

A STUDY OF LEVELS OF hs-CRP AND LIPID PROFILE IN PATIENTS WITH DIABETIC NEPHROPATHY

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Abstract

BACKGROUND: Diabetes mellitus (DM), which has a high morbidity and mortality rate, has emerged as a significant worldwide health issue. One of the most significant long-term microvascular consequences of diabetes mellitus (DM) is type 2 diabetic nephropathy (T2DN), which is a major contributor to end-stage renal disease (ESRD) globally. There is data indicating that T2DM is a mild inflammatory condition. Inflammatory markers including high-sensitivity C-reactive protein (hs-CRP) have been linked to an increased risk of diabetes, and its role in diabetic nephropathy (DN) pathogenesis is yet unknown. **AIM:** This study aimed to measure the levels of serum high sensitivity C-reactive protein (hs-CRP) and lipid profile in patients with diabetic nephropathy and to compare with that of normal subjects. **MATERIAL AND METHODS:** In this study, T2DM patients with nephropathy were enrolled in the study group (n=70) and healthy subjects were enrolled in the control group (n=70). Serum hs-CRP levels and lipid profile were assessed in both the groups. The data was analysed using SPSS version 26. **RESULTS:** When compared to controls, patients with DN had significantly higher levels of hs-CRP as compared to that of controls. The mean value total cholesterol, LDL, and triglycerides is also significantly more in patients with diabetic nephropathy as compared to controls with p-value <0.001. **CONCLUSION:** According to our findings, patients with diabetic nephropathy frequently experience an increase in hs-CRP levels along with dyslipidemia. Regular monitoring of these markers may help to reduce the incidence of unfavourable outcomes in patients with diabetic nephropathy.

Keywords: Diabetic Nephropathy, hs-CRP, Lipid Profile.

INTRODUCTION

Diabetic nephropathy [DN] is amongst the most significant medical concerns that affects people with diabetes. It currently constitutes as the primary cause of cardiovascular morbidity and mortality as well as the primary cause of end-stage renal disease in patients with diabetes [1]. DN affects 20–40% of T2DM patients [2], mostly because Type 2 diabetes is becoming more common and is linked to obesity [3,4]. Earlier Diabetic Nephropathy was not thought of as an inflammatory illness. However, according to current research, the development and progression of DN are aided by kidney inflammation. The established metabolic, biochemical, and hemodynamic abnormalities in the diabetic kidney may be a major component that activates inflammation [5].

According to studies, those who develop diabetic nephropathy have low-grade inflammation for years before the disease manifests itself [6]. Numerous human investigations have confirmed similar results, and numerous cross-sectional studies have shown an association between diabetic nephropathy and elevated levels of inflammatory markers such IL-6, fibrinogen, or hs-CRP [7].

A highly sensitive indicator of inflammation is an acute phase reactant known as C-reactive protein (CRP). When there is inflammation, its level drastically increases [8]. It is considered as the best markers of vascular inflammation because of its extended half-life, low cost of estimation, and level stability without diurnal change [9]. Disorders including diabetes mellitus (DM), cardiovascular disease, metabolic syndrome, renal failure, etc. are linked to CRP [10,11,12]. A highly sensitive variant of CRP is called high-sensitivity CRP (hs-CRP). It is detected using highly sensitive assays, which are capable of detecting

CRP levels with a sensitivity range of 0.01 mg/L to 10 mg/L. In the absence of obvious inflammation, these assays can therefore detect even low-grade inflammation [13].

Patients with Type 2 diabetes have greater serum levels of high sensitivity CRP (hs-CRP) than healthy individuals, and this factor is crucial to the onset and progression of T2DM [9]. It has also been demonstrated that the amount of this inflammatory marker correlates with glycemic control markers such glycosylated haemoglobin A1c (HbA1c) [14]. However, the association of hs-CRP with development and progression of Diabetic nephropathy is yet not very clear.

Therefore, the present study was conducted in order to assess serum hs-CRP levels and lipid profile in patients with DN.

MATERIAL AND METHODS:

STUDY DESIGN:

This cross-sectional and observational study was carried out at Index Medical College and Hospital, Indore from January 2021 to January 2022. The ethical clearance was obtained from the Ethical Committee of Index Medical College and Hospital, Malwanchal University, Indore.

