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QUANTITATIVE MEASUREMENT OF SERUM UREA, URIC ACID, CREATININE AND HEPATIC BIOMARKERS LEVELS TO ASSESS THE INTENSITY OF PRE-ECLAMPSIA AND ECLAMPSIA AMONG POPULATION IN TRIPURA, NORTH-EAST INDIA.

Biochemistry

Dr. Soumyadip Sarma* Senior Resident, Department of Biochemistry, Shija Academy Of Health Sciences, Imphal, Manipur. *Corresponding Author

Dr. Bappaditya Som Associate Professor, Department of Obstetrics and Gynecology, Tripura Medical College and Dr B.R Ambedkar Memorial Teaching Hospital, Tripura.

ABSTRACT

Background: Pre-eclampsia and eclampsia are often referred as hypertensive disorders of pregnancy or pregnancy induced hypertension (PIH), which is a prevalent disorder of gastrointestinal tract associated with increased fetal and maternal morbidity and mortality. The etiology of the aforementioned disease is yet to be discovered properly. There are often fluctuations in levels of liver function and kidney function markers that have been portrayed as a suggestive of link of severity of PIH. Current study is aimed to estimate serum Urea, Uric acid, Creatinine and hepatic biomarkers levels and their impact upon the severity of pre-eclampsia and eclampsia respectively. **Materials And Methods:** 50 patients admitted in Obstetrics and Gynecology wards of Tripura Medical College constituted case group and 50 normotensive pregnant women constituted control group. Serum samples were collected and analyzed for Urea, Uric acid and Creatinine levels predicting renal functions and liver transaminases (SGOT and SGPT) predicting liver functions. The time period for this study was from August 2019 to July 2020 in Tripura Medical College. **Results:** The levels of serum Uric acid are increased significantly ($p<0.001$), levels being 4.26±0.82 mg/dl in normal pregnant women and 5.58±0.79 mg/dl in patients of pre-eclampsia or eclampsia. There was no visible difference in levels of Urea and Creatinine among study and control groups. There is increase in the levels of both SGOT and SGPT in case group as compared to control group which is significant in nature (p value=0.008). **Conclusions:** Liver transaminases and serum Uric acid are strongly accompanied with pre-eclampsia or eclampsia, and plays a pivotal role for prediction of hepatorenal damage in patients with pregnancy induced hypertension (PIH).

KEYWORDS

SGOT, SGPT, Urea, Creatinine, Uric acid, pregnancy induced hypertension.

INTRODUCTION

Hypertensive disorders of pregnancy are considered to be the most frequent disorders happened in human pregnancies.[1] It may also be categorised as Pre-eclampsia, Eclampsia syndrome and Gestational hypertension.[2] Though they are comparatively non-malignant in nature; yet about half of the cases of benign Gestational hypertension may convert to Pre-eclampsia, which is a fatal condition that can prove dangerous to expectant mothers.[3] Mostly an improperly implanted placenta is commonly believed to be the major underlying cause of the formation of gestational hypertension among humans.[1] There are presence of various research that have been executed to obtain any relation of Urea, Uric acid and Creatinine with Pre-eclampsia or Eclampsia.

Usually in normal uncomplicated pregnancy the Urate clearance is more where in Pre-eclampsia the tubular function of kidney is affected first than the glomerular function and reduced Uric acid clearance produces changes in glomerular filtration rate that leads to hyperuricemia. Though majority of authors agreed that Pre-eclampsia is always accompanied with hyperuricemia, there are also others who differ from this opinion.[4,5,6] There are also presence of contradictory results regarding Urea and Creatinine. Majority of research expressed that there is no significant difference in the obtained mean value of Urea and Creatinine in normotensive pregnant women and patients of Pre-eclampsia. However, few research studies expressed increased Urea and Creatinine levels in the study group.[7]

Placental dysfunction produces ischaemia caused by systemic vasospasm and thrombosis that harms the maternal organs in gestational hypertension. Haemorrhage and thrombosis formation cause injury to the liver parenchyma associated with hepatocellular necrosis which in the long run causes hepatic enzymes elevation.[8]

The current study focussed at estimating the levels of various hepatorenal biomarkers according to the severity of pregnancy induced hypertension (PIH).

AIMS AND OBJECTIVES

A) To Evaluate:

i) Concentrations of serum Urea, Uric acid and Creatinine

ii) Concentrations of SGOT and SGPT in patients with PIH and healthy controls .

B) To Establish:

i) Inter-relationships between hepatic and renal biomarkers among the patients.

