Association between serum uric acid and triglyceride-glucose index in adult hypertensives

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ABSTRACT:

Background: The incidence of hypertension increases with age, which leads to an increased risk of morbidity and death due to complications such as stroke, myocardial infarction, renal failure, and heart failure. The purpose of research to find Association between serum uric acid and triglyceride-glucose index in adult hypertensives.

Methods: This is a observational cross sectional study of 200 cases of Diagnosed hypertensive patients in the age group of 20-30 yrs attending to the Department of General Medicine, SVRR Govt. General Hospital S.V. Medical College, Tirupati. Blood pressure, serum uric acid, fasting blood glucose, Fasting Lipid profile, triglyceride-glucose index were measured.

Results: Hypertensives showed greater mean uric acid concentrations (P<0.0001). Uric acid correlated positively with systolic and diastolic blood pressure (P<0.0001). Increased Triglyceride–Glucose index was associated with increased uric acid levels in Hypertensives. Correlation analysis of triglyceride-glucose index with other parameters shows positive correlation with age, SBP, DBP, Serum uric acid, fasting glucose, serum Triglyceride.

Conclusion: In people with hypertension, there was a positive correlation between the TyG index and serum uric acid.

Key words: Uric acid, Triglyceride-glucose index, Adult hypertensives, Hyperuricemia.

Introduction: The incidence of hypertension increases with age, which leads to an increased risk of morbidity and death due to complications such as stroke, myocardial infarction, renal failure, and heart failure. Hypertension is the most common form of cardiovascular disease found in adults. Uric acid is a byproduct of the purine metabolism that is formed in the blood from either endogenous purine (2/3) compounds or from purines that are consumed in the diet (1/3). Consuming alcohol and foods high in purines, drinking inadequate amounts of water, and engaging in insufficient physical activity are all significant factors that lead to hyperuricemia. Based on monosodium urate's solubility in serum at 36.8°C, men's and women's normal levels are 7 and 6 mg/dl, respectively. As a result of hyperuricemia, the water-insoluble urate salt can become saturated and precipitate out as monosodium urate crystals, which can lead to gout and kidney stones. In addition, hyperuricemia has been linked to a wide variety of disorders, such as hypertension significant cardiovascular and coronary events, type 2 diabetes, chronic renal disease, obesity, and metabolic syndrome. Because of the consistent rise in the number of people who are affected by hyperuricemia, this condition is increasingly becoming a concern in every region of the world. It is believed that uric acid has a pathogenic role in hypertension, which may be mediated by a number of mechanisms including inflammation, vascular smooth muscle cell proliferation in renal microcirculation, endothelial dysfunction, and activation of the renin-angiotensin-aldosterone system. Triglyceride-glucose (TyG) index was first reported in 2008 and compared with the HOMA-IR index, demonstrating that the TyG index can be used as a reliable, economical, and easy alternative for insulin resistance. It is possible that hyperinsulinemia brought on by insulin resistance will promote the reabsorption of uric acid.
acid, and this will add to the correlation between hyperuricemia and high blood pressure. Recent research has shown that there is a correlation between the TyG index and hyperuricemia. Furthermore, this correlation was shown to be stronger than the one between obesity indices and hyperuricemia in general Chinese populations.

Therefore in the present study aimed to investigate the Association between serum uric acid and triglyceride-glucose index in adult hypertensives.

**Aims and Objectives:**
**Aim:** To evaluate the Association between serum uric acid and triglyceride-glucose index in adult hypertensives

**Objectives:**
- Among the adult hypertensives in a tertiary care hospital, the followed parameters estimated and analysed.
  - To estimate serum uric acid levels
  - To estimate fasting blood glucose levels
  - To estimate triglyceride levels
  - To calculate Triglyceride-glucose index
- To determine association between serum uric acid and triglyceride-glucose index.

**Materials and Method:** This is a observational cross sectional study of 200 cases in the age group of 20-30 yrs attending to the Department of General Medicine, SVRR Govt. General Hospital S.V. Medical college, Tirupati, for a period of three months after obtaining the date of approval from Institutional scientific and ethics comitee. after gaining each participant's informed consent. The interviews were conducted one-on-one with each participant, and a standardised questionnaire was used to collect data on the participants' gender, age, weight, height, waist circumference, hip circumference, smoking status, level of physical activity, and diet.