INCLUSION CRITERIA:[15,16,17]

The study group included 70 diagnosed cases of T2DM (FPG > 126 mg/dl and HbA1c > 6.5%) with nephropathy who were between the ages of 30 and 70. The urine albumin/creatinine ratio was used to compute the UACR. ACR 30 mg/g and/or eGFR 60 mL/min/1.73 m² were used to define DN.

Age as well as sex-matched healthy subjects who attended the OPD and IPD of the Medicine Department of Index Medical College and Hospital were enrolled as the control group.

EXCLUSION CRITERIA: [16,17]

The study excluded participants who had a history of active infections, trauma, malignancy, smoking, alcoholism, cancer, rheumatoid arthritis, and those taking any anti-inflammatory medications or who had a body mass index (BMI) >30. The study excluded patients with type 1 diabetes, gestational diabetes mellitus, diabetic ketoacidosis, smoking, alcoholism, chronic illnesses, and those receiving treatment with lipid-lowering medications, anti-inflammatory drugs, multivitamins, and aspirin.

All eligible subjects who took part in the study were informed of its objectives. Informed consent was taken from all the participants. Physical examination along with previous history taking was done for all patients. All anthropometric measurements, including waist circumference, body mass index, height and weight were obtained. To conduct several biochemical investigations, blood samples were collected.

SAMPLE COLLECTION:

After fasting of eight to twelve hours, 7 ml of venous blood was drawn from the antecubital vein. 4 ml of blood was placed into a plain vacutainer and, 1.5ml was transferred into the fluoride vial whereas last 1.5 ml was transferred into an EDTA vacutainer. Centrifugation was used to separate the sera from the blood samples.

The GOD-POD method was used to measure plasma glucose levels[18]; the High Performance Liquid Chromatography technique was used to calculate HbA1c[19]; the cholesterol oxidase peroxidase method was used to measure serum cholesterol[20]; the Glycerol oxidase-Trinder method was used to measure serum TGs[21]; and the modified polyethylene glycol precipitation method was used to measure high-density lipoprotein (HDL) levels [22]. To calculate LDL, the Fridelwald equation was used [23]. Estimation of serum creatinine was done by the modified Jaffe's method [24] and urea was estimated by Urease Berthelot's method [25] by using Cobas Integra (Roche) fully automated analyser. Urine ACR was measured from random urine sample. Urinary albumin was measured by 'Immuno Turbidometry method' while urinary creatinine was measured by 'Jaffe's spectrophotometric method'[26]eGFR was calculated using Cockcroft and Gault equation[27]. Immunoturbidometric analysis of serum hs-CRP was performed using a COBAS-501 fully automated analyzer. (28,29).

RESULTS

STATISTICAL ANALYSIS

The data was arranged in tabular form and SPSS software 26.0 was used to analyse the data . According to age, gender, and BMI, descriptive statistics (frequency and percentage) were obtained for the distribution of diabetic nephropathy cases and controls.

The mean values of constant variables were compared between cases and controls using the independent-student t test.

Interpretation of results:

p-values below 0.05 were considered significant, while those below 0.01 were considered very significant.

TABLE 1: DISTRIBUTION OF PATIENTS WITH DIABETIC NEPHROPATHY ACCORDING TO AGE AND GENDER

AGE GROUP(YEARS)	NUMBER OF CASES		TOTAL
	(N=70)		
	MALES	FEMALES	
30-40	13	8	21
41-50	14	7	21
51-60	17	11	28

The distribution of patients with diabetic nephropathy according to gender and age is shown in Table 1. In the age group of 31 to 40, there were 21 patients (13 men and 8 women), 41 to 50, there were 21 patients (14 men and 7 women), and 51 to 60, there were 28 patients (17 men and 11 women), for a total of 70 patients (44 males and 26 females).

TABLE 2: DISTRIBUTION OF HEALTHY CONTROLS ACCORDING TO AGE AND GENDER

AGE GROUP(YEARS)	NUMBER OF SUBJECTS	TOTAL
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	(N=70)		
	MALES	FEMALES	
30-40	8	8	16
41-50	14	13	27
51-60	16	11	27

The age and gender distribution of the control group is shown in Table 2. Between the ages of 31 and 40, there were 16 healthy subjects (8 men and 8 women), between the ages of 41 and 50, there were 27, (14 men and 13 women), and between the ages of 51 and 60, there were 26, (16 men and 11 women), for a total of 70 healthy subjects (38 males , 32 females).

