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Elevated Cardiac Biomarkers, Erectile Dysfunction, and Mortality in U.S. Men:

NHANES 2001 to 2004

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Abstract

BACKGROUND—The prevalence of elevated cardiac biomarkers and their link to mortality in men with erectile dysfunction in the U.S. population are unknown.

OBJECTIVES—The purpose of this study was to evaluate the prevalence of elevations in N-terminal prohormone B-type natriuretic peptide, high sensitivity troponin (hs-troponin) T, and 3 hs-troponin I assays and their associations with mortality in U.S. men with and without erectile dysfunction.

METHODS—We conducted cross-sectional analyses using logistic regression to examine associations of elevated cardiac biomarkers (>90th percentile) with erectile dysfunction in 2,971 male participants aged 20 years or older in the National Health and Nutrition Examination Survey (NHANES) from 2001 to 2004. We conducted prospective analyses using Cox regression to examine the mortality implications of elevations in cardiac biomarkers in the setting of erectile dysfunction.

RESULTS—Elevations in hs-troponin T and the 3 hs-troponin I assays were associated with erectile dysfunction, with the strongest association for hs-troponin T (adjusted OR: 2.01; 95% CI: 1.22–3.30). Elevated N-terminal prohormone B-type natriuretic peptide was not significantly associated with erectile dysfunction (OR: 1.22; 95% CI: 0.74–2.03). There were 673 deaths during a median of 16 years of follow-up. Men with erectile dysfunction were at an elevated risk of

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APPENDIX For supplemental tables and a figure, please see the online version of this paper.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

death (adjusted HR: 1.23; 95% CI: 1.04–1.46). Those men with elevated cardiac biomarkers in the setting of erectile dysfunction were at highest risk of all-cause and cardiovascular mortality (adjusted HRs ranging from ~1.5 to 2.4).

CONCLUSIONS—In this national study, the association of erectile dysfunction with elevated hs-troponin and excess mortality risk suggests that men with erectile dysfunction should be evaluated and targeted for intensive cardiovascular risk management.

Keywords

cardiac biomarkers; erectile dysfunction; high sensitivity troponin assays; NHANES; NTproBNP

Erectile dysfunction affects more than 20 million adult men in the U.S.[1] and can have an underlying neurological, endocrinological, or psychological etiology.[2] Vascular disease can also cause erectile dysfunction via abnormalities in penile blood supply.[3] Cardiovascular disease and its risk factors are strongly linked to erectile dysfunction,[4] and men with a history of erectile dysfunction are at elevated risk for all-cause and cardiovascular mortality.[5–7] Despite the strong link to cardiovascular disease events, there are few studies that have examined the association of subclinical cardiovascular disease with erectile dysfunction.[8]

There is growing interest in the use of the cardiac biomarkers N-terminal prohormone B-type natriuretic peptide (NTproBNP) and high sensitivity cardiac troponin (hs-troponin) to monitor risk in the general adult population and to guide treatment and management of cardiovascular risk in high-risk groups.[9] Elevations in hs-troponin in the general adult population reflect chronic subclinical myocardial damage.[10,11] NTproBNP is a biomarker of cardiac wall stress and is routinely used in the evaluation of congestive heart failure.[12] NTproBNP and hs-troponin are potent markers of mortality and future cardiovascular risk, especially heart failure.[13–16] However, no prior studies have examined a possible link between these cardiac biomarkers and erectile dysfunction in the general male population.

Because erectile dysfunction can have an underlying vascular basis, we hypothesized that hstroponin and NTproBNP would be associated with prevalent erectile dysfunction in the general population of U.S. men. To address this question, we conducted a cross-sectional study of adult men who participated in the National Health and Nutrition Examination Survey (NHANES) from 2001 to 2004. We examined the prevalence of subclinical cardiovascular disease defined by elevations in high-sensitivity cardiac troponin and/or NTproBNP in men with erectile dysfunction. We also examined the risk of mortality associated with having elevated cardiac biomarkers in the setting of erectile dysfunction.

METHODS

STUDY POPULATION.

Erectile dysfunction was assessed in all male participants aged 20 years or older in the 2001 to 2004 NHANES survey cycle. We measured cardiac biomarkers in all eligible participants with stored blood in this survey cycle. Our study population included 2,971 men without a history of cardiovascular disease (self-reported coronary heart disease, angina, heart attack,

stroke, or heart failure), who were not missing any covariates of interest, and who had valid assessments of erectile dysfunction and cardiac biomarkers.

MEASUREMENT OF CARDIAC BIOMARKERS.

