Journal of Chemical Health Risks

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JCHR (2025) 15(6), 53-59 | ISSN:2251-6727

Emerging Biomarkers in Diabetic Kidney Disease: The Role of Serum Renalase and Fatty Acid Binding Proteins in T2DM Patients

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(Received: 27 September 2025 Revised: 05 October 2025 Accepted: 01 November 2025)

KEYWORDS

Serum Renalase, Fatty Acid Binding Protein, Type 2 Diabetes Mellitus, Diabetic Kidney Disease

ABSTRACT:

Background: Diabetic kidney disease (DKD) remains a major cause of end-stage renal disease, with current diagnostic methods often failing to detect damage in its early stages. This has led to an increased focus on novel biomarkers that can provide a more comprehensive view of the disease's progression. Renalase, a circulating enzyme primarily secreted by the kidneys, functions to metabolize catecholamines and possesses anti-inflammatory and anti-fibrotic properties. While its levels rise in DKD as a compensatory, protective response to systemic and renal stress, this elevation negatively correlates with kidney function, making it a potential biomarker for monitoring disease severity. The FABP family, particularly liver-type FABP (L-FABP) and adipocyte-type FABP (A-FABP), plays a crucial role in lipid metabolism. In DKD, lipid overload in the renal tubules leads to cellular injury and increased expression of L-FABP. The release of L-FABP into the urine serves as a highly sensitive indicator of early tubular damage, often preceding the onset of microalbuminuria. Similarly, elevated serum A-FABP levels are linked to increased inflammation and a higher risk of renal function decline. This paper examines the significance of two such protein families, renalase and fatty acid-binding proteins (FABPs), which are significantly increased in DKD

Methods: About 60 study participants were selected from the General Medicine OPD based on the age and gender matched depending on the on the inclusion and exclusion criteria. All the biochemical parameters were analysed by using biochemistry auto analyser and FABP-1and Renalase by sandwitch ELISA method. Statistical Analysis: Data analysis involved descriptive statistics, analysis of variance (ANOVA), Tukey HSD post hoc tests. We employed pearman's correlation to examine relationships between relevant variables. The p value significant at <0.05.Finally, receiver operating characteristic (ROC) analysis was performed to evaluate the diagnostic performance of Renalase and FABP.

Results: Serum RNLS levels and FABP levels were significantly higher in DKD patients compared to T2DM and controls (0.042, 0.77 & 0.65), $p < 0.01$). Receiver operating characteristic curve analysis demonstrated the diagnostic value of RNLS and FABP in DKD is (1 (95% confidence interval (CI) = 0.70–0.82, $p < 0.01$)) and 0.77 (95% CI = 0.66–0.84, $p < 0.01$) respectively

Conclusion: The increased levels of renalase and FABPs in DKD highlight the complex interplay

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JCHR (2025) 15(6), 53-59 | ISSN:2251-6727

of compensatory and injurious mechanisms in the disease's pathogenesis. These proteins offer promising avenues for more effective early diagnosis and prognosis by providing insights into both systemic responses and specific cellular damage within the kidney.

1. Introduction

Diabetic kidney disease (DKD) is a severe complication of diabetes mellitus and a leading cause of end-stage renal disease (ESRD). Its progression is often linked to both glomerular and tubulointerstitial damage in the kidneys.(1) Traditional markers like albuminuria have limitations, leading to a search for new biomarkers that can provide earlier and more precise indicators of renal damage. In this context, two notable protein families, renalase and fatty acid-binding proteins (FABPs), have been found to be significantly increased in DKD and are being investigated for their roles in its pathogenesis and as potential diagnostic markers(2)

Renalase in Diabetic Kidney Disease

Renalase is a recently discovered flavoprotein, an enzyme primarily produced by the kidney. Its main function is to metabolize and degrade circulating catecholamines, such as adrenaline and dopamine (3). This role is significant because high levels of catecholamines are associated with increased sympathetic activity, hypertension, and cardiovascular complications, all of which are common in patients with chronic kidney disease (CKD).

