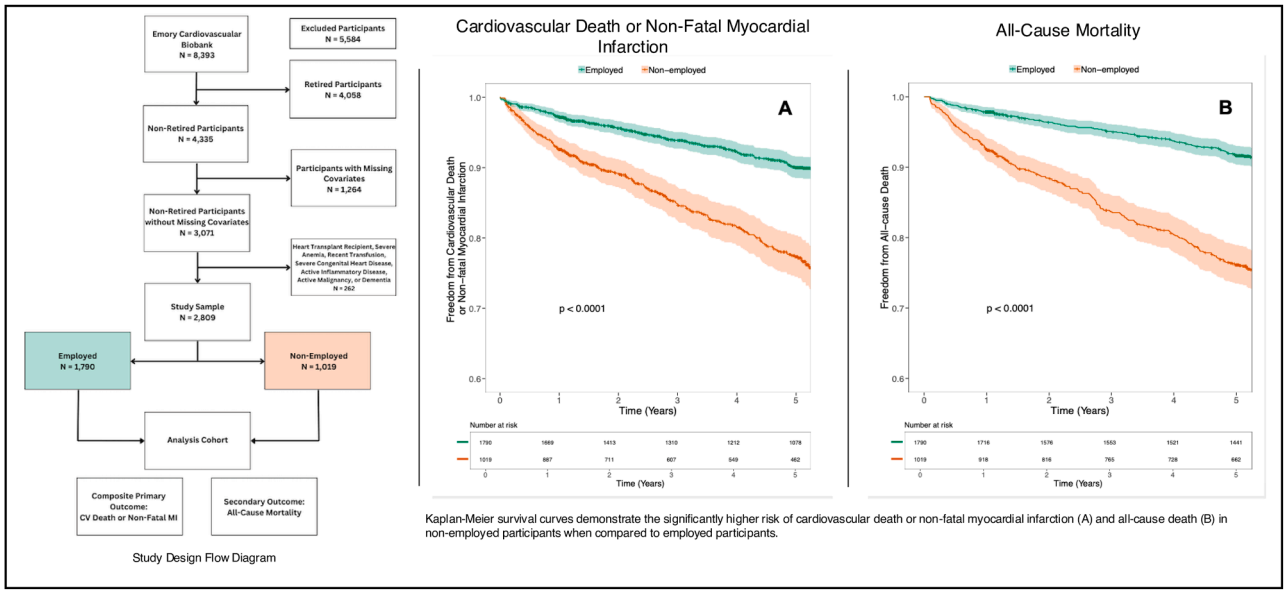
At the time of recruitment into EmCAB, participants indicated their employmentstatus as: full time (≥35 hours per week), part time (<35 hours per week), unemployed, disabled, or retired. Throughout the EmCAB enrollment process, participants' definitions of "retired" were found to be subjective and to vary significantly between individuals. As such, given the heterogeneity of this definition and the importance of the fidelity of the employment status variable in this analysis, participants who selected "retired" on the employment status survey were excluded from this study. Participants who selected full time or part time were considered "employed", and participants who selected unemployed or disabled were considered "non-employed." A total of 2809 EmCAB participants met either of these definitions and were included into the following analyses (see Central Illustration, Flow Diagram).

2.3. Cardiovascular risk factors

During the recruitment process into EmCAB, participants were interviewed at a baseline visit to document their demographics, medical history, medication use, and socio-behavioral habits. The presence of cardiovascular risk factors, such as diabetes mellitus, hypertension, hypercholesterolemia, chronic kidney disease (CKD), heart failure, and history of myocardial infarction (MI) were documented per physician diagnosis or treatment. Medical records and International Classification of Diseases, Ninth (ICD-9) and Tenth (ICD-10) diagnostic codes were reviewed to corroborate participant-reported medical history. Height and weight were measured at the time of recruitment into EmCAB, and body mass index (BMI) was calculated by dividing weight in kilograms by the square of height in meters-squared. Left ventricular ejection fraction (LVEF) was documented by review of the most recent transthoracic echocardiogram nearest to the time of recruitment or by left ventricular angiography during cardiac catheterization. Estimated glomerular filtration rate (eGFR) was calculated via the Chronic Kidney Disease Epidemiology Collaboration Equation [10].

2.4. Follow-up and adverse outcomes

The primary outcome was defined as the composite of cardiovascular death or incident non-fatal MI. The secondary outcome was defined as all-cause death. Participants in EmCAB were prospectively followed for outcome occurrence by phone contact at 1 year, 5 years, and every subsequent 5 years following enrollment, by electronic medical record review, and by surveillance of the social security death index and state records. Cause of death was ascertained from review of medical records or from contact with participants' relatives. Cardiovascular death or



Central Illustration. Study Design Flow Diagram (Left). Kaplan-Meier survival curves demonstrate the significantly higher risk of cardiovascular death or non-fatal myocardial infarction (A) and all-cause death (B) in non-employed participants when compared to employed participants (Right).

non-fatal MI were adjudicated separately by two cardiovascular medicine physicians who were blinded to EmCAB data. Cardiovascular death was defined as death secondary to cardiovascular ischemia, stroke, or sudden death as result of a presumed cardiovascular cause [11]. The outcome of non-fatal MI was adjudicated using the third and fourth universal definition of MI [12,13]. Participants without any available outcome data during the follow-up period were considered lost to follow-up and excluded from time-to-event analyses.

