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Prediabetes is an incremental risk factor for adverse cardiac events: A nationwide analysis

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ABSTRACT

Background and aims: Prediabetes is defined as a state of impaired glucose metabolism with hemoglobin A1c (HbA1c) levels that precede those of a diabetic state. There is increasing evidence that suggests that hyperglycemic derangement in prediabetes leads to microvascular and macrovascular complications even before progression to overt diabetes mellitus. We aim to identify the association of prediabetes with acute cardiovascular events.

Methods: We utilized the National inpatient sample 2018-2020 to identify adult hospitalizations with prediabetes after excluding all hospitalizations with diabetes. Demographics and prevalence of other cardiovascular risk factors were compared in hospitalizations with and without prediabetes using the chi-square test for categorical variables and the *t*-test for continuous variables. Multivariate regression analysis was further performed to study the impact of prediabetes on acute coronary syndrome, acute ischemic stroke, intracranial hemorrhage, and acute heart failure.

Results: Hospitalizations with prediabetes had a higher prevalence of cardiovascular risk factors like hypertension, hyperlipidemia, obesity, and tobacco abuse. In addition, the adjusted analysis revealed that hospitalizations with prediabetes were associated with higher odds of developing acute coronary syndrome (OR-2.01; C.I:1.94-2.08; P<0.001), acute ischemic stroke (OR-2.21; 2.11-2.31; p<0.001), and acute heart failure (OR-1.41; C.I.: 1.29-1.55; p<0.001) as compared to hospitalizations without prediabetes.

Conclusions: Our study suggests that prediabetes is associated with a higher odds of major cardiovascular events. Further prospective studies should be conducted to identify prediabetes as an independent causative factor for these events. In addition, screening and lifestyle modifications for prediabetics should be encouraged to improve patient outcomes.

1. Introduction

Prediabetes is defined as a metabolic state in between normoglycemia and diabetes. According to the American Diabetes Association (ADA), prediabetes is described as impaired fasting glucose with fasting plasma glucose of 5.6-6.9 mmol/L, 2h glucose during a 75g oral glucose tolerance test of 7.8-11.0 mmol/L, or HbA1c in the range of 39-47 mmol/mol (5.7-6.4%) [1]. A study by Selvin et al. revealed that in a population of nondiabetic adults, glycated hemoglobin was strongly associated with increased risks of cardiovascular diseases and all-cause mortality as compared to fasting glucose [2]. According to the Centers for Disease Control and Prevention (CDC), more than 1 in 3 individuals have prediabetes, i.e., 96 million adult Americans, and approximately 80% of these individuals are unaware of their glycemic status [3].

Abbreviations: CDC, Centers for Disease Control and Prevention; HBA1c, hemoglobin A1c; ADA, American Diabetes Association; T2DM, Type 2 diabetes mellitus; CVS, cardiovascular diseases; ACS, Acute Coronary Syndrome; AIS, Acute ischemic stroke; NIS, National inpatient sample; OR, odds ratio; C.I., Confidence-intervals.

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Prediabetes may eventually progress to overt Type 2 diabetes mellitus (T2DM), a condition proven to pose significant cardiovascular risks. However, the concept of cardiovascular pathology already in development prior to the onset of diabetes is gaining traction [4]. Research has suggested that even a high normal glucose tolerance at 1 h during an oral glucose tolerance test (OGTT) may have a cardiometabolic profile comparable to those individuals with impaired glucose tolerance. Specifically, these individuals have demonstrated atherogenic lipid patterns and early markers of vascular damage [5]. Prediabetes might be a stand-alone parameter for increased risk of cardiovascular complications. It is implicated in the pathogenesis of cardiovascular diseases (CVD) through its effects on micro-and macro-vasculature [6]. Our study aims to identify an association of prediabetes with cardiovascular complications, namely Acute Coronary Syndrome (ACS), acute heart failure, acute ischemic stroke (AIS), and intracranial hemorrhage in adults.

2. Methods

2.1. National inpatient sample

The Agency for Healthcare Research and Quality (Healthcare Cost and Utilization Project) has developed a de-identified database called the National Inpatient Sample (NIS). It is the largest publicly available inpatient database in the United States, including data on about 7 million unweighted hospitalizations annually. The national estimate can be calculated using discharge weights, accounting for 35 million hospitalizations annually.

