Study of Liver Enzymes and Proteinuria in Pregnancy Induced Hypertension

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Abstract:

Background: Pregnancy induced hypertension is a leading cause of morbidity and mortality in pregnant women. Preeclampsia and Eclampsia, Sepsis and Haemorrhage are the prime killers in pregnancy.

Aim: The aim of the study is to assess the antepartum severity and complications of preeclampsia by estimating liver enzymes and proteinuria.

Materials And Methods: The study comprises of 60, third trimester pregnant women, among those 24 were clinically diagnosed as Preeclamptic cases, 16 were Eclamptic cases and 20 were Normotensive antenatal controls. Liver enzymes and 24 hrs proteinuria levels were estimated and compared in them.

Statistical Analysis: Results were analysed using epi info 3.5.4 by students t-test

Results:— Mean values of LDH in cases is 699.42 U/L,SGOT is 43.95 U/L,SGPT is 39.82 U/L,ALP is 167.75 U/L,24Hrs Urinary protein in cases is 926.22 mg/day, and in controls the mean values were 347.5 U/L, 21U/L,19.7U/L,119.9U/L, 124.35mg/day respectively.

Conclusion: Mean values of LDH, SGOT, SGPT, ALP, 24Hrs Urinary protein levels were significantly higher in pregnancy induced hypertension cases when compared with normotensive antenatal controls

Keywords: Liver enzymes, Pregnancy induced hypertension, Proteinuria

I. Introduction

Pregnancy induced hypertension (PIH) is the most common hypertensive disorder of pregnancy. It is the 2nd most common killer disease in pregnancy.[1] It complicates almost 10% of pregnancies [2]. PIH is a pregnancy specific multiorgan syndrome. It increases the maternal and perinatal morbidity and mortality. PIH occurs due to imbalance between thromboxane A2 (TXA2) and prostacyclin (PGI2) trophoblastic hypoperfusion and endothelial dysfunction.[3]

It is categorized into Preeclamptic and Eclamptic toxemia. Preeclampsia is characterized by a clinical triad of Hypertension (>140/90 mm of Hg), proteinuria and edema developed after 20 weeks of gestation. Eclampsia is its severe form with superadded convulsions.

Abnormal placentation, vasospasm and placental vascular insufficiency are the core features in PIH. Angiotensin mediated contraction of endothelial cells result in leakage and subendothelial deposition of platelets and fibrinogen which in turn leads to hemorrhage, necrosis, hypoxia of surrounding tissues and endorgan disturbances.

PIH occurs mainly due to insufficient invasion of trophoblast into uterine wall which converts the uterine spiral arteries into low resistance vessels due to low levels of maternal HCG and CG hormones. This increases the resistance to uterine blood flow and results in occurrence of gestational hypertension.[4]

Intense spasm of hepatic arterioles produce hepatocyte damage,hemorrhage,subcapsular hematomas, periportal hemorrhagic necrosis and fibrin deposits in liver resulting in elevated liver enzymes lactate dehydrogenase(LDH),serum glutamyl oxaloacetate transaminase (SGOT),serum glutamyl pyruvate transaminase (SGPT), Alkaline phosphatase(ALP) in serum.

HELLP syndrome is a complication that occurs in 0.5-0.9% pregnancies and 10-20% women with severe preeclampsia. It is common in third trimester. It is a triad of Hemolysis, elevated liver enzymes and low platelet count.. Elevated LDH levels is an indicator of hemolysis,so by estimating liver enzymes, LDH and 24 hrs proteinuria we are trying to assess the severity and complications of PIH as per criteria developed by university of Mississippi as of 1999.[5]

Increased turnover of trophoblastic tissue, tissue ischemia, decreased renal clearance, glomerular endotheliosis results in protein leakage in urine.

II. Aim

This study is aimed to assess the severity and complications in PIH by estimating liver enzymes and 24 hrs urinary protein in both cases and controls and comparing

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III. Materials And Methods

Present study was carried out in Department of Biochemistry and Department of Obstetrics and Gynaecology of S.V Medical college Tirupati on a total of 60 pregnant woman in third trimester. Among those 24 were clinically diagnosed preeclamptic cases, 16 were eclamptic cases and 20 were normotensive age matched antenatal controls.

3.1 Inclusion Criteria
Cases: Pregnant woman in third trimester clinically diagnosed to have PIH with blood pressure >140/90 mm of Hg on 2 separate occasions more than 6 hrs apart with bilateral pedal edema, proteinuria and convulsions
Controls: Age matched normotensive third trimester pregnant woman with no pedal edema proteinuria and convulsions

3.2 Exclusion Criteria
Pregnant woman in first and second trimesters. Woman with previous H/O Hypertension, Diabetes, chronic renal failure, congestive cardiac failure, convulsions. Pregnant woman with anemia, chronic infections, sepsis, asthma, etc.,

3.3 Sample Collection
After taking informed consent, 6ml of venous blood sample was collected from antecubital vein in aseptic conditions immediately after admission, before commencement of treatment and serum was separated and serum LDH, SGOT, SGPT, ALP were estimated immediately by using pyruvate as substrate, modified IFCC method, p-NPP method (Dr.Reddy’s lab kit) respectively in ERBA semiautoanalyser. 24 hrs urinary samples were collected in thymol crystals containing sterile cans and protein was estimated in them using 3% sulphasalicylic acid method/TCA Precipitation method.

