RESEARCH PAPER

Association of Serum Uric Acid and Hs-CRP with Early Vascular Changes in Newly Diagnosed Type 2 Diabetes Mellitus

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ABSTRACT

Type 2 Diabetes mellitus (T2DM) feels more like a situation that travels with a bigger burden of cardiovascular problems, it often shows up when the chronic inflammation keeps running, and there's also insulin resistance in the background, plus endothelial dysfunction… you know that whole pathway web. The current study was kind of aimed at figuring out what happens between SUA (serum uric acid) and hs-CRP (high sensitivity C reactive protein) together, and how that lines up with early vessel modifications such as CIMT (carotid intima-media thickness) in people who were newly diagnosed with T2DM, more or less in a straightforward manner.

This work was carried out in the Department of General Medicine, CMC Hisar, and it was a hospital based study, like entirely. The total duration was 18 months, nothing more. A total of 151 newly diagnosed T2DM cases were included and they all fit the inclusion, as well as the exclusion criteria properly. Clinical and anthropometric assessment were performed and along with that biochemical testing was also done. SUA was measured using the enzymatic colorimetry method, and hs-CRP was evaluated by immunoturbidimetry

The mean age of the group was 33.64 ± 4.23 years. There was almost equal representation of both genders, so the balance was kind of there. The average CIMT was 0.92 ± 0.20 mm, and elevated CIMT showed up in 53.6% of subjects, this kinda points to a higher occurrence of silent atherosclerosis before it fully turns into something obvious.

The average hs-CRP came out around 11.00 ± 4.01 mg/L, meanwhile the average serum uric acid was 5.81 ± 1.23 mg/dL. For the correlation part, analysis showed, a rather slight positive association between CIMT and hs-CRP (r = 0.088) and also a slight inverse relation between CIMT and serum uric acid (r = -0.036)

In conclusion, this study kinda hints at a high occurrence of systemic, low grade inflammation along with subclinical atherosclerosis in newly diagnosed T2DM patients. It also kind of emphasizes that hs-CRP, SUA, and CIMT may work as early indicators, or early warnings, for cardiovascular risk.

Keywords: *Atherosclerosis, endothelial dysfunction, hs-CRP, serum uric acid, Type 2 Diabetes Mellitus*

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INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) has become a huge global health issue, because the load of sickness and early death from it keeps rising. Tang et al., (2022) mention that in 2021 around 536.6 million people were already diagnosed with diabetes, and by 2045 this figure might keep going up to 783.2 million. T2DM accounts for nearly 90% of all diabetes cases and it is often framed as insulin resistance, with insulin insufficiency tagging along as well, sort of like the same set. But it's not only a metabolic disorder situation—T2DM also drags in long-term vascular problems. This is why the risk of cardiovascular events increases, and why other complications can appear too, such as nephropathy, retinopathy and neuropathy.

Even though there have been a lot of advances in how T2DM is treated, cardiovascular events are still, basically, the leading reason people with T2DM die. Vascular complications in diabetes can even emerge very early, and this is largely linked to higher glucose levels, oxidative stress, endothelial dysfunction, and inflammatory activity. So, it becomes really important to find biomarkers that can

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signal early vascular disease in diabetes, before things get more severe.

Among the newer biomarkers that have gotten a lot of research attention, serum uric acid (SUA) and high sensitivity C reactive protein (hs-CRP) kind of stand out. In humans, SUA is the last metabolite made from purines metabolism, basically. For a while, people assumed it was biologically inert, like just an end product of metabolism, nothing else really, no big deal. But more recent studies suggest higher SUA levels are tied with insulin resistance and endothelial dysfunction and this whole bundle of cardiometabolic disorders. It's not one single pathway you can point to and call it a day, more like a chain of events with overlap.

