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RETROSPECTIVE ANALYSIS OF HOSPITAL LABORATORY RECORDS TO EVALUATE THE CORRELATION BETWEEN HBA1 C LEVELS AND ESTIMATED GLOMERULAR FILTRATION RATES IN TYPE 2 DIABETES MELLITUS PATIENTS IN CENTRAL KERALA

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is often associated with dysglycemia and chronic kidney disease (CKD). HbA1c is an indicator of long-term glycaemic control, and estimated glomerular filtration rate (eGFR) is an indicator of renal function. Real-world hospital laboratory data from South India are scarce. **Material and Methods:** We conducted a retrospective cross-sectional study using de-identified laboratory data from three tertiary hospitals in central Kerala (January 2025 to August 2026). Adults (≥ 18 y) with confirmed T2DM and concurrent measurements of HbA1c and serum creatinine were included. eGFR was calculated with the CKD-EPI formula. Linear relationships were assessed with Pearson and monotonic relationships with Spearman correlations. Multivariable linear regression adjusted for age, sex, BMI, duration of diabetes, hypertension and class of medication. **Results:** There were 1872 patients (55.3% male), with mean HbA1c 8.2% and eGFR 84.3 mL/min/1.73 m². Pearson r = -0.21 ($p < 0.001$). Following adjustment, regression demonstrated that a 1% rise in HbA1c was associated with a 2.4 mL/min/1.73 m2 decline in eGFR (95% CI -3.1 to -1.7). Stronger correlations were observed in age > 65 y (r = -0.35) and baseline eGFR < 60 mL/min/1.73 m² (r = -0.42). SGLT2 inhibitor users had a higher eGFR. **Conclusion:** In T2DM patients from central Kerala, a modest inverse association between HbA1c and eGFR persists after adjustment. Targeted glycaemic control and routine renal monitoring are warranted, particularly in older and CKD-prone groups.

INTRODUCTION

Diabetes is one of the greatest health challenges across the world in the 21st century, and more than 90% of all diabetes cases are type 2 diabetes (T2DM). The International Diabetes Federation (IDF) says there are an estimated 537 million persons living with diabetes globally. to reach 643 million by 2030.[1] India is home to the second-largest diabetic population and is a significant contributor to the worldwide diabetes epidemic.[2] Kerala, a state in India, has become a diabetes hub, with urban prevalence rates in the adult population topping 20%, which is considerably higher than the national norm.[3,4] The growing frequency is the result of a complex interaction of variables such as transition to energy-rich meals, sedentary lifestyles, genetic susceptibility and an ageing population.[5,6] T2DM is well known for its microvascular consequences, including diabetic kidney disease (DKD), which remains a major cause of end-stage renal disease (ESRD) and cardiovascular morbidity and acute glycaemic dysregulation.[7,8]

DKD has a complex pathogenesis including hyperglycaemia-induced metabolic and haemodynamic changes, oxidative stress,

inflammation and fibrosis in the glomerular and tubulointerstitial compartments.[9,10] Persistent hyperglycaemia triggers these pathogenic processes, as shown by high HbA1c. HbA1c, the main index of long-term glycaemic control and of treatment decision, provides an integrated measure of mean glycaemia over the previous 8–12 weeks.[11] Renal function and glycaemic control have been investigated in landmark clinical trials and prospective cohort studies. The United Kingdom Prospective Diabetes Study (UKPDS) showed that intensive glucose control in newly diagnosed T2DM patients reduced the occurrence of microvascular sequelae such as nephropathy.[12] The Action to Control Cardiovascular Risk in Diabetes (ACCORD) and ADVANCE trials also demonstrated that intensive glycaemic control reduces the decline of renal function. However, the proper HbA1c target and benefit are still debatable, particularly in older or comorbid people.[13]

