A Study of Serum Uric acid levels with Acute Myocardial Infarction and its correlation with Killip’s classification

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The aim of this study is to estimate the levels of serum uric acid in patients with acute myocardial infarction and also to study the correlation with Killip’s Classification of Cardiac failure. This is a cross sectional study, we selected the patients from ICCU and emergency department. 30 patients with AMI and age matched controls taken for study. Uric acid levels measured in Autoanalyser Beckman Coulter AU 480. The mean serum uric acid among the study group and the control group on the day of admission (Day 0) was 7.6 ± 1.6 and 5.4 ± 1.2 respectively. There was a gradual increase in the SUA levels on Day 0, 3 and 7 and was proportionate with the increasing Killip’s class. It is concluded that the serum uric acid (SUA) levels are increased significantly among patients diagnosed with acute myocardial infarction (AMI) when compared with the controls. It is also evident that there is a significant correlation between SUA levels and the severity of the AMI based on the Killip’s classification. Higher the Killip’s class, higher the uric acid levels. Therefore, SUA shall be used as a marker in diagnosing and as a cofactor useful along with other clinical examinations and investigations to interpret the severity of AMI as well. However, additional work must be done to understand the full clinical potential of Uric acid and correlation with other cardiac failure parameters.

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INTRODUCTION
Coronary heart disease (CHD) is the single most cause of death in the developed countries and is one of the leading causes of disease burden in developing countries as well. The CHD death rate however varies dramatically across the developing countries both as a proportion of cardiovascular disease (CVD) deaths and as a proportion of all deaths, (Gaziano et al., 2010) Clinical studies have proved that serum uric acid (SUA) is significantly associated with cardiovascular disease (Baker et al., 2005; Brodov et al., 2009). Uric acid is considered to be an independent predictor of major adverse cardiovascular events (MACE) in patients with CHD (Wannamethee et al., 1997; Culleton et al., 1999). Uric acid, the catabolic end product of purines and the levels are sex-dependent (Liu et al., 2017). Hyperuricemia is a condition when serum uric acid is above 6 mg/dL in women and 7 mg/dL
in men (Shani et al., 2016; So and Thorens, 2010).

An increased concentration of serum uric acid is responsible for platelet reactivity mediating inflammation and stimulation of smooth muscle cell proliferation which probably worsens acute thrombosis (Patetsios et al., 2001). Uric acid induces endothelial dysfunction by activating the HMGB1/RAGE signaling pathway, says a study (Cai et al., 2017). This mechanism plays an important role in the pathogenesis of thrombosis leading to ischemia and thereby proceeding to infarction.

This study estimates the levels of serum uric acid in patients with acute myocardial infarction at three different days since the day of infarction (0, 3 and 7 days) and correlates the serum uric acid levels along with the Killip’s classification, thereby highlighting the importance of SUA levels in AMI.

MATERIALS AND METHODS

This study is a prospective study, which included 60 patients between the age group 20-55 attending the ICU and the Emergency department and diagnosed as AMI compared to 60 age and sex-matched controls. Study Group A included the patients who presented with a history of chest pain and diagnosed as ST-segment elevation acute myocardial infarction (STEMI) or non-ST segment elevation acute myocardial infarction (NSTEMI) on the basis of history, examination, ECG changes and biochemical markers like troponin T, CK-MB and CK-Total. The control group was named as Group B. Patients with history of chronic kidney disease, gout, hypothyroidism, haematological malignancy and those on drugs like diuretics, salicylates affecting the SUA levels were excluded from the study.

Blood sample was collected and estimated for serum uric acid (SUA) by uricase method in Beckman Coulter AU480 auto analyzer.

RESULTS AND DISCUSSION

The mean age of the study Group A was 46 ± 8 and the mean age of the control Group B was 52 ± 2. The study group consisted of 18 women and 12 men in Group A, patients diagnosed with STEMI and NSTEMI, and 14 women and 16 men in Group B, controls.

With the help of the collected data, the mean age Statistical analysis by Independent ‘t’ test and Pearson’s correlation analyses by standard statistical methods determines the relationship between variables. p value<0.05 was considered statistically significant. The mean SUA among the study group and the control group on the day of admission (Day 0) was 7.6 ± 1.6 and 5.4 ± 1.2 respectively shown in Table 1.

Tables 2, 3 and 4 explain the mean SUA of the patients on Day 0, 3 and 7. From the tables, it is evident that the SUA are higher in concentration with an increase in the Killip’s classification.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean SUA on Day 0</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>7.6 ± 1.6</td>
</tr>
<tr>
<td>B</td>
<td>5.4 ± 1.2</td>
</tr>
</tbody>
</table>

From the study, it was evident that the SUA was increased in patients diagnosed with AMI. Moreover, it was also found that there was a comparative increase in SUA among each class of Killip. To be more precise, there was a gradual increase in the SUA levels as the Killip’s class was also increasing.

