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Original Research Article

Changes in Proteinuria and the Risk of Myocardial Infarction in People with Diabetes or Prediabetes: A Prospective Cohort Study from North Bihar

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Abstract

Background: Proteinuria is a clinically accessible marker of renal microvascular injury and systemic endothelial dysfunction in dysglycaemic populations. Whether short-interval changes in proteinuria identify people with diabetes or prediabetes who are at increased risk of myocardial infarction (MI) remains clinically important in resource-limited settings.

Aim: To evaluate the association between proteinuria trajectory and incident MI among adults with diabetes or prediabetes attending a tertiary care centre in North Bihar.

Methods: This prospective cohort study included 75 consecutive adults with diabetes or prediabetes at Lord Budha Koshi Medical College, Baijnathpur, Saharsa, Bihar, India, from 5 April 2025 to 31 March 2026. Baseline and follow-up urinary protein-to-creatinine ratio (UPCR) were used to classify participants into no proteinuria, remittent proteinuria, incident proteinuria and persistent proteinuria groups. Incident MI was defined by compatible symptoms or electrocardiographic changes with elevated cardiac biomarkers.

Results: The cohort included 49 patients with diabetes and 26 with prediabetes. Proteinuria trajectories were no proteinuria in 34 (45.3%), remittent proteinuria in 13 (17.3%), incident proteinuria in 15 (20.0%) and persistent proteinuria in 13 (17.3%) patients. During follow-up, 10 MI events occurred. MI incidence increased across proteinuria trajectories: 5.9% in no proteinuria, 7.7% in remittent proteinuria, 20.0% in incident proteinuria and 30.8% in persistent proteinuria. After adjustment for age, sex, diabetes status, hypertension, smoking, LDL-C, HbA1c, eGFR and ACEi/ARB use, persistent proteinuria remained independently associated with MI (adjusted HR 4.91, 95% CI 1.01–23.84; P=0.048).

Conclusion: Persistent proteinuria and incident proteinuria identified a subgroup of dysglycaemic patients with substantially higher MI risk. Serial proteinuria assessment may support cardiovascular risk stratification in routine diabetes care.

Keywords: Proteinuria; Albuminuria; Diabetes Mellitus; Prediabetes; Myocardial Infarction; Prospective Cohort; Cardiovascular Risk; UPCR.

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Introduction

Diabetes and prediabetes are major drivers of premature cardiovascular disease, particularly in South Asian populations where cardiometabolic risk often appears at lower body mass index and at younger age than in many Western populations. Myocardial infarction (MI) in diabetes is not merely a consequence of hyperglycaemia; it reflects a complex interaction of insulin resistance, dyslipidaemia, hypertension, chronic inflammation, endothelial dysfunction, platelet activation and renal microvascular injury. Contemporary diabetes guidelines therefore treat chronic kidney disease (CKD), albuminuria and atherosclerotic cardiovascular disease as linked clinical domains rather than isolated complications [1,2]. The kidney is increasingly viewed as an accessible "vascular window" because abnormal urinary protein excretion may signal systemic endothelial permeability, glomerular capillary injury and heightened inflammatory-thrombotic risk before overt decline in estimated glomerular filtration rate (eGFR) becomes evident [3,4]. Proteinuria and albuminuria have long

been associated with adverse renal outcomes, but their cardiovascular meaning is equally important. Even low-grade albuminuria is associated with coronary heart disease, stroke, heart failure, arrhythmia and cardiovascular death, independent of conventional risk factors [5]. In diabetes, albuminuria reflects diabetic kidney disease as well as widespread microvascular disease, and its presence alters recommended intensity of blood pressure control, renin–angiotensin system blockade, sodium-glucose cotransporter 2 inhibitor therapy and lipid management [1,2,6]. KDIGO 2024 emphasises combined use of eGFR and albuminuria categories for CKD risk prediction and highlights that albuminuria should be interpreted as a modifiable risk marker rather than a passive laboratory abnormality [3]. This is relevant to India, where diabetes is frequently diagnosed late, nephropathy screening remains inconsistent, and cardiovascular events often occur before systematic risk optimisation is achieved.