2.5. Statistical analysis

Participant demographics and comorbidities were reported as frequencies for categorical variables. Medians [interquartile range] were reported for all continuous variables. The Pearson Chi-squared test was utilized to assess for differences in categorical variables between employment status groups, and the Wilcoxon rank-sum test was utilized to assess for differences in continuous variables between employment status groups. Propensity matching was not performed in order to better represent the implications of SES as a contributor to individual-level cardiovascular risk. We assessed missing data across all covariates included in multivariable models. Visual inspection was used to evaluate patterns and potential mechanisms of missingness. Based on this assessment, missingness was determined to be consistent with a Missing At Random (MAR) mechanism. Therefore, we conducted a complete case analysis for all primary and secondary outcomes.

Kaplan-Meier survival curves were utilized to visualize the survival data. The independent association between employment status and the primary outcome of cardiovascular death or non-fatal MI was assessed with stepwise multivariable Fine and Gray competing-risk regression modeling, while accounting for non-cardiovascular death [14]. The independent association between employment status and the secondary outcome of all-cause death was assessed with multivariable Cox proportional hazards regression modeling. Models were adjusted for demographics (age, sex, race [Black versus non-Black]), educational attainment level (EAL, stratified by completion of college), and traditional cardiovascular risk factors (BMI, smoking history, diabetes mellitus, hypertension, hypercholesterolemia, heart failure, history of MI, LVEF, eGFR, aspirin use, and statin use). Additional analyses were performed to examine the risk of adverse event occurrence between part-time employed, unemployed, and disabled subgroups when compared to the full-time employed subgroup.

Sensitivity analyses were performed to examine for effect modification between employment status and other significant predictors of cardiovascular death or non-fatal MI identified in multivariable competing-risk regression modeling. For these analyses, LVEF was dichotomized at 50 %, and eGFR was dichotomized at 60 mL/min per 1.73 m^2. To assess the potential impact of missing data, we utilized multiple imputation (with 20 iterations) by chained equations (MICE) and fit multivariable competing risk regressions for the primary composite outcome within each imputed dataset. Risk estimates were then pooled across imputed datasets using Rubin's rules and compared to the estimates of the primary analysis. Additional post-hoc analyses comparing the risk of cardiovascular death or non-fatal MI between the Black and non-Black cohorts of our sample were also performed to explore the impact of race on the relationship between employment status and adverse cardiovascular outcomes.

All analyses were performed using R version 4.2.2 (https://www.r-project.org/).

3. Results

3.1. Baseline characteristics

Participants' baseline demographics and pertinent clinical characteristics are included in Table 1. The study cohort included 2809 participants with a mean age of 56.5 (SD 10.3) years, 65 % men, and 27 % individuals of self-identified Black race. The mean age of the excluded retired cohort was 71.7 (SD 7.9) years. Among the employed cohort, 1515 (84.6 %) were employed full-time and 275 (15.4 %) were employed part-time. The non-employed cohort comprised 36.3 % (N = 1019) of the entire study cohort. Within the non-employed cohort, 289 (28.4 %) were unemployed, and 730 (71.6 %) were disabled. When compared to participants in the employed cohort, participants in the non-employed cohort were significantly younger, more frequently female and Black, had a lower EAL, and had a higher prevalence of smoking, diabetes mellitus, hypertension, and history of MI and HF. The non-employed cohort was also found to have a significantly lower LVEF and eGFR, as well as lower aspirin and statin usage.

3.2. Employment status and adverse outcomes

During a median follow-up time of 5.1 [IQR 4.4] years, 406 (14 %) cardiovascular death or non-fatal MI events and 537 (19 %) all-cause death events occurred. Follow-up data were available for 95 % of the cohort, with 5 % of participants being lost to follow-up and excluded from time-to-event analyses. Kaplan-Meier survival analysis demonstrated a significantly higher rate of cardiovascular death or non-fatal MI (Central Illustration, A) and all-cause death (Central Illustration, B) in the non-employed group compared to the employed group. Kaplan-Meier survival analysis by employment status subgroup demonstrated

Table 1
Baseline Characteristics of Study Sample.