TABLE 3: DISTRIBUTION OF THE STUDY POPULATION ACCORDING TO BMI

BMI RANGE (kg/m ²)	DIABETIC NEPHROPATHY CASES		CONTROLS	
	N(70)	%	N(70)	%
Below 18	1	1.42%	5	7.14%
18-24.99	22	31.4%	41	58.57%
25-29.99	37	52.85%	21	30%
30 and above	10	14.28%	3	4.28%
Mean \pm SD	25.56 \pm 3.50		23.71 \pm 3.72	

According to the above mentioned table, 41 (58.57%) of the controls and 22 (31.4%) of the patients with diabetic nephropathy both had normal BMI.

37(52.85%) of the cases were found to be overweight (BMI 25–29.99), compared to 21(30%) controls who fell into this category.

10 (14.28%) of the diabetic nephropathic cases had a BMI of 30 or above, compared to just 3 (4.28 %) of the controls.

Table 4: COMPARISON OF BLOOD PRESSURE OF THE STUDY POPULATION

PARAMETERS	DIABETIC NEPHROPATHY CASES (Mean \pm SD)	CONTROLS (Mean \pm SD)	p-Value
Systolic BP(mm of Hg)	139.40 \pm 12.35	127.49 \pm 11.6	<0.001

Diastolic BP(mm of Hg)	89.89 ± 7.54	78.81 ± 7.68	<0.001
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In patients with diabetic nephropathy, the mean systolic blood pressure is 139.40 ± 12.35mm Hg, which is substantially higher than the mean systolic blood pressure of controls, which is 127.49 ± 11.6 mm Hg (p< 0.001).

Diastolic blood pressure in cases with diabetic nephropathy is higher than in controls, with mean values of 89.89 ± 7.54mm Hg and 78.81 ± 7.68 mm Hg, respectively.

Table 5: COMPARISON OF GLYCEMIC PARAMETERS BETWEEN PATIENTS WITH DIABETIC NEPHROPATHY AND HEALTHY CONTROLS

PARAMETERS	DIABETIC NEPHROPATHY CASES (Mean ± SD)	CONTROLS (Mean ± SD)	p-Value
FPG(mg/dl)	175.84± 44.71	83.77± 10.12	<0.001
PP(mg/dl)	254.29± 91.68	122.09 ± 12.25	<0.001
HbA1c (%)	9.09± 1.92	5.30 ± 0.61	<0.001

When compared to the controls with mean value of FPG 83.77± 10.12 mg/dl, patients with diabetic nephropathy had a considerably higher mean FPG of 175.84± 44.71mg/dl.

When compared to controls, who had mean PP glucose levels of 122.09 ± 12.25mg/dl, patients with diabetic nephropathy had mean PP glucose levels of 254.29± 91.68mg/dl.

In cases of diabetic nephropathy, the mean HbA1c value was 9.09± 1.92%, whereas in controls, it was 5.30 ± 0.61%. This difference was highly significant with a p value < 0.001.

TABLE 6: COMPARISON OF BLOOD UREA, SERUM CREATININE, ACR AND eGFR BETWEEN PATIENTS WITH DIABETIC NEPHROPATHY AND HEALTHY CONTROLS

PARAMETERS	DIABETIC NEPHROPATHY CASES (Mean ± SD)	CONTROLS (Mean ± SD)	p-Value
BLOOD UREA(mg/dl)	155.11±59.45	30.19±10.43	<0.001
SERUM CREATININE(mg/dl)	5.17±3.02	0.96±0.27	<0.001
UACR (mg/g)	725.90±577.89	18.20±5.24	<0.001

eGFR(ml/min/1.73m ²)	22.71±12.77	89.41±32.25	<0.001
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According to the above table, diabetic nephropathy cases had significantly higher blood urea, serum creatinine, and urine albumin-creatinine ratios (UACR) than controls.

When compared to controls, who had blood urea levels of 30.19 ± 10.43 mg/dl, patients with diabetic nephropathy had blood urea levels that were considerably higher, with a mean value of 155.11±59.45 mg/dl.