3.4 Statistical Analysis
Data analysed by epi info 3.5.4 software by student’s t test.

IV. Results

Table 1: Mean & S.D Values In Pregnancy Induced Hypertension Cases And Normotensive Controls

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>UNITS</th>
<th>CASES</th>
<th>CONTROLS</th>
<th>t-VALUE</th>
<th>p-VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDH</td>
<td>U/L</td>
<td>699.42±252.23</td>
<td>347.5±98.8</td>
<td>5.9</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>SGOT</td>
<td>U/L</td>
<td>43.95±14.02</td>
<td>21±6.66</td>
<td>6.8</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>SGPT</td>
<td>U/L</td>
<td>39.82±12.56</td>
<td>19.7±6.5</td>
<td>6.6</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>ALP</td>
<td>U/L</td>
<td>1191.44±862.98</td>
<td>167.75±44.42</td>
<td>4.0</td>
<td>&lt;0.01*</td>
</tr>
</tbody>
</table>

Table 2: Mean & S.D Values In Preeclampsia And Eclampsia Cases

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>UNITS</th>
<th>PREECLAMPSIA</th>
<th>ECLAMPSIA</th>
<th>t-VALUE</th>
<th>p-VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDH</td>
<td>U/L</td>
<td>690.75±241.8</td>
<td>706.04±268.16</td>
<td>0.2</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>SGOT</td>
<td>U/L</td>
<td>45.69±13.52</td>
<td>42.79±14.81</td>
<td>1.8</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>SGPT</td>
<td>U/L</td>
<td>41.63±11.92</td>
<td>38.63±13.34</td>
<td>0.7</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>ALP</td>
<td>U/L</td>
<td>178.69±52.69</td>
<td>160.46±38.51</td>
<td>1.6</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>24hrs urinaryproteins</td>
<td>mg/day</td>
<td>926.22±685.76</td>
<td>124.35±59.54</td>
<td>5.1</td>
<td>&lt;0.01*</td>
</tr>
</tbody>
</table>

* significant p-value <0.05

Fig 1: Bar diagram showing mean values of liver enzymes in cases and controls:
Fig 2: Bar diagram showing mean values of 24 hrs urinary protein in cases and controls:

V. Discussion

Our study was carried over pregnancy induced hypertension cases (n=40) and normotensive antenatal controls (n=20) both in third trimester. Among 40 PIH cases taken 24(60%) were preeclamptic cases and 16 (40%) were eclamptic cases.

Mean and S.D values of LDH in cases and controls were 699.42±252.23 and 347.5± 98.8 U/L respectively. So there is a significant increase in serum LDH levels in PIH cases when compared to controls. It is a good indicator of hemolysis in PIH cases. In preeclampsia cases it is 706.04± 268.16 and in eclampsia cases it is 690.75±241.8. No significant variation was found in LDH raise between preeclampsia and eclampsia cases.

Mean and S.D values of SGOT, SGPT, ALP in cases and control are shown in Table 1. And there levels in preeclampsia and eclampsia cases were shown in Table 2. These enzyme levels are significantly raised in PIH cases when compared to normotensive controls and there is no significant variation in the increase in eclampsia cases when compared to preeclampsia cases.

In preeclampsia hypervascularization and vasoconstriction of liver leads to liver cell injury and alteration of cell membrane permeability and damage to the cell which allows intracellular enzyme to leak into the blood leading to elevated liver enzymes like SGOT, SGPT, ALP[6].

Other studies have reported an elevation of liver enzymes in PIH [1,7,8].

Significant 24 hrs urinary proteinuria is seen in PIH cases when compared to controls. Increased levels are significantly higher in eclampsia cases than preeclampsia cases.

In our study there was a significant raise in the liver enzymes and 24hrs urinary protein in PIH cases when compared to normotensive controls of 3rd trimester.

VI. Conclusion

Our study shows that pregnancy induced hypertension is associated with increase in level of liver enzymes like LDH, SGOT, SGPT and ALP and the levels are directly related to the increase in 24 hrs urinary protein. In our study it is once again proved that pregnancy induced hypertension is accompanied by increase in 24 hrs urinary protein and it indicates the severity of PIH. Our study shows no significant variation in the increased levels of liver enzymes and 24 hrs urinary protein between preeclampsia and eclampsia cases. Early diagnosis of PIH and its complications can bring down the maternal and fetal morbidity and mortality and can improve the fetoal outcome.

References