These results are corroborated by observational studies in real-world patients, which have demonstrated an inverse association between estimated glomerular filtration rate (eGFR) and HbA1c across different ethnicities and geographic regions.[2,14] However, the clinical importance and severity of this HbA1c-eGFR relationship may differ dramatically amongst populations. South Asian communities, notably those in India, often present with a unique phenotype of T2DM, with a younger age of onset, higher insulin resistance and increased risk of microvascular sequelae, despite relatively low levels of glycaemic exposure.[15,16] The epidemiological change in India has resulted in the merging of CKD and diabetes epidemics. Recently, the ICMR–INDIAB study documented that a considerable proportion of people with T2DM show indications of renal damage at the time of diagnosis.[2,17] In this setting, Kerala is unique with a high prevalence of T2DM and improving, but still limited access to expert nephrology care. Thus, it is a good setting to study the real-life correlates of renal function in T2DM.[18,19]

While there is a large amount of data from Western cohorts and an increasing literature from metropolitan Indian centres, there is a lack of studies specifically examining the HbA1c–eGFR link utilising routinely collected hospital laboratory records from tertiary care centres in Central Kerala. These real-world data are essential given that they reflect the clinical practice patterns, patient demographics and treatment decision-making that impact outcomes in everyday care, not the highly controlled environments of clinical trials. Most previous regional studies were limited to certain metropolitan populations, had small sample numbers, or did not consider important confounders such as hypertension, obesity, and class of medicine.[6,20,21] Indian T2DM cohorts have not been properly studied for the potential protective effects of SGLT2 inhibitors and GLP-1 receptor agonists on the kidneys; thus, our knowledge of the effects of these agents on the glycemic–renal axis is incomplete.[22]

Hence, the present study intends to address this void by undertaking a comprehensive retrospective analysis of laboratory data obtained from hospitals at three tertiary care hospitals in central Kerala. The objective is to quantify the correlation between HbA1c and eGFR, determine the main demographic and clinical subgroups in which this correlation is the strongest, quantify the independent association after adjusting for age, sex, BMI, duration of diabetes, hypertension and antihyperglycemic medication class, and assess the potential modifying effect of SGLT2 inhibitor therapy. The study will be conducted using a large, de-identified dataset of T2DM patients with simultaneous HbA1c and serum creatinine measurements. Our research is intended to give clinically useful information for healthcare providers in Kerala and other South Asian locations and hence emphasise the importance of integrated glycaemic and renal monitoring in the normal management of T2DM.

MATERIALS AND METHODS

Study Design and Setting: Retrospective cross-sectional examination of anonymised laboratory records of tertiary health care facilities of Central Kerala, over the period January 2025- August 2026.

Population

Inclusion: Adults (≥18 y) with proven T2DM, simultaneous HbA1c and serum creatinine tested ≤30 days apart, and comprehensive demographic data.

Exclusion: Type 1 diabetes, secondary diabetes, recent contrast, advanced renal damage, missing data.

Data Extraction: Age, sex, weight/height (BMI), diabetes duration, hypertension status, current antihyperglycemic medications (metformin, sulfonylureas, insulin, SGLT2 inhibitors, GLP-1 RAs), HbA1c (%), serum creatinine (mg/dL), eGFR (CKD-EPI 2021 equation, race-modified according to local protocol).

Statistical Analyses

Descriptive data were given as mean ± SD for continuous variables and number (percentage) for categorical variables. Correlations were computed using Pearson (linear) and Spearman (monotonic) with 95% confidence intervals. eGFR was analysed as an outcome with HbA1c as a predictor using multivariable linear regression analysis with covariates age, sex, BMI, duration of T2DM, hypertension, and class of medication. Modelling was performed in a stepwise manner, from unadjusted to demographic and clinical adjustment to full adjustment. Subgroup interaction was evaluated by stratified correlations. All analyses were done using R version 4.4.0 (R Foundation for Statistical Computing, Vienna, Austria) with a significance level of two-sided $\alpha = 0.05$.

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RESULTS

The cohort included 1 872 patients (55.3 % men). Baseline characteristics are shown in Table 1. Glycaemic control was poor overall (mean HbA1c 8.2%). Most eGFRs were ≥90 mL/min/1.73 m2 (48.7 %), with 16.3 % eGFR < 60.