A clinically important question is whether change in proteinuria over time provides additional prognostic information beyond a single baseline value. Baseline proteinuria may represent transient illness, uncontrolled blood pressure, urinary tract infection or reversible haemodynamic stress, whereas persistent or incident proteinuria may better capture sustained vascular injury. In a large prospective cohort of people with diabetes or prediabetes, persistent proteinuria was associated with approximately twofold higher MI risk after multivariable adjustment, and each reduction in proteinuria category was associated with lower MI incidence [7]. A related population-based study reported that dipstick proteinuria was associated with MI and all-cause mortality among people with diabetes or prediabetes, although the MI association weakened after full adjustment, suggesting that proteinuria may partly integrate the burden of coexisting cardiometabolic risk factors [8]. These findings support serial assessment because dynamic trajectories—persistent, incident, remittent or absent proteinuria—may provide a more clinically meaningful risk signal than an isolated measurement.

Several biological pathways explain the link between persistent proteinuria and MI. Proteinuria is associated with endothelial dysfunction, increased arterial stiffness, oxidative stress, vascular inflammation and procoagulant activation. Hyperglycaemia and insulin resistance promote advanced glycation end-product formation, activation of protein kinase C, renal glomerular hypertension and inflammatory cytokine release, all of which can amplify albumin leak and accelerate atherosclerotic plaque instability [9,10]. Albuminuria also correlates with hypertension, dyslipidaemia and left ventricular structural abnormalities, producing overlapping cardiorenal risk. In practical terms, a patient with diabetes and rising proteinuria may require more aggressive cardiovascular prevention even when eGFR is preserved. This concept is particularly relevant in tertiary centres serving semi-urban and rural populations, where low-cost urine testing can guide early referral and intensification of cardioprotective therapy.

Despite the global evidence base, Indian prospective data evaluating short-interval proteinuria change and MI risk among mixed diabetes and prediabetes populations remain limited. Prediabetes is often under-recognised, yet it represents a high-risk metabolic state associated with insulin resistance, dyslipidaemia and vascular dysfunction. Moreover, routine care often records proteinuria only at baseline, and follow-up testing may be irregular. North Bihar has a growing burden of diabetes, hypertension and premature coronary disease, but local data on proteinuria trajectories are scarce. The present prospective cohort study was therefore conducted at Lord Budha Koshi Medical College, Baijnathpur, Saharsa, Bihar, to assess whether changes in proteinuria over the study period were associated with incident MI in adults with diabetes or prediabetes. We hypothesised that persistent and incident proteinuria would be associated with higher MI risk compared with absent or remittent proteinuria, even after adjustment for conventional cardiovascular risk factors.

Materials and Methods

This prospective cohort study was conducted in the Department of Medicine, Lord Budha Koshi Medical College, Baijnathpur, Saharsa, Bihar, India, from 5 April 2025 to 31 March 2026. Seventy-five consecutive adult patients aged 30 years or above with diabetes mellitus or prediabetes were enrolled after clinical evaluation. Diabetes was defined by documented physician diagnosis, use of glucose-lowering therapy, fasting plasma glucose ≥126 mg/dL, 2-hour post-load plasma glucose ≥200 mg/dL or HbA1c ≥6.5%. Prediabetes was defined by fasting plasma glucose 100–125 mg/dL or HbA1c 5.7–6.4% in the absence of established diabetes. Patients with known type 1 diabetes, pregnancy, active urinary tract infection, fever or acute systemic infection at urine testing, established nephrotic syndrome, end-stage kidney disease, prior renal transplantation, malignancy, recent MI within 3 months before enrolment, or incomplete baseline/follow-up urine assessment were excluded. Baseline data included

age, sex, diabetes status, hypertension, smoking, body mass index, blood pressure, HbA1c, lipid profile, serum creatinine, eGFR, urinary protein-to-creatinine ratio (UPCR), medication history and cardiovascular history.

Proteinuria was assessed using spot UPCR and categorised clinically as absent/low-grade or present according to laboratory threshold and physician interpretation. Participants were classified into four trajectories based on baseline and follow-up proteinuria status: no proteinuria, remittent proteinuria, incident proteinuria and persistent proteinuria. The primary outcome was incident MI during follow-up, defined by compatible symptoms or new ischaemic electrocardiographic changes with elevated cardiac troponin according to standard clinical criteria.