In the context of DKD, research has revealed a complex and seemingly paradoxical relationship with renalase. While renalase is generally seen as having a protective, anti-inflammatory, and anti-fibrotic effect, its serum levels are found to be significantly elevated in patients with DKD and other forms of CKD(4). This increase appears to be a compensatory mechanism. As kidney function declines, the body attempts to upregulate renalase production to counteract the rising levels of catecholamines and other stressors.

Studies in animal models of diabetes have shown that a deficiency in renalase exacerbates renal injury, while overexpression of the protein can ameliorate DKD symptoms, including a reduction in albuminuria and mesangial expansion(5). This suggests that renalase, despite being a marker of disease progression when elevated, is actually attempting to protect the kidney from further damage. The elevation of circulating renalase in DKD patients is therefore considered a response to the underlying pathology, rather than a direct cause of the disease. The degree of this elevation often correlates negatively with the estimated glomerular filtration rate (eGFR) and positively with the severity of the disease, making it a potential biomarker for monitoring DKD progression(6).

Fatty Acid-Binding Proteins (FABPs) in Diabetic Kidney Disease

Fatty acid-binding proteins are a family of small intracellular proteins that play a crucial role in lipid metabolism, transporting fatty acids to different cellular compartments.(7) Specific isoforms of FABPs, particularly liver-type FABP (L-FABP, also known as FABP1) and adipocyte-type FABP (A-FABP, also known as FABP4), are of particular interest in DKD.

The kidneys have a high metabolic demand and rely heavily on fatty acid oxidation for energy. In a healthy state, L-FABP is expressed in the proximal tubules of the kidney, where it assists in the reabsorption and metabolism of fatty acids.(8) However, in diabetic conditions, the kidney is subjected to a state of lipid overload due to hyperglycemia and dyslipidemia. This overload leads to increased uptake of free fatty acids by the renal tubules, which in turn causes cellular stress and damage.(9)

In response to this stress, the expression of L-FABP in the renal tubules is upregulated. When the tubule cells are injured, L-FABP is released into the urine(10). Consequently, increased urinary L-FABP levels are a strong indicator of early renal tubular injury, often appearing before the onset of microalbuminuria, the traditional first-line marker for DKD. This makes urinary L-FABP a highly sensitive biomarker for detecting DKD at its earliest stages and for predicting its progression (11).

Similarly, serum levels A-FABP, which is primarily produced by adipocytes, are also found to be elevated in patients with type 2 diabetes and DKD(12). High A-

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JCHR (2025) 15(6), 53-59 | ISSN:2251-6727

FABP levels are associated with increased insulin resistance, inflammation, and a higher risk of rapid renal function decline (13). Both L-FABP and A-FABP thus serve as valuable biomarkers reflecting different aspects of the metabolic and inflammatory stress on the kidney in a diabetic context. Their measurement, particularly urinary L-FABP, offers a more direct assessment of tubulointerstitial damage, complementing the information provided by albuminuria which primarily reflects glomerular injury(14).

In summary, the significant increase in both renalase and FABPs in DKD highlights the multifaceted nature of the disease. Renalase levels rise as a compensatory, protective response to systemic and renal stress, while FABPs, especially L-FABP, reflect the direct cellular injury and metabolic overload occurring in the kidney. Together, these proteins offer a more comprehensive view of DKD pathology and hold promise as novel biomarkers for early diagnosis, prognosis, and therapeutic monitoring.

2. MATERIALS AND METHODOLOGY

This study was conducted at Mahatma Gandhi Medical College and Research Institute (MGMCRI), in the Department of Biochemistry in collaboration with the Department of General Medicine, under the jurisdiction of Sri Balaji Vidyapeeth, Puducherry. This study was approved by the Institutional Research Council (IRC) and Institutional of Human Ethical Committee (IHEC).

Inclusion criteria

T2DM with and without Nephropathy patients with confirmation of urinary dipstick positive for albumin subjects with age of 35-65 years irrespective of gender will be taken from General medicine OPD, followed obtaining written informed consent from the study participants. 4ml of venous blood will be collected under aseptic precautions and medical supervision.

