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In conclusion, the neutrophil and platelet activation, known predisposing factors to thrombosis, together with increased CRP production and reduced NO production, might share in causing the hyperuricemia-associated endothelial dysfunction and atherosclerotic plaque formation. Therefore, it could be recommended that physicians should be aware of the role of elevated uric acid in inducing cardiovascular insult, and that individuals suffering from hyperuricemia should be advised to have a strict follow-up for their platelet function, which could participate in the cardiovascular pathology.

Figure 2.

Changes in plasma nitrate in the normouricemic and the different hyperuricemic groups.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>5-days hyperuricemia</th>
<th>2-weeks hyperuricemia</th>
<th>4-weeks hyperuricemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control rats</td>
<td>3.2±0.38 (7)</td>
<td>3.0±0.56 (9)</td>
<td>NS</td>
</tr>
<tr>
<td>Hyperuricemic rats</td>
<td>3.0±0.59 (9)</td>
<td>2.8±0.39 (7)</td>
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<td>Neutrophil (%)</td>
<td>31.6±2.93 (7)</td>
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<td>Lymphocyte %</td>
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Table 1. Results of the changes in leucocyte parameters in the different studied groups.

P: Significance of difference from matched control rats calculated by Student's ″t″test for unpaired data. NS: Not significant.
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<th>TLC (x10^3/µl)</th>
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Is Hyperuricemia A Risk Factor to Cardiovascular Disease?

http://dx.doi.org/10.5772/54871
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Figure 4. Graphs showing correlations of plasma uric acid versus neutrophil %, lymphocyte %, and platelet aggregation in all the studied groups of rats.
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References