We conducted measurements of NTproBNP, hs-troponin T, and hs-troponin I (3 different assays) as part of a stored serum study utilizing NHANES biospecimens.[17] Hs-troponin T and NTproBNP (Roche Diagnostics) were measured on a Cobas e601 analyzer using Elecsys reagents. Hs-troponin I (Abbott Laboratories) was measured using the ARCHITECT i2000SR, hs-troponin I (Siemens Healthcare Diagnostics) was measured using the Centaur XPT, and hs-troponin I (Ortho Clinical Diagnostics) was measured using the Vitros 3600.

All participants provided written informed consent. All study protocols including this stored serum study were approved by the ethics review board of the National Center for Health Statistics.

ASSESSMENT ERECTILE DYSFUNCTION.

Erectile dysfunction was only assessed during the 2001 to 2004 NHANES survey cycles. In these survey years, all men aged 20 or older were asked to self-report erectile dysfunction during a computer-assisted self-interview system in response to the prompt: “How would you describe your ability to get and keep an erection adequate for satisfactory intercourse?” Possible responses included “always or almost always able; usually able; sometimes able; or never able.” In this study, we defined erectile dysfunction as ‘never able to achieve an erection’ to increase the specificity of classification and allow us to focus on organic causes of erectile dysfunction (the other major category of erectile dysfunction, psychologic, is characterized by intermittent ability to achieve erections such as nocturnal erections).[18]

ASSESSMENT OF COVARIATES.

Age, sex, education, marital status, race, and ethnicity were self-reported by participants. All interview questions were conducted using a computer-assisted self-interview system conducted by trained personnel. In this study, race and ethnicity were categorized according to responses to survey questions on race and Hispanic origin. Body mass index (kg/m^2) was calculated based on measured height and weight. Hypertension was defined as mean blood pressure ≥140/90 mm Hg or current use of blood pressure-lowering medications, and hypercholesterolemia was defined as total cholesterol ≥240 mg/dL or current use of cholesterol-lowering medications. Diabetes was defined as a self-reported physician diagnosis, current use of glucose-lowering medications, or HbA1c ≥6.5%.

MORTALITY.

Deaths are captured in NHANES via probabilistic linkage to the National Death Index (end of follow-up, December 31, 2019). Cardiovascular mortality was defined as heart disease or cerebro-vascular disease as the underlying cause of death.[19]

STATISTICAL ANALYSES.

We compared the characteristics of U.S. adult men with and without erectile dysfunction. We evaluated the crude and age-adjusted prevalence of elevated cardiac biomarkers among

men with and without erectile dysfunction. Age-adjustment was conducted using logistic regression (direct approach). To put the NTproBNP and different hs-troponin assays on equal footing, we defined 'elevated' NTproBNP or hs-troponin as a value above the weighted 90th percentile calculated in the study population without self-reported cardiovascular disease. We conducted secondary analyses using typical clinical cut-points for elevated NT-proBNP of ≥125 pg/mL for those aged <75 years and ≥450 pg/mL for those ≥75 years.[20,21] Manufacturer-defined 99th percentiles or recommended 'risk' thresholds for hs-troponin: hs-troponin T (Roche) ≥14 ng/L; hs-troponin I (Abbott) ≥6 ng/L; hs-troponin I (Siemens) ≥47.34 ng/L; and hs-troponin I (Ortho) ≥11 ng/L.[22–25]

We evaluated the independent associations of categories of elevated cardiac biomarkers with prevalent erectile dysfunction using logistic regression. We also modeled each of the cardiac biomarkers as restricted cubic splines with knots located at the 5th, 35th, 65th, and 95th percentiles. We used Cox regression models to generate HRs for the association of erectile dysfunction with all-cause and cardiovascular mortality overall and in the presence and absence of elevated cardiac biomarkers. Follow-up time was calculated from baseline until death or administrative censoring (December 31, 2019). In analyses of cardiovascular mortality, deaths due to other causes were censored.

Model 1 included an adjustment for age (years). Model 2 included age (years), race (non-Hispanic White, non-Hispanic Black, Mexican American, or other/other Hispanic), education (college graduate, some college, or less than a high school education), marital status (married or not married or cohabitating), smoking (never former, current), body mass index (<25, 25-<30, or ≥30 kg/m^2), hypertension (measured blood pressure 140/90 mm Hg or current use of blood pressure-lowering medications), hyper-cholesterolemia (total cholesterol ≥240 mg/dL or current use of cholesterol-lowering medications), and diabetes (current use of glucose-lowering medications or hemoglobin A1c ≥6.5%).