Table 1: Demographic, clinical and laboratory characteristics of T2DM patients (n = 1 872) stratified by HbA1 c category

Variable	Overall (n = 1 872)	HbA1c < 7.0 % (n = 31 2)	HbA1c 7.0–8.0 % (n = 521)	HbA1c > 8.0 % (n = 1 039)	p-value
Age, years (mean ± SD)	58.6 ± 9.2	55.1 ± 8.7	57.8 ± 9.0	60.2 ± 9.1	<0.001
Sex – Male, n (%)	1 035 (55.3)	1 68 (53.8)	289 (55.5)	578 (55.6)	0.78
BMI, kg/m² (mean ± SD)	26.8 ± 4.5	26.2 ± 4.1	26.7 ± 4.4	27.1 ± 4.6	0.003
T2DM duration, years (mean ± SD)	1 0.3 ± 7.1	6.4 ± 5.2	9.1 ± 6.5	1 2.0 ± 7.4	<0.001
Hypertension, n (%)	641 (34.2)	72 (23.1)	1 68 (32.2)	401 (38.6)	<0.001
Current smoker, n (%)	21 4 (1 1 .4)	31 (9.9)	58 (1 1 .1)	1 25 (1 2.0)	0.42
HbA1 c, % (mean ± SD)	8.2 ± 1 .5	6.4 ± 0.4	7.5 ± 0.3	9.3 ± 1 .1	<0.001
Serum creatinine (mean ± SD mg/dL)	1 .02 ± 0.38	0.91 ± 0.22	0.97 ± 0.28	1 .1 0 ± 0.43	<0.001
eGFR, mL/min/1 .73 m² (mean ± SD)	84.3 ± 22.1	92.4 ± 1 8.7	87.1 ± 20.3	79.2 ± 22.6	<0.001
eGFR category, n (%)	G1 (≥90): 91 2 (48.7) G2 (60-89): 654 (34.9) G3a (45-59): 21 8 (1 1 .6) G3b-5 (<45): 88 (4.7)				<0.001
Medication class, n (%)	Metformin only: 587 (31 .4) Metformin + SU: 421 (22.5) Insulin-based: 51 2 (27.4) SGLT2i (± others): 1 98 (1 0.6) GLP-1 RA (± others): 54 (2.9)				<0.001 (overall)

Baseline demographics (Table 1) show a clear gradient of older age, longer duration of T2DM, higher BMI and more hypertension in patients with higher HbA1c. Progressive renal dysfunction is also evident with glycaemic stratification (mean eGFR: 92.4 → 87.1 → 79.2 mL/min/1 .73 m²). Medication patterns align with therapeutic intensity (most SGLT2-inhibitor use in the well-controlled and insulin regimens in the poorly controlled groups).

Table 2: Bivariate correlations between HbA1 c and eGFR stratified by key patient characteristics

Subgroup	n	Pearson r (95 % CI)	Spearman ρ (95 % CI)	p-value
Overall	1 872	-0.21 (-0.26, -0.1 6)	-0.1 9 (-0.24, -0.1 4)	<0.001
Age ≤65 y	1 324	-0.1 6 (-0.21 , -0.1 1)	-0.1 4 (-0.1 9, -0.09)	<0.001
Age >65 y	548	-0.35 (-0.42, -0.27)	-0.32 (-0.39, -0.24)	<0.001
Male	1 035	-0.1 9 (-0.25, -0.1 3)	-0.1 7 (-0.23, -0.1 1)	<0.001
Female	837	-0.24 (-0.30, -0.1 7)	-0.22 (-0.28, -0.1 5)	<0.001
Diabetes duration ≤1 0 y	987	-0.1 7 (-0.23, -0.1 1)	-0.1 5 (-0.21 , -0.09)	<0.001
Diabetes duration >1 0 y	885	-0.26 (-0.32, -0.20)	-0.24 (-0.30, -0.1 8)	<0.001
eGFR ≥90 (G1)	91 2	-0.1 2 (-0.1 8, -0.06)	-0.1 0 (-0.1 6, -0.04)	<0.001
eGFR 60-89 (G2)	654	-0.1 8 (-0.25, -0.1 1)	-0.1 6 (-0.23, -0.09)	<0.001
eGFR <60 (G3-5)	306	-0.42 (-0.50, -0.33)	-0.39 (-0.47, -0.30)	<0.001
Hypertension absent	1 231	-0.1 9 (-0.24, -0.1 3)	-0.1 7 (-0.22, -0.1 1)	<0.001
Hypertension present	641	-0.27 (-0.34, -0.1 9)	-0.25 (-0.32, -0.1 7)	<0.001
SGLT2 inhibitor use – No	1 674	-0.22 (-0.27, -0.1 7)	-0.20 (-0.25, -0.1 5)	<0.001
SGLT2 inhibitor use – Yes	1 98	-0.1 1 (-0.25, 0.03)	-0.09 (-0.23, 0.05)	0.1 2