2.2. Patient population

The NIS 2018-2020 was queried for all hospitalizations with a primary or secondary diagnosis of prediabetes. Of these, only hospitalizations with an age greater than 18 years were selected. All hospitalizations with diabetes were excluded from the study. Demographics (age, sex, race, socioeconomic status, and insurance), hospital characteristics (hospital bed size, teaching status and location, and hospital region), and other cardiac risk factors (hypertension, hyperlipidemia, obesity, and tobacco abuse) were compared in hospitalizations with and without prediabetes. International Classification of Disease- Tenth Edition- Clinical Modification (ICD-10-CM) was used to identify all the different diagnoses.

2.3. Statistical analysis

STATA/MP 17.0 software was used for statistical analysis. The chi-square test was used to calculate p values for categorical variables, and the *t*-test was used for continuous variables. P values < 0.05 were considered statistically significant. Univariate logistic regression analysis was first performed to calculate the odds for acute coronary syndrome, AIS, intracranial hemorrhage, and acute heart failure with prediabetes in the subpopulation of adults after excluding diabetic hospitalizations. Multivariate logistic regression analysis was further performed to validate the findings after adjusting for relevant confounding variables. The analysis was adjusted for demographics, hospital characteristics, the Charlson comorbidity index [7], and other cardiac risk factors, including tobacco abuse, hypertension, hyperlipidemia, and obesity.

3. Results

3.1. Baseline characteristics

A total of 1,121,385 (1.005%) index admissions with a diagnosis of prediabetes were identified, which were compared with 110,371,330 (98.9%) non-prediabetics. Hospitalizations with prediabetes were older (median age 63 years vs. 55 years; P<0.001) with relatively uniform sex distribution. Most hospitalizations with prediabetes were whites (61.6%), followed by blacks (16.2%), Hispanics (13.6%), Asians/Pacific Islanders (4.9%) and Native Americans (0.6%). This was comparable to adults without prediabetes, except that the proportion of whites, blacks, and Hispanics with prediabetics was slightly higher. Notably, the prevalence of prediabetes increased with an increase in median household incomes by zip code. The urban hospitals had significantly more hospitalizations with prediabetes than without prediabetes (95% vs. 91%; p<0.001). Hospitalizations with prediabetes were higher in the Western hospitals (35%) of the United States, while hospitalizations without prediabetes were found to be higher in the southern hospitals (39.9%) (Table 1).

3.2. Cardiovascular risk factors

The prevalence of cardiac risk factors was compared in hospitalizations with and without prediabetes. Hospitalizations with prediabetes had a higher prevalence of hypertension (47.6% vs. 30.1%; P<0.001), hyperlipidemia (52.9% vs25.6%; p<0.001), obesity (29% vs. 21.9%; P<0.001) and tobacco abuse (33.5% vs 12.6%; P<0.001). (Table 2).

3.3. Univariate regression analysis

Univariate logistic regression analysis was performed prior to adjusted analysis to study the association between acute cardiovascular events and prediabetes. Unadjusted analysis revealed increased odds of ACS (OR-2.93; C.I: 2.86-3.01; P<0.001) and AIS (OR-2.74; C.I: 2.67-2.82; P<0.001); a decreased odds was seen with acute heart failure

Table 1
Demographics and hospital characteristics of hospitalizations with and without Prediabetes.

Baseline characteristics		Adult Patients with Prediabetes (N = 1121385) (1.005%)	Adult Patients without Prediabetes (N = 110371330) (98.99%)	P value
AGE (in years)		63.0±0.8	55.0±0.05	<0.001
Female sex		51.6%	60.2%	<0.001
Race	White	61.6%	68.6%	<0.001
	Black	16.2%	14.2%	
	Hispanic	13.6%	10.8%	
	Asian/ Pacific islander	4.9%	2.7%	
	Native American	0.7%	0.6%	
	Others[a]	2.9%	3.0%	
Quartile of median household income for zip code	0−25 th	23.9%	29.2%	<0.001
	26 th -50 th	23.8%	26.2%	
	51 st -75 th	25.9%	24.0%	
	76 th -100 th	26.4%	20.1%	
Primary payer	Medicare	52.4%	44.3%	<0.001
	Medicaid	13.9%	20.7%	
	Private	30.1%	30.3%	
	Others	3.8%	4.8%	
Hospital teaching status and location	Rural	4.9%	8.9%	<0.001
	Urban non-teaching	16.2%	21.2%	
	Urban teaching	78.9%	69.9%	
Hospital bed-size	Small	20.5%	21.0%	0.25
	Medium	28.5%	28.9%	
	Large	51.1%	50.1%	
Hospital region	Northeast	17.5%	18.8%	<0.001
	Midwest	20.6%	22.2%	
	South	26.8%	39.9%	
	West	35.0%	19.7%	

Table 2
Prevalence of comorbidities in hospitalizations with and without pre-diabetes.

