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ASSESSMENT OF SERUM URIC ACID AND C-REACTIVE PROTEIN LEVELS IN TYPE 2 DIABETIC PATIENTS IN A TERTIARY CARE HOSPITAL

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ABSTRACT: Introduction: The incidence and prevalence of diabetes and its complications has been significantly increased in recent times. C-reactive protein and hyperuricemia are early inflammatory markers associated with type 2 diabetes. Increased uric acid levels are known to be associated with endothelial dysfunction. The relationship between serum uric acid and inflammation in type 2 diabetes has not been explored thoroughly. **Aim:** The study estimates the levels of High sensitivity (hs) C-reactive protein and serum uric acid in type 2 diabetic patients. To find the correlation between serum uric acid levels and C-reactive protein levels in diabetic patients. **Material and Methods:** The observational study was conducted on 100 patients suffering from type 2 diabetic patients more than 40 years of age of either sex, coming to the biochemistry lab for investigations for six months. The serum uric acid, hs-C-reactive protein, and fasting blood glucose levels were analyzed. **Results:** It was observed that hs-CRP was significantly correlated with uric acid (with r=0.276, p-value= 0.005). It was also observed that there was no statistically significant difference in the mean levels of age, serum glucose, uric acid, and hs-C-reactive protein in both genders. **Conclusion:** There was a significant correlation between serum hs-CRP and serum uric acid levels in diabetic patients. Further research should be done to investigate the effect of other inflammatory markers in diabetes.

INTRODUCTION: The incidence of diabetes is ascending globally. As predicted by the International Diabetes Federation, the global pandemic of diabetes will occur by 2035, and out of every 10 people 1 will be diabetic. Diabetes mellitus is associated with several factors [1]. The incidence of diabetes rose from 108 million in 1980 to 422 million in 2014. The prevalence has been increasing more rapidly in unprivileged countries than in privileged countries.

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Between 2000 and 2016, there was a 5% increase in premature mortality due to diabetes. Diabetes was the ninth leading cause of death in 2019, with an estimated 1.5 million deaths due to diabetes [2]. C-reactive protein (CRP), is an acute-phase protein that is synthesized and released by liver macrophages as well as adipocytes.

It is a sensitive marker in the early phase of inflammation with a half-life of up to 19 hours. It is mainly synthesized in the liver by stimulating inflammatory cytokines such as interleukin-6 [3]. A high sensitivity (hs)-CRP measurement method was developed with the advancement of technology, which enables the measurement of even low concentrations of CRP with high precision [4]. In atherosclerotic lesions, there is local production of CRP by lymphocytes or smooth

muscle cells and monocytes [3]. It had been observed in some previous studies that the blood concentration of hs-CRP may help in predicting the development of diabetes and metabolic syndrome in adults. In a study by Hoorn et al., the group with elevated CRP levels had more fasting blood glucose levels compared to other groups and type 2 diabetes [5]. This study helped in examining the relationship between inflammation and diabetes. Higher C-reactive protein (CRP) levels and hyperuricemia are independent markers of inflammation. An increase in uric acid levels is also associated with endothelial dysfunction [6].

In humans, uric acid is the final product of purine metabolism, which is synthesized in the liver. The purine nucleotides get metabolized to hypoxanthine and guanine, some of which can be reconverted and phosphorylated into hypoxanthine nucleotides. In contrast, the residual part is metabolized by xanthine dehydrogenase/oxidase (XDH/XO) enzymatic reaction to the end product uric acid. The enzyme XDH/XO is mainly expressed in the liver and small intestine parenchymal cells. The production of uric acid primarily depends on the amount of substrate availability and the activity of XO. The kidneys also play a significant role in the regulation of levels of blood uric acid.