Variable	All (N = 2809)	Employment Status		P-value
		Employed (N = 1790)	Non-employed (N = 1019)	
Age (Years)	57.2 (12.8)	57.8 (13.0)	56.2 (12.7)	<0.001
Male	1818 (65 %)	1292 (72 %)	526 (52 %)	<0.001
Black	749 (27 %)	341 (19 %)	408 (40 %)	<0.001
Smoking History	1821 (65 %)	1120 (63 %)	701 (69 %)	<0.001
Low EAL (By College)	1765 (63 %)	934 (52 %)	831 (82 %)	<0.001
Body Mass Index (kg/m^2)	29.6 (8.0)	29.5 (7.6)	29.8 (9.6)	0.300
History of MI	636 (22 %)	364 (20 %)	272 (27 %)	<0.001
Heart Failure	744 (26 %)	367 (21 %)	377 (37 %)	<0.001
Diabetes Mellitus[a]	975 (35 %)	529 (30 %)	446 (44 %)	<0.001
Hypertension	2086 (74 %)	1269 (71 %)	817 (80 %)	<0.001
Hypercholesterolemia	1866 (66 %)	1193 (67 %)	673 (66 %)	0.740
LVEF (%)	55.0 (10.0)	55.0 (10.0)	55.0 (15.0)	<0.001
eGFR (mL/min/1.73 m^2)	81.1 (30.6)	81.7 (27.7)	75.7 (42.5)	<0.001
Aspirin Use	2070 (74 %)	1369 (76 %)	701 (69 %)	<0.001
Statin Use	1900 (68 %)	1265 (71 %)	625 (62 %)	<0.001
Cardiovascular Death or Non-Fatal MI	406 (14 %)	187 (10 %)	219 (21 %)	<0.001
All-Cause Death	537 (19 %)	225 (13 %)	312 (31 %)	<0.001

Abbreviations: EAL (educational attainment level), MI (myocardial infarction), LVEF (left ventricular ejection fraction), eGFR (estimated glomerular filtration rate).

[a] Continuous variables are expressed as medians (IQR) and were compared by the Wilcoxon rank-sum test, and categorical variables are expressed as counts (percentage) and were compared by the Pearson Chi-squared test.

similar findings, with employed subgroups exhibiting a similar, lesser risk of adverse cardiovascular outcomes when compared to non-employment due to unemployment or disability (Fig. 1).

When compared to employed participants, non-employment was a significant predictor of cardiovascular death or non-fatal MI (sHR 2.42, 95 % CI 1.99–2.94, $P < 0.001$) in an unadjusted competing-risk regression model accounting for non-CV death (Table 2, Model 1). Similarly, unadjusted Cox regression demonstrated higher risk of all-cause mortality in the non-employed compared to the employed groups (HR 3.06, 95 % CI 2.58–3.64, $P < 0.001$) (Table 2, Model 1). After adjustment for demographics (age, sex, and Black race), non-employment remained a significant predictor of cardiovascular death or non-fatal MI (sHR 2.34, 95 % CI 1.90–2.89, $P < 0.001$) and all-cause death (HR 3.31, 95 % CI 2.76–3.98, $P < 0.001$) (Table 2, Model 2). Adjustment for EAL resulted in mild attenuation of the hazard of non-employment compared to employment as a predictor of cardiovascular death or non-fatal MI (sHR 2.10, 95 % CI 1.69–2.61, $P < 0.001$) and all-cause death (HR 3.10, 95 % CI 2.57–3.75, $P < 0.001$) (Table 2, Model 3). After full adjustment for traditional cardiovascular risk factors (BMI, history of MI, heart failure, diabetes mellitus, hypertension, hypercholesterolemia, LVEF, eGFR, aspirin use, and statin use), non-employment remained a significant predictor of cardiovascular death or non-fatal MI (sHR 1.78, 95 % CI 1.41–2.19, $P < 0.001$) and all-cause death (HR 2.46, 95 % CI 2.02–2.99, $P < 0.001$) when compared to the employed group (Table 2, Model 4).

Table 2
Multivariable Analyses of Employment Compared to Non-employment as a Predictor of Adverse Outcomes.

Models	Cardiovascular Death or Non-Fatal Myocardial Infarction[c]		All-Cause Death[d]	
	sHR (95 % CI)	P-Value	HR (95 % CI)	P-Value
Model 1: Non-employment (Unadjusted)	2.42 (1.99–2.94)	<0.001	3.06 (2.58–3.64)	<0.001
Model 2: Model 1 + Demographics[a]	2.34 (1.90–2.89)	<0.001	3.31 (2.76–3.98)	<0.001
Model 3: Model 2 + EAL	2.10 (1.69–2.61)	<0.001	3.10 (2.57–3.75)	<0.001
Model 4: Model 3 + traditional risk factors[b]	1.78 (1.41–2.19)	<0.001	2.46 (2.02–2.99)	<0.001

[a] Model adjusted for age, sex, and race.
[b] Model adjusted for educational attainment level, body mass index, history of myocardial infarction, heart failure, diabetes mellitus, hypertension, hypercholesterolemia, left ventricular ejection fraction, estimated glomerular filtration rate, aspirin use, statin use.
[c] Examined by Fine and Gray competing-risk regression, accounting for competing non-cardiovascular death.
[d] Examined by Cox proportional-hazards regression.

