Comparative study of serum LDH and uric acid in hypertensive versus normotensive pregnant woman

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Abstract

Aims & Objectives: Our aim is to compare the serum levels of Lactate dehydrogenase (LDH) and Uric Acid in hypertensive versus normotensive pregnant woman and correlate their levels with the severity of the disease.

Material & Methods: 25 antenatal patients having hypertension (Group A) & equal no. of normotensive patients (Group B) were chosen. Three readings of blood pressure were recorded after 5 minutes rest, the interval between the measurements and the average value was recorded. About 3 ml of blood was drawn under aseptic precautions from selected subjects in a plain vial for serum. Serum was separated by centrifugation and used for estimation of serum levels of LDH and UA.

Observation & Results: Demographic parameters and baseline values were nonsignificant between two groups. We have observed significant difference in serum LDH and Uric acid in hypertensive group patients in comparison with normotensive patients.

Conclusion: We conclude that serum LDH and Serum Uric acid are reliable and inexpensive markers to predict severity and outcome of hypertensive disorders of pregnancy

Keywords: LDH, Uric acid, hypertensive disorders.

1. Introduction

Pregnancy is a physiological state associated with many alterations in metabolic, biochemical, physiological, hematological and immunological processes. If there are no complications, all these changes are reversible following a few days to a few months after delivery[1].

Hypertension during pregnancy is a major health problem. It is one of the leading causes of perinatal morbidity and mortality.[2][3][4] Preeclampsia (PE) is a theoretical disease with a pathogenesis that is not clearly understood yet. Lately vascular system pathology and vasoconstriction have been blamed as causes for preeclampsia.

During early pregnancy, there is increased body fat accumulation associated with increased lipogenesis, while in late pregnancy, there is accelerated breakdown of fat depots which play an important role in fetal development.[5] Early pregnancy dyslipidemia is associated with an increased risk of preeclampsia[6].

Several studies have been carried out till date to understand the pathophysiological basis of this disease. But still the exact pathophysiology of this disease is not known. Lactate Dehydrogenase (LDH) is mainly an intracellular enzyme. It is responsible for interconversion of pyruvate and lactate in the cells. Its levels are several times greater inside the cells than in the plasma.[7] Uric acid (UA) is an end product of purine metabolism. It is filtrated through the glomeruli and almost completely reabsorbed in the proximal convoluted tubules (PCT) by both active and passive carrier mediated processes. It is also actively secreted into the tubules. 85% of total excreted UA is derived by tubular secretion. Hyperuricemia is found to be one of the earliest laboratory manifestations of preeclampsia.[8]
Our aim is to compare the serum levels of Lactate dehydrogenase (LDH) and Uric Acid in hypertensive versus normotensive pregnant woman and correlate their levels with the severity of the disease.

2. Material & Methods

After approval from the Institutional Ethical Committee and informed written consent, this prospective randomized study was carried out. 50 patients who satisfied inclusion & exclusion criteria were recruited from OPD / Obstetric ward of Dhiraj General Hospital, SBKS MIRC through computer randomization and equally divided into two groups.

Group A: Hypertensive antenatal patients (n=25)
Group B: Normotensive antenatal patients (n=25)

2.1 Inclusion criteria
1) Gestational age >20 weeks
2) Primi / Multigravida
3) Antenatal patients of age 20-35 yrs
4) All Antenatal patients normotensive as well as hypertensive, which do not fall under exclusion criteria.

2.2 Exclusion criteria
1) Patients with all maternal and/or fetal abnormalities, known renal disease, diabetes, hepatic dysfunction, alcoholism, dyslipidaemia.
2) Preexisting hypertension before pregnancy except PIH or on any type of anti-hypertensive treatment.

After recruitment, blood pressure (BP) will be measured and on the basis of BP, all the participants will be divided into two groups i.e. normotensive pregnant women and hypertensive pregnant women.

2.3 Blood pressure measurements

Blood pressure was recorded by Residents of Obstetrics & Gynecology in the Obstetric ward & OPD using a mercury sphygmomanometer and stethoscope from the upper arm after the subjects had been sitting for more than 5 minutes according to the guidelines of the American Heart Association. Three readings were recorded after 5 minutes rest interval between the measurements and the average value was recorded.

2.4 Collection of blood samples

About 3 ml of blood was drawn under aseptic precautions from selected subjects in a plain vial for serum. Serum was separated by centrifugation and used for estimation of serum levels of LDH and UA.

The concentration of serum LDH and UA was analysed by using analytical kits from ERBA Diagnostics Mannheim GmbH in semi-autoanaylzer (CHEM-5 Plus V2, Erba Mannheim). Values were calculated as mean ± SD and the statistical analysis was done using GRAPHPAD PRISM V.6.0 software. Student’s unpaired t-test was used for comparison between two groups. The p-value of less than 0.05 was considered as statistically significant.