The uric acid that is filtered by the glomeruli, about 90% of that is reabsorbed by the proximal convoluted tubule, mainly by urate transporter 1 (URAT1) and glucose transporter 9 (GLUT9), and the remaining 10% is responsible for 60–70% of total excretion of body's uric acid. A small amount of uric acid, about 30-40%, is secreted in the intestines. In healthy individuals, uric acid's production and excretion rate is relatively constant. Any change in the amount of uric acid in body fluids can imitate the condition of metabolism, immunity and other functions of the human body. The body retains excessive uric acid if the production of uric acid is more or its excretion is less. Hyperuricemia was defined as the circulating uric acid levels of more than 7.0 mg/dl for men and 5.7 mg/dl for women [7]. High serum uric acid levels may be used as a predictor for obesity, hypertension, T2DM, cardiovascular diseases, metabolic syndrome, and reduced glomerular filtration rate (GFR). The uric acid enters vascular smooth muscles to activate an inflammatory cycle and endothelial dysfunction, which in turn, causes the release of CRP, further increasing the risk of albuminuria in DM cases [8]. While acting as an inflammatory factor, UA increases oxidative stress and enhances the activation of the renin-angiotensin-aldosterone system (RAAS) [9]. Thus, Uric acid act as an independent risk factor for early kidney disease and is associated with the occurrence and development of Diabetic nephropathy [10].

It was evident in previous studies that serum uric acid levels could directly introduce increased cytokines like CRP, INF, and IL-6 in the bloodstream to cause renal dysfunction and increases albumin excretion in urine [11]. The high concentration of uric acid in the blood plays a major role in kidney stones and can also indicate various diseases, like diabetes, gout, hyperparathyroidism, and acute kidney failure. In previous studies, higher levels of CRP have been associated with diabetes, albuminuria, reduced eGFR and increased incidence of cardiovascular disease. In some recent clinical studies, it had been found that CRP was associated with diabetic nephropathy in both type 1 and type 2 diabetes [12]. Hyperuricemia is a metabolic abnormality that is closely related to type 2 diabetes. The association between serum uric acid and inflammation in type 2 diabetes has not been reported. Thus, the following study was conducted to estimate the levels of hs-C-reactive protein and serum uric acid in type 2 diabetic patients and to find the correlation between serum uric acid and hs-C-reactive protein.

MATERIAL AND METHODS: The study was conducted in the department of Biochemistry, Government Medical College, Jammu, for a period of six months *i.e*., from October 2021 to March 2022, on 100 already diagnosed type 2 diabetic patients more than 40 years of age of either sex, coming to the biochemistry laboratory for investigations. The detailed history of the patient was taken prior to sample collection. The study was carried out after obtaining due approval from the College's Institutional Ethics Committee (Reference no. IEC/GMC/2021/597 dated: 09.09.2021). 5 ml of fasting blood samples were withdrawn from the antecubital vein under aseptic conditions from each individual with their consent,

duly following the guidelines and norms of the hospital. Blood samples were collected in plain and sodium fluoride vacutainers. The serum uric acid, C-reactive protein, and fasting blood glucose levels were analyzed. Abbott Architect c-Systems estimated blood glucose levels by hexokinase method.

Abott Architect c-Systems estimated serum uric acid levels by uricase method. The normal range of serum uric acid in adult's males is 3.5-7.2 mg/dl and in females is 2.6 -6.0 mg/dl [13]. Abott Architect c-Systems estimated serum CRP levels by an immunoturbidimetric method. The normal range of serum CRP is 0-5mg/L [13]. The statistical analysis was conducted using SPSS version 20.

Inclusion Criteria: Patients suffering from type 2 diabetes, more than 40 years of age, of either sex, with a duration of diabetes, not more than 5 years.

Exclusion Criteria: Patients less than 40 years of age, Pregnant women, Patients suffering from type 1 diabetes, dehydration, urinary tract obstruction, muscular dystrophy, or diseases other than type 2 diabetes.

RESULTS: Table 1 shows the percentage distribution of patients in different age groups. Out of the total 100 patients, 49 were less than 50 years of age, 28 patients were in the age group of 51-60 years, and 23 were in the age group of more than 60 years.

Table 2 shows the percentage distribution of males and females in different age groups. Out of the 100 patients, 43 were males, and 57 were females. Out of 43 males, 20 were in the age group of less than 50 years, 12 were in the 51-60 years age group and 11 were in the more than 60 age group.

Out of the total 57 females, 29 were in the age group of less than 50 years, 16 were in the 51-60 years of age group and 12 were in the more than 60 age group. **Table 3** shows the gender-wise distribution of patients according to their hs-CRP levels. Out of 100 patients, 77 had hs-CRP levels between 0-5mg/L, 9 had hs-CRP levels between 5.1 mg/L to 10mg/L, and 14 had hs-CRP levels more than 10mg/L. **Table 4** shows **the** Age-wise distribution of patients according to hs-CRP levels. Out of 100 patients in the age group of less than 50 years, 39 patients had hs-CRP levels between 0-5mg/L, 4 patients had hs-CRP levels between 5.1-10mg/L and 6 patients had hs-CRP levels more than 10mg/L.