RESULTS

The prevalence of severe erectile dysfunction (never able to achieve an erection) was 4.5%, corresponding to 4.4 million men aged 20 years or older in the U.S. in 2001 to 2004, but with a much higher prevalence among men aged 65 or older (26.2%). The mean age among men with severe erectile dysfunction was 65 years, 23 years older than men without erectile dysfunction (Supplemental Table 1). The crude prevalence of elevated NTproBNP or hs-troponin (exceeding the 90th percentile in the general adult male population aged 20 or older) ranged from 25.3% to 47.9% among men with erectile dysfunction, depending on the assay (Supplemental Table 1). Given the substantial differences in age comparing men with and without erectile dysfunction, we included age adjustment in all subsequent analyses.

When comparing age-adjusted prevalence estimates, men who reported erectile dysfunction were less likely to be White and more likely to be Mexican American, to have lower education levels, and to have a substantially higher burden of cardiovascular risk factors such as hypertension, high cholesterol, or diabetes (Table 1). The age-adjusted prevalence of elevated NTproBNP was similar in the 2 groups, whereas elevated hs-troponin was more common in men with erectile dysfunction (Figure 1).

In age- and multivariable-adjusted models, elevations in hs-troponin T and the 3 hs-troponin I assays were associated with erectile dysfunction (Table 2), although the associations for elevated hs-troponin I (Abbott) and hs-troponin I (Siemens) were not statistically significant after adjustment for other risk factors (Model 2) (Central Illustration). Similar results were observed using clinical cut-points (Supplemental Table 2). Elevated NTproBNP was not associated with erectile dysfunction (Table 2, Supplemental Table 2). Hs-troponin T was more strongly associated with erectile dysfunction as compared to the hs-troponin I assays. In analyses with the cardiac biomarkers modeled continuously using restricted cubic splines, the stronger association with hs-troponin is particularly evident (Supplemental Figure 1).

There were 673 deaths (191 from cardiovascular causes) in this population during a median of 16 years of follow-up. Men reporting erectile dysfunction were at a significantly elevated risk of all-cause mortality (HR: 1.23; 95% CI: 1.04–1.46) and a nonsignificantly elevated risk of cardiovascular mortality (HR: 1.35; 95% CI: 0.92–1.10) after accounting for other risk factors (Table 3). Men experiencing erectile dysfunction and elevations in any of the cardiac biomarkers were at the highest risk of subsequent mortality, with a consistently >2-fold increased risk of cardiovascular death (Table 3, Supplemental Table 3).

DISCUSSION

We found that subclinical myocardial damage, as assessed by elevated concentrations of hs-troponin T or I, was associated with erectile dysfunction, whereas NTproBNP was not. In this general population of men aged 20 years or older without a history of cardiovascular disease, the crude prevalence of elevated cardiac biomarkers was extremely high (~25%-50%), primarily due to the older age of these men. However, even after adjustment for age and other risk factors, the prevalence of elevated hs-troponin was significantly higher in men with erectile dysfunction compared to those without. The high prevalence of subclinical vascular disease even after accounting for age and other cardiovascular risk factors suggests that men with erectile dysfunction should be evaluated and targeted for intensive cardiovascular risk management.

The specific association of hs-troponin with erectile dysfunction suggests a common etiology. Hs-troponin is released into the blood from cardiomyocytes in the setting of myocardial disease, which can occur due to structural heart disease (eg, from hypertension or diabetes) or ischemic heart disease (eg, from coronary atherosclerosis). These same underlying causes of myocardial disease are associated with erectile dysfunction.[26,27] We observed stronger associations of hs-troponin T with erectile dysfunction as compared to the hs-troponin I assays. Hs-troponin T and hs-troponin I are only modestly correlated in the general population,[28] and prior studies have also reported distinct associations of hs-troponin T vs hs-troponin I with different vascular outcomes.[29] The more robust associations of hs-troponin T with erectile dysfunction are consistent with the stronger associations of hs-troponin T with skeletal muscle disease and frailty.[30,31] Unlike hs-troponin, NTproBNP is a hemodynamic biomarker that is secreted by cardiomyocytes in response to myocardial wall stretch and may be less relevant to the disease processes that underlie erectile dysfunction.[32]

Erectile dysfunction was an independent risk factor for mortality in this general U.S. adult population.[5,6] We also found that those men who had erectile dysfunction and elevated cardiac biomarkers were at a markedly higher risk of cardiovascular death. These results are consistent with a large body of literature demonstrating that NTproBNP and hs-troponin provide useful prognostic information in the general population and can identify those persons at highest risk for all-cause and cardiovascular mortality. Our results suggest that clinicians should be aware of erectile dysfunction as a marker of mortality risk in adult men and that cardiac biomarkers can identify a subset at highest risk.

STUDY LIMITATIONS.

