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A Study of Liver Profile in Pregnancy Induced Hypertensive Women and Compare with Normal Pregnant Women

Varsha Butolia[1], Ashok.L. Bajentri[1], Sumitra Kumari[1,] Babita Garhwal[1]
[1]Department of Physiology, Pacific Institute of Medical Sciences,
Sai Tirupati University, Udaipur, Rajasthan, India

***Corresponding Author:**
Varsha Butolia
Ph.D. Scholar, Department of Physiology, Pacific Institute of Medical Sciences,
Sai Tirupati University, Udaipur, Rajasthan, India

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Abstract

Background: Pregnancy-induced hypertension (PIH) is a form of high blood pressure in pregnancy. It occurs in about 7 to 10 percent of all pregnancies. Another type of high blood pressure is chronic hypertension - high blood pressure that is present before pregnancy begins. Pregnancy-induced hypertension is also called toxemia or preeclampsia. It occurs most often in young women with a first pregnancy. It is more common in twin pregnancies, and in women who had PIH in a previous pregnancy.

Methods: This paper focuses on Identify the importance of biochemical parameter in pregnancy induced hypertension including liver profile and their implications in the evolution of the disease.

Results: The present study showed that the mean and standard deviation of ALP, SGOT and SGPT significantly high in Pregnancy induced Hypertension patients compare to normal pregnant women with p value <0. 0001. And mean standard deviation of Albumin is significantly low in Pregnancy induced Hypertension patients compare to normal pregnant women with p value <0. 0001.

Conclusions: Our study also shows that ALP, SGOT, SGPT value is higher and low albumin in preeclampsia patient especially in sever preeclampsia. The Liver Profile might be a useful laboratory marker for clinical prediction and disease severity evolution of preeclampsia.

Keywords: ALP, SGOT, SGPT,ALB

Introduction

In obstetrics, hypertensive disorders of pregnancy (HDP) are the most important issue. Pre-eclampsia rates in Indian hospitals range from 5% to 15%, while eclampsia rates are at 1.5% [1]. Pregnancy-related hypertension problems can occur anywhere from 1 to 35% of the time [2]. They rank among the most prevalent pregnancy issues and are associated with higher rates of maternal and perinatal morbidity and mortality.

Cellular death occurs as a result of the multisystemic, multifactorial condition known as preeclampsia. Women with hypertensive disorders during pregnancy frequently have their serum AST, ALT, LDH, and uric acid tested. In the research, there is disagreement on the relationship between these factors and the severity of preeclampsia. Boissier de Sauvages distinguished between epilepsy and eclampsia in the 18th century. De Sauvages provided his opinions on the cause of convulsions in addition to the distinction he made in disease classification. According to Temkin (1971), he thought that convulsions were the result of nature attempting to purge the organism of any unhealthy elements. Dr. Robert Johns also realized in 1843 that there was a link between premonitory symptoms in the final

trimester of pregnancy and the onset of puerperal convulsions. According to Johns (1843), these premonitory symptoms included a headache, brief loss of eyesight, excruciating stomach pain, and edema of the hands, arms, neck, and face.

Stroganoff's major goal was to stop convulsions because they interfered with the heart, lungs, kidneys, and liver's normal activities (Speert, 1958). He managed the eclampsia and disregarded An anti-angiogenic factor is the endoglin protein, which regulates placental trophoblast development in the uterus (Caniggia, Taylor, Ritchie, Lye, & Letarte, 1997) and maintains vascular tone (Jerkic et al., 2004; Toporsian et al., 2005).

Given that placental and blood pressure anomalies are seen in pre-eclampsia, they may be possibly involved in the development of the condition [3].

Virchow first reported hepatic abnormalities in women with fatal eclampsia in 1856. The periportal bleeding in the liver's periphery was the hallmark lesion. In 1973, Sheehan and Lynch reported that half of the women who died from eclampsia had some degree of hepatic infarction with bleeding. These matched up with publications from the 1960s that mentioned higher serum hepatic transaminase levels. Infarction can occasionally result in hepatic failure, often known as shock liver, and it can be made worse by hypotension brought on by obstetrical bleeding.

Hyperuricemia is one of the earliest laboratory signs of preeclampsia (Powers, 2006). It most likely occurs from impaired glomerular filtration, increased tubular reabsorption, and decreased secretion, which all contribute to reduced uric acid clearance (Lindheimer, 2008a). Preeclampsia has a complicated pathophysiology, with an aberrant placentation as its main cause. Ernest first proposed the idea that the placenta's reduced perfusion is the pathophysiology of pregnancy-induced hypertension in 1939. There are two stages in the progression of pregnancy-induced hypertension, according to Oxford Group in 1991 and supported by Roberts. Reduced placental perfusion characterizes stage 1, and maternal endothelial cell activation characterizes stage 2. [4],[5]

The decidua and myometrium are invaded by cytotrophoblastic cells during a typical pregnancy. These cells take the place of the spiral arteries' endothelium and media, resulting in the formation of low resistance, big diameter arterioles. Vasopressors no longer work as well on them, and the placenta receives more blood flow. [6]

Only the decidual vessels in PIH experience incomplete endovascular trophoblastic invasion; the myometrial vessels do not. These vascular changes result in endothelial damage, myointimal cell proliferation, medial necrosis, and lipid buildup in myointimal cells and macrophages. Hertig (1945) referred to this as arthrosis. Hyperinflation, hypoxia, and the discharge of placental debris are caused by superimposed thrombosis.