In the age group of 51-60 years, 20 patients had hs-CRP levels between 0-5mg/L, 4 patients had hs-CRP levels between 5.1-10mg/L and 4 patients had hs-CRP level more than 10mg/L. In the age group of more than 60 years, 18 patients had hs-CRP levels between 0-5mg/L, 1 patient had hs-CRP levels between 5.1-10mg/L, and 4 patients had hs-CRP levels more than 10mg/L. **Table 5** shows the mean levels of age, serum glucose, serum uric acid, and serum hs-CRP levels and their minimum and maximum values.

It had been observed that the mean serum Glucose levels, serum uric acid levels, and serum hs-CRP levels in type 2 diabetic patients were 112.77 mg/dl, 6.23 mg/dl, and 6.65 mg/L. **Table 6** shows the mean levels of different parameters in both genders. It was observed that the mean levels of age, serum glucose, uric acid and hs-C-reactive protein were higher in males as compared to females, but the difference was not statistically significant.

Table 7 shows a correlation between various parameters. It was observed that hs-CRP was significantly correlated with serum glucose and uric acid. No significant correlation was observed between age and serum glucose, uric acid, and hs-CRP levels. The serum uric acid levels significantly correlate with fasting serum glucose levels. The scatter plot, **Fig. 1**, shows a significant correlation between serum hs-CRP levels and serum uric acid levels.

TABLE 1: PERCENTAGE DISTRIBUTION OF PATIENTS IN DIFFERENT AGE GROUPS

Age Groups	Frequency	Percent	Cumulative Percent
<=50 Years	49	49.0	49.0
51-60 Years	28	28.0	77.0
>60 Years	23	23.0	100.0
Total	100	100.0	

TABLE 2: GENDER-WISE DISTRIBUTION OF PATIENTS IN DIFFERENT AGE GROUPS

		Age groups			
		<=50 Years	**51-60 Years**	**>60 Years**	**Total**
SEX	Count	20	12	11	43
Male	% within SEX	46.5%	27.9%	25.6%	100.0%
	% within age groups	40.8%	42.9%	47.8%	43.0%
Females	Count	29	16	12	57
	% within SEX	50.9%	28.1%	21.1%	100.0%
	% within age groups	59.2%	57.1%	52.2%	57.0%
Total	Count	49	28	23	100
	% within SEX	49.0%	28.0%	23.0%	100.0%
	% within age groups	100.0%	100.0%	100.0%	100.0%

TABLE 3: GENDER-WISE DISTRIBUTION OF PATIENTS ACCORDING TO HS-CRP LEVELS

			hs-CRP groups			
			0-5	**5.1- 10**	**More than 10.**	**Total**
	F	Count	46	6	5	57
		% within SEX	80.7%	10.5%	8.8%	100.0%
		% within hs-CRP groups	59.7%	66.7%	35.7%	57.0%
	M	Count	31	3	9	43
		% within SEX	72.1%	7.0%	20.9%	100.0%
		% within hs-CRP groups	40.3%	33.3%	64.3%	43.0%
Sex	Total	Count	77	9	14	100
		% within SEX	77.0%	9.0%	14.0%	100.0%
		% within hs-CRP groups	100.0%	100.0%	100.0%	100.0%

TABLE 4: AGE-WISE DISTRIBUTION OF PATIENTS ACCORDING TO HS-CRP LEVELS

			Hs-CRP groups			
			0-5	**5.1- 10**	**more than 10.**	**Total**
Age groups	<=50 Years	Count	39	4	6	49
		% within age groups	79.6%	8.2%	12.2%	100.0%
		% within hs-CRP groups	50.6%	44.4%	42.9%	49.0%
	51-60 Years	Count	20	4	4	28
		% within age groups	71.4%	14.3%	14.3%	100.0%
		% within hs-CRP groups	26.0%	44.4%	28.6%	28.0%
	>60 Years	Count	18	1	4	23
		% within age groups	78.3%	4.3%	17.4%	100.0%
		% within hs-CRP groups	23.4%	11.1%	28.6%	23.0%
	Total	Count	77	9	14	100
		% within age groups	77.0%	9.0%	14.0%	100.0%
		% within hs-CRP groups	100.0%	100.0%	100.0%	100.0%

