



International Journal of Reproductive Medicine & Gynecology

Research Article

Biochemical Changes in Pregnancy-Induced Hypertension at a Tertiary Care Hospital - 8

Devika Gupta^{1*}, Swarn Kanta² and Rita Thakur³

¹Department of Obstetrics and Gynaecology, MVJ Medical College and Research Hospital, Bengaluru, Karnataka, India

²Associate Professor, Department of Obstetrics and Gynaecology, Government Medical College, Jammu and Kashmir, India

³Department of Obstetrics and Gynaecology, Government Medical College, Jammu and Kashmir, India

***Address for Correspondence:** Devika Gupta, Department of Obstetrics and Gynaecology, MVJ Medical College and Research Hospital, Bengaluru, Karnataka, India, Tel: +91-941-922-0583; E-mail: drdevikagupta@gmail.com

Submitted: 25 October 2022; **Approved:** 30 October 2022; **Published:** 31 October 2022

Cite this article: Gupta D, Kanta S, Thakur R. Biochemical Changes in Pregnancy-Induced Hypertension at a Tertiary Care Hospital. Int J Reprod Med Gynecol. 2022 Oct 31;8(1): 007-013. doi: 10.37871/ijrmg.id62

Copyright: © 2022 Gupta D, et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.



ABSTRACT

Aim: To study the biochemical changes in hypertensive disorders in pregnancy in our hospital.

Background: Hypertensive Disorders in Pregnancy (HDP) remains a major global health issue because of significant perinatal mortality and morbidity, which effect 6-8% of pregnancies.

Methodology: A non-randomized prospective observational study was conducted in the postgraduate department of obstetrics and gynecology, SMGS hospital Jammu from November 2018 to October 2019 after getting approval from the ethical committee. The study is aimed to evaluate the biochemical changes in 300 pregnant females with hypertensive disorders of pregnancy.

Results and Conclusion: Out of 300 patients, 179 had gestational hypertension, 100 had preeclampsia, and 21 had eclampsia. Hypertensive disorders of pregnancy were significantly associated with low platelet count (60.6%), deranged liver function tests (SGOT (32.6%), SGPT (35.6%), S. bilirubin (18.6%), Serum Lactate dehydrogenase (28%)), deranged Renal function tests (S. creatinine (35%), serum uric acid (25.3%)), deranged coagulation profile (PT (29.3%)) and albuminuria. 119 (39.6%) patients were induced.

Keywords: Hypertensive disorders; Pregnancy; Albuminuria; Serum uric acid

ABBREVIATIONS

LFT: Liver Function Tests; SGOT: Serum Glutamic Oxaloacetic Transaminase; SGPT: Serum Glutamic Pyruvate Transaminase; LDH: Serum Lactate Dehydrogenase; RFT: Renal Function Tests; PT: Prothrombin Time

INTRODUCTION

Hypertensive disorders of pregnancy were reported in 6-8% of pregnancies [1]. THE WORKING GROUP of the National High Blood Pressure Education Programme (NHBPEP) (2000) NHBPEP classified hypertensive disorders of pregnancy into four types: Gestational Hypertension, Preeclampsia – eclampsia, Chronic Hypertension, and preeclampsia superimposed on chronic hypertension [2]. Gestational hypertension is the new onset of hypertension after 20 weeks of gestation with BP > 140/90 with no proteinuria. Preeclampsia is defined as hypertension after 20 weeks of gestation with one or more of the following: proteinuria, maternal end-organ dysfunction (including renal, hepatic, hematological, or neurological complications), or fetal growth restriction. Eclampsia is the development of convulsions in a pre-existing preeclampsia, or it may appear unexpectedly in a patient with minimally elevated blood pressure and no proteinuria. Chronic hypertension with superimposed pre-eclampsia includes new onset proteinuria in hypertensive women but no proteinuria before 20 weeks of gestation. Risk of perinatal mortality and morbidity increases in patients of pre-eclampsia and eclampsia. About 70% of hypertensive disorders are due to gestational hypertension and preeclampsia whereas 30% are due to pre-existing or undiagnosed hypertension. The incidence ranges from 61% in primigravida and 39% in multigravida. Maternal complications include HELLP (4.54%), acute renal failure (7.27%), postpartum hemorrhage (23.63%), abruptio placentae (7.27%), pulmonary edema (0.90%), cerebral hemorrhage (0.7%) [3].