Exclusion criteria

People with any acute and chronic illness (except T2DM and Nephropathy), gastrointestinal disorders, pregnancy, urinary tract infection and dyslipidemia

Biochemical assessments

All the estimations were enabled by established methods/procedures duly approved by the International Federation of Clinical Chemistry and laboratory Medicine (IFCC). The Internal quality Control was maintained through samples provided by M/sBiorad USA. External Quality Assessment, was facilitated through the Clinical Biochemistry laboratory of Christian Medical College (CMC), Vellore which has been accredited by the National Accreditation Board for Testing and Calibration Laboratories (NABL). Venous blood was collected from the subjects. Insulin, HOMA-IR (a measure of insulin resistance), lipid profile, glycated haemoglobin were estimated, based on glucose oxidase-peroxidase method Fasting insulin was enabled by automated chemiluminescence. Glycated hemoglobin (HbAIC) was quantitated by HPLC method. Fasting insulin (venous plasma) levels was determined by automated electro Chemiluminescence. The insulin resistance index was assessed by the homeostatic model assessment of Insulin resistance (HOMA-IR) and computed using the formula: Fasting insulin (mU/L) x fasting glucose (mmol/L)/22.5. Triacylglycerols (TAG) n serum was measured by glycerol kinase method. Total cholesterol was quantitated by the enzymatic method. HDL cholesterol was measured by polyanion precipitation. LDLcholesterol was computed using Friedwald equation i.e., LDL cholesterol= Total cholesterol-(HDL cholesterol + VLDL) where VLDL = TAG/5. Serum Renalase and FABP-1 were estimated by ELISA kit method.

STATISTICAL ANALYSIS

MS Excel was used to record the data set. JASP-16.0 was used for statistical analysis namely, percentages that was computed for categorical variables. Mean and standard deviation was calculated for numerical

variables. Pearson's correlation was analysed among the study groups. Receiver operating characteristic (ROC) curves was enabled to determine area under curve (AUC) for assessing the utility of a biochemical predictor and also a means of comparing two or more predictive models.

3. Results

In this study we observed higher levels of serum Renalase and serum L-FABP was associated with increased risk of renal function decline in patients with

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JCHR (2025) 15(6), 53-59 | ISSN:2251-6727

T2DM and renal dysfunction from the following statistical tools.

Table 1: Karl Pearson's correlation coefficient of Renalase with other parameters(r value) and p value

	Control		T2DM		DKD	
	r value	p value	r value	p value	r value	p value
Insulin	-0.12	0.5391	-0.11	0.5496	0.05	0.7862
FBS	0.29	0.1260	0.32	0.0445*	0.17	0.3670
HbA1C	0.01	0.9640	0.14	0.04571*	0.04	0.0429*
S.Creat	-0.02	0.9293	-0.41	0.1192	-0.29	0.0255*
Egfr mL	-0.17	0.3643	0.18	0.3530	0.36	0.0474*
CHOLES	0.01	0.9605	0.16	0.3888	0.17	0.3648
TG	-0.19	0.3189	0.04	0.8491	0.02	0.9098
HDL	-0.31	0.0997	0.02	0.9298	0.21	0.2664
TG/HDL	-0.07	0.7210	0.04	0.8451	-0.08	0.6727
ALB	-0.21	0.2639	0.17	0.3773	0.09	0.6341
Cret/alb	-0.17	0.3774	-0.44	0.0665	-0.34	0.0147*
FABP-1	0.08	0.6586	0.05	0.7727	-0.15	0.0423*

T2DM- type 2 diabetes mellitus, DKD-Diabetic Kidney Disease, DFU- Diabetic Foot ulcer, FBS- Fasting Blood Glucose, TGL- Triglycerides, HDL- High density Lipoproteins, Alb- Albumin , FABP- Fatty acid binding proteins
*p value significant at 0.05
Table 1 represents the significant correlation of renalase with FBS and HbA1C in T2DM patients, renalase with HbA1C, S.creatinine, eGFR, Creat/Alb ratio and FABP-1 in DKD patients, Renalase with FBS and Cholesterol in DFU patients