TABLE 5: THE MEAN VALUES, STANDARD DEVIATION AND RANGE OF VARIOUS PARAMETERS IN TYPE 2 DIABETIC PATIENTS

Parameter	Mean	Standard Deviation	Range
Age	53.83 years	11.13	40-86
S. Glucose F	112.77 mg/dl	35.61	78-278
S.Uric Acid	6.23 mg/dl	1.58	2.80-10.6
S. hs-CRP	6.65 mg/L	12.60	0.09-60.30

TABLE 6: GENDER WISE COMPARISON OF DIFFERENT PARAMETERS

Parameter	Males (N=43) Mean±SD	Females (N=57) Mean±SD	p-value*
Age	54.37±12.60 years	53.42±9.97 years	0.958 (Mann Whitney Test)
S. Glucose F	118.83±41.47mg/dl	108.19±30.05mg/dl	0.112 (Mann Whitney Test)
S. Uric Acid	6.43±1.65 mg/dl	6.08±1.51 mg/dl	0.273
S. hs-CRP	8.71±15.06 mg/L	5.09±10.23 mg/L	0.575 (Mann Whitney Test)

*p-value <0.05, is considered significant

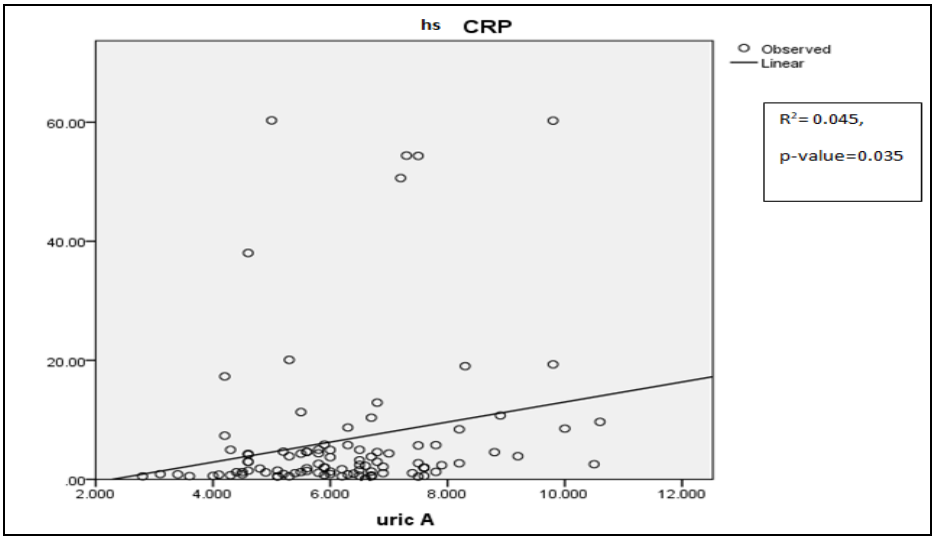

FIG. 1: SCATTER PLOT SHOWING THE CORRELATION BETWEEN SERUM HS-CRP LEVELS AND SERUM URIC ACID LEVELS

TABLE 7: SPEARMAN'S CORRELATION BETWEEN DIFFERENT PARAMETERS IN TYPE 2 DIABETIC PATIENTS

		hs-CRP	AGE	S.Glucose(F)	S.uric Acid
hs-CRP	Correlation Coefficient	1.000	.128	.342**	.276**
	Sig. (2-tailed)	.	.203	.001	.005
	N	100	100	100	100
AGE	Correlation Coefficient	.128	1.000	.077	.134
	Sig. (2-tailed)	.203	-	.449	.183
	N	100	100	100	100
S.Glucose (F)	Correlation Coefficient	.342**	.077	1.000	.260**
	Sig. (2-tailed)	.001	.449	-	.009
	N	100	100	100	100
S. uric Acid	Correlation Coefficient	.276**	.134	.260**	1.000
	Sig. (2-tailed)	.005	.183	.009	-
	N	100	100	100	100