METHODOLOGY

A non-randomized prospective observational study was conducted in the department of obstetrics and gynaecology at SMGS Hospital, Jammu, from November 2018 to October 2019. A total of 300 patients with hypertensive disorders in pregnancy were included in the present study after satisfying the inclusion and exclusion criteria. Inclusion criteria: patients beyond 20 weeks of pregnancy with pregnancy-induced hypertension irrespective of age and parity. Exclusion Criteria: Chronic Hypertension, Chronic renal disease, Coarctation of aorta, Endocrine disorder (diabetes mellitus, pheochromocytoma, thyrotoxicosis), Connective tissue diseases

(lupus Erythematosus) and patient refusal. All the patients were subjected to detailed history taking, general physical examination, thorough systemic and obstetric examination. The women were grouped into three main categories: Gestational hypertension, Preeclampsia, and Eclampsia based on the clinical presentation at admission. BP was measured using the auscultatory method with standard calibrated, validated instrument. Two BP readings taken 4 hours apart with a value \geq 140/90 mmHg were considered hypertension. Proteinuria of 300 mg/dl (1+ dipstick) protein in a random urine sample was considered significant proteinuria. The onset of convulsions in a woman with pre-eclampsia that cannot be attributed to other causes was considered Eclampsia. Patient with BP reading \geq 140/90 mmHg without significant proteinuria was considered as gestational hypertension. The laboratory evaluation of biochemical parameters in HDP were assessed, which includes platelet count, LFT, RFT, Prothrombin time and urine for albumin.

The data were analyzed using software Microsoft Excel, SPSS version 23 for windows and OPENEPI app. Data were reported as mean \pm standard deviation and proportions as deemed for quantitative and qualitative variables, respectively. The qualitative data were compared using the chi-square test. A *p*-value of < 0.05 was considered statistically significant.

RESULTS

The majority of the females, 242 (80.6%) were in the age group of 21-30 years with a mean age of 26.34 years (26 years 4 months) ranging from 16 to 48 years. Only 9 (3%) females were in the age group > 35 years. About 18 (6%) females were in the age group \leq 20 years (Figure 1).

The majority of females were primigravida (154; 51.3%) followed by second gravida (93; 31%), and only 5 (1.6%) patients were gravida 5 or more (Figure 2).

Maximum number of patients were \geq 37 Weeks gestational age (213; 71%) followed by 34-36 weeks (57; 19%) and \leq 34 weeks (30; 1%) (Figure 3).

Most of the patients in this study had Gestational hypertension (179; 59.7%) followed by Preeclampsia (100; 33.3%) and only 21 (7%) patients had antepartum eclampsia (Figure 4).

The majority of patients with platelet count \leq 1.5 lakhs/mm³ seen in patients with eclampsia followed by preeclampsia and gestational hypertension, respectively. The result is significant at *p* < 0.05 (Table 1).

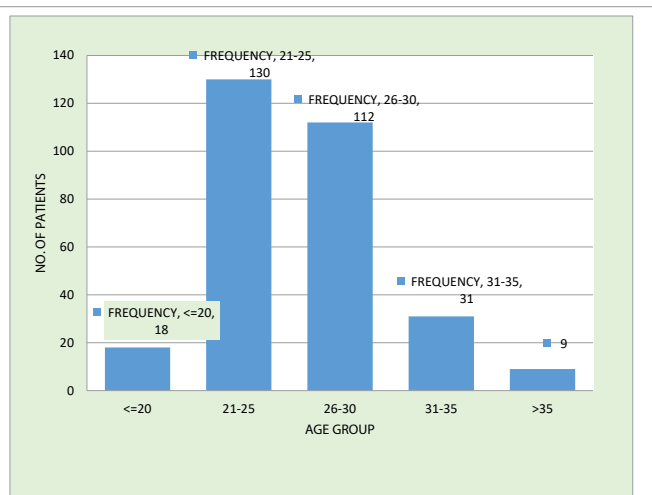


Figure 1: Bar chart showing no. of patients according to age.