MATERIALS AND METHODS

This prospective study was performed in Department of Biochemistry in collaboration with Department of Obstetrics and Gynaecology, Tripura Medical College and Dr B.R Ambedkar Memorial Teaching Hospital, Tripura upon 50 patients with gestational hypertension which constituted the study group. Out of these 50 patients, 15(30%) were associated with mild Pre-eclampsia,25 (50%) were severe Pre-eclamptic and 10 (20%) were with Eclampsia. 50 normotensive pregnant women in the wards of Obstetrics and Gynaecology department were among the control group. The time period for this study was from August 2019 to July 2020 in Tripura Medical College.

We have confirmed the diagnosis of Pre-eclampsia by using the "Report of the American College of Obstetricians and Gynaecologists' Task Force on Hypertension in Pregnancy" criteria. According to these, the patients with systolic blood pressure ≥140 mm Hg along with diastolic blood pressure ≥90 mm Hg (measured after four hours of complete rest, twice daily) associated with proteinuria (≥300 mg protein/24 hours) were considered as Pre-eclamptic. The diagnosis of Eclampsia was confirmed with the association of tonic-clonic seizures here.

Inclusion Criteria:-

It included pregnant females up to 40 years of age and with 20 weeks of gestation and more.

Exclusion Criteria:-

These include any associated neurological, hepatic, cardiac or renal disorders not because of pregnancy induced hypertension, PCOS, multiple pregnancy, associated molar pregnancy, known cases of Diabetes Mellitus and all cases with previous history of essential hypertension or chronic hypertension because of any other causes.

Plain vacutainers were used for collection of venous samples collected for both groups. SGOT and SGPT levels were determined by using IFCC kinetic method on autoanalyzer.[9] Serum Creatinine and Urea levels were estimated by colorimetric Brod and Sirota and Urease method respectively.[10,11] Serum Uric acid levels were estimated by colorimetric method of Caraway.[12] The obtained data was analysed statistically. Received results were statistically explained using student's t-test. A p value of less than 0.05 was taken as significant.

Study Method:-

Ethical committee approval was obtained for the study. Written

informed consent was obtained from all participants of the study.

Statistical Method Of Analysis:-
Statistical analysis was done using SPSS statistical package version 21. Data is expressed in terms of Mean±SD. Unpaired student's t-test was used to compare the results between study group and control group. Statistically significant variation was perceived where p value was less than 0.05.

RESULTS :-
The differences found in age, height, weight, parity, gravity and demographic data were statistically insignificant, Systolic and diastolic blood pressure have been significantly higher among the Pre-eclamptic and Eclamptic patients compared to healthy pregnant women ($p<0.001$ and $p<0.001$, respectively).

The levels of serum Uric acid were significantly increased ($p<0.001$) associated with PIH, levels being 4.26±0.82 mg/dl in normal pregnant women and 5.58±0.79 mg/dl in PIH patients. The mean blood Urea level in the case group was 31.45±6.49 mg% against 30.15±6.1 mg% in the control group, mean serum Creatinine level was 0.99±0.265 mg% in the case group as compared to 0.99±0.255 mg% in the control group. The p value of blood Urea and serum Creatinine was 0.881 and 0.2985 respectively and hence non-significant. (Table No.1).

Mean value of SGOT in the case and control group were 33.37±41.06 IU/L and 28.43±6.33 IU/L (normal range is 6 to 40 IU/L) respectively. The mean value of SGPT in the case and control group were 29.95±10.46 IU/L and 25.25±5.84 IU/L (normal range is 6 to 40 IU/L) respectively. The increase in SGOT levels among cases as compared to controls shows statistically significant association (p value=0.008). Similarly increase in SGPT levels among cases in comparison to controls show statistically significant association (p=0.008). (Table No.2).

Table No.1

Parameter	Case Group	Control Group	P- Value	Significance
Uric acid (mg/dl) Normal range=2-6 mg%	5.58±0.79	4.26±0.82	0.000	S**
Urea (mg/dl) Normal range=20-40 mg%	31.45±6.49	30.15±6.1	0.881	NS*
Creatinine (mg/dl) Normal range=0.7-1.4 mg%	0.99±0.265	0.99±0.255	0.2985	NS*

NS*= Non Significant S**=Significant

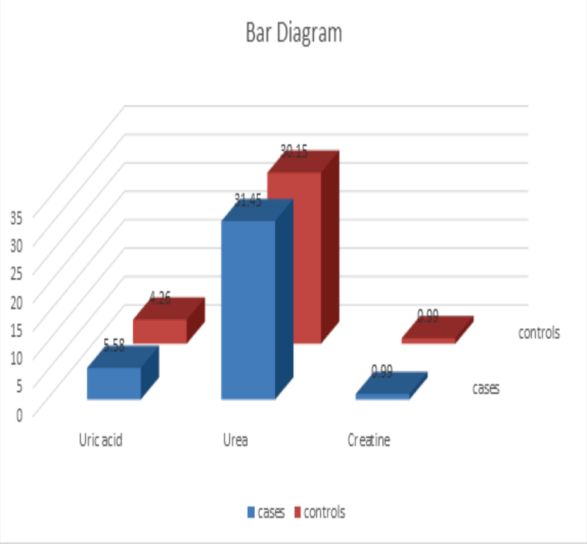

Figure 1

Table No.2

Parameter	Case Group	Control Group	P- Value	Significance
SGOT (IU/L) Normal range=6-40 IU/L	33.37±41.06	28.43±6.33	0.008	S**
SGPT (IU/L) Normal range= 6-40 IU/L	29.95±10.46	25.25±5.84	0.008	S**