Limitations of this study included the relatively small sample size as erectile dysfunction was only assessed during the 2001 to 2004 survey cycles of NHANES, possible under-reporting of erectile dysfunction, which could have resulted in some misclassification, and the cross-sectional nature of NHANES, which precludes drawing conclusions about the directionality of the observed associations with cardiac biomarkers.

This study benefited from the rigorous design and methods employed in NHANES. Strengths include the nationally representative sample of adult men in the U.S., the assessment of erectile dysfunction using standard questions via a computer-assisted self-interview system, and the availability of NTproBNP and hs-troponin assessed using 4 different assays. All measurements in this study were conducted by trained staff according to standardized protocols.

In conclusion, we found a high prevalence of subclinical cardiac dysfunction, as assessed by cardiac biomarkers, in men with erectile dysfunction. The associations of hs-troponin with erectile dysfunction but not NTproBNP are consistent with the involvement of an underlying vascular etiology and suggest that interventions to prevent microvascular disease (eg, blood pressure control, physical activity, diabetes prevention) may prevent erectile dysfunction.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Reagents for the NTproBNP and hs-troponin assays were donated by the manufacturers.

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ABBREVIATIONS AND ACRONYMS

hs-troponin I	high sensitivity troponin I
hs-troponin T	high sensitivity troponin T
NTproBNP	N-terminal prohormone B-type natriuretic peptide

REFERENCES

1. Selvin E, Burnett AL, Platz EA. Prevalence and risk factors for erectile dysfunction in the US. Am J Med. 2007;120(2):151–157. [PubMed: 17275456]
2. Yafi FA, Jenkins L, Albersen M, et al. Erectile dysfunction. Nat Rev Dis Primers. 2016;2:16003. [PubMed: 27188339]
3. Billups KL. Erectile dysfunction as a marker for vascular disease. Curr Urol Rep. 2005;6(6):439–444. [PubMed: 16238918]
4. Mostafaei H, Mori K, Hajebrahimi S, Abufaraj M, Karakiewicz PI, Shariat SF. Association of erectile dysfunction and cardiovascular disease: an umbrella review of systematic reviews and meta-analyses. BJU Int. 2021;128(1):3–11. [PubMed: 33260254]
5. Araujo AB, Travison TG, Ganz P, et al. Erectile dysfunction and mortality. J Sex Med. 2009;6(9):2445–2454. [PubMed: 19538544]
6. Loprinzi PD, Nooe A. Erectile dysfunction and mortality in a national prospective cohort study. J Sex Med. 2015;12(11):2130–2133. [PubMed: 26559652]
7. Dong JY, Zhang YH, Qin LQ. Erectile dysfunction and risk of cardiovascular disease: meta-analysis of prospective cohort studies. J Am Coll Cardiol. 2011;58(13):1378–1385. [PubMed: 21920268]
8. Omland T, Randby A, Hrubos-Strøm H, Røsjø H, Einvik G. Relation of erectile dysfunction to sub-clinical myocardial injury. Am J Cardiol. 2016;118(12):1821–1825. [PubMed: 27780552]
9. Pop-Busui R, Januzzi JL, Bruemmer D, et al. Heart failure: an underappreciated complication of diabetes. A consensus report of the American Diabetes Association. Diabetes Care. 2022;45(7):1670–1690. [PubMed: 35796765]
10. Sabatine MS, Morrow DA, de Lemos JA, Jarolim P, Braunwald E. Detection of acute changes in circulating troponin in the setting of transient stress test-induced myocardial ischaemia using an ultrasensitive assay: results from TIMI 35. Eur Heart J. 2009;30(2):162–169. [PubMed: 18997177]
11. Canty JM Jr. Myocardial injury, troponin release, and cardiomyocyte death in brief ischemia, failure, and ventricular remodeling. Am J Physiol Heart Circ Physiol. 2022;323(1):H1–H15. [PubMed: 35559722]
12. Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2013;62(16):e147–e239. [PubMed: 23747642]
13. Di Angelantonio E, Chowdhury R, Sarwar N, et al. B-type natriuretic peptides and cardiovascular risk: systematic review and meta-analysis of 40 prospective studies. Circulation. 2009;120(22):2177–2187. [PubMed: 19917883]
14. Willeit P, Welsh P, Evans JDW, et al. High-sensitivity cardiac troponin concentration and risk of first-ever cardiovascular outcomes in 154,052 participants. J Am Coll Cardiol. 2017;70(5):558–568. [PubMed: 28750699]
15. Bonora E, Kiechl S, Willeit J, et al. Plasma glucose within the normal range is not associated with carotid atherosclerosis: prospective results in subjects with normal glucose tolerance from the Bruneck study. Diabetes Care. 1999;22(8):1339–1346. [PubMed: 10480780]
16. Jia X, Sun W, Hoogeveen RC, et al. High-sensitivity troponin I and incident coronary events, stroke, heart failure hospitalization, and mortality in the ARIC study. Circulation. 2019;139(23):2642–2653. [PubMed: 31030544]