Due to elevated levels of the majority of coagulation factors and decreased anticoagulant action, normal pregnancy is a hypercoagulable state. Because the endothelium has been damaged, there is an accentuation of the hypercoagulable condition in pregnancy-induced hypertension.

Platelet counts, bleeding times, clotting times, prothrombin times, and activated partial thromboplastin times are all aspects of the coagulation profile that are investigated in pregnancy-induced hypertension. Prior to the development of complications such HELLP Syndrome, disseminated intravascular coagulation, and cerebrovascular issues, these measurements are helpful in determining the severity of coagulation abnormalities in pregnancy-induced hypertension at an earlier stage.

Vasospasm and endothelial dysfunction, fibrin deposition, varying degrees of hepatic ischemia damage, microangiopathic hemolytic anemia, and thrombocytopenia are the hallmarks of the HELLP syndrome.

The Sibai et al. [7]. suggested triad of hemolysis, increased liver enzymes, and thrombocytopenia. One of the main symptoms of the disorder is hemolysis, which is brought on by a microangiopathic haemolytic anemia that can be identified by the presence of fragmented (schizocytes) or contracted red cells with spicula (Burr cells) in peripheral blood smears, increased reticulocyte counts, elevated serum lactate dehydrogenase levels (LDH, >600U/L), and decreased hemoglobin concentrations. A more accurate predictor of hemolysis and the presence of unconjugated bilirubin (>1.2 mg/100 ml) is a low

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haptoglobin concentration (1 g/L - 0.4). Elevated liver enzyme levels can indicate both liver involvement and the hemolytic process. Increased aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels are primarily brought on by liver damage, whereas elevated LDH levels are largely caused by hemolysis. Due to their increasing consumption, platelets' count is decreased in people with the HELLP syndrome; activated platelets stick to injured vascular endothelial cells.[8] The diagnosis of the whole form of this disorder requires the presence of all three major components, while partial or incomplete HELLP syndrome only includes one or two aspects of triad [9]. This condition is thought to be a variation or complication of severe preeclampsia.

Endothelium changes from being antithrombotic to prothrombotic when the production of vasodilators and antiplatelet agents, such as prostacyclin and nitric oxide, is reduced and endothelin levels are elevated. The fibrinolytic system is downregulated.

The cause of thrombocytopenia is

1. a shorter platelet lifespan
2. Increased consumption of platelets
3. Reduced prostacyclin production
4. Immunological processes.

Materials and Method

This is an observational analytical case control study conducted to find the changes that occur in the neutrophil-lymphocyte ratio in pregnancy induced hypertension as compared to that in normal pregnancy. The liver profile was determined among pre-eclampsia patients, (who are admitted to the maternity ward and labour room of the Obstetrics and Gynaecology department, Pacific Institute of Medical Sciences Umarda Udaipur, and in the control groups, having age and anthropometrically matched apparently healthy pregnant women for a period of two years. A total number of 100 patients admitted at Pacific Institute of Medical Sciences Udaipur, was form the subjects of the present study. Out of these 50 patients were suffering from pre-eclampsia and eclampsia., and 50 were normal Pregnant women. Efforts will be made to match all anthropometric factors comparable to both the groups of patients.

In Inclusion Criteria Pregnant women between 37 and 42 weeks of gestation with pre-eclampsia and eclampsia and Normotensive pregnant women between 37 and 42 weeks of gestation.

In exclusion criteria Pre-existing medical disorders - diabetes mellitus, chronic kidney disease, any coagulopathies, chronic hypertension, and thyroid disorder, Smokers, alcoholics, Multifetal gestation. Placental abruption or previa.

After obtaining written informed consent from all patients and healthy control, 5 ml of venous blood was collected in Plain vial, and separate the serum using centrifuge machine and run in fully biochemistry analyser EM 360.

Clinical Methodology: Symptoms (hypertension, nausea, vomiting), liver profile was recorded by using Autoanalyzer EM 360.

Statistical Analysis: For the quantitative analysis, we used the software SPSS software. In this meta-analysis, all p values reported were two-tailed with the statistical significance set at $\leqslant$ 0.05.