3.3. Employment status subgroups and adverse outcomes

Analyses examining the risk of cardiovascular death or non-fatal MI and all-cause death between individual employment status subgroups (full-time employed, part-time employed, unemployed, and disabled subgroups) in regression models adjusted for demographics, EAL, and traditional cardiovascular risk factors are shown in Table 3. No significant difference in adverse event risk was observed between full-time and part-time employed participants in multivariable regression modeling. When compared to full-time employed participants, unemployment was a significant predictor of all-cause death (HR 1.71, 95 % CI 1.25–2.35, $P = 0.001$) but only trended towards significance for the prediction of cardiovascular death or non-fatal MI (sHR 1.36, 0.96–2.00, $P = 0.082$). Disabled status was significantly predictive of both cardiovascular death or non-fatal MI (sHR 1.70, 95 % CI 1.32–2.19, $P < 0.001$) and all-cause death (HR 2.93, 95 % CI 2.33–3.69, $P < 0.001$) when compared to full-time employment. Lastly, within non-employed participants, when compared to unemployed participants, disabled status was not a significant predictor of cardiovascular death or non-fatal MI (sHR 1.22, 95 % CI 0.87–1.73, $P = 0.250$) but was of all-cause death (HR 1.71, 95 % CI 1.28–2.30, $P < 0.001$).

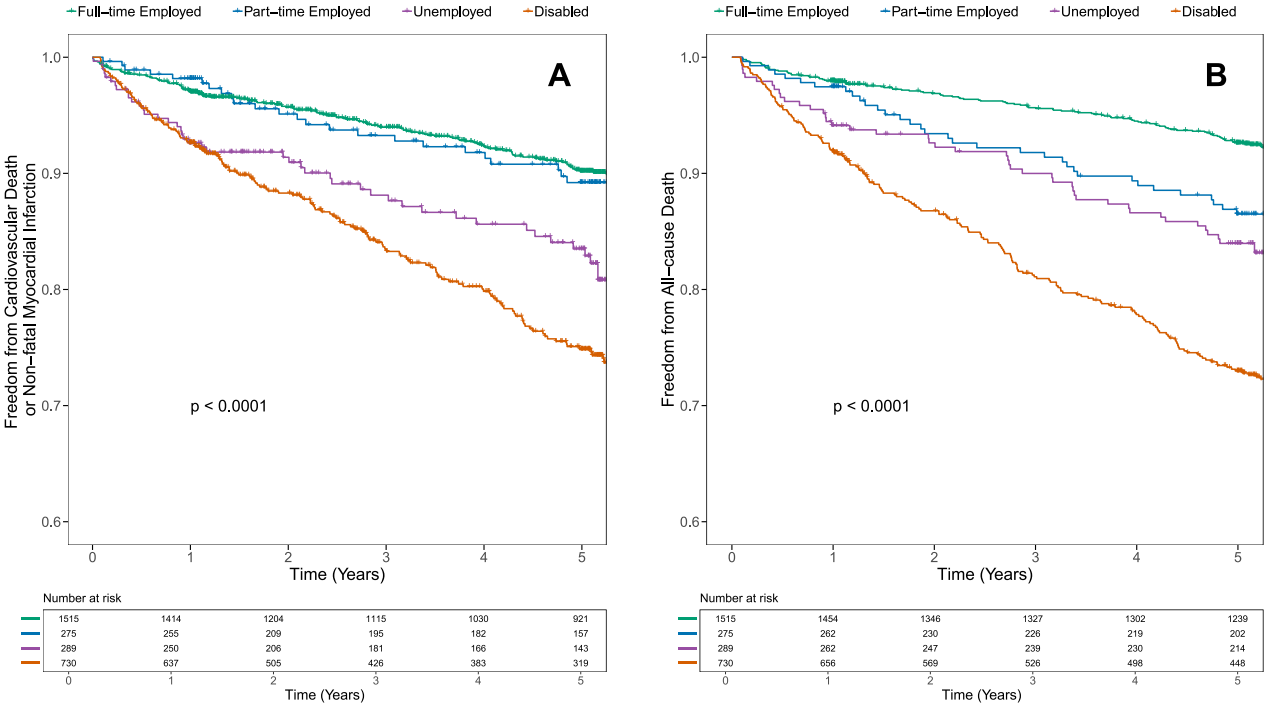


Fig. 1. Adverse Outcome Risk by Employment Status Subgroups. Kaplan-Meier survival curves demonstrate the significant difference in the risk of cardiovascular death or non-fatal myocardial infarction (A) and all-cause death (B) between employment status subgroups.