17. National Health and Nutrition Examination Survey 1999–2004 data documentation, high-sensitivity cardiac troponins (Surplus). Accessed October 26, 2022. https://wwwn.cdc.gov/Nchs/Nhanes/1999-2000/SSTROP_A.htm
18. Segraves KA, Segraves RT, Schoenberg HW. Use of sexual history to differentiate organic from psychogenic impotence. Arch Sex Behav. 1987;16(2):125–137. [PubMed: 3592960]
19. National Center for Health Statistics. The LINKAGE of National Center for Health Statistics Survey Data to the National Death Index—2015 Linked Mortality File (LMF): Methodology Overview and Analytic Considerations. National Center for Health Statistics; 2019.
20. Maisel AS, Krishnaswamy P, Nowak RM, et al. Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. N Engl J Med. 2002;347(3):161–167. [PubMed: 12124404]
21. Januzzi JL Jr, Chen-Tournoux AA, Christenson RH, et al. N-terminal pro-B-type natriuretic peptide in the emergency department: the ICON-RELOADED study. J Am Coll Cardiol. 2018;71(11):1191–1200. [PubMed: 29544601]
22. High-sensitivity cardiac troponin I and T assay analytical characteristics designated by manufacturer 2019. Accessed September 9, 2022. https://www.ifcc.org/media/477656/high-sensitivity-cardiac-troponini-and-t-assay-analytical-characteristics-designated-by-manufacturer-v012019.pdf
23. N-terminal pro-brain natriuretic peptide - 510(k) SUBSTANTIAL EQUIVALENCE DETERMINATION. 2008. Accessed August 3, 2022. https://www.accessdata.fda.gov/cdrh_docs/reviews/K063662.pdf
24. VITROS high sensitivity troponin I assay. 2022. Accessed May 30, 2023. https://www.orthoclinicaldiagnostics.com/assets/pdf/EMEA/Brochure_CLM_hsTroponin_PR-04701%20(2).pdf
25. Abbott high sensitivity troponin I. 2018. Accessed October 26, 2022. https://www.corelaboratory.abbott/int/en/offerings/assays/risk-stratification
26. McEvoy JW, Chen Y, Nambi V, et al. High-sensitivity cardiac troponin T and risk of hyper-tension. Circulation. 2015;132(9):825–833. [PubMed: 26152706]
27. Selvin E, Lazo M, Chen Y, et al. Diabetes mellitus, prediabetes, and incidence of subclinical myocardial damage. Circulation. 2014;130(16): 1374–1382. [PubMed: 25149362]
28. Welsh P, Preiss D, Shah ASV, et al. Comparison between high-sensitivity cardiac troponin T and cardiac troponin I in a large general population cohort. Clin Chem. 2018;64(11):1607–1616. [PubMed: 30126950]
29. Welsh P, Preiss D, Hayward C, et al. Cardiac troponin T and troponin I in the general population. Circulation. 2019;139(24):2754–2764. [PubMed: 31014085]
30. Schmid J, Liesinger L, Birner-Gruenberger R, et al. Elevated cardiac troponin T in patients with skeletal myopathies. J Am Coll Cardiol. 2018;71(14):1540–1549. [PubMed: 29622161]
31. Strandberg LS, Roos A, Holzmann MJ. Stable high-sensitivity cardiac troponin T levels and the association with frailty and prognosis in patients with chest pain. Am J Med Open. 2021;1–6:100001.
32. Laufer EM, Mingels AM, Winkens MH, et al. The extent of coronary atherosclerosis is associated with increasing circulating levels of high sensitive cardiac troponin T. Arterioscler Thromb Vasc Biol. 2010;30(6):1269–1275. [PubMed: 20299689]

PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE:

Subclinical cardiac dysfunction is common in men with erectile dysfunction. Men who have erectile dysfunction are at high risk for cardiovascular disease and mortality, even after accounting for other risk factors.

TRANSLATIONAL OUTLOOK:

Men with erectile dysfunction should be evaluated and targeted for intensive cardiovascular risk management.