3. Observation & Results

A total of 50 patients was recruited for the study. There were no significant differences between the two groups in demographic data and such as Age, Height, Weight and Parity (Table 1).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group A</th>
<th>Group B</th>
<th>P value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>23.60 ± 3.43</td>
<td>23.43 ± 2.56</td>
<td>P &gt; 0.05 NS</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>52.8 ± 3.5</td>
<td>52.6 ± 3.37</td>
<td>P &gt; 0.05 NS</td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>154.9 ± 3.94</td>
<td>156.43 ± 3.39</td>
<td>P &gt; 0.05 NS</td>
<td></td>
</tr>
<tr>
<td>Gestational Age (weeks)</td>
<td>34.3 ± 2.18</td>
<td>34.03 ± 1.19</td>
<td>P &gt; 0.05 NS</td>
<td></td>
</tr>
<tr>
<td>Baseline SBP (mmHg)</td>
<td>157.41 ± 8.36</td>
<td>114.52 ± 6.32</td>
<td>P &lt; 0.05 S</td>
<td></td>
</tr>
<tr>
<td>Baseline DBP (mmHg)</td>
<td>96.24 ± 6.65</td>
<td>70.13 ± 5.21</td>
<td>P &lt; 0.05 S</td>
<td></td>
</tr>
</tbody>
</table>

NS – Non Significant  S- Significant

Figure 1: Comparison of Serum LDH (IU/L)

<table>
<thead>
<tr>
<th>Serum LDH</th>
<th>Group A</th>
<th>Group B</th>
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<tbody>
<tr>
<td></td>
<td>328.4</td>
<td>146.2</td>
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</table>

Table 1: Demographic Profile
We have observed significant difference in serum LDH and Uric acid in hypertensive group patients in comparison with normotensive patients. (Figure 1 and Figure 2)

4. Discussion

Pre-eclampsia is a common medical complication of pregnancy. In India, the incidence of pre-eclampsia is reported to be 8-10% of the pregnancies[9]. It contributes significantly to maternal and fetal mortality and morbidity. Pre-eclampsia is a multisystem disorder characterized by hypertension to the extent of 140/90 mm Hg or more, proteinuria (≥300mg/day) and edema induced by pregnancy after 20th week[10]. Without intervention, pre-eclampsia progresses to eclampsia, this is characterized by malignant hypertension and epileptiform convulsions requiring emergency caesarean section[11].

Many theories have suggested that endothelial dysfunction caused by factors released from ischemic placenta may be a causative factor for disease pathogenesis[12].

In our study, we have observed a significant increase in serum LDH and uric acid level in women with hypertension in comparison with normotensive women. These findings were in accordance with a study done by Qublan et al[13] and Kozic et al[14]. They concluded that serum LDH can be a useful marker for the prediction of adverse outcome of pregnancy in severe preeclampsia. Serum LDH has also found to be a useful predictor of birth of small for gestational age infants in preeclamptic pregnancy[15]. Previous studies demonstrated the importance of amniotic serum LDH level for the prediction of fetal growth restriction.

It is found that LDH-A(4) isoenzyme is immunolocalized primarily in the fetal endothelial cells while LDH-B(4) isoenzyme is predominantly present in syncytiotrophoblasts. The LDH-A(4) isoenzyme activity increased approximately by 1.6-fold in preeclampsia when compared with normal pregnancy. This may also suggest that endothelial dysfunction present at uteroplacental vessels can lead to hypoperfusion to the growing fetus & may lead to elevation of LDH isoform[16].

Hypertensive disorders of pregnancy are commonly associated with a decrease in renal function due to damage done by hypertension and widespread endothelial dysfunction. Glomeruli undergo structural changes with pronounced endothelial cell swelling, vacuolization and hypertrophy of the cytoplasmic organelles known as “glomerular endotheliosis”. The net effects have reduced renal blood flow, reduced GFR, impaired tubular reabsorption & secretory function[17][18]. In our study, we found that the mean serum UA levels were significantly higher in cases when compared with controls. This finding is in accordance with the study done by Punthumapol et al[19]. It is found that estimation of serum UA is as important as proteinuria in identifying the risk of renal involvement and fetal compromise[20]. Maternal hyperuricemia is found to be a strong predictor of maternal disease progression and fetal outcome. Thus, it can be used as a useful and inexpensive marker for predicting disease severity, renal function status and fetal growth retardation in women presenting with HDP[21]. In our study, mean levels of serum LDH & UA were significantly higher in group A when compared with group B (p<0.05). These findings indicate that increased levels of these parameters are seen as the disease severity increases.

5. Conclusion

We conclude that serum LDH and Serum Uric acid are reliable and inexpensive markers to predict severity and outcome of hypertensive disorders of pregnancy.
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References