Table 3
Multivariable Analyses of Employment Status Subgroups Compared to Full-Time Employment as a Predictor of Adverse Outcomes.

Employment Status Subgroup	N	Cardiovascular Death or Non-Fatal Myocardial Infarction[a]		All-Cause Death[b]	
		sHR (95 % CI)[c]	P-Value	HR (95 % CI)[c]	P-Value
Full-Time Employment	1515	Reference	–	Reference	–
Part-Time Employment	275	0.99 (0.67–1.46)	0.950	1.22 (0.87–1.71)	0.240
Unemployment	289	1.39 (0.96–2.00)	0.082	1.71 (1.25–2.35)	0.001
Disabled	730	1.70 (1.32–2.19)	<0.001	2.93 (2.33–3.69)	<0.001

[a] Examined by multivariable-adjusted Fine and Gray competing-risk regression, accounting for competing non-cardiovascular death.
[b] Examined by multivariable-adjusted Cox proportional-hazards regression.
[c] Model adjusted for age, sex, Black race, smoking history, educational attainment level, body mass index, history of myocardial infarction, heart failure, diabetes mellitus, hypertension, hypercholesterolemia, left ventricular ejection fraction, estimated glomerular filtration rate, aspirin use, statin use.

3.4. Sensitivity analyses

A multiplicative, multivariable interaction analysis was performed to explore for effect modification of the association between employment status and cardiovascular death or non-fatal MI. A significant interaction between Black race and employment status was observed, such that non-employment was associated with a greater risk of cardiovascular death or non-fatal MI in non-Black participants (sHR 1.87, 95 % CI 1.42–2.46, $P < 0.001$) when compared to Black participants (sHR 1.19, 95 % CI 0.81–1.75, $P = 0.380$). Further post-hoc analyses, however, revealed a higher 5-year incidence of cardiovascular death or non-fatal MI in the Black cohort of our sample when compared to the non-Black cohort (18.8 % vs. 12.9 %, $P < 0.001$) (Table S1). Multivariable-adjusted competing-risk regression analysis similarly demonstrated a significantly higher risk of adverse cardiovascular outcomes in employed Black participants compared to employed non-Black participants irrespective of employment status and other clinical variables (sHR 1.66, 95 % CI 1.19–3.32, $P = 0.003$) (Table S2). There were no significant interactions observed between employment status and sex, EAL, history of MI, diabetes mellitus, LVEF, or eGFR for the prediction of adverse cardiovascular outcomes (Figure S1).

The counts and percentage of missing data for all variables included in multivariable analyses are shown in Table S3. In the imputed analysis, both the direction and magnitude of the association between non-employment and incident cardiovascular death or non-fatal myocardial infarction (sHR 1.65, 95 % CI 1.34–2.02, $P < 0.001$) (Table S4) were consistent with the complete-case analysis (sHR 1.78, 95 % CI 1.41–2.19, $P < 0.001$), supporting the robustness of the observed associations.

4. Discussion

In this large cohort study of non-retired patients with CAD, we show that non-employment, either due to unemployment or disability, was associated with a significantly higher risk of adverse cardiovascular outcomes and all-cause mortality when compared to those who were employed. This association was independent of demographics, educational attainment level, and traditional cardiovascular risk factors, with a greater than 75 % higher risk of adverse cardiovascular events observed in the non-employed group when compared to employed participants. Given that the majority of non-employed participants in our sample were disabled, and that disability was associated with the highest risk of adverse outcomes in subgroup analyses, it is likely that the observed associations in our primary analysis may have been driven by individuals with significant health-related work limitations. Although the association between non-employment and all-cause mortality has previously been described in the general population, our findings extend these observations to patients with CAD and furthermore encompass important adverse cardiovascular outcomes [15–18].

Perhaps the most striking finding in our analyses was that disability-related non-employment carried the greatest risk of adverse outcomes among all employment subgroups. Compared to full-time employed individuals, those with disability had a 70 % higher risk of cardiovascular death or non-fatal MI and nearly a three-fold higher risk of all-cause death. These findings persisted after adjustment for demographics, SES, and traditional risk factors. In contrast, unemployment without disability was associated with elevated risk of all-cause mortality but did not reach significance for cardiovascular events. Overall, these associations between non-employment and higher risk of adverse cardiovascular outcomes appeared to be greater in non-Black, predominantly White participants, although Black participants had overall higher event rates.