Fig 1: ROC curve analysis between RNLS and FABP in T2DM patients

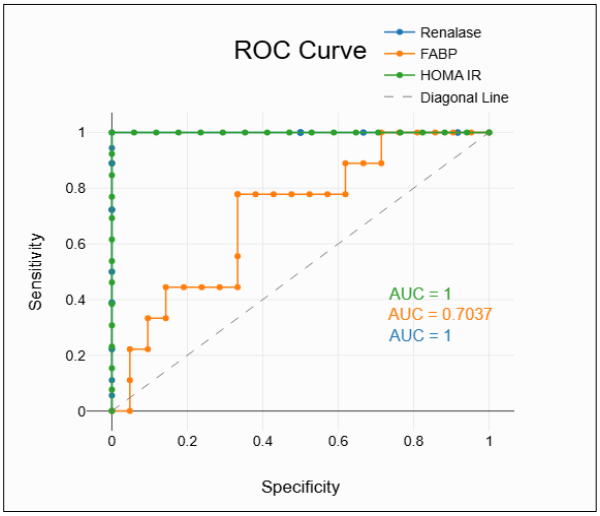

Fig.1 represents ROC of RNLS and FABP with HOMA –IR shows cut off, sensitivity and specificity with area under curve values for group 1 T2DM as, 1 and 0.70

Fig 2: ROC curve analysis between RNLS and FABP in DKD patients

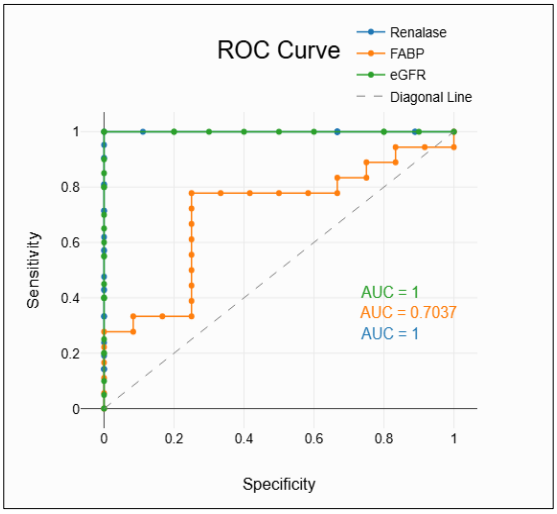

Fig.2: Represents ROC of RNLS and FABP with e GFR shows cut off, sensitivity and specificity with area under curve values for group 1 DKD as, 1 and 0.70

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4. Discussion

Diabetes mellitus is a metabolic disorder characterized by chronic hyperglycemia and hyperlipidemia that predisposes affected individuals go long term micro and macro vascular complications[15]. The overall risk of dying among people with diabetes is at least double the risk of their peers without diabetes

The traditional approach to assessing kidney function in patients with Type 2 Diabetes Mellitus (T2DM) has long centered on serum creatinine (SCr) and estimated glomerular filtration rate (eGFR).(16) While these markers are foundational, their well-documented limitations in the context of diabetic kidney disease (DKD) highlight the critical need for more specific and sensitive biomarkers. The emergence of serum renalase (RNLS) presents a significant paradigm shift, offering a more precise tool for the early detection and management of DKD. Current treatments for DKD offer limited success in preventing the progression to kidney failure.[17]

Renalase, a FAD-dependent oxidase primarily secreted by the kidney, has dual enzymatic and cytoprotective roles—possibly regulating catecholamine metabolism and cellular survival via receptor-mediated pathways (18,19,20,21) Also seen in the heart, skeletal muscles, and small intestine. RNLS is the name of the gene that produces this protein (otherwise called C10 or f59 or FLJ11218). It is likely to be interested in controlling blood pressure and cardiovascular function. Renalase has four distinct isoforms (hRenalase1 to hRenalase4), although only hRenalase1 is primarily found in human blood (22)

Clinically, serum renalase levels are significantly elevated in DKD patients compared to those with type 2 diabetes without complications (median ~34.8 vs. ~30.5, $p < 0.01$).(23) Elevated renalase correlates inversely with glomerular filtration rate (eGFR) and positively with albuminuria and carotid intima-media thickness, reinforcing its potential as both a severity marker and vascular risk indicator.(24)

Beyond DKD, a longitudinal study in pre-dialysis CKD (predominantly non-DKD patients) found higher circulating renalase independently predicted renal disease progression, hospital admissions, and all-cause mortality, underscoring its prognostic value.(25) These findings collectively suggest that renalase elevation likely reflects a compensatory response to nephron stress and sympathetic over activity—a physiological adaptation that becomes a marker of declining renal function.