Hyperuricemia has been often seen in patients who have T2DM plus metabolic syndrome, it feels like they commonly show up together in real life. It supports oxidative stress by stimulating the enzyme NADPH oxidase and then, after that, drives the formation of reactive oxygen species, ROS. This, in turn, reduces nitric oxide availability, so you end up with endothelial dysfunction. On top of that, uric acid boosts inflammation, through mechanisms like NF-κB, and it also promotes an increase in pro inflammatory cytokines plus vascular smooth muscle proliferation, not at all in a small way either.

Inflammation plays a key role in how diabetic complications end up showing, especially when vascular disorders are involved, and it's sort of tied into a lot of downstream events. hs-CRP is an acute-phase protein that is synthesized by the liver, and it's usually used as a practical "temperature check" for overall systemic inflammation. When hs-CRP concentration is higher, it has been linked with insulin resistance, metabolic syndrome, and endothelial dysfunction in people living with T2DM. In multiple reports, diabetic patients had clearly increased hs-CRP levels compared with non-diabetics, in a way that's pretty consistent, even if you adjust for other covariates, or a similar set of factors. (Seo and Shin et al., 2017; Tutuncu et al., 2016, Gao et al., 2024; Sibianun and Slevin, 2025).

Hyperuricemia is also often seen in patients with T2DM, especially when metabolic syndrome is present, almost like they tend to appear together in real life. It kind of backs up oxidative stress because it activates the enzyme NADPH oxidase, and then it promotes the generation of reactive oxygen species, ROS. This in turn decreases nitric oxide availability so endothelial dysfunction follows. And at the same time, uric acid enhances inflammatory signaling via pathways like NF-κB , and that then encourages higher levels of pro-inflammatory cytokines plus vascular smooth muscle proliferation, not in a small, calm way

Inflammation itself takes a key role in how diabetic complications tend to show up, especially when we talk about vascular disorders. hs-CRP is an acute phase protein that is synthesized by the liver, and it is usually used as a useful marker for overall systemic inflammation. When hs-CRP concentrations are higher, it has been linked to insulin resistance, metabolic syndrome and endothelial dysfunction in people with T2DM. In several reports, diabetic patients show up with significantly increased hs-CRP levels compared to non diabetics, like it stays consistently higher even after you adjust for other variables, or at least that's what it looks like.

Once diagnosed with T2DM, the person kind of enters a critical window where early vascular changes might still be avoided if treatment is adequate. Put differently, building some kind of connections between metabolic markers and inflammatory factors during this initial stage can help identify the profile subgroup, with a higher probability of later cardiovascular complications.

So, in line with that, the present study was set up to look at the relationship between these variables. The findings might help pinpoint patients who are at higher risk for cardiovascular disease and could back the development of measures that prevent more vascular harm in T2DM patients.

MATERIAL AND METHOD

This present cross sectional kind of study was carried on in the Department of General Medicine at CMC Hisar for about 18 months. Newly diagnosed T2DM patients, who were visiting the Medicine OPD, and who met the inclusion, along with exclusion requirements, were brought into the study only after obtaining prior informed consent from them, as well.

Inclusion Criteria

1. T2DM who are treatment naïve
2. Without any evidence of CAD/CVA
3. Age group 18-40 years

Exclusion Criteria

1. Past history of CAD.
2. Past history of any ischemic neurological illness or ischemic CVA.
3. Patients not suffering from endocrine disorders like hypothyroidism and other diseases
4. potentially affecting the variables
5. Patients with history of smoking or consuming alcohol.
6. Drugs induced diabetes like steroids
7. Any history of malignancy, present or past.
8. Patients with history of gout or drug treatment for hyperuricemia
9. Pregnancy induced Diabetes Mellitus

Sample Size

Calculation of the sample size was done by using the formula explained by Hulley et al., (2013).

$N=[(Z\square+Z\square)/C]^2+3$

This formula needs an entry of $\alpha = 0.05$ (type 1 error) and also $\beta = 0.10$ (type 2 error). The correlation coefficient for hs-CRP with CIMT was taken as 0.26, kinda based on the work by Lachine et al., (2016). So then the resulting sample size was, calculated to be 151 subjects.