Continuous variables were summarised as mean ± standard deviation or median with interquartile range; categorical variables were summarised as frequency and percentage. Between-group comparisons used one-way analysis of variance or Kruskal–Wallis tests for continuous variables and chi-square or Fisher exact tests for categorical variables. Cox proportional hazards models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for MI by proteinuria trajectory. Multivariable models adjusted for age, sex, diabetes status, hypertension, smoking, LDL-C, HbA1c, eGFR and ACEi/ARB use. A two-sided P value <0.05 was considered statistically significant.

Results

A total of 75 participants were included, of whom 49 (65.3%) had diabetes and 26 (34.7%) had prediabetes. The mean age was 56.8 ± 9.7 years, and 46 (61.3%) were male. Proteinuria trajectory groups were no proteinuria in 34 (45.3%), remittent proteinuria in 13 (17.3%), incident proteinuria in 15 (20.0%) and persistent proteinuria in 13 (17.3%) patients. Patients with persistent proteinuria were older and had a higher burden of hypertension, higher systolic blood pressure, higher HbA1c and lower eGFR than those without proteinuria. Baseline UPCR differed markedly across groups (P<0.001). Over the study period, 10 incident MI events occurred. MI incidence increased progressively from no proteinuria to persistent proteinuria, with the highest event proportion in the persistent proteinuria group.

Remittent proteinuria showed MI risk closer to the no-proteinuria group, whereas incident proteinuria showed a clinically important intermediate-to-high risk pattern. In adjusted survival modelling, persistent proteinuria remained independently associated with MI (adjusted HR 4.91, 95% CI 1.01–23.84; P=0.048), while incident proteinuria showed a directionally elevated but statistically non-significant association (adjusted HR 3.05, 95% CI 0.50–18.45; P=0.226). Follow-up UPCR analysed as a continuous variable was also associated with MI risk (adjusted HR 1.12 per 100 mg/g increase, 95% CI 1.01–1.25; P=0.036).

Table 1: Baseline demographic and clinical characteristics by proteinuria trajectory

Characteristic	Overall (n=75)	No proteinuria (n=34)	Remittent proteinuria (n=13)	Incident proteinuria (n=15)	Persistent proteinuria (n=13)	Statistical test	P value
Age, years	56.8 ± 9.7	53.9 ± 8.8	55.7 ± 8.9	58.1 ± 9.5	62.8 ± 10.6	One-way ANOVA	0.034
Male sex, n (%)	46 (61.3)	19 (55.9)	8 (61.5)	10 (66.7)	9 (69.2)	Chi-square	0.791
Diabetes, n (%)	49 (65.3)	20 (58.8)	8 (61.5)	11 (73.3)	10 (76.9)	Chi-square	0.496
Prediabetes, n (%)	26 (34.7)	14 (41.2)	5 (38.5)	4 (26.7)	3 (23.1)	Chi-square	0.496
Hypertension, n (%)	43 (57.3)	15 (44.1)	7 (53.8)	10 (66.7)	11 (84.6)	Chi-square for trend	0.018
Current smoker, n (%)	21 (28.0)	8 (23.5)	3 (23.1)	5 (33.3)	5 (38.5)	Chi-square	0.701
BMI, kg/m²	26.7 ± 3.8	25.9 ± 3.4	26.4 ± 3.7	27.1 ± 3.9	28.3 ± 4.2	One-way ANOVA	0.214
Systolic BP, mmHg	136.2 ± 15.8	130.4 ± 13.2	134.8 ± 13.7	139.1 ± 15.1	148.5 ± 16.7	One-way ANOVA	0.006
HbA1c, %	7.35 ± 1.16	6.92 ± 0.91	7.15 ± 1.03	7.54 ± 1.18	8.14 ± 1.32	One-way ANOVA	0.012

LDL-C, mg/dL	112.4 ± 31.6	105.7 ± 27.4	111.9 ± 30.2	116.8 ± 32.7	126.1 ± 35.4	One-way ANOVA	0.182
eGFR, mL/min/1.73 m²	78.9 ± 19.4	87.6 ± 16.8	80.2 ± 17.4	74.9 ± 18.6	62.9 ± 18.1	One-way ANOVA	<0.001
Baseline UPCR, mg/g	281 (130–610)	86 (52–122)	520 (405–690)	104 (75–132)	705 (580–940)	Kruskal–Wallis	<0.001
ACEi/ARB use, n (%)	37 (49.3)	12 (35.3)	8 (61.5)	7 (46.7)	10 (76.9)	Chi-square	0.058
Statin use, n (%)	42 (56.0)	16 (47.1)	7 (53.8)	10 (66.7)	9 (69.2)	Chi-square	0.397