**Inclusion Criteria:**
- Diagnosed hypertensive patients with systolic blood pr 2140mm of Hg and diastolic blood pressure ≥ 90 mm of Hg or I Subjects who have given written informed consent

**Exclusion Criteria:**
- Study participants with Pregnancy, myocardial infarction, stroke
- Subjects with chronic kidney disease, chronic liver disease, cancer, Hypertensive patients on lipid lowering drugs

**Sample size calculation:** The prevalence of hypertension (cumulative previously diagnosed and new cases) among individuals aged 15 to 49 years was 35.1%.

It was estimated that 35.1% of people suffer from hypertension. The size of the sample was determined based on the acquired prevalence, which was found to be 64.9%, at a confidence interval of 95% and an absolute precision of 10%.

\[ n = \frac{4pq}{L^2} \]

- \( n \) = Minimum sample size
- \( p \) = Prevalence in percentage
- \( q = 100-p \)
- \( L \) = Allowable error in percentage of prevalence.

Using the above formula and data,

- \( p = 35.1\% \)
- \( q = (100-p) =100-35.1= 64.9 \)  \( L = 20\% \)

Minimum sample \( n \) = \( \frac{4 \times 64.9 \times 35.1}{7 \times 7} \) = 188
The minimum sample size 188 case by the following sampling method. But the convenience purpose 200 sample was obtained.

After an overnight fast of 8-12 hours, venous blood samples were collected from all of the participants. After that, the samples were allowed to clot at room temperature for 1-3 hours, and then the serum was separated by centrifuging the samples for 15 minutes at 3000 rpm. Uric acid, triglycerides were assessed after being extracted from these samples. A sample of venous blood was drawn into a sodium fluoride tube for the purpose of glucose measurement.

Fasting glucose will be estimated by Glucose oxidase peroxidase method

Triglycerides estimated by glycerol phosphate oxidase/ peroxidase method

Serum uric acid estimated by Uricase POD method

TyG index was calculated with established formulas according to the previous studies: \( \text{TyG} = \ln \left( \frac{\text{TG (mg/ml)} \times \text{FPG (mg/ml)}}{2} \right) \)

Blood pressure measured by sphygmanomaneter

**Statistical Analysis**

Statistical software SPSS version 20.0 was used for statistical analysis. The data presented as mean, median, standard deviation, Correlation analysis done and pearsons calculated.

**Results:**

**Table no: 1.Baseline Characteristics of study participants by Gender**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Total (n=200)</th>
<th>Male (n=112)</th>
<th>Female (n=88)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>31.5±15.2</td>
<td>34.2±14.2</td>
<td>28.5±12.5</td>
<td>0.003</td>
</tr>
<tr>
<td>SBP ,mm hg</td>
<td>114.6±12.4</td>
<td>124.6±12.8</td>
<td>110.2±11.4</td>
<td>P &lt; 0.0001</td>
</tr>
<tr>
<td>DBP ,mm hg</td>
<td>72.8±9.5</td>
<td>79.6±5.4</td>
<td>75.6±6.8</td>
<td>P &lt; 0.0001</td>
</tr>
<tr>
<td>Serum Uric acid</td>
<td>5.18±1.3</td>
<td>5.12±0.9</td>
<td>4.1±1.2</td>
<td>P &lt; 0.0001</td>
</tr>
<tr>
<td>Fasting Glucose</td>
<td>174.2±32.5</td>
<td>154.5±45.2</td>
<td>135.2±23.5</td>
<td>P = 0.0004</td>
</tr>
<tr>
<td>Serum Triglyceride</td>
<td>198.78±75.23</td>
<td>152±45.3</td>
<td>113.64±76.32</td>
<td>P &lt; 0.0001</td>
</tr>
<tr>
<td>Triglyceride and Glucose index</td>
<td>9.64±0.52</td>
<td>8.66±0.54</td>
<td>7.23±0.26</td>
<td>P &lt; 0.0001</td>
</tr>
</tbody>
</table>

In total 200 participants, 112 were men, 88 were females. Table 1 shows the mean ± SD of age, SBP, DBP, Serum uric acid, fasting Glucose, Serum triglyceride, triglyceride and Glucose index.