**correlation is significant at the 0.01 level (2-tailed).

DISCUSSION: In the present study, it was observed that the serum CRP levels are significantly correlated with serum glucose and serum uric acid levels. There was a significant correlation between serum glucose levels and serum uric acid. No significant difference was found when various parameters were compared in both genders. Besides, the antioxidant role of uric acid, many authors suggested that the high levels of uric acid act as a promoter for systemic inflammation, endothelial dysfunction, CRP expression, hypertension and cardiovascular diseases [8]. In a study conducted by Kang *et al*, to investigate the role of uric acid in CRP synthesis and to find any pathological connection between uric acid and CRP in diabetes, cardiovascular diseases and CKD. It had been observed that since evidence shows that CRP has direct effect to promote atherosclerotic process and endothelial cell dysfunction [14]. In a study conducted on patients having cardiovascular disease and Metabolic Syndrome, it was observed that the primary mechanism of the low-grade inflammation and insulin resistance in these patients was the increased uric acid levels which may be partly responsible for the pro-inflammatory endocrine imbalance in the adipose tissue and vascular smooth muscle cells [15]. In another study, when allopurinol was administered to mice, it caused uric acid lowering, which further increased adiponectin's production and decreased monocyte chemoattractant protein-1, thus improving the pro-inflammatory endocrine imbalance in the adipose tissue. In addition, lowering uric acid in obese mice decreased macrophage infiltration in the adipose tissue and reduced insulin resistance [9]. In addition, uric acid has also been shown to induce the synthesis of other inflammatory cytokines like

interleukin 1β, interleukin 6, tumor necrosis factor α in mononuclear cells and CRP in cultured vascular cells [7]. In a study conducted by Matsuoka *et al.,* after adjusting other potential confounders, significant correlation between serum uric acid and CRP levels was observed [16].

The various pathological mechanisms like inflammation, oxidative stress, endothelial dysfunction, and inhibition of insulin pathway are involved in diabetes, as shown in various studies [14]. In another study conducted on healthy individuals, a positive correlation was observed between serum uric acid and TNF-*α*, interleukin-6, and C-reactive protein [15]. Uric acid increases the production of reactive oxygen species, like hydrogen peroxide, causing inflammation and shows pro-oxidant effects in vascular tissue. It also acts as a potent antioxidant that can eliminate superoxide and hydroxyl radicals from plasma. Uric acid also leads to cellular damage by causing activation of various inflammatory factors, oxidative stress-induced lipid peroxidation and DNA damage [17]. Oxidative stress also causes a reduction in insulin secretion by affecting the expression of insulin gene [16]. Uric acid reduces nitric oxide secretion and inhibits the proliferation and migration of endothelial cells [14]. At the receptor level, uric acid causes recruitment of ectonucleotide pyrophosphatase / phosphodiesterase 1 (ENPP1), thus directly inhibiting the insulin signaling pathway [18]. All these factors interfere with insulin sensitivity and the homeostasis of glucose, thereby promoting the development of diabetes. The changes like desulfation of glycosaminoglycans, formation of advanced glycation end products and receptors for advanced glycation end products (AGE-RAGE complexes) in diabetes are also linked to the metabolic disorder [19].

In contradiction to our study, Li *et al.* observed an inverse association of serum uric acid with diabetic parameters. This can be due to increased renal excretion of uric acid in the presence of hyperglycemia. As the duration of diabetes increases, the function of β cell worsens, hence the glycemic control and gradual increase in the rate of renal filtration. In the previous studies, it was observed that the high levels of uric acid in metabolic syndrome had been ascribed due to increased insulin levels. Elevated insulin levels are related to impaired renal excretion of uric acid, thus leading to its retention. But their study had a limitation: they could not eliminate the effect of certain diseases and the medications given for those diseases [20].

Limitations of the Study: There are several limitations to this study. Firstly, this study was conducted with small sample size. Secondly, the duration of diabetes was not taken into consideration. Thirdly, the other factors involved in inflammation or inflammatory markers were not thoroughly explored.

CONCLUSION: The present study's findings suggest a significant correlation between serum CRP and serum uric acid levels in diabetic patients. Further, the study also demonstrates that no significant difference was found when a gender-wise comparison of serum CRP and serum uric acid levels was made. Further research should be done to investigate the mechanisms involving the effect of uric acid and CRP on insulin secretion and by decreasing inflammatory activity and lowering serum uric acid levels in the prevention and treatment of diabetes and its complications.

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