Our findings lay further groundwork for understanding the relationship between employment status and adverse health outcomes in vulnerable populations. It is vitally important to recognize that there exists a bidirectional relationship between lower SES, herein represented by lack of employment, and traditional cardiovascular risk factors, such that those who are not employed are more likely to be afflicted by comorbidities that lead to CVD and contribute to adverse outcome occurrence [19–23]. Moreover, patients with these comorbidities are more likely to be non-employed, as in our cohort. This phenomenon has previously been described by both the Coping and Latent Sickness hypotheses [15]. The former suggests that non-employment may give rise to adverse behavioral changes, which then contribute to deterioration in health and the acquisition of comorbid conditions, such as hypertension, hypercholesterolemia, diabetes mellitus, and obesity. The latter posits that these adverse health behaviors and risk factors likely always existed and contributed to the acquisition of non-employed status. In practice, both theories likely contribute to the employment status-mortality relationship observed by us and others and assist in understanding the association that employment status has with the development of traditional cardiovascular risk factors [15,16,24].

Our findings support the notion that lower SES may drive the acquisition of these aforementioned cardiovascular risk factors to some extent. Non-employed EmCAB participants were found to possess a disproportionately higher burden of traditional cardiovascular risk factors and significantly greater burden of CVD, such as history of prior MI and heart failure, compared to those who were employed. These findings suggest that non-employed individuals with CAD might be at greater risk of developing further traditional cardiovascular risk factors and additional forms of CVD than employed individuals with CAD. Although individual-level income data was unavailable for our sample, it is likely that our non-employed cohort earned a lower income when compared to our employed cohort. Moreover, if we consider that those who are non-employed also have limited access to higher-level employer-provided financial benefits, such as employer-provided health and dental insurance, retirement plans, and paid leave, this cohort's socioeconomic burden becomes clearer [25]. We argue that the over-representation of adverse socioeconomic factors including, but not limited to, employment status may at least partially drive the disparity in traditional cardiovascular risk factors that we observe in our non-employed cohort.

The relationship between employment status and adverse cardiovascular outcomes in our registry does appear to be significantly modified by race. Our findings suggest that non-employment yields a greater risk of cardiovascular death or non-fatal MI among non-Black participants when compared to Black participants. To our knowledge, however, no prior evidence has replicated or endorsed similar results, and this finding is largely contradictory to the majority of prior studies that suggest that Black individuals are at greater risk of adverse

cardiovascular events, even among lower SES, Black and non-Black cohorts [26–29]. There are several potential explanations that may be underlying this finding. We identified a greater burden of traditional cardiovascular risk factors, prevalent CVD, and cardiovascular mortality in Black participants compared to non-Black participants in our sample (Table S1). Sensitivity analyses also importantly revealed a significantly higher adverse cardiovascular outcome risk in employed Black participants when compared to employed non-Black participants in competing-risk regression modeling, even after full adjustment (Table S2). As such, the protective effect of employment does not appear to be as robust in employed Black individuals with CAD when compared to employed non-Black individuals with CAD. This finding may have obscured the additional impact of non-employment in Black participants of our registry.

The presence of effect modification by sex was also examined and not observed (Fig. S1). Although our findings demonstrated a significantly higher percentage of female participants in the non-employed cohort, a significant statistical interaction was not present, suggesting that individuals in our cohort share a similar risk of adverse outcome occurrence attributable to employment status regardless of sex.

4.1. Strengths

Our study has several strengths. This study is, to our knowledge, the first to evaluate the association between employment status and cardiovascular outcomes among patients with CAD. Our large study sample with extended follow-up and high event rates offered ample statistical power to adequately assess the effects of non-employment. Additionally, our sample's representation of women (35 %, Table 1) and participants of self-identified Black race (27 %, Table 1) allowed for focused sensitivity analyses regarding these important demographic characteristics. Individual-level EAL data furthermore allowed for granular consideration of this essential metric of SES in our analyses and allowed for a targeted approach to examine for effect modification by EAL.

4.2. Limitations

This study is limited by its observational design, which precludes causal inferences and may introduce bias. EmCAB recruited from three Emory-affiliated hospitals all located in an urban, high-risk Southeastern US patient population, which could limit the overall generalizability of our findings. We performed a complete case analysis, which may introduce selection bias. However, our assessment supported a MAR mechanism, reducing the likelihood of substantial bias from missing data. Given the heterogeneity of responses to our employment status variable, we resolved to exclude all participants from the study who selected "retired" for their employment status, which limits the translation of these findings to retired individuals. Additionally, participants' employment status was assessed only at the time of recruitment into EmCAB and not followed longitudinally. Consequently, transitions between employment and non-employment, as well as the duration of non-employment, were not captured and may have introduced exposure misclassification. In a minority of cases, cause of death was based on family member report when medical records were unavailable, which could have introduced potential misclassification bias. Granular angiographic data, such as the number and severity of diseased vessels, were not consistently available and thus not included in adjusted models. Lastly, as participant-level income and mental health data were not routinely collected, we were unable to examine the direct effect of financial insecurity or psychological health on this relationship. Although we conducted extensive sensitivity analyses and attempted to adjust for a variety of appropriate confounders, the possibility for residual confounding remains, given the observational nature of the study.

