Original Research Article

Study of Hs Troponin I & uric acid in patients of myocardial infarction

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ABSTRACT

Objective: We aimed to provide Correlation of Hs Troponin I & Uric Acid in patients of Myocardial Infarction.

Materials and Methods: 100 patients who came to cardiac emergency in Shri Mahant Indresh Hospital. Serum samples taken for Hs Troponin I and Uric Acid for patients of Myocardial Infarction and run on VITROS 5600/7600 which is based on dry chemistry.

Results: With 100 patients of more than 40 years of age 61 were males & 39 were females. For both males & females age mean & SD was 59.8±10.77.

In our study we took 100 random patients coming to cardiac emergency out of which 50 patients had raised trop I and 45 patients had raised uric acid levels. Out of those 50 patients with raised HS Trop I 25 patients had raised values for uric acid. For Hs Trop I males – 21.88±48.8 & females 1676±57.58.

For uric acid for males-6.545±3.75 & for females- 6.315±1.86.

Therefore Hs Trop I & uric acid were both significant when compared with age T value was 2.7001 and P value was 0.0075. Whereas when compared with sex that is male and female to both Hs Trop I and uric acid then Hs Trop I was more significant with P value 0.0001.

Conclusion: Uric acid is an economical marker that is readily, quickly and reliably obtainable & can be one of the predictable prognostic indicator in acute Myocardial Infarction.

A R T I C L E  I N F O

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1. Introduction

Coronary artery disease (CAD) is a worldwide health epidemic. Coronary artery disease develops when the major blood vessels that supply our heart become damaged or diseased. Cholesterol containing deposits in our coronary arteries and inflammation are usually to blame for coronary arteries.

Symptoms of CAD-

1. Chest pain
2. Shortness of breath

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3. Heart attack

The Global burden of disease Study reported that in 1990 there were 5.2 million deaths from cardiovascular diseases in economically developed countries and 9.1 million deaths from the same causes in developing countries.1 The prevalence of CAD in India increased from 1% in 1960 to 9.7% in 1995 in urban populations & in rural population it is most doubled in past decade.2

There has been growing interest in the link between uric acid levels, xanthine oxidoreductase and cardiovascular disease. Previous studies have reported that a high concentration of uric acid is a strong marker of an unfavourable prognosis of moderate to severe heart failure.
and cardiovascular disease. Uric acid levels may be elevated in heart failure and provide important prognostic information.

A failing heart due to acute MI may cause tissue hypoperfusion and hypoxia, which trigger xanthine oxidase activation and oxidative stress. Xanthine oxidase and oxidative stress as reflected by uric acid levels may form a vicious cycle that promotes severe heart failure.

Myocardial Infarction (MI) is the leading cause of mortality from cardiovascular disease and accounts for around 1 million deaths in China annually. Thus prevention of MI through greater understanding and reduction of risk factors has significant implications for public health and clinical practice. Serum uric acid the end product of purine metabolism has been proved to be associated with hypertension, diabetes mellitus, obesity & dyslipidemia all of which are principal contributors in development and progression of MI & may reduce longevity of affected individuals however whether serum Uric Acid is an independent risk factor for MI and all cause mortality has been under debate.

Following Myocardial infarction(MI) some proteins and enzymes labelled cardiac markers (CPK-MB, Troponin T&I) are released in blood in large quantity from necrotic heart muscle. Epidemiological studies have recently shown that uric acid may be a risk factor for cardiovascular diseases and a negative prognostic marker for mortality in subjects with pre existing heart failure. Adenosine synthesized locally by vascular smooth muscle in cardiac tissues rapidly degraded by the endothelium to uric acid, which undergoes rapid efflux to the vascular lumen due to low intracellular pH and negative membrane potential. Xanthine oxidase activity and uric acid synthesis are increased in vivo under ischaemic conditions and therefore elevated serum uric acid may act as a marker of underlying tissue ischaemia. Although the mechanism by which uric acid may play pathogenetic role in cardiovascular disease is unclear, hyperuricemia is associated with deleterious effects on endothelial dysfunction, oxidative metabolism, platelet adhesiveness & aggregation.

2. Aims & Objective

We aimed to provide correlation of Hs Troponin I & Uric Acid in patients of Myocardial Infarction. And provide significance of uric acid in patients of Myocardial Infarction.

3. Materials and Methods

100 patients who came to cardiac emergency in Shri Mahant Indresh Hospital. Serum samples taken for Hs Troponin I and Uric Acid for patients of Myocardial Infarction and run on VITROS 5600/7600 which is based on dry chemistry.

4. Result

Among the 100 patients of more than 40 years of age 61 were males & 39 were females. For both males & females age mean & SD was 59.8±10.77.

Out of which 50 patients had raised trop I and 45 patients had raised uric acid levels. Out of those 50 patients with raised HS Trop I 25 patients had raised values for uric acid.

For Hs Trop I males 21.88 ± 48.8 & females 1676 ± 57.58.

For uric acid for males 6.545 ± 3.75 & for females 6.315 ± 1.86.

Therefore Hs Trop I & uric acid were both significant when compared with age T value was 2.7001 and P value was 0.0075.Whereas when compared with sex that is male and female to both Hs Trop I and uric acid then Hs Trop I was more significant with P value 0.0001.

5. Discussion

The study showed that the MI risk and all cause mortality depended on both Serum uric acid exposure and on the time course of Serum uric acid accumulation. The relationship between Serum uric acid and MI has been debated with conflicting results in previous studies. The AMORIS study and the Rotterdam study have demonstrated a significant association between Serum uric acid & MI. In contrast, the Tromso study and the NHANES (National Health and Nutrition Examination Survey) III study have failed to establish an independent association between Serum uric acid and MI. First, Serum uric acid is a product of xanthine oxidoreductase, which is known to be one of the most important sources of reactive oxygen species, High Serum uric acid is therefore associated with increased vascular endothelial function, vascular smooth muscle cell proliferation and oxidative stress thereby increasing the risk of MI and all-cause mortality. Second, high Serum uric acid exerts a plethora of deleterious effects in cells and thus may be directly involved in the pathophysiological characteristics of MI and all-cause mortality. Third, high Serum uric acid is correlated with almost all known cardiovascular risk factors, such as metabolic syndrome and chronic kidney disease thus, a higher level of Serum uric acid may be seen as correlation of cardiovascular risk or an epiphenomenon of coexisting cardiometabolic risk factor.

6. Conclusion

In acute MI, patients with hyperuricemia had higher mortality. Serum uric acid levels correlated with Killip classification in acute MI. Serum uric acid can be used as a marker of short term mortality in patients. Hyperuricemia is an indicator of poor prognosis in acute MI.

Uric acid is an economical biomarker that is readily, quickly and reliably obtainable, it can be one of the
The authors declare no conflict of interest.

Table 1:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>H.S Tropin Mean±SD</th>
<th>Uric acid Mean±SD</th>
<th>T value</th>
<th>P value</th>
<th>Significant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>59.8±10.77</td>
<td>59.8±10.77</td>
<td>0</td>
<td>1</td>
<td>NS(P≥0.05)</td>
</tr>
<tr>
<td>Observed value</td>
<td>20.59±52.55</td>
<td>6.375±3.184</td>
<td>2.7001</td>
<td>0.0075</td>
<td>S(P&lt;0.05)</td>
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Table 2:

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<td>0.0001</td>
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<td>Uric Acid</td>
<td>6.545±3.751</td>
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Predictable prognostic indicator in acute Myocardial Infarction.

7. Source of Funding

None.

8. Conflict of Interest

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References


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