METHODOLOGY

After enrolment, all patients were put through clinical assessment, and a physical check up. Anthropometric figures such as height weight and Body Mass Index BMI were obtained. Then a venous blood sample was taken aseptically for biochemistry, and the serum levels later served for the analyses. The serum uric acid concentration was quantified using an enzymatic colorimetric approach, while hs-CRP was assessed using an immuno-turbidimetric method.

For Carotid Intima-Media Thickness, CIMT, ultrasonography was done with high frequency resolution B-mode imaging, using a linear probe set at 7.5 to 10 MHz. CIMT was evaluated at the far wall of the distal common carotid artery, roughly 1 cm before the carotid bifurcation. On each side, three CIMT readings were collected and then the average of those readings was used as the final CIMT value.

All clinical, as well as biochemical parameters were recorded in a predesigned proforma.

Statistical Analysis

All the statistical analyses were carried out using the SPSS software package, version 26.0 so yeah. Quantitative data was written as mean ± SD. As for the qualitative part, it was shown as the number along with the percentage. When it felt reasonable, they applied the Chi-square test, and also the Student's t-test. A p-value lower than 0.05 was considered statistically significant.

RESULT AND DISCUSSION

So, in this present study, we enrolled 151 newly diagnosed T2DM patients to see how SUA and hs-CRP might be linked with the early phases of vessel harm, measured through CIMT. Then we tested various demographic, anthropometric, biochemical, inflammatory, and vascular items, to figure out if there was some connection, even a subtle one, to subclinical atherosclerosis among people who were just newly diagnosed with diabetes mellitus.

Demographic Profile

The average age of the subjects in the study was 33.64 ± 4.23 years, which kinda suggests that a good number of the patients were having T2DM at a relatively young age (Fig 1). In general, the gender distribution was pretty close, 51.0% of the participants were male while 49.0% were female, (Table 1). The mean BMI came out to be 25.99 ± 5.10 kg/m², and this seems to show a noticeable share of obesity, plus also people in the overweight range (Table 2). On top of that, the waist circumference and the waist-to-hip ratio both looked high, so there was strong sign of central fat accumulation, and an even more serious cardiometabolic risk as well.

Table 1: Demographic Characteristics in Newly Diagnosed T2DM Patients.

Parameter		Frequency (n=151)	Percentage (%)
Age (years)		33.64 ± 4.23	
18–24		42	27.8
25–29		36	23.8
30–34		32	21.1
35–40		34	22.5
Gender	**Male**	77	51.0
	Female	74	49.0

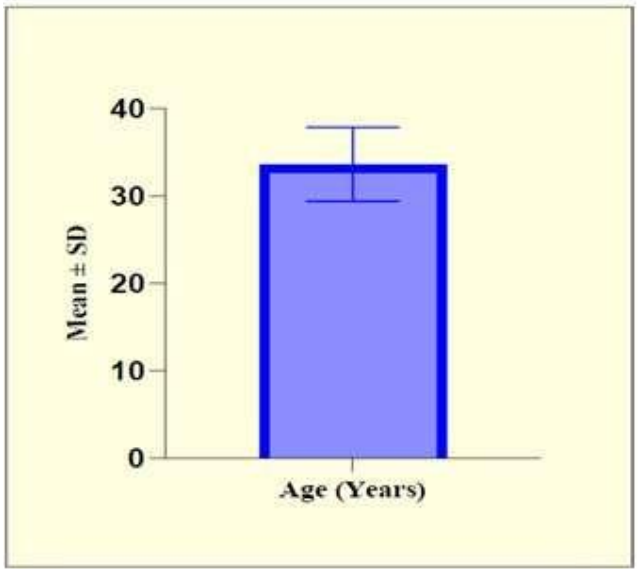

Figure 1. Graphical Representation of Mean Age among patient population.