5. Conclusions

The present study demonstrates that among non-retired patients with CAD, non-employment was independently associated with adverse cardiovascular outcomes and all-cause mortality when compared to employed participants. Overall, these findings provide important context regarding the impact of social determinants of health in populations with CVD. Routine screening for employment might serve as an important means of identifying those at higher risk and trigger targeted allocation of resources and more frequent surveillance in an effort to improve outcomes in this inherently vulnerable population. Notably, individuals with disability-related non-employment represented the highest-risk subgroup in our study, highlighting a need for focused clinical attention within this population. Future studies should aim to explore mediators of this relationship, such as psychological health, in order to evaluate for potential causative links between non-employment and adverse cardiovascular outcomes in patients with CAD.

Ethical review statement

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Ethical approval was obtained from the Emory University Institutional Review Board. Informed consent was obtained from all participants at the time of enrollment.

Author agreement

We confirm that all authors have seen and approved the final version of the manuscript titled "Employment Status as a Predictor of Adverse Outcomes in Patients with Coronary Artery Disease" being submitted for publication in the American Journal of Preventive Cardiology. We warrant that the manuscript is our original work and that, to the best of our knowledge, it has not been published previously nor is it under consideration for publication elsewhere.

All co-authors have contributed significantly to the work and have agreed to be listed as authors. We further confirm that we have disclosed any potential conflicts of interest and have complied with ethical guidelines for the conduct of research.

CRediT authorship contribution statement

Travis M. Wilson: Writing – review & editing, Writing – original draft. **Adithya K. Yadalam:** Writing – review & editing, Writing – original draft, Methodology, Funding acquisition, Formal analysis, Conceptualization. **Shaimaa Sakr:** Data curation. **Matthew E. Gold:** Funding acquisition. **Vardhmaan Jain:** Funding acquisition. **Alexander C. Razavi:** Funding acquisition. **Nishant Vatsa:** Funding acquisition. **Daniel A. Gold:** Funding acquisition. **Yi-An Ko:** Data curation. **Yunyun Chen:** Data curation. **Chang Liu:** Data curation. **Nisreen Haroun:** Resources. **Muhammad Owais:** Resources. **Ishan Nadkarni:** Resources. **Ozair Khawaja:** Resources. **Hassan Allaqaband:** Resources. **Laurence S. Sperling:** Supervision. **ArshedA. Quyyumi:** Writing – review & editing, Supervision, Methodology, Investigation, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Disclosures

The authors report no relevant disclosures.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ajpc.2025.100997.

Data availability

The data that support the findings detailed in this study are available upon reasonable request to the co-corresponding authors.

References

[1] Tsao CW, Aday AW, Almarzooq ZI, Anderson CAM, Arora P, Avery CL, Baker-Smith CM, Beaton AZ, Boehme AK, Buxton AE, et al. Heart Disease and Stroke statistics—2023 update: a report from the American Heart Association. Circulation. 2023;147. https://doi.org/10.1161/CIR.0000000000001123.

[2] Centers for Disease Control and Prevention and National Center for Health Statistics. Multiple cause of death, CDC WONDER online database. Accessed August 1, 2023. Available at https://wonder-cdc-gov/mcd-icd10.html.

[3] Schultz WM, Kelli HM, Lisko JC, Varghese T, Shen J, Sandesara P, Quyyumi AA, Taylor HA, Gulati M, Harold JG, et al. Socioeconomic status and cardiovascular outcomes: challenges and interventions. Circulation 2018;137:2166–78.

[4] Stringhini S, Carmeli C, Jokela M, Avendaño M, Muennig P, Guida F, Ricceri F, d'Errico A, Barros H, Bochud M, et al. Socioeconomic status and the 25 × 25 risk factors as determinants of premature mortality: a multicohort study and meta-analysis of 1·7 million men and women. The Lancet 2017;389:1229–37.

[5] Mosquera PA, San Sebastian M, Waenerlund A-K, Ivarsson A, Weinehall L, Gustafsson PE. Income-related inequalities in cardiovascular disease from mid-life to old age in a Northern Swedish cohort: a decomposition analysis. Soc Sci Med 2016;149:135–44.

[6] Méjean C, Droomers M, Van Der Schouw YT, Sluijs I, Czernichow S, Grobbee DE, Bueno-de-Mesquita HB, Beulens JWJ. The contribution of diet and lifestyle to socioeconomic inequalities in cardiovascular morbidity and mortality. Int J Cardiol 2013;168:5190–5.

[7] Dupre ME, George LK, Liu G, Peterson ED. The cumulative effect of unemployment on risks for acute myocardial infarction. Arch Intern Med 2012;172:1731.

[8] Meneton P, Kesse-Guyot E, Méjean C, Fezeu L, Galan P, Hercberg S, Ménard J. Unemployment is associated with high cardiovascular event rate and increased all-cause mortality in middle-aged socially privileged individuals. Int Arch Occup Environ Health 2015;88:707–16.