S**= Significant

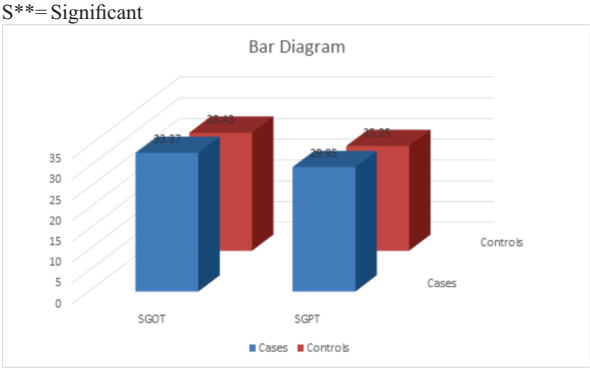

Figure 2

DISCUSSION
The mean serum Uric acid levels in the aforementioned study were 5.58 mg% in the case group and 4.26 mg% in the control group ($p<0.001$) (Figure 1). The obtained findings were in symmetry with the studies conducted by Tsukimori et al.[5](2008) who got 6.6±1.5 mg% serum Uric acid in the case group. Similar findings were obtained by Magna Manjareeka et al[4] (2013) and Jeyabalan et al1[3] (2007). The rise in the mean serum Uric acid concentration value in the Pre-eclamptic women was highly significant as compared to the normotensive pregnant women because in women associated with Pre-eclampsia, glomerular filtration rate and renal plasma flow are reduced as a consequence of enhanced afferent arteriolar resistance or decreased ultrafiltration coefficient. In another study conducted by Shannon A et al (2007) observed that the increase of Uric acid among Pre-eclamptic women mostly produces proteinuria and hypertension. Increased production of Uric acid obtained from placental, maternal and foetal tissues by enhanced tissues breakdown could also prove this enhanced concentration.[15]

Increased Uric acid reduces proliferation of endothelial cells, placental development and migration, hampers placental amino acid uptake, invasion and incorporation of trophoblast into endothelial monolayers.[16]

So it can be speculated that levels of serum Uric acid are predictive of pregnancy induced hypertension. Therefore, it can also be speculated that Uric acid may play direct roles in the pathogenesis of Pre-eclampsia at both the level of maternal vasculature and the placenta itself.

In this current study, the result of Urea and Creatinine levels were insignificant. These are comparable to Magna Manjareeka et al[14] (2013). On the contrary, the study conducted by Israa A MJ et al[7] (2012) showed increase in blood Urea and Creatinine levels in patients with Pre-eclampsia as compared to normotensive pregnant women. These parameters are considered to be of little value in the diagnosis of Pre-eclampsia.

SGOT and SGPT levels had mean value as 33.37 IU/L and 29.95 IU/L respectively with p value= 0.008 which is significantly indicative of Pre-eclampsia (Figure 2).

The clinical trial organized by Seymour consisting 85 patients with PIH; ALT and AST were increased in 54% patients with Pre-eclampsia, whereas the gestational hypertensive cases were raised in 14% cases only. It can Also be indicated that patients with abnormal liver function tests had bad maternal and foetal consequences.[17]

There are no difference in AST and ALT levels between women with

Pre-eclampsia and normal pregnant women in the study conducted by Peralta et al.[18] The liver enzymes are found to be elevated due to the obstruction of hepatic blood flow by sinusoidal fibrin deposit. Peripheral necrosis is happened by this obstruction. There is also severe Pre-eclampsia which is associated with haemolysis and thrombocytopenia which causes elevated serum transaminase levels indicative of hepatocellular necrosis.[8]

LIMITATION

Study samples have been obtained of 20 weeks of gestational period. So these biomarkers do not project proper follow of trends in this disease. However, this study acts as an adjuvant to the present data.

CONCLUSION

In the aforementioned study, we can conclude that liver transaminases and Uric acid are cheaper and feasible markers which provide early signals of hepatorenal complications among patients with gestational hypertension. Proper history obtaining along with proper screening of this condition should be done as soon as possible which will be beneficial in reducing maternal and foetal morbidity and mortality.

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