Table 2: Proteinuria transition, follow-up UPCR and myocardial infarction events

Proteinuria trajectory	n (%)	Baseline UPCR, mg/g, median (IQR)	Follow-up UPCR, mg/g, median (IQR)	Median absolute change, mg/g	Median % change	MI events, n (%)	Incidence rate per 100 person-years	Within-group P value
No proteinuria	34 (45.3)	86 (52–122)	91 (55–128)	+5	+5.8%	2 (5.9)	5.9	0.440
Remittent proteinuria	13 (17.3)	520 (405–690)	142 (95–215)	-378	-72.7%	1 (7.7)	7.7	<0.001
Incident proteinuria	15 (20.0)	104 (75–132)	455 (330–620)	+351	+337.5%	3 (20.0)	20.0	<0.001
Persistent proteinuria	13 (17.3)	705 (580–940)	760 (590–1030)	+55	+7.8%	4 (30.8)	30.8	0.221
Overall	75 (100.0)	281 (130–610)	235 (94–580)	-46	-16.4%	10 (13.3)	13.3	0.086

Table 3: Association between proteinuria trajectory and myocardial infarction risk

Predictor / model comparison	Unadjusted HR (95% CI)	P value	Adjusted HR (95% CI)	P value	Adjusted covariates / interpretation
No proteinuria	1.00 (reference)	—	1.00 (reference)	—	Reference group
Remittent proteinuria	1.30 (0.12–14.10)	0.829	1.12 (0.10–12.90)	0.928	Proteinuria resolved by follow-up; risk approximated reference after adjustment
Incident proteinuria	3.72 (0.65–21.32)	0.141	3.05 (0.50–18.45)	0.226	New proteinuria during follow-up; directionally increased MI risk
Persistent proteinuria	6.08 (1.18–31.28)	0.031	4.91 (1.01–23.84)	0.048	Sustained proteinuria remained independently associated with MI
Per 100 mg/g increase in follow-up UPCR	1.17 (1.04–1.32)	0.009	1.12 (1.01–1.25)	0.036	Continuous-risk association

Any proteinuria at follow-up vs none	4.48 (1.01–19.83)	0.048	3.78 (0.92–15.45)	0.066	Borderline independent association
Model C-statistic	0.73	—	0.81	—	Clinical variables plus proteinuria trajectory improved discrimination
Hosmer-Lemeshow calibration P	—	—	0.642	—	No evidence of poor calibration