When compared to controls with mean value 0.96±0.27 mg/dl individuals with diabetic nephropathy had a significantly higher mean blood creatinine level 5.17±3.02 mg/dl

Cases with diabetic nephropathy had mean value of UACR 725.90 ± 577.89 mg/gm, as compared to controls with mean value 18.20±5.24 mg/dl , which is highly significant with a p value of less than 0.001.

In patients of diabetic nephropathy, the mean eGFR was 22.71±12.77 ml/min/1.73 m², which is substantially lower than the mean eGFR of controls, which was 89.41±32.25 ml/min/1.73 m² (p<0.001).

TABLE 7: COMPARISON OF LIPID PROFILE BETWEEN PATIENTS WITH DIABETIC NEPHROPATHY AND HEALTHY CONTROLS

PARAMETERS	DIABETIC NEPHROPATHY CASES (Mean ± SD)	CONTROLS (Mean ± SD)	p-Value
TOTAL CHOLESTEROL (mg/dl)	216.21±40.02	168.60±38.24	<.001
HDL (mg/dl)	37.03±8.85	47.8±9.91	0.058
LDL (mg/dl)	139.17±41.21	90.44±33.14	<.001
VLDL (mg/dl)	40.00±11.29	30.35±12.67	<.001
TG (mg/dl)	200.04±56.45	151.77±63.38	<.001

In patients of diabetic nephropathy, the mean serum cholesterol level was 216.21 ± 40.02mg/dl, whereas in controls, it was 168.60±38.24 mg/dl. The mean value of serum LDL in cases with diabetic nephropathy was 139.17 ± 41.21mg/dl, compared to 90.44±33.14 mg/dl in controls.

When compared to controls, who had a mean value of 30.35±12.67 mg/dl for serum VLDL, cases of diabetic nephropathy had a mean value of 40.00 ± 11.29 mg/dl, which was noticeably higher.

The results of TG likewise followed the same pattern, with a mean value of 151.77±63.38 mg/dl in controls and 200.04 ± 56.45 mg/dl in patients of diabetic nephropathy.

When compared to controls, who had mean value of HDL levels 47.8±9.91 mg/dl patients with diabetic nephropathy cases had a mean value of 37.03 ± 8.85 mg/dl.