	Adult Patients with Prediabetes	Adult Patients without Prediabetes	P value
Cardiac risk factors			
Hypertension	47.6%	30.1%	<0.001
Hyperlipidemia	52.9%	25.6%	<0.001
Obesity	29.0%	21.9%	<0.001
Tobacco abuse	33.5%	12.6%	<0.001

(0.79; OR- 0.74-0.84; P<0.001) while the odds of intracranial hemorrhage (OR-1.11; C.I: 0.88-1.40; P = 0.37) was not statistically significant.

3.4. Multivariate regression analysis

Multivariate logistic regression analysis was performed by taking acute cardiovascular events (i.e., ACS, AIS, intracranial hemorrhage, and acute heart failure) as outcomes of interest. Analysis was adjusted for demographics (age, gender, race, and income quartile based on zip code), cardiac risk factors (hypertension, diabetes, obesity, hyperlipidemia, and tobacco abuse), other comorbidities included in the Charlson comorbidity index, and other hospital characteristics (hospital location, teaching status, bed size, and region). Adjusted analysis revealed that hospitalizations with prediabetes were associated with higher odds of developing ACS (OR-2.01; C.I:1.94-2.08; P<0.001), AIS (OR-2.21; 2.11-2.31; p<0.001) and acute heart failure (OR-1.41; C.I.: 1.29-1.55; p<0.001) as compared to hospitalizations without prediabetes. However, the odds of developing intracranial hemorrhage was not statistically significant (OR-1.11; C.I: 0.77-1.59; P = 0.25) (Table 3).

4. Discussion

Our study concluded that the odds of developing ACS, acute ischemic stroke, and acute heart failure was higher in hospitalizations with prediabetes after adjusting for relevant confounders. In contrast, the odds of intracranial hemorrhage was not statistically significant (Table 3).

It is well established that T2DM is prevalent comorbidity among individuals with other traditional cardiovascular disease risk factors such as dyslipidemia, hypertension, and obesity [8]. Our study shows that even hospitalizations with prediabetes have a higher degree of associated comorbidities.

There is growing evidence that prediabetes's effects on the cardiovascular system may be as detrimental as its successor, T2DM [8]. Persons with hyperglycemia far below the threshold for diagnosis of T2DM are known to be at high risk for cardiovascular events [9]. This may be due to the early initiation of damaging effects from chronic hyperglycemia on the cardiovascular system. Literature reveals that dysglycemia increases the risk of vascular disease at all levels. Prediabetes and metabolic syndrome often coexist, leading to a higher propensity to develop a vascular atheroma [10]. A primary component of metabolic syndrome, obesity, mainly found in the central visceral fat distribution, is a source of inflammatory cytokines associated with vascular damage [11]. On the other hand, hyperglycemia induces coronary microvascular dysfunction via activating pro-inflammatory cytokines and endothelial dysfunction, leading to atherosclerotic plaque formation, eventually leading to myocardial injury [8,9]. Hyperglycemia also contributes to hyperlipidemia via glycation of lipoproteins, leading to their delayed breakdown. It also causes the enhanced uptake of these lipoproteins through scavenger receptors on inflammatory cells, which further accelerates atherosclerosis, inflammation, vascular disease, and endothelial dysfunction [12].

Primarily, the macrovascular complications of prediabetes are thought to be a consequence of atherosclerosis (Fig. 1).

Hypertension as a sequela of prediabetes is a significant risk factor for atherosclerotic coronary vascular disease (ASCVD) [11]. Furthermore, it is postulated that insulin resistance and hyperinsulinemia, which lead to prediabetes and later T2DM, play significant roles in the pathogenesis of high blood pressure due to reduced atrial natriuretic peptide, upregulation of renin-angiotensin-aldosterone system activity and increased sympathetic tone [11].