Correlation analysis (Table 2) showed a general weak inverse Pearson r = -0.21 (p < 0.001). Subgroup analysis suggests effect modification; the association is stronger with older age, longer duration of T2DM, baseline eGFR < 60 mL/min/1 .73 m², and presence of hypertension. Conversely, the correlation is blunted by SGLT2-inhibitor therapy (non-significant).

The overall weak inverse correlation (r = 0.21) indicates a modest glycemic renal link. Effect modification analysis showed a significantly stronger association in older patients (> 65 y, r = -0.35), those with a longer diabetes duration (> 10 y, r = -0.26), and especially among those already with reduced baseline eGFR (< 60 mL/min/1.73 m2, r = -0.42). Hypertension increases the association, while ongoing SGLT2-inhibitor therapy appears to decrease the association (non-significant trend), indicating a renoprotective effect. Multivariable regression (Table 3) showed that HbA1c was an independent predictor of lower eGFR after full adjustment. More covariates improve the fit of the

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model (R^2=23.6 %). Medication analysis shows a protective association for SGLT2 inhibitors and an adverse association for insulin-based regimens (probably severity bias).

Table 3: Multivariable linear regression of eGFR (dependent variable) on HbA1 c and covariates in T2DM patients

Variable	Model 1 (Unadjusted)	Model 2 (Demographics + Clinical)	Model 3 (Full Adjustment)
HbA1 c (per 1 % increase)	-2.80 (-3.48, -2.1 2)	-2.38 (-3.04, -1 .72)	-2.31 (-2.97, -1 .65)
Age (per year)		-0.42 (-0.51 , -0.33)	-0.39 (-0.48, -0.30)
Female sex (ref: Male)		2.1 4 (0.87, 3.41)	1 .98 (0.72, 3.24)
BMI (per kg/m²)		-0.31 (-0.44, -0.1 8)	-0.28 (-0.41 , -0.1 5)
Diabetes duration (per year)		-0.28 (-0.36, -0.20)	-0.24 (-0.32, -0.1 6)
Hypertension (ref: No)		-5.62 (-7.1 2, -4.1 2)	-4.87 (-6.35, -3.39)
Medication class (ref: Metformin only)			Metformin + SU: -1 .1 2 (-2.78, 0.54) Insulin-based: -3.45 (-5.1 2, -1 .78) SGLT2i (± others): 3.21 (1 .1 8, 5.24) GLP-1 RA (± others): 1 .87 (-1 .42, 5.1 6)
Model R^2	0.044	0.21 8	0.236
F-statistic (p)	85.6 (<0.001)	72.3 (<0.001)	58.9 (<0.001)

Model 1: crude association. Model 2: adjusted for age, sex, BMI, duration of diabetes and hypertension. The coefficient is slightly enhanced. In Model 3, HbA1 c remained an independent predictor (β = −2.31) when medication class was included. SGLT2 inhibitors improved eGFR (+3.21 mL/min/1.73 m^2), indicating renal protection beyond glycaemic management. Conversely, insulin-based regimens decrease eGFR, presumably reflecting disease severity. GLP-1 RA trends are flat.

Sensitivity Analyses: Excluding eGFR < 30 mL/min/1.73 m^2 (n = 34) did not significantly modify the adjusted β (-2.28). The effect was more pronounced in duration of diabetes ≥5 years (n = 1,421) (β = −2.56). The correlation was comparable for the MDRD equation for eGFR (r = -0.20). The results are therefore resilient to alternative definitions and exclusions.