TABLE 7: COMPARISON OF LIPID PROFILE BETWEEN PATIENTS WITH DIABETIC NEPHROPATHY AND HEALTHY CONTROLS

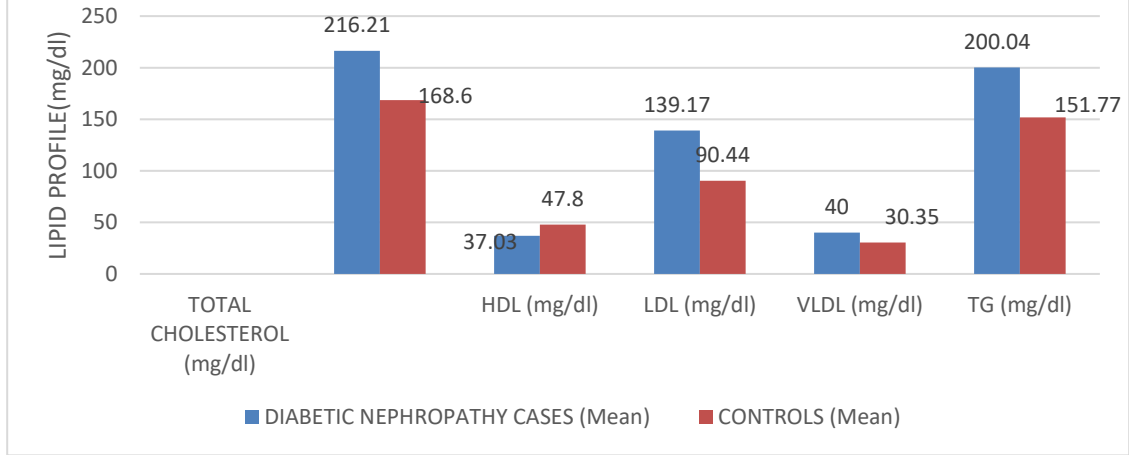
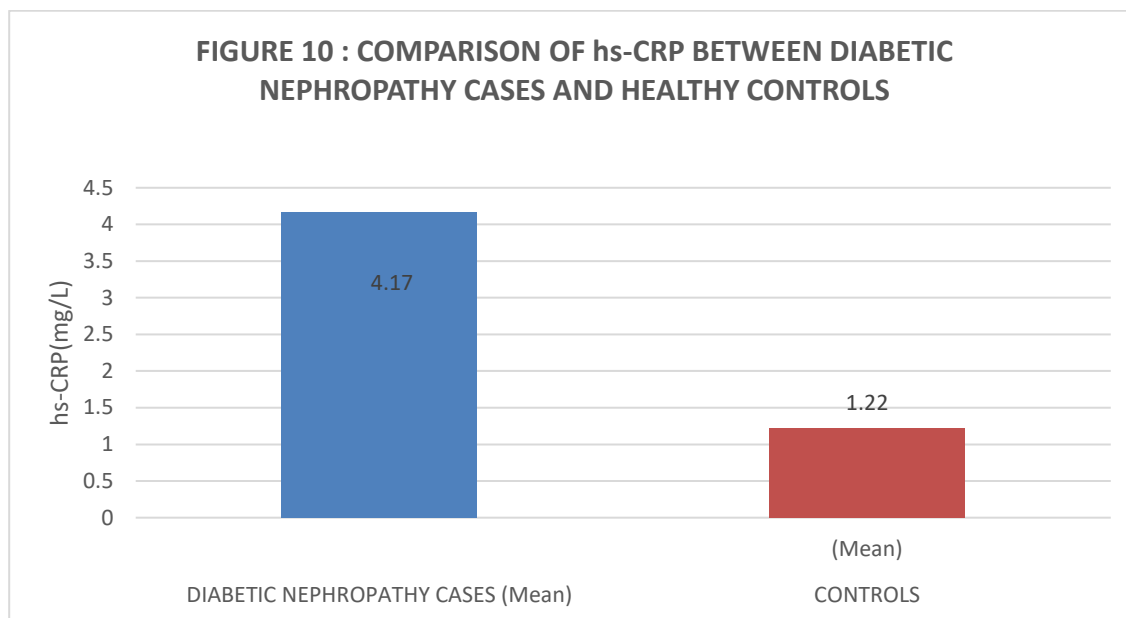


TABLE 7: COMPARISON OF hs-CRP BETWEEN PATIENTS WITH DIABETIC NEPHROPATHY AND HEALTHY CONTROLS

PARAMETERS	DIABETIC NEPHROPATHY CASES (Mean ± SD)	CONTROLS (Mean ± SD)	p-Value
hs-CRP(mg/L)	4.17±1.57	1.22±0.54	<0.001

In compared to controls, who had a mean value of hs-CRP 1.22±0.54 mg/L, patients with diabetic nephropathy had a mean value 4.17 ± 1.57 mg/L, which was highly significant (p < 0.001).



DISCUSSION:

Diabetic nephropathy (DN) and End-stage kidney disease (ESKD) are becoming more common among those with type 2 diabetes mellitus (DM)[30]. Growing data emphasises how important inflammation is to the development of DN. The influence of growth factors, proinflammatory cytokines including IL-1, IL-6, IL-18, and TNF[31-33], and other chemokines may have direct or indirect contributions to the pathogenesis of DN[34,35]. In both DN patients as well as experimental animal models of DN increased expression of these cytokines have been observed[36-38]. However, the cost of measuring these variables restricts their therapeutic use.

On the other hand, hs-CRP can conveniently and cheaply offer useful information regarding the status of inflammation [39]. It is susceptible to inflammation because of the multiple inflammatory stimuli that affect it [40]. Additionally, glycation, oxidation, and insulin resistance in type 2 diabetes mellitus are linked to obesity, hypertension, dyslipidemia, and altered levels of lipoproteins [41,42]. In patients with diabetic nephropathy, low-grade systemic inflammation and lipid profiles may be used to anticipate the development of cardiovascular disease[43].

In order to determine the association between serum hs-CRP & lipid profile in patients with diabetic nephropathy, the current study was conducted. In this work, we focused on the possibility that early detection of changes in these parameter and timely treatment could slow the development and progression of diabetic nephropathy and reduce the risk of consequences like cardiovascular disease.

In this present study, 70 participants as healthy controls & 70 participants diagnosed with diabetic nephropathy attending OPD & IPD of Index Medical College & Research centre were selected randomly.