**Table no.2 : Correlation between Tryglyceride index and other parameters**

<table>
<thead>
<tr>
<th>Regression Statistics - TyG</th>
<th>Pearson R</th>
<th>R square</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>0.21</td>
<td>0.04</td>
<td>0.0232</td>
</tr>
<tr>
<td>SBP ,mm hg</td>
<td>0.17</td>
<td>0.03</td>
<td>0.0625</td>
</tr>
<tr>
<td>DBP ,mm hg</td>
<td>0.17</td>
<td>0.03</td>
<td>0.0576</td>
</tr>
<tr>
<td>Serum Uric acid</td>
<td>0.30</td>
<td>0.09</td>
<td>0.0007</td>
</tr>
<tr>
<td>Fasting Glucose</td>
<td>0.85</td>
<td>0.73</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Serum Triglyceride</td>
<td>0.70</td>
<td>0.49</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>
Correlation analysis of triglyceride glucose index with other parameters shows positive correlation with age, SBP, DBP, Serum uric acid, fasting glucose, serum Triglyceride.

Discussion: The present study suggested that the relationship between serum uric acid and triglyceride-glucose index is limited to young hypertensive patients. A number of epidemiological studies have reported that HUA is accompanied with hypertension. The positive correlation between elevated Serum uric acid levels and hypertension has been described in many populations, and increasing amounts of evidence suggests that sUA is a causal contributor to hypertension. In a large cohort study involving 2062 participants with a mean follow-up of 21.5 years it was reported that high Serum uric acid levels concentration was independently associated with the incidence of developing hypertension (RR: 1.05, 95% CI, 1.01–1.10, P = .02). Renal insufficiency and liver disease are associated with HUA, and they are also well-established mechanisms for secondary hypertension. We, however, excluded patients with renal impairment (eGFR < 60 mL/min/1.73 m²) and liver disease (serum glutamic pyruvic transaminase > 80 IU/L). Therefore, the association between HUA and hypertension in the present study is not due to renal insufficiency or liver disease.

Increased Serum uric acid levels in asymptomatic and uncomplicated people with essential hypertension may indicate early renal vascular abnormalities, including a reduction in cortical blood flow and a depressed tubular secretion of urate caused by its reduced delivery to the tubular secretory sites. This depressed tubular secretion of urate is caused by the fact that essential hypertension causes a reduced delivery of urate to the tubular secretory sites. The potential relevance of Serum uric acid levels in reflecting and predicting the vicious cycle that leads to increased renal impairment and raised blood pressure needs to be clarified through investigations. These studies are required. An increase in the activity of the sympathetic nervous system has also been linked to a decrease in the amount of uric acid that is excreted by the kidneys. On the other hand, the fundamental mechanics are a mystery. A high level of insulin in the blood can lead to a decrease in the amount of uric acid and salt that is excreted in the urine. This might happen as a result of a reduction in tubular secretion, an increase in reabsorption, or both. Elevated Serum uric acid levels may be a reflection of both of these mechanisms due to the fact that hyperinsulinemia may increase the activity of the sympathetic nervous system. In addition, insulin may act as a mediator in the direct relationship that exists between Serum uric acid levels and proximal tubular sodium reabsorption.

It was shown that when insulin resistance is present, glycolysis intermediates are converted to 5-phosphoribose and phosphorhic acid ribose pyrophosphate. This results in an increase in the synthesis of serum uric acid. Additionally, high levels of insulin brought on by insulin resistance drive sodium-hydrogen exchange in renal tubules, which in turn leads to an increase in the excretion of hydrogen ions as well as a rise in the amount of uric acid that is reabsorbed. whereas the activation of the renin-angiotensin system caused by hyperinsulinemia lowers renal blood flow, increases urate reabsorption, and generates xanthine oxidase, all of which lead to an increase in uric acid production. xanthine oxidase is also responsible for the formation of uric acid.

Conclusion: In adults who had hypertension, researchers discovered that there were positive relationships between the TyG index and serum uric acid, as well as between the TyG index and hyperuricemia. The TyG index is a simple, inexpensive, and independent risk factor that has the potential to serve as an attainable indicator in the treatment of hypertension and hyperuricemia.

References:
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