DISCUSSION

In this large retrospective, hospital-based study in central Kerala, HbA1c levels of T2DM patients were independently associated with eGFR independent of age, sex, obesity, duration of diabetes, hypertension or medication class. The clinically relevant but modest loss of ~2.3 mL/min/1.73 m^2 per 1% increase in HbA1c could expedite CKD progression over many years. These results are consistent with the literature yet provide important insights for South Asia.

Comparison with Indian Studies. Our correlation coefficient (Pearson r = 0.21) is comparable overall to a few recent Indian studies. A significant negative correlation between HbA1c and eGFR (r = -0.32) was reported by Waheed et al,[8] in a North Indian cohort of 100 T2DM patients. Evidence from eastern India is relevant too. Roy and Bhattacharjee analysed clinic records of 241 patients and showed decreased eGFR in 20.73% of the study population, while only 31.12% of the study population achieved the study-defined target HbA1c. HbA1c and eGFR differed significantly depending on the glycaemic and renal status in the four groups. Their analysis was not a simple correlation study, but it highlights the usefulness of the joint assessment of glycaemic control and renal function in routine clinical records.[23] However, these studies were limited by a lack of subgroup analysis, a single-centre design or a small sample size. The multi-centre design with stratified analyses and robust adjustment allows a better understanding of the effect modification in the Indian population in comparison to other studies.

Comparison with International Cohorts: The strength of the correlation between HbA1c and eGFR in our study is somewhat weaker than that reported in a few landmark international trials. For example, the UKPDS demonstrated a strong linear relationship between glycaemic exposure and microvascular complications, with a 1 % reduction in HbA1c resulting in a substantial reduction in the risk of progression of nephropathy.[12] The ACCORD and ADVANCE trials also supported the renal benefits of intensive glycaemic control, but the effect sizes were dependent on baseline renal function and age.[13] Correlation coefficients from observational investigations in European and North American populations have been reported to be in the range of r = -0.30 to r = -0.40.[24] The attenuated correlation observed in our cohort might be due to several factors inherent to the South Asian T2DM phenotype: early age of onset of disease, higher prevalence of concomitant hypertension, genetic predisposition altering the renal response to hyperglycemia and the real-world nature of our data consisting of patients with advanced complications and heterogeneous treatment patterns.[25,26] Additionally, the use of concurrent rather than longitudinal measurements may underestimate the true association, as cumulative glycaemic exposure over time is likely to be a stronger predictor of renal decline than a single HbA1c value.[27]

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Effect Modification by Age and Baseline Renal Function: An interesting finding of our study was the substantial increase in the correlation between HbA1c and eGFR in elderly patients (> 65 years; r = -0.35) and in patients with pre-existing renal impairment (eGFR < 60 mL/min/1.73 m2; r = -0.42). This supports the "renal reserve" and "two-hit" models of DKD: older age and decreased nephron mass predispose to the nephrotoxic consequences of hyperglycemia as the remaining functional glomeruli are under increased metabolic stress [28,29]. This reflects the observation that glycaemic control is more strongly associated with outcomes in T2DM patients with CKD than those with normal renal function.[30] In the clinical setting, lower glycaemic goals and more frequent monitoring of renal function may be beneficial in these groups, but the risk of hypoglycaemia should be taken into consideration, particularly in the elderly with limited life expectancy or comorbid conditions.[31]

Hypertension and Drug Effects: The negative correlation was accentuated by hypertension (r = -0.27), implying a synergistic effect of diabetes and hypertension on renal function. The pathophysiological view is that these disorders produce glomerular hypertension, endothelial dysfunction, and proteinuria, which progress to DKD.[32] The UKPDS demonstrated that tight blood pressure control significantly reduced microvascular complications independently of blood glucose reduction, and current guidelines stress the need for dual risk factor treatment.[24]

Noteworthy is the attenuation and non-significance of the HbA1c–eGFR correlation among SGLT2 inhibitor-treated patients ($r \approx -0.11$, $p = 0.12$). This finding is in line with the renoprotective effects seen in large cardiovascular-kidney outcome clinical trials. The CREDENCE trial (canagliflozin) and DAPA-CKD trial (dapagliflozin) demonstrated that SGLT2 inhibitors markedly decreased the risk of kidney failure and improved eGFR trajectories in patients with T2DM and CKD, independent of glycaemic control.[33,34] In our cohort, SGLT2 inhibitor use was independently associated with a 3.21 mL/min/1.73 m^2 higher eGFR, supporting the concept that these agents offer direct renal benefits that may partially uncouple the relationship between glycemia and renal function. This finding has significant clinical implications as it suggests that early use of SGLT2 inhibitors as per current Indian and international guidelines may prevent the renal sequelae of poor glycaemic control among T2DM patients [35]. Our data are observational and cannot be causally inferred, and selection bias (SGLT2 inhibitors may be preferred in healthier individuals) may partly explain the protective effect.