DISTRIBUTION OF DN PATIENTS & CONTROLS ACCORDING TO GENDER AND AGE

The study population included participants who had been diagnosed with DN and were between the age group of 30 and 60 years. Number of patients in age group from 31 to 40 years, were 21 (13 men and 8 women), 41 to 50 years, were 21(14 men and 7 women), and 51 to 60 years were 28 (17 men and 11 women) patients, for a total of 70 patients (44 males and 26 females). For healthy controls between the age group of 31 and 40, there were 16 healthy individuals (8 men, 8 women), between the age group of 41 and 50, there were 27, (14 men, 13 women), and between the age group of 51 and 60, there were 27, (16 men, 11 women), for a total of 70 healthy subjects (38 males, 32 females).

DISTRIBUTION OF THE STUDY POPULATION ACCORDING TO BMI

According to table 3, 41 (58.57%) controls and 22 (31.4%) patients with diabetic nephropathy both had normal BMIs. 37 cases (52.85%) were found to be overweight (BMI 25–29.99), compared to 28 controls (40%) who fell into this category. 10 (14.28%)

of the diabetic nephropathic cases had a BMI of 30 or above, compared to just 1 (1.42%) of the controls.

Intraglomerular hypertension, which elevates renal blood flow and fractional urine albumin clearance, is a major contributor to obesity-related albuminuria[44-46]. The resulting mechanical stress damages a crucial cellular layer of the glomerular filtration barrier, causing glomerular hypertrophy and an increase in the distance between nearby podocytes. This could possibly lead to podocyte mortality with focal segmental glomerulosclerosis[47,48].

A study also showed that in patients with an eGFR of at least 60 mL/min per 1.73 m² and a BMI of 30 kg/m² or above, there is a rapid loss of renal function; this trend is increased in older patients [49].

COMPARISON OF BLOOD PRESSURE OF THE STUDY POPULATION

In patients with diabetic nephropathy, the mean systolic blood pressure is 137.91 ± 12.90 mm Hg, which is substantially higher than the mean systolic blood pressure of controls, which is 124.03 ± 10.08 mm Hg (p < 0.001). Diastolic blood pressure in cases with diabetic nephropathy is higher than in controls, with mean values of 88.20 ± 8.58 mm Hg and 79.69 ± 7.28 mm Hg, respectively.

Albuminuria or overt nephropathy frequently occur in type 1 diabetic patients before hypertension [50]. Due to common risk factors such as obesity, dyslipidemia, and the cardiorenal metabolic syndrome, however, the development of albuminuria and decrease in estimated GFR (eGFR) in type 2 diabetes typically occur before the onset of hypertension.

Patients with DN may develop hypertension for a variety of reasons, including inappropriate renin-angiotensin aldosterone system (RAAS) and sympathetic nervous system activation, volume expansion is brought on by increased sodium reabsorption, peripheral vasoconstriction, endothelin 1 upregulation, inflammation, the production of reactive oxygen species, and nitric oxide downregulation[51]. Numerous of these elements raise the incidence of CVD in individuals with diabetes and hypertension as well as speed up the onset of renal disease[50].

A meta-analysis of 16 cohort studies involving 3,15,321 participants found that persons in general were more likely to experience lower eGFR levels when their blood pressure was between 120/80 and 139/ 89 mmHg. [52] A similar link between repeated SBP between 125 and 134 mmHg and the likelihood of developing chronic kidney disease was shown by an investigation involving roughly 267 469 people from Hong Kong[53].

COMPARISON OF LEVELS OF BLOOD UREA, SERUM CREATININE AND URINARY ALBUMIN-CREATININE RATIO IN PATIENTS WITH DIABETIC NEPHROPATHY AND HEALTHY CONTROLS

In the current investigation, we found that patients with diabetic nephropathy had significantly higher levels of blood urea, serum creatinine, and ACR than controls (p< 0.001).

Additionally, study by Shin DI et al. in 2013 showed an independent relationship between ACR and hs-CRP (r = 0.62, p<0.001). Our results are consistent with earlier research on the relationships of hs-CRP with ACR in diabetes as in the present study[54]

COMPARISON OF LIPID PROFILE BETWEEN PATIENTS WITH DIABETIC NEPHROPATHY AND HEALTHY CONTROLS

The levels of TC, LDL, TG, and VLDL were found to be considerably higher in diabetic nephropathy cases as compared to controls (p <0.001), whereas the levels of HDL were found to be lower in diabetic nephropathy patients as compared to controls.