Table 2: Comparison of SUA with Killip’s class on Day 0

<table>
<thead>
<tr>
<th>Killip’s class</th>
<th>N</th>
<th>SUA (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>13</td>
<td>7.2 ± 1.1</td>
</tr>
<tr>
<td>II</td>
<td>8</td>
<td>7.7 ± 1.6</td>
</tr>
<tr>
<td>III</td>
<td>4</td>
<td>8.1 ± 0.9</td>
</tr>
<tr>
<td>IV</td>
<td>5</td>
<td>7.9 ± 1.2</td>
</tr>
</tbody>
</table>

According to a study conducted by Ehmoda et al. and Okazaki H et al., it was evident that the SUA levels were high among patients with AMI and correlated with the adverse effects post-AMI (Elneihoum et al., 2014; Okazaki et al., 2016). This was in concordance with our study.

Table 3: Comparison of SUA with Killip’s class on Day 3

<table>
<thead>
<tr>
<th>Killip’s class</th>
<th>N</th>
<th>SUA (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>15</td>
<td>7.6 ± 0.8</td>
</tr>
<tr>
<td>II</td>
<td>9</td>
<td>8.1 ± 1.6</td>
</tr>
<tr>
<td>III</td>
<td>3</td>
<td>8.6 ± 1.1</td>
</tr>
<tr>
<td>IV</td>
<td>3</td>
<td>8.7 ± 1.4</td>
</tr>
</tbody>
</table>

The sample outcome was also observed in a study was the increased SUA levels correlated with the risk of incident heart failure. The SUA levels were also correlated to the adverse outcomes as well. This study was conducted by (Huang et al., 2014).

Regarding the concept of using SUA as a predictor of mortality, there are several studies conducted explaining the increased SUA concentration among higher classes of Killip. The study conducted by Nadkar M et al. is one among them where it was found
that SUA were increased in AMI patients and were correlated with Killip’s class. This was in accordance with our study. Nadkar et al. also found that SUA is a good predictor of mortality after AMI (Nadkar and Jain, 2008).

<table>
<thead>
<tr>
<th>Killip’s class</th>
<th>N</th>
<th>SUA (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>17</td>
<td>8.3 ± 1.3</td>
</tr>
<tr>
<td>II</td>
<td>9</td>
<td>8.5 ± 1.2</td>
</tr>
<tr>
<td>III</td>
<td>2</td>
<td>8.9 ± 1.7</td>
</tr>
<tr>
<td>IV</td>
<td>2</td>
<td>9.2 ± 0.7</td>
</tr>
</tbody>
</table>

This fact is also supported by the study conducted by (Patil et al., 2015; Biswas et al., 2016). Patil L et al. found that among the 20 patients who expired post-AMI, 16 had higher SUA levels. 19 of them belonged to Killip’s class IV. On all days (Day 0, 3, 7) SUA were high in higher Killip’s classes. This was in contrast to our study, where we found that the SUA is gradually increasing from Day 0 to Day 3 and Day 3 to Day 7.

The study of Biswas K et al. concludes that Killip’s class III and IV had higher SUA levels than II and I. This was in accordance with our study.

Yan L et al. studied SUA among 5686 patients and concluded that it is a good prognostic marker in patients with AMI (Yan et al., 2014). This was matching to the results of our study where the mean SUA was 7.6 md/dl and high compared to the control group with a mean SUA of 5.4 mg/dl.

There were also studies on Clinical SYNTAX score (CSS) and SUA. Xiong Z et al. found that SUA level was significantly proportional to the severity and complexity of Coronary heart disease. He also found that SUA was high with high CSS with a mean of 8.3 mg/dl (Xiong et al., 2011).

This study was also supported by the one conducted by Ekici B et al., which concluded that there is a positive correlation between SUA and CSS. The mean SUA in severe CSS was 6.5 mg/dl, (Ekici et al., 2015).

Thus, our study concludes that the SUA levels are higher in AMI when compared to the controls with a significance of p < 0.001. Moreover, the SUA level gradually increases on Days 0, 3 and 7. Further, there was a positive correlation of the SUA levels with the Killip’s classes I, II, III and IV with a higher significance of p < 0.001. Thus, the higher the Killip’s class, the higher the SUA levels.

CONCLUSIONS

From the study, it is concluded that the serum uric acid (SUA) levels are increased significantly among patients diagnosed with acute myocardial infarction (AMI) when compared with the controls. It is also evident that there is a significant correlation between SUA levels and the severity of the AMI based on the Killip’s classification. Higher the Killip’s class, higher the uric acid levels. Therefore, SUA shall be used as a marker in diagnosing and as a cofactor useful along with other clinical examinations and investigations to interpret the severity of AMI as well.

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Ethical Clearance

Taken from Institutional Ethical Committee No — 1361/IEC/2018.

Conflict of Interest

The authors declare that they have no conflict of interest.

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