Result

Table 1: Comparison of Liver Profile between Normal Pregnant Women and Pregnancy induced Hypertension patients

S. No	Test	Normal Patient		Pih Cases		P Value
		Mean	SD	Mean	SD	
1	Alkaline Phosphates	36.70	17.80	344.50	62.28	<0.0001
2	Albumin	4.42	0.44	3.90	0.64	<0.0001

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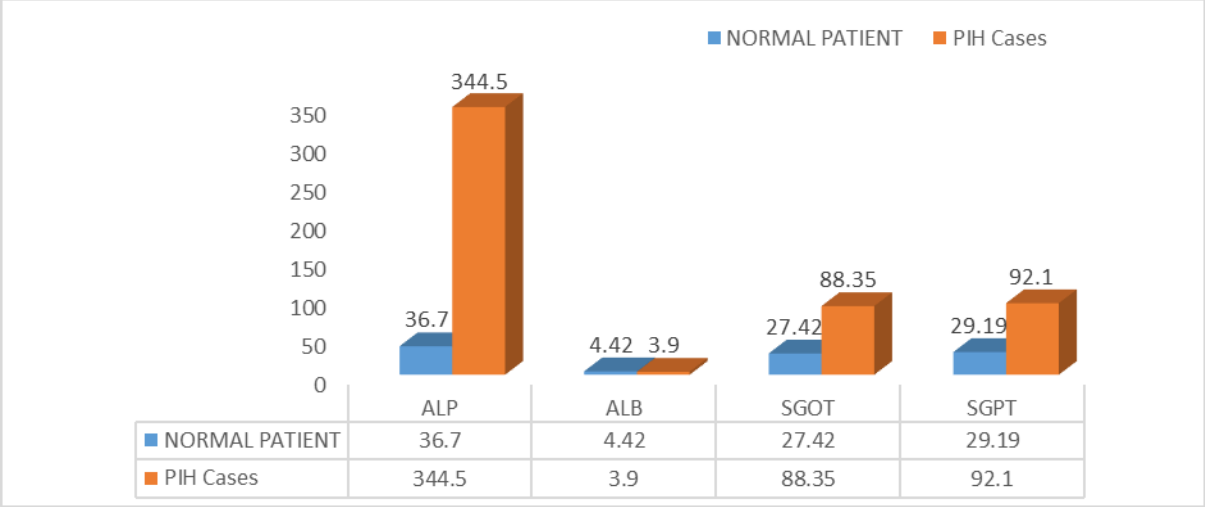
3	SGOT	27.42	8.48	88.35	127.82	<0.0001
4	SGPT	29.19	8.37	92.10	121.82	<0.0001

Fig 1: Comparison of Liver Profile between Normal Pregnant Women and Pregnancy induced Hypertension patients.



The present study shows that the mean and standard deviation of ALP, SGOT and SGPT significantly high in Pregnancy induced Hypertension patients compare to normal pregnant women with p value <0. 0001and mean standard deviation of Albumin is significantly low in Pregnancy induced Hypertension patients compare to normal pregnant women with p value <0. 0001. (Table 1, Fig 1)

Discussion

When compared with Group 2 (cases) and Group 1 (controls), AST, ALT were statistically very significantly raised in cases. This is inconsistent with N. R. Hazari et al study which showed significant increase. This was in relation to another study, increased levels of AST and ALT was found in severe preeclampsia, whereas in mild preeclampsia the increase was within normal range [4,5]. Magnitude of liver dysfunction in the present study as determined by AST was increased in 102 (68%) cases in group 2 whereas it was raised in only 2 control (1.33%) in group 1. AST elevations may be secondary to damage of other organs like heart, kidney, brain, intestine and placenta. ALT was raised in 115 (76.66 %) patients with hypertensive disorder of pregnancy. A rise in Alanine transaminase is known to be more specific to liver damage. Also the increase was statistically significant, AST and ALT were found to be increased in group 2. preeclampsia with HELLP syndrome, end organ damage compared to normotensive control and gestational hypertension patients and non-severe preeclampsia. Our results are in agreement to that of Knapen et al., and Elad Mei-Dan et al. [6,7,8]. Also Girling JC et al reported the higher prevalence of elevated liver function tests (LFT) in preeclamptic group (54%) than gestational hypertension[9].

Conclusion

The present study done on Normal Pregnant women and pregnancy induced hypertension patient admitted in Pacific Institute of Medical Sciences, Udaipur. Total 100 women were included for this study .50 were Normal Pregnant women and 50 were pregnancy induced hypertension patient. 19-35 age group was taken for this study, the study shows that the mean value and standard deviation of ALP, SGOT,SGPT were significantly high in pregnancy induced hypertension patient compare to Normal Pregnant women. Mean standard deviation of Albumin is significantly low in Pregnancy induced Hypertension patients compare to normal pregnant women with p value <0. 0001.

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