Table 2. Anthropometric Characteristics in Newly Diagnosed Type 2 Diabetes Mellitus Patients.

Parameter		Frequency (n=151)	Percentage (%)
Height (cm)		165.20 ± 7.89	
Weight (kg)		70.54 ± 12.93	
BMI (kg/m²)		25.99 ± 5.10	
BMI Category	**Normal (18.5–24.9)**	65	43.0
	Overweight (25–29.9)	50	33.1
	Obese (≥30)	36	23.8
Waist Circumference (cm)		90.88 ± 11.23	
Waist-to-Hip Ratio		0.95 ± 0.05	

Similar findings have been mentioned earlier studies too, like Gur et al., (2013) showed that the thickening of the intima-media layer was positively tied to uric acid level, hs-CRP, and also to a less favorable biochemical profile, kinda the same direction. Also, Saito et al., (2023) in the PRIZE study suggested that age, BMI and a prior history of cardiovascular atherosclerosis actually had a big say in CIMT progression.

In this current study, even if these patients did not have a previously diagnosed diabetes, or a cardiovascular disease, more than half still showed elevated CIMT values, so this seems to hint at silent or subclinical atherosclerosis. Therefore, overall, these results suggest that vascular alterations could already be underway by the time T2DM is formally diagnosed, and it supports the concept that early cardiovascular risk screening is important, together with prompt preventative steps.

Vascular Changes, Serum Uric Acid and hs-CRP

In the study population the average CIMT came out to 0.92 ± 0.20 mm, more or less. In other words higher CIMT levels meaning >0.9 mm, showed up in about 53.6% of the patients while "normal'' CIMT values were found in only a small set of cases (Table 3). These results suggest that endothelial impairment, and rather hidden atherosclerotic changes could appear early, long before the actual visible clinical manifestation of T2DM related complications.

Table 3. Carotid Intima-Media Thickness (CIMT) Distribution in Newly Diagnosed Type 2 Diabetes Mellitus Patients.

Parameter	Frequency (n=151)	Percentage (%)
CIMT (mm)	0.92 ± 0.20	
Normal (<0.7 mm)	12	7.9
Borderline (0.7–0.9 mm)	58	38.4
Increased (>0.9 mm)	81	53.6

Similarly, the mean hs-CRP level turned out to be pretty elevated (11.00 ± 4.01 mg/L), and almost everyone had an inflammatory status that landed in the high risk bracket (>3 mg/L), so it feels like theres a noticeable amount of gentle low-grade inflammation in these folks (Table 4). On the other hand, the average SUA level was 5.81 ± 1.23 mg/dL, and this measure looked higher in roughly one third of the patients. Even if the general uric acid amount stayed in physiologic range, the raised values observed in a clear subset of participants hints at metabolic dysregulation, oxidative stress, and possible endothelial injury in these people.

In the current study, SUA showed a sort of weak negative relation with CIMT (r = -0.036), and that sort of doesn't line up with earlier work, which had found a more noticeable correlation between SUA and the thickness of the vessel wall, in some way. In a study with T2DM patients, Venishetty et al., (2018) noted a significant link between SUA and CIMT (r = 0.779, p = 0.001), implying that elevated uric acid levels might be contributing to carotid atherosclerotic changes. Likewise, Wu et al., (2019) found a meaningful association between SUA and CIMT among individuals with metabolic syndrome and stated that assessing uric acid together with oxidative stress markers, plus the metabolic syndrome score, helped improve forecasting of vascular alteration. More importantly, Gur et al., (2013) showed that the increase in SUA along with hs-CRP levels was independently connected with thoracic aortic intima media thickness.

Table 4. hs-CRP Levels in Newly Diagnosed Type 2 Diabetes Mellitus Patients.