According to Neves et al., insulin resistance and dysglycemia are associated with a hypercoagulable state [11] through alterations in the level of coagulating protein and impaired fibrinolytic activity. A prothrombotic state further contributes to ASCVD (Fig. 1). According to a study by Yuan et al., fibrinogen was independently associated with long-term all-cause and cardiac mortality among coronary artery disease (CAD) patients undergoing percutaneous coronary intervention (PCI), especially in those with pre-diabetes and diabetes [13].

In addition to prediabetes being involved in the pathogenesis of CAD, few studies have demonstrated how it is associated with worse outcomes as compared to normoglycemic individuals in CAD patients. A study by Yong et al. revealed that prediabetes was independently associated with an increased risk of major adverse cardiovascular events (MACEs) after PCI as compared with individuals with normoglycemia, even after adjusting for severity of coronary lesions [14]. These studies demonstrate that prediabetes may independently predict poor prognosis after PCI in CAD patients.

In a study by Selvin et al., prediabetes and T2DM were independently associated with the development of subclinical myocardial damage, as assessed by high-sensitivity troponin, and those individuals were at the highest risk of clinical events. Furthermore, the elevation in high sensitivity troponin among these individuals was associated with future clinical outcomes, particularly heart failure and death [9], suggesting that cardiac damage may occur via a non-atherosclerotic mechanism. Another non-atherosclerotic mechanism that contributes to adverse cardiovascular events, including ischemia, infarction, and heart failure, is cardiac autonomic neuropathy (CAN). Multiple studies have shown a higher prevalence of CAN in patients with prediabetes than those with normal glucose tolerance [15].

Prediabetes has also been studied as a risk factor for stroke in previous studies. In a meta-analysis study by Yeusong et al., prediabetes was associated with an increased risk of new strokes in patients with ischemic stroke and transient ischemic attack compared to patients with normal glucose metabolism [16]. The pathophysiology may be related to the metabolic and cellular changes that insulin resistance has in promoting atherosclerosis in the carotid arteries [16]. In a cohort meta-analysis by Huang et al., prediabetes was associated with an increased risk of CAD, stroke, and all-cause mortality [17]. In another meta-analysis of 38 prospective studies that studied CVD as an outcome, it was revealed that increasing glucose levels displayed a linear relationship to CVD risk [18].

The epidemiological link between T2DM and heart failure has been established through studies such as the Framingham Heart Study [10]; however, there is no established direct link between both comorbidities.

As seen in prediabetes, chronic hyperinsulinemia may be a key risk

Table 3
Multivariate regression analysis showing odds ratio of acute cardiovascular events.

	Odds ratio	Confidence intervals	P value
Acute coronary syndrome			
Prediabetes	2.01	1.94-2.08	<0.001
Acute ischemic stroke			
Prediabetes	2.21	2.11-2.31	<0.001
Intracranial hemorrhage			
Prediabetes	1.11	0.77-1.59	0.56
Acute heart failure			
Prediabetes	1.41	1.29-1.55	<0.001

[a] Adjusted for demographics, cardiac risk factors, charlson comorbidity index, and other hospital characteristics.