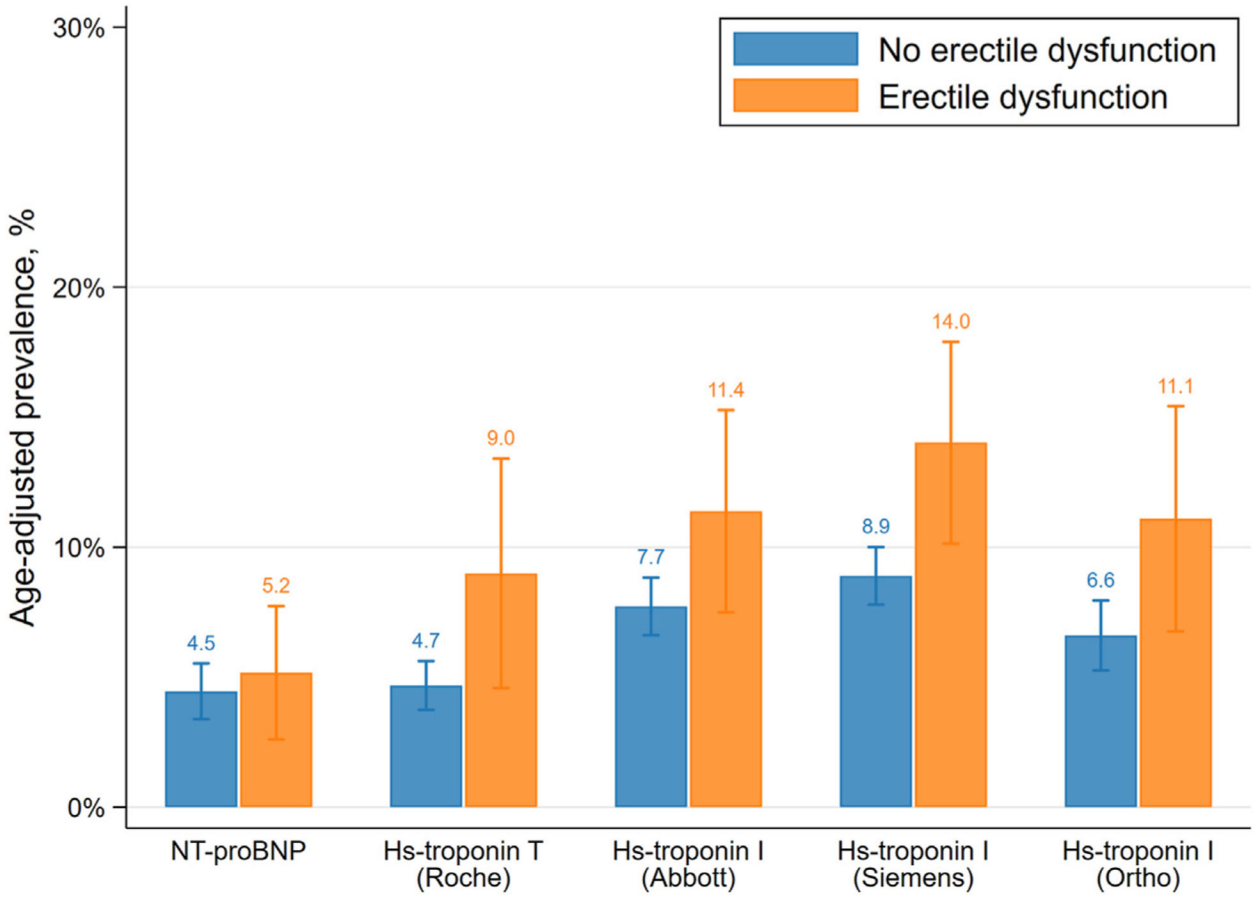
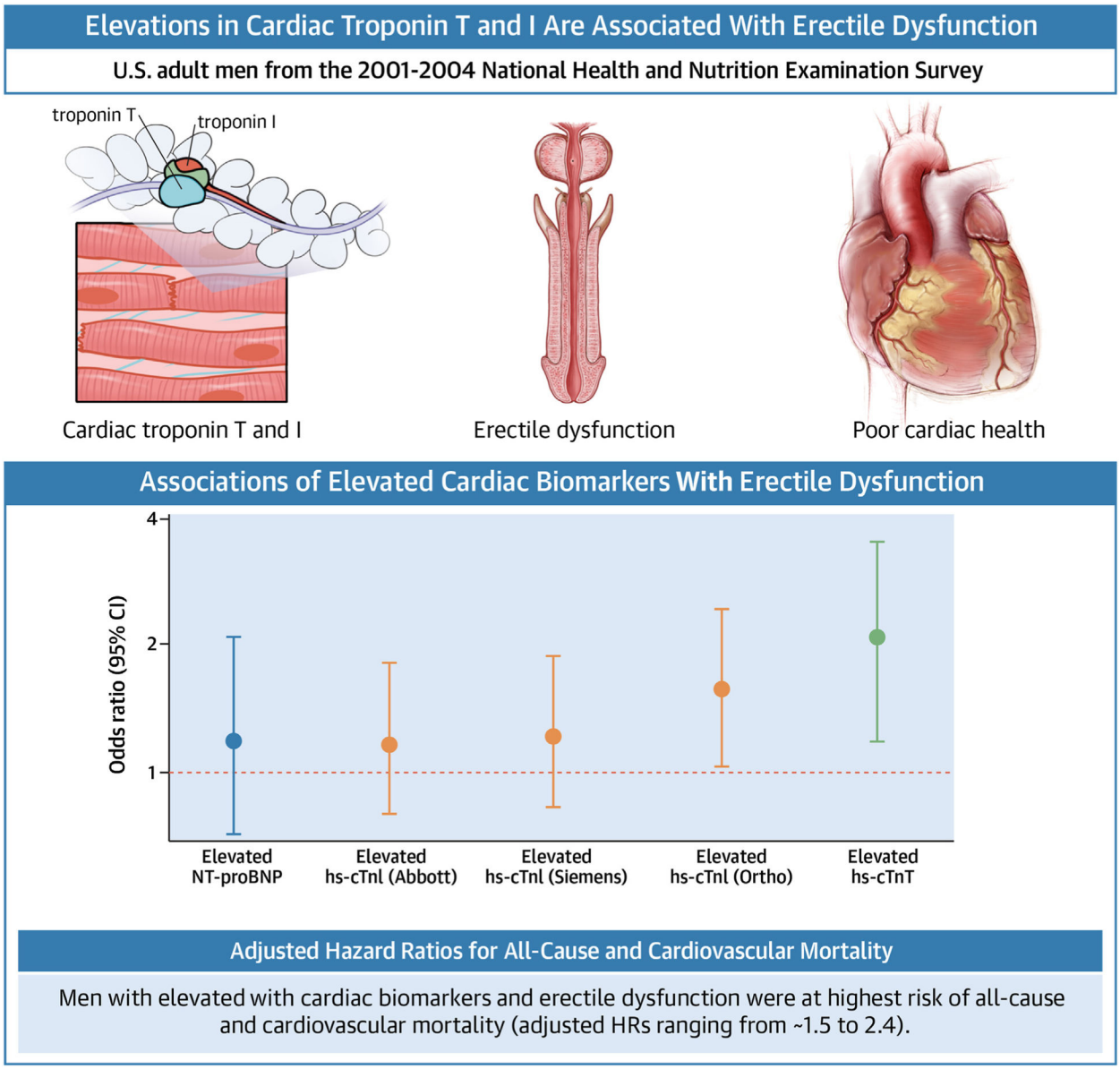