It's possible that insulin resistance and poor insulin action on lipoprotein metabolism are responsible for the altered lipid profile in T2DM. Increased lipolysis will enhance the production of VLDL and LDL-c, which is high in triglycerides. It will also speed up the breakdown of HDL-c and accelerate the synthesis of triglycerides [55]. Diabetes mellitus results in diabetic nephropathy, which is marked by albumin excretion in the urine, elevated blood pressure, decreased glomerular filtration rate, and a higher risk of cardiovascular disease [56]. An altered lipid profile could make the condition worse and lead the disease towards kidney dysfunction [57] Elevated lipoproteins and lipids may aggravate diabetic nephropathy by resulting in glomerular and tubulointerstitial damage [58].

In a study conducted by NN Jisieike-Onuigbo et al., DN was significantly associated with high TC and high TG (p<0.001) but

not with LDL-C or HDL-C. (p value 0.49 and 0.26 respectively) [59] Our findings are similar to Kamran Mahmood Ahmed Aziz's findings, [60] who demonstrated a substantial ($p < 0.001$) correlation between DN and High LDL-C. Similar findings were obtained by Noura Al-Jameil et al in their study. [62]

COMPARISON OF hs-CRP BETWEEN PATIENTS WITH DIABETIC NEPHROPATHY AND HEALTHY CONTROLS

In contrast to controls, who had a mean value of 1.00 ± 0.48 mg/L, patients with diabetic nephropathy had a mean value of 4.17 ± 1.57 mg/L, which was highly significant ($p < 0.001$) [Table 7]

There are a number of potential processes that could result in chronic low-grade inflammation in diabetes and associated consequences. The concentration of advanced glycation end products rises in a hyperglycemic state. Advanced glycation end products have been demonstrated to activate macrophages, upregulate the manufacture of interleukin-1, interleukin-6 (IL-6), and tumour necrosis factor, which results in the generation of CRP. These end products have also been proven to increase oxidative stress. [62] Another explanation is that the rise in CRP levels is due to cytokines produced by adipose tissue. [63].

Inflammation and hyperglycemia have been linked in studies. [64] Glycation is known to start the inflammatory process, which raises hs-CRP levels. Since poor glycemic control causes glycation-induced inflammation, hs-CRP can indicate when it will start. [65] The pathophysiology of DN may not only be influenced by the load of long-term glucose intake (diabetes), but also by impaired insulin sensitivity. Patients with type-2 diabetes may be at risk for nephropathy due to low-grade inflammation. In their investigation, M. S. Roopakala et al. came to the same conclusion as us that hs-CRP levels gradually rise in DN patients [66]

In order to investigate the relationship between the concentration of hs-CRP and the prevalence of DN, a meta-analysis was conducted. It was confirmed that elevated hs-CRP levels were associated with the prevalence of DN, which is also consistent with our findings. It is still debatable, though, whether hs-CRP is a separate risk factor for diabetic nephropathy.

As the current investigation was an observational study rather than a randomised controlled trial, the possibility of residual confounding factors cannot be completely ruled out when interpreting the results. Second, the study sample size was small and all the data came from a single hospital, which could have a negative impact on the accuracy of the findings. To investigate this, additional studies with a wider population are necessary. Third, the underlying processes of hs-CRP and DN were not examined in our investigation, and it is important to determine if elevated hs-CRP levels are a cause or an effect of DN. Additionally, a number of inflammatory cytokines, including IL-6, IL-18, and TNF-, which are linked to DN and could potentially the results, were not included in our study.

CONCLUSION:

In the current investigation, we discovered that patients with diabetic nephropathy had significantly higher values of the lipid profile (Total cholesterol, HDL, VLDL and TG), hs-CRP, BMI, and blood pressure when compared to controls. As a result, it was shown from this study that hs-CRP was related to and may have influenced the development of DN. As a result, doctors may use hs-CRP as an independent indicator when assessing diabetics who are developing complications like DN. Potentially, hs-CRP can act as a biomarker for the emergence of DN. In the meanwhile, improving our knowledge of the mechanism(s) by which inflammatory molecules like hs-CRP may contribute to the onset of DN can aid in the development of new treatment strategies and, if successful, lower the incidence of DN.

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