**Risk of Coronary Heart Disease with Raised Serum Uric Acid**

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A possible association of hyperuricemia with risk of Coronary Heart Disease (CHD) has been noted in several studies. In this study it is reported that in 222 patients who had experienced myocardial infarction, 68.5% had serum uric acid levels above 6 mg/dl and 31.5% had normal serum uric acid. Thus, raised serum uric acid level may be associated with Coronary Heart Diseases.


**Key words:** Coronary heart disease, uric acid.

**Introduction**

Uric acid (urate), an organic compound comprised of carbon nitrogen, oxygen and hydrogen, is the final oxidation product of purine metabolism. For decades it has been hypothesized that the oxidant properties of uric acid might be protective against aging, oxidative stress and oxidative cell injury. However, recently epidemiological and clinical evidences suggest that hyperuricaemia might be a risk factor for cardiovascular disease where enhanced oxidative stress play an important pathophysiological role. It has also hypothesized that hyperuricaemia might be involved in chronic heart failure and metabolic syndrome.1 The high incidence of coronary artery disease (CHD) in patients suffering from gout has been known for over 50 years, but the question of the more specific relationship of hyperuricemia to CHD has been raised comparatively recently. The increased incidence of hyperuricemia in patients with CHD had been shown by several investigators.2-4 It is shown that there is a significant relationship between hyperuricemia and hypertension.5 Hansen found significant relationship in 36% of 115 patients with acute cerebral infarction.6 Others found hyperuricemia in 32% of 59 patients suffering from cerebrovascular disease.7 A wider range of normal values has recently been established by epidemiological studies.8

Despite these important findings, little attention has been paid to the possible relationship between hyperuricemia and CHD. The present study was undertaken to further clarify the apparent association between raised serum uric acid level and coronary heart disease.

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Methods
An unselected series of 222 consecutive patients with various types of CHD admitted in Coronary Care Unit, Mymensingh Medical College Hospital from January, 1991 to December, 1992 and In Dinajpur Medical College Hospital during the year 2007-2008, were investigated.

The diagnosis of CHD was based on detailed clinical examinations, ECG and Serum Enzyme study (e.g. SGOT, Serum LDH). Conditions such as hypothyroidism, hyperthyroidism, polycythemia and other blood dyscrasias and dysproteinaemias were excluded, since these are known to alter serum uric acid levels. In addition to routine laboratory tests, measurements of serum uric acid, blood urea and serum creatinine were carried out in each patient. Patients with acute renal failure were not included in this study and those with moderate or advanced renal disease were excluded. Serum uric acid (SUA) level was determined by Phosphotungstic Sodium Cyanide Method.9

Results
Table I summarizes the pertinent data in 222 patients. The order of listing and numbering the value has been arbitrarily arranged according to age. In 152 (68.5%) patients out of 222 patients. SUA level was above 6 mg/dl and 70 (31.5%) was below 6 mg/dl. Patients having SUA level below 6 mg/dl, 20 were between the age of 40-49 years, 32 were between 50-59 years and 18 were between 60 and above years, and those of above 6 mg/dl 10 were between 30-39 years, 34 were between 40-49 years, 46 were between 50-59 years and 62 were between 60 and above years.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>SUA levels</th>
<th>Total</th>
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<tbody>
<tr>
<td></td>
<td>&lt;6</td>
<td>&gt;6</td>
</tr>
<tr>
<td>30-39</td>
<td>10</td>
<td>20</td>
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<tr>
<td>40-49</td>
<td>20</td>
<td>34</td>
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<tr>
<td>50-59</td>
<td>32</td>
<td>46</td>
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<tr>
<td>&gt; 60</td>
<td>18</td>
<td>62</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>152</td>
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Discussion
Although our results clearly demonstrate the present co-existence of high SUA level and CHD, the explanation for this association is not obvious. Gertlen and associates10 have incorporated the SUA level in their so-called CUP ratio: K equals cholesterol level (mg/dl) multiplied by SUA level (mg/dl) and divided by phospholipid level (mg/dl). This is used in assessing the proneness to CHD of a given person and thus, these workers recognized that the higher the SUA level, just as the higher the cholesterol, the greater is the probability that the CHD would eventuate. It is known that hyperuricemia is inherited as a simple Mendelian dominant characteristic.11 Genetic or hereditary predisposition has been considered to be a most significant factor in the development of CHD itself, one might speculate to the genetic linkage of hyperuricaemic and CHD.12,13 Trasak AM et al demonstrated in a 21 years follow up study in 28,613 elderly women that SUA is an independent risk factor for all major forms of deaths from CVD including acute, subacute and chronic forms of CHD, CHF and stroke in elderly, post-meopausal women.14

Regardless of the explanation for the association of hyperuricemia and CHD, the predictive value, of this parameter seems apparent. Of greater importance is the question of what part, if any, uric acid plays in the pathogenesis of coronary atherosclerosis. Here again little is known of the role played by uric acid in the
development of vascular disease. Others demonstrated deposits of urate crystals in the proliferated intima of arteries or in organized thrombi, and this, of course, represents one mechanism by which uric acid may participate in the process of vascular degeneration. Another possible factor is that uric acid is a surface active agent and may facilitate the deposition of lipids in the subintimal tissues of atherosclerotic vessels.

Gur M et al in a study of 851 cases, having CAD in 495 patients and 356 individuals with normal coronary angiogram, concluded that uric acid level is associated with the presence but not with the severity of the coronary artery disease.

Conclusion

It would appear that more investigative work along these lines is indicated for the elucidation of the role of uric acid in CHD. This is particularly important, since there is already available a most satisfactory uricosuric agent for the control of elevated SUA levels, namely, probenecid.

References