FIGURE 1.

Age-Adjusted Prevalence of Elevations in Cardiac Biomarkers (>90th Percentile) in U.S. Men According to Erectile Dysfunction Status, NHANES 2001 to 2004



CENTRAL ILLUSTRATION.
Cardiac Biomarkers and Erectile Dysfunction

TABLE 1

Age-Adjusted Characteristics of U.S. Men Aged 20 or Older According to ED Status, NHANES 2001 to 2004

	No ED	ED
Unweighted N	2,716	255
Age, y (unadjusted)	42.3 (0.3)	65.3 (1.1)
Race/ethnicity		
Non-Hispanic White	74.9 (2.1)	62.4 (5.2)
Non-Hispanic Black	10.1 (1.1)	8.5 (2.0)
Mexican American	7.0 (1.1)	20.1 (5.1)
Other or other Hispanic	8.0 (1.1)	9.0 (4.0)
Education		
College or higher	27.9 (1.6)	22.3 (3.5)
Some college	30.5 (1.1)	15.3 (1.8)
High school equivalent or less	41.6 (1.4)	62.4 (3.8)
Not married or cohabitating	27.9 (1.4)	39.9 (5.3)
Smoking status		
Never	46.7 (1.6)	48.0 (5.4)
Former	24.9 (1.0)	27.7 (3.8)
Current	28.4 (1.4)	24.3 (4.2)
Body mass index categories, kg/m^2		
<25	28.6 (1.2)	32.7 (3.6)
25-<30	42.0 (1.3)	35.0 (4.0)
≥30	29.4 (1.0)	32.3 (4.4)
Hypertension	21.5 (1.3)	28.6 (4.6)
Hypercholesterolemia	22.7 (1.1)	18.7 (2.9)
Diabetes	6.1 (0.6)	9.6 (1.9)

Estimates are age-adjusted percentage (SE) unless otherwise indicated.