[9] Ko Y-A, Hayek S, Sandesara P, Samman Tahhan A, Quyyumi A. Cohort profile: the Emory Cardiovascular Biobank (EmCAB). BMJ Open 2017;7:e018753.

[10] Levey AS, Stevens LA, Schmid CH, Zhang Y (Lucy), Castro AF, Feldman HI, Kusek JW, Eggers P, Van Lente F, Greene T, et al. A new equation to estimate glomerular filtration rate. Ann Intern Med 2009;150:604.

[11] Patel RS, Ghasemzadeh N, Eapen DJ, Sher S, Arshad S, Ko Y, Veledar E, Samady H, Zafari AM, Sperling L, et al. Novel biomarker of oxidative stress is associated with risk of death in patients with coronary artery disease. Circulation 2016;133:361–9.

[12] Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, Thygesen K, Alpert JS, White HD, Jaffe AS, et al. Third Universal definition of myocardial infarction. J Am Coll Cardiol 2012;60:1581–98.

[13] Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, White HD. The Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth Universal Definition of Myocardial Infarction (2018) Circulation 2018;138. https://doi.org/10.1161/CIR.0000000000000617.

[14] Fine JP, Gray RJ. A proportional hazards model for the subdistribution of a competing risk. J Am Stat Assoc 1999;94:496–509.

[15] Roelfs DJ, Shor E, Davidson KW, Schwartz JE. Losing life and livelihood: a systematic review and meta-analysis of unemployment and all-cause mortality. Soc Sci Med 2011;72:840–54.

[16] Rico MS, Plessz M, Airagnes G, Ribet C, Hoertel N, Goldberg M, Zins M, Meneton P. Cardiovascular burden and unemployment: a retrospective study in a large population-based French cohort. PLoS One 2023;18:e0288747.

[17] Picchio M, Ubaldi M. Unemployment and health: a meta-analysis. J Econ Surv 2024. joes.12588.

[18] d'Errico A, Piccinelli C, Sebastiani G, Ricceri F, Sciannameo V, Demaria M, Di Filippo P, Costa G. Unemployment and mortality in a large Italian cohort. J Public Health (Bangkok) 2021;43:361–9.

[19] Mattiasson I, Lindgärde F, Nilsson JA, Theorell T. Threat of unemployment and cardiovascular risk factors: longitudinal study of quality of sleep and serum cholesterol concentrations in men threatened with redundancy. BMJ 1990;301:461–6.

[20] Lynch JW, Kaplan GA, Cohen RD, Tuomilehto J, Salonen JT. Do cardiovascular risk factors explain the relation between socioeconomic status, risk of all-cause mortality, cardiovascular mortality, and acute myocardial infarction? Am J Epidemiol 1996;144:934–42.

[21] Kozakiewicz K, Podolecka E, Kwaśniewska M, Drygas W, Pająk A, Tendera M. Association between socioeconomic status and cardiovascular risk. Polish Heart Journal (Kardiologia Polska) 2016;74:179–84.

[22] Clark AM, DesMeules M, Luo W, Duncan AS, Wielgosz A. Socioeconomic status and cardiovascular disease: risks and implications for care. Nat Rev Cardiol 2009;6:712–22.

[23] Albert MA, Glynn RJ, Buring J, Ridker PM. Impact of traditional and novel risk factors on the relationship between socioeconomic status and incident cardiovascular events. Circulation 2006;114:2619–26.

[24] Nie J, Wang J, Aune D, Huang W, Xiao D, Wang Y, Chen X. Association between employment status and risk of all-cause and cause-specific mortality: a population-based prospective cohort study. J Epidemiol Community Health 2020;74:428–36.

[25] Schudde L, Bernell K. Educational attainment and nonwage labor market returns in the United States. AERA Open 2019;5:233285841987405.

[26] Garcia M, Almuwaqqat Z, Moazzami K, Young A, Lima BB, Sullivan S, Kaseer B, Lewis TT, Hammadah M, Levantsevych O, et al. Racial disparities in adverse cardiovascular outcomes after a myocardial infarction in young or middle-aged patients. JAHA 2021;10:e020828.

[27] Graham GN, Jones PG, Chan PS, Arnold SV, Krumholz HM, Spertus JA. Racial disparities in patient characteristics and survival after acute myocardial infarction. JAMA Netw Open 2018;1:e184240.

[28] Spertus JA, Jones PG, Masoudi FA, Rumsfeld JS, Krumholz HM. Factors associated with racial differences in myocardial infarction outcomes. Ann Intern Med 2009;150:314–24.

[29] Bucholz EM, Ma S, Normand S-LT, Race KrumholzHM. Socioeconomic status, and life expectancy after acute myocardial infarction. Circulation 2015;132:1338–46.