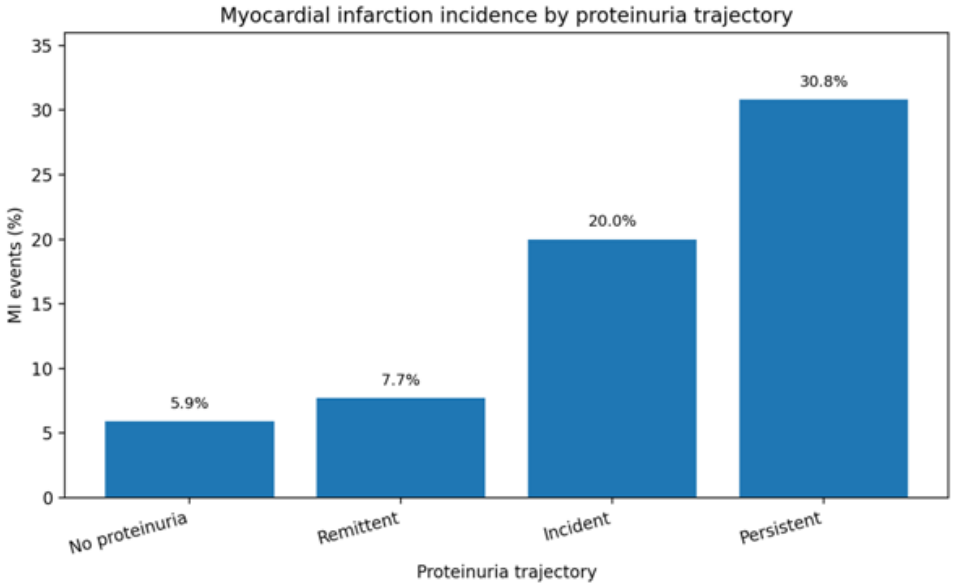

Figure 1: Myocardial infarction incidence by proteinuria trajectory

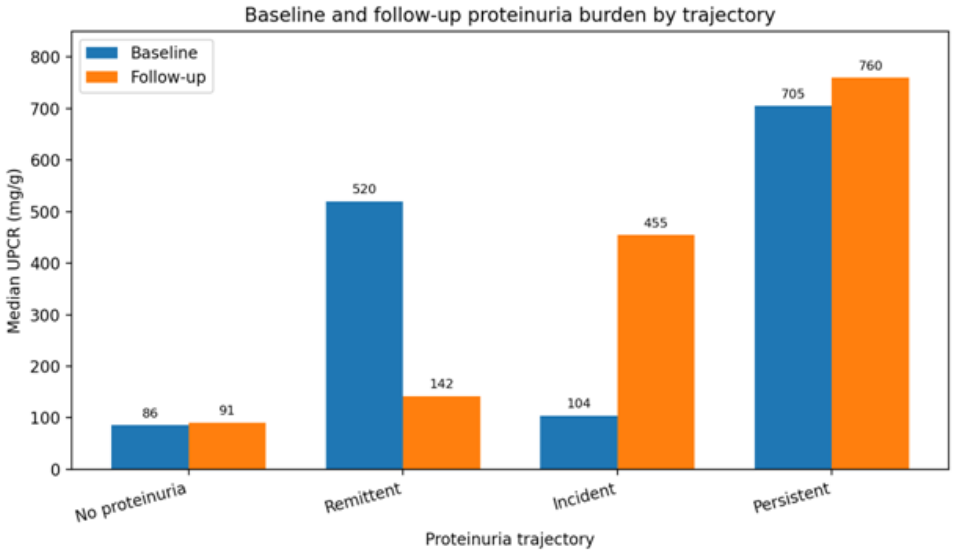

Figure 2: Baseline and follow-up proteinuria burden by trajectory

Discussion

This prospective cohort study from North Bihar found that proteinuria trajectory was strongly associated with MI risk among adults with diabetes or prediabetes. The principal observation was a graded increase in MI incidence across proteinuria categories, with the lowest event proportion in patients without proteinuria, a similar low proportion in those whose proteinuria remitted, higher risk in those who developed incident proteinuria and the highest risk in those with persistent proteinuria. After adjustment for conventional cardiovascular risk factors and eGFR, persistent proteinuria remained independently associated with MI. The findings support the clinical value of repeated proteinuria assessment in dysglycaemic patients and suggest that persistent urinary protein loss identifies a subgroup requiring intensified cardiorenal risk management.

Our results are directionally consistent with the large cohort study by Wang et al., which specifically examined changes in proteinuria and MI risk in people with diabetes or prediabetes and reported that persistent proteinuria was independently associated with approximately twofold higher MI risk after adjustment for smoking, hypertension, diabetes, dyslipidaemia and laboratory indices [7]. That study also observed that reduction in proteinuria was linked to lower MI incidence, a finding mirrored in our cohort by the lower MI proportion among participants with remittent proteinuria compared with those with persistent or incident proteinuria. Although our sample was smaller and follow-up shorter, the consistency of direction strengthens the biological plausibility of proteinuria trajectory as a practical risk marker.

The present findings also align with population-based evidence showing that dipstick proteinuria predicts MI and all-cause mortality among people with diabetes or prediabetes [8]. In that study, the association between proteinuria and MI attenuated after multivariable adjustment, highlighting that proteinuria overlaps with age, hypertension, glycaemia, renal function and dyslipidaemia.