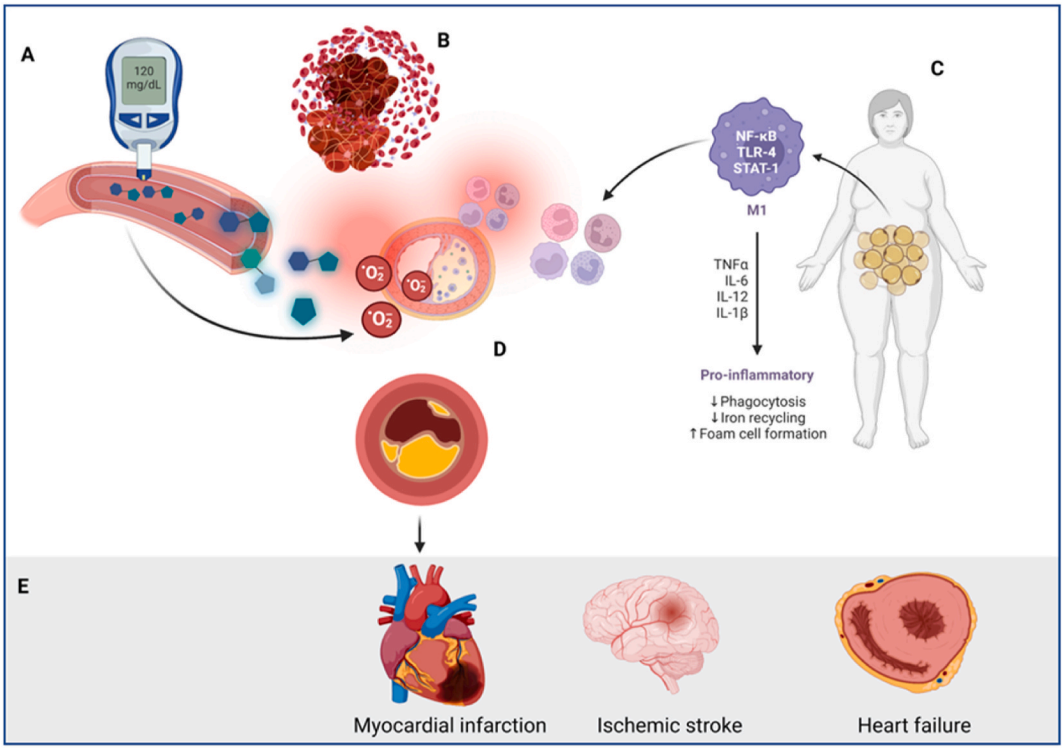

Fig. 1. Implications of pre-diabetes on cardiovascular macrovasculature
Hyperglycemic state (A), hypercoagulable state (B) and metabolic syndrome (C) may lead to higher propensity to develop vascular atheroma (D). All these factors lead to increased risk of myocardial infarction, stroke and heart failure (E).

factor in promoting myocardial hypertrophy and concentric remodeling [19]. Prediabetes is associated with left ventricular diastolic dysfunction [19], which is considered to be the first stage of heart failure development. In a study by Di Palo et al., the effects of prediabetes on diastolic function in 167 patients with HbA1c between 5.7% and 6.4% revealed that patients with prediabetes had significantly lower peak mitral inflow in early diastole (E wave), to late diastolic atrial velocity (A wave) ratio ($P<0.05$), a higher L.A. volume (LAV) ($P<0.05$), and higher spherical index (S.I.) ($P<0.05$) when compared to controls. All three indices reveal early signs of diastolic dysfunction [20]. All these studies demonstrate how the phenotype of heart failure is affected by prediabetes. Our study goes a step further by finding an association between acute heart failure exacerbation and prediabetes.

5. Conclusion

Our study draws many vital conclusions. It reveals that hospitalizations with prediabetes have a higher burden of comorbidities. It also shows how prediabetes may be independently associated with ACS, acute heart failure, and AIS, irrespective of the comorbidities.

Prior literature also suggests how microvascular and macrovascular damage due to hyperglycemia can occur well before an overt diabetes diagnosis. We thus recommend screening for prediabetes and aggressively managing modifiable risk factors in these patients to prevent cardiovascular events. Further prospective studies are required to establish the causation of our findings, and head-to-head trials comparing the cardiovascular burden in prediabetics vs. normoglycemics would be imperative to consider starting early treatment in these patients.

6. Limitations

Our study is a retrospective study using NIS, a coding database. It does not have granular-level data like medications, lab values, etc. So, the diagnosis of prediabetes was based on its ICD-10 billing code rather than on lab values. This may have led to missing diagnosis of prediabetics in hospitalized patients. This limitation also did not allow us to assess the degree of glycemic control in these patients and stratify them based on severity.

NIS accounts only for hospitalized patients. Prediabetes and diabetes are mainly encountered in the outpatient setting unless these patients are admitted for complications. Hence, it is difficult to extrapolate the findings to the general population. Nonetheless, given the study's power, our findings pose an essential question on the treatment cut-offs for diabetes.

The cross-sectional design of the study limits its utility in predicting the time to cardiovascular events in the study population of interest, i.e., prediabetics.

Further prospective trials are required to prove prediabetes as an independent predictor of major acute cardiovascular events.

Author contributions

All named authors meet the International Committee of Medical Journal Editors (ICMJE) criteria for authorship for this article, take responsibility for the integrity of the work as a whole, and have given their approval for this version to be published.

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.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.athplu.2023.08.002.

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