Clinical Implications for the Indian Healthcare Setting: The present study has various practical implications for clinical practice in Kerala and South Asia. The high prevalence of renal impairment (16.3% with eGFR<60) and poor glycaemic control (8.2% mean HbA1c) suggests a need for more active screening and therapy of hyperglycaemia and CKD in routine diabetes care. The Indian Diabetes Consensus recommends annual testing of eGFR and urine albumin-creatinine ratio in all T2DM patients, but compliance is low, especially in primary and secondary care settings.[36] Our results underscore the impact of monitoring eGFR at each follow-up visit for diabetes, particularly in the elderly and those with long-standing diabetes. Secondly, the strong effect modification by age and baseline renal function supports individualising glycaemic targets. According to the American Diabetes Association and Indian guidelines, stricter control (HbA1c < 7.0%) may be beneficial in younger patients with preserved renal function, whereas older patients and those with established CKD might benefit from less stringent targets (HbA1c < 8.0%) to avoid hypoglycaemia.[36,37]

Additional therapeutic options are available for high-risk patients with the availability of SGLT2 inhibitors and GLP-1 receptor agonists with cardiovascular and renal benefits beyond glucose lowering. However, cost, access and prescriber awareness remain significant barriers to their extensive use in India, especially in non-metropolitan areas.[38] Third, the epidemics of diabetes and CKD are converging in India as demonstrated by the ICMR-INDIAB study, which requires a multidisciplinary approach by physicians, nephrologists, dietitians, and primary care providers.[19,20] Public health initiatives should concentrate on raising awareness of the importance of regular renal function testing, promoting lifestyle modifications (e.g., dietary sodium restriction, exercise) and ensuring equal access to newer renoprotective drugs. The successful model of community-based screening in Kerala for detection of non-communicable diseases could be adapted for early detection of T2DM patients at high risk for CKD in the disease trajectory.[39]

Limitations: The following are the limitations of the present study. First, the retrospective design of the study limits causal inference and is more prone to selection bias, information bias and residual confounding. Second, data on urine albumin-to-creatinine ratio are not available, so we cannot assess the contribution of proteinuria, a major indicator of DKD progression. Third, information on blood pressure control, socioeconomic status, dietary patterns, or genetic factors that might affect HbA1c and eGFR was unavailable. Fourth, one measurement of HbA1c may not represent long-term variability of blood glucose, which has been implicated in the development of diabetic complications. Fifth, the CKD-EPI equation is validated to estimate eGFR, but may be less accurate with higher renal function, biasing the correlation toward the null. Finally, the study cohort was recruited from tertiary care facilities, which may exaggerate people with more severe disease

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and limit generalisability to community-dwelling T2DM patients.

Future Research Directions: Creating causation and intervention windows needs long-term prospective studies. Collection of albuminuria, blood pressure, medication compliance, and lifestyle data should be warranted along with monitoring of eGFR and HbA1c. India needs renal investigations of glucose-lowering medicines, especially SGLT2 inhibitors and GLP-1 receptor agonists. Genetic markers for DKD susceptibility may improve risk assessment and drug targeting. Further implementation research is needed to make these clinical care models sustainable in the Indian health system, especially in resource-poor locations.

CONCLUSION

Inverse and independent correlation of eGFR and HbA1c with T2DM patients in Central Kerala hospitals were observed. The strongest association was with age, long-term diabetes and baseline renal impairment. This association appears to be attenuated by SGLT2 inhibitors, suggesting renal protection. Real-world studies have shown that intensive glycaemic management, proactive renal function monitoring and early use of renoprotective medicines can halt the progression of diabetic kidney disease in this high-risk location.

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