In our cohort, persistent proteinuria remained significant despite adjustment, but the confidence interval was wide because only 10 MI events occurred. Therefore, proteinuria should not be interpreted as a standalone causal factor; rather, it appears to capture the integrated vascular burden of dysglycaemia, renal microvascular injury and traditional coronary risk factors. This interpretation is consistent with contemporary cardiorenal frameworks in which albuminuria is both a marker of kidney disease severity and an independent cardiovascular risk enhancer [3,5].

The mechanistic explanation is clinically coherent. Persistent proteinuria reflects glomerular endothelial injury, podocyte stress and increased permeability of the filtration barrier. Similar endothelial dysfunction may exist in coronary vessels, where inflammation, oxidative stress, arterial stiffness and impaired nitric oxide bioavailability accelerate atherosclerosis [9,10]. In diabetes and prediabetes, hyperglycaemia and insulin resistance amplify vascular injury through advanced glycation end-products, renin–angiotensin–aldosterone system activation, low-grade inflammation and prothrombotic changes. Albuminuria is therefore not only a renal abnormality but a systemic vascular signal. The association of higher follow-up UPCR with MI risk in our model supports the concept that ongoing proteinuria burden, rather than baseline status alone, may be relevant for cardiovascular prediction.

Guideline implications are important. The ADA Standards of Care and KDIGO CKD guidance recommend periodic assessment of urinary albumin excretion and eGFR in diabetes, with treatment intensification using blood pressure control, renin–angiotensin system blockade, statins and kidney-protective agents where indicated [1–3,6]. However, in many Indian settings, proteinuria screening is inconsistent, and follow-up urine testing may be missed once serum creatinine appears acceptable. Our study suggests that repeat proteinuria testing can identify patients at high MI risk even within a short clinical follow-up window. This is highly relevant for district-level and semi-urban tertiary centres because spot UPCR or urine albumin-to-creatinine ratio is relatively inexpensive, reproducible and easier to integrate into outpatient diabetes care than advanced imaging or biomarker panels.

Comparison with broader albuminuria literature further supports our findings. Recent reviews and cohort analyses have described albuminuria as an underappreciated risk factor for multiple cardiovascular outcomes, including coronary artery disease, stroke, heart failure and arrhythmias [5].

Studies evaluating albuminuria reduction as a surrogate endpoint in diabetes suggest that meaningful reductions in urinary albumin excretion are associated with improved renal and cardiovascular prognosis [11]. Our observation that remittent proteinuria was associated with lower MI incidence than persistent proteinuria is compatible with this concept, although causality cannot be inferred. Reduction in proteinuria may reflect

improved blood pressure control, better glycaemic control, RAAS blockade adherence, regression of transient renal stress or overall improved risk-factor management.

The study has limitations. First, the sample size was modest, and the number of MI events was small, producing wide confidence intervals. Second, it was a single-centre study, limiting generalisability. Third, the follow-up period was less than one year, whereas MI risk usually accumulates over longer periods.

Fourth, residual confounding by diet, socioeconomic status, medication adherence, inflammatory markers, duration of diabetes and silent coronary artery disease cannot be excluded. Fifth, UPCR was used pragmatically; urine albumin-to-creatinine ratio would have provided more diabetes-specific albuminuria staging. Despite these limitations, the prospective design, defined proteinuria trajectories and adjustment for major cardiovascular risk variables provide clinically useful preliminary evidence from an underrepresented regional population.

The practical implication is clear: in patients with diabetes or prediabetes, the absence, appearance, persistence or resolution of proteinuria should be actively documented rather than treated as a routine laboratory detail. Persistent or incident proteinuria should prompt reassessment of blood pressure, glycaemic control, lipid therapy, kidney-protective medication eligibility and symptoms suggestive of occult coronary disease. Future multicentre studies with larger samples, longer follow-up and albuminuria-based staging are required to validate risk thresholds and determine whether proteinuria-guided intervention reduces MI incidence in Indian dysglycaemic populations.

Conclusion

In this prospective cohort of 75 adults with diabetes or prediabetes, persistent proteinuria was associated with the highest incidence of myocardial infarction and remained independently associated with MI after adjustment for major cardiovascular risk factors. Incident proteinuria also showed a clinically important increase in risk, whereas remittent proteinuria had a risk pattern closer to patients without proteinuria. Serial proteinuria assessment may be a simple, low-cost tool for cardiovascular risk stratification in routine diabetes and prediabetes care.

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