Parameter	Frequency (n=151)	Percentage (%)
hs-CRP (mg/L)	11.00 ± 4.01	
Low (<1 mg/L)	1	0.7
Moderate (1–3 mg/L)	0	0.0
High (>3 mg/L)	150	99.3

A weak positive association between hs-CRP and CIMT (r = 0.088) was observed in this study, which kind of hints at a possible link between whole body inflammation and the emergence of vascular abnormalities in newly diagnosed T2DM patients. Even if the relationship is not that strong, the relatively elevated hs-CRP concentrations found in nearly all participants suggest inflammation may already be present right at the start of diabetes and could be helping drive endothelial dysfunction.

Sort of similar results were mentioned by Lachine et al., (2016), where they described higher hs-CRP and CIMT values in T2DM patients than in control subjects, and they also showed hs-CRP as an independent factor linked with CIMT. Gur et al., (2013) , meanwhile, also recognized hs-CRP as an independent correlate for intima-media thickness. Then Kawamoto et al., (2013) reported that SUA, along with hs-CRP, could be used to foresee metabolic syndrome and insulin resistance.

CORRELATION ANALYSIS

In the current study, the correlation analysis showed a weak, slightly positive link between CIMT and hs-CRP (r = 0.088), suggesting systemic inflammation might be part of what drives vascular changes in newly diagnosed T2DM patients. There was also a weak positive association between CIMT and HOMA-IR (r = 0.080), which kind of implies that insulin resistance could contribute to subclinical atherosclerosis (Table 5). Still, we observed a weak negative correlation between CIMT and the SAU level (r = -0.036).

Table 5. Correlation Matrix Showing Association of CIMT with Metabolic and Inflammatory Parameters.

Variables	CIMT	Uric Acid	hs-CRP	HOMA-IR	LDL
CIMT	1.000	-0.036	0.088	0.080	0.004
Uric Acid	-0.036	1.000	0.074	0.112	0.166
hs-CRP	0.088	0.074	1.000	0.003	-0.007

Previous studies have pointed out that there seems to be a more noticeable connection, between inflammatory and metabolic biomarkers, and overall vascular health. Wu et al., (2019), Lachine et al., (2016), and Kawamoto et al., (2013) showed that SUA, hs-CRP, insulin resistance, and metabolic irregularities are linked with higher CIMT and higher vascular hazards as well.

In this study, though, we observed only a weak correlation between SUA, hs-CRP, and CIMT. One possible reason is that the participants were relatively young, and had newly diagnosed T2DM", so the exposure to high blood glucose, inflammatory activity, and oxidative stress may not yet be strong enough to create a clearer signal. In other words, once obesity, insulin resistance, dyslipidemia, and inflammatory disruptions start to appear, they may collectively indicate the gradual build-up of vascular dysfunction, which feels more multi-angled than being driven by a single marker by itself.

So, overall, these results imply that subclinical atherosclerosis and systemic inflammation are shared features in people with newly diagnosed T2DM. That is why SUA and hs-CRP could still function as fairly good markers for assessing vascular disease risk.

CONCLUSION

In the newly diagnosed cases of T2DM, a pretty high prevalence of subclinical atherosclerosis and systemic inflammation was seen, kinda like a usual routine pattern, honestly. CIMT came out higher in many patients, so it seems like vascular dysfunction was already there at diagnosis rather than showing up later. Also, the high hs-CRP levels hinted at ongoing low grade inflammation that, it's not that it truly stops.

Meanwhile, higher SUA levels showed up in a noticeable number of these patients too. But when we ran the statistical correlation, only weak correlations were observed between CIMT, hs-CRP and serum uric acid. So early vascular changes in people with newly diagnosed

T2DM likely come from multiple intertwined causes, including obesity, insulin resistance, inflammation, metabolic disruptions and a few other factors as well.

Even if the correlation between serum uric acid, hs-CRP and CIMT was low these measures can still work as markers pointing toward higher cardiometabolic and vascular risk. Overall, these findings really underline why cardiovascular risk assessment should start early, and preventive measures should be initiated on time, right after T2DM diagnosis.

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