However, caution is warranted. The elevated renalase could signal worsening pathology rather than protection. Its mechanisms—whether via catecholamine clearance or activation of anti-apoptotic pathways (PI3K/MAPK)—remain an area of active research (26)

Liver-type FABP (L-FABP) is abundantly expressed in proximal tubular cells and is upregulated in response to ischemic or oxidative stress. L-FABP (L-FABP) thus reflects early proximal tubular injury.(27) Multiple cross-sectional studies show significantly elevated uL-FABP in patients with diabetes—even during normoalbuminuria—compared to healthy controls, indicating its performance as an early biomarker.(28)

Longitudinal evidence strengthens its prognostic validity. Reviews pooling 21 studies (including some longitudinal arms) show baseline L-FABP predicts DKD onset and progression independently of albuminuria or eGFR in both type 1 and type 2 diabetes.(26) In a large Japanese cohort, L-FABP predicted significant eGFR decline (≥30%) even among albuminuric patients after adjustments.(29)

Meta-analyses and population data confirm that L-FABP correlates positively with albumin-to-creatinine ratio (ACR) and systolic blood pressure, and negatively with eGFR across diabetic groups. Its relationship with cardiovascular risk is also emerging: in CKD cohorts, higher urinary L-FABP independently predicted cardiovascular events and ESRD progression (30)

Our findings elegantly reflect a dual-pathway model: renalase captures systemic and hemodynamic stress responses (catecholaminergic load, inflammation, vascular burden), while L-FABP provides a window into localized tubular injury—often preceding overt glomerular dysfunction. Together, they offer a more nuanced, multilayered biomarker strategy than albuminuria or eGFR alone.

This aligns with the renal tubular hypothesis, which posits that early DKD involves metabolic overwork, oxidative stress, and inflammation at the tubular level—even before glomerular damage becomes apparent (23). FABP is a known marker for renal dysfunction, from our findings we conclude that that both RNLS and FABP can be used as a early diagnostic marker for the diabetic kidney disease.

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JCHR (2025) 15(6), 53-59 | ISSN:2251-6727

Clinical Implications & Future Directions

Early Diagnosis: Renalase and L-FABP may facilitate identification of DKD at earlier stages—even before albuminuria—supporting timely intervention. Risk Stratification: Elevated levels of these biomarkers could help categorize patients at higher risk of rapid progression or cardiovascular complications, guiding individualized therapy. Monitoring & Treatment Response: Serial tracking may reflect response to renoprotective interventions (e.g., RAS inhibitors, SGLT2 inhibitors), though this requires further validation. Research Horizons: Prospective, multicenter trials should validate cutoffs, assess additive value over conventional markers, and explore whether biomarker-guided therapy improves outcomes. Exploring renalase's therapeutic potential—as agonist or antagonist—also merits attention

Limitations of the study

Many studies are retrospective or cross-sectional with small cohorts (e.g., 233 patients in renalase study) Heterogeneity in L-FABP assay methods and cutoff thresholds may affect comparability Longitudinal data linking these biomarkers to hard clinical endpoints in DKD are still limited. Therefore, larger, well-controlled, and standardized studies will help translate these findings into clinical practice.

Acknowledgment

Authors thank Prof. Seethesh Ghose, Dean, Mahatma Gandhi Medical College and Research Institute, a constituent teaching unit of Sri Balaji Vidyapeeth, Dr. K. S. Reddy, Dean Research, Sri Balaji Vidyapeeth and Dr. Nihar Ranjan Biswas, Vice-Chancellor, Sri Balaji Vidyapeeth for their support and patronage in enabling this research communication

Conflict of Interest

Authors declare there is no conflict of Interest

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