TABLE 2

Odds Ratios (95% CI) for Elevated[a] NT-proBNP or hs-Troponin With Erectile Dysfunction in U.S. Men Aged 20 Years or Older, NHANES 2001 to 2004

	OR (95% CI)	
	Model 1	**Model 2**
NT-proBNP		
Not elevated	1.00 (Reference)	1.00 (Reference)
Elevated	1.15 (0.71–1.86)	1.22 (0.74–2.03)
Hs-troponin T (Roche)		
Not elevated	1.00 (Reference)	1.00 (Reference)
Elevated	2.10 (1.36–3.25)	2.01 (1.22–3.30)
Hs-troponin I (Abbott)		
Not elevated	1.00 (Reference)	1.00 (Reference)
Elevated	1.39 (1.01–1.92)	1.21 (0.86–1.71)
Hs-troponin I (Siemens)		
Not elevated	1.00 (Reference)	1.00 (Reference)
Elevated	1.47 (1.09–1.97)	1.29 (0.91–1.82)
Hs-troponin I (Ortho)		
Not elevated	1.00 (Reference)	1.00 (Reference)
Elevated	1.71 (1.20–2.43)	1.56 (1.04–2.34)

Model 1: adjusted for age. Model 2: adjusted for age, race, education, marital status, smoking, body mass index, hypertension, hypercholesterolemia, and diabetes.

[a]Elevated NT-proBNP and troponin were defined as >90th percentile of each assay: 106.3 pg/mL for NT-proBNP; 12.45 ng/L for hs-troponin T (Roche); 4.80 ng/L for hs-troponin I (Abbott); 8.67 ng/L for hs-troponin I (Siemens); and 2.00 ng/L for hs-troponin I (Ortho).

TABLE 3

Adjusted[a] Hazard Ratios (95% CI) for All-Cause and Cardiovascular Mortality According to ED and Elevated[b] NT-proBNP or hs-Troponin at Baseline, U.S. Men Aged 20 or Older, NHANES 2001 to 2004

	All-Cause Mortality		Cardiovascular Mortality	
	N Deaths/N	HR (95% CI)	N Deaths/N	HR (95% CI)
No ED	508/2,716	1.00 (Reference)	135/2,716	1.00 (Reference)
ED	165/255	1.23 (1.04–1.46)	56/255	1.35 (0.92–1.10)
ED and NT-proBNP				
No ED	508/2,716	1.00 (Reference)	135/2,716	1.00 (Reference)
ED, not elevated	62/131	1.15 (0.88–1.51)	17/131	0.96 (0.53–1.74)
ED, elevated	103/124	1.31 (0.99–1.72)	39/124	1.73 (1.16–2.56)
ED and hs-troponin T (Roche)				
No ED	508/2,716	1.00 (Reference)	135/2,716	1.00 (Reference)
ED, not elevated	46/116	0.82 (0.59–1.12)	11/116	0.55 (0.26–1.13)
ED, elevated	119/139	1.60 (1.29–1.99)	45/139	2.08 (1.34–3.25)
ED and hs-troponin I (Abbott)				
No ED	508/2,716	1.00 (Reference)	135/2,716	1.00 (Reference)
ED, not elevated	91/170	1.10 (0.88–1.38)	27/170	0.90 (0.53–1.52)
ED, elevated	74/85	1.51 (1.08–2.10)	29/85	2.26 (1.45–3.54)
ED and hs-troponin I (Siemens)				
No ED	508/2,716	1.00 (Reference)	135/2,716	1.00 (Reference)
ED, not elevated	104/182	1.10 (0.89–1.36)	31/182	0.99 (0.59–1.64)
ED, elevated	61/73	1.61 (1.23–2.10)	25/73	2.40 (1.57–3.68)
ED and hs-troponin I (Ortho)				
No ED	508/2,716	1.00 (Reference)	135/2,716	1.00 (Reference)
ED, not elevated	67/143	1.04 (0.79–1.37)	15/143	0.78 (0.40–1.49)
ED, elevated	98/112	1.47 (1.16–1.87)	41/112	2.07 (1.35–3.19)

[a]Adjusted for age, race, education, marital status, smoking, body mass index, hypertension, hypercholesterolemia, and diabetes.

[b]Elevated NT-proBNP and troponin were defined as >90th percentile of each assay: 106.3 pg/mL for NT-proBNP; 12.45 ng/L for hs-troponin T (Roche); 4.80 ng/L for hs-troponin I (Abbott); 8.67 ng/L for hs-troponin I (Siemens); and 2.00 ng/L for hs-troponin I (Ortho).