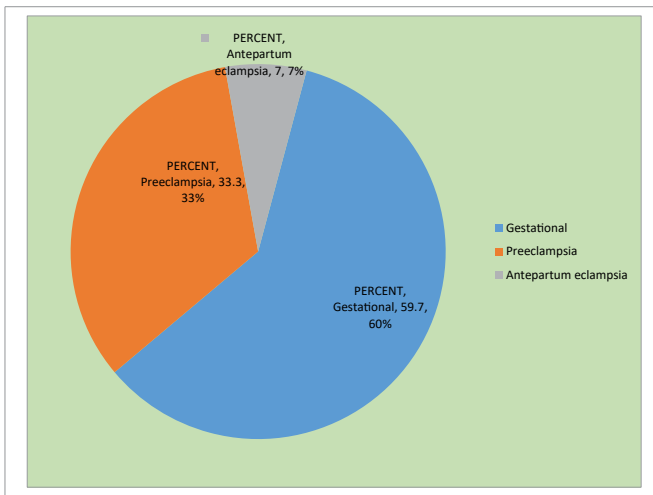


Figure 4: Pie diagram showing the percentage-wise distribution of patients according to the type of hypertension.

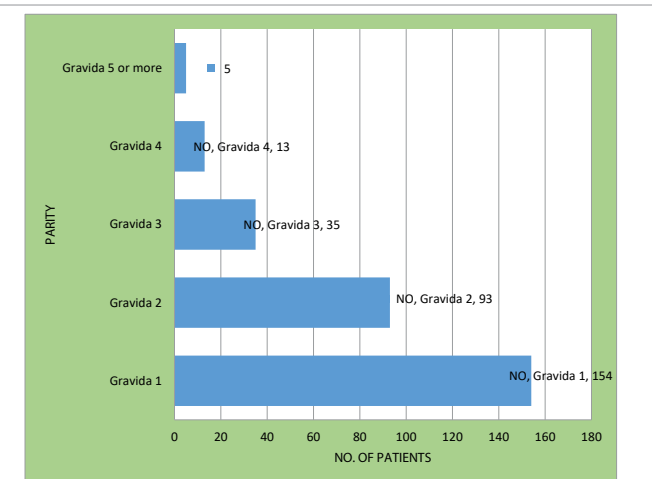


Figure 2: Bar chart showing no. of patients according to parity.

Table 1: Frequency distribution of patients according to platelets count and correlation of platelet count with type of HTN.

Platelet Count (l/mm ³)	G. HTN n = 179	Preeclampsia n = 100	Eclampsia n = 21	Total (n = 300)
≤ 1100.000?	6 (3.3%)	21 (21%)	5 (23.8%)	32(10.6%)
1-1.5150.000?	98 (54.7%)	42 (42%)	10(47.6%)	150(50%)
> 1.5	75 (41.8%)	37 (37%)	6(28.57%)	118 (39.3%)
Total	179 (100%)	100 (100%)	21 (100%)	300

The chi-square statistic is 25.5575. The p-value is .000039. The result is significant at p < .01.

Out of 300, 195 (65%) patients had S. Creatinine < 0.8 mg/dl and 105 (35%) had S. Creatinine ≥ 0.8 mg/dl. The maximum patients with S. creatinine > 0.8 mg/dl were seen with eclampsia followed by preeclampsia followed by gestational hypertension. The p value is < 0.00001 (Table 2).

Why 0.8? (Classification and Diagnosis is greater than 1.1).

The majority of patients with S. bilirubin > 1.1 mg/dl were present in eclampsia followed by preeclampsia and gestational hypertension, respectively. The result is significant at p < 0.05 (Table 3).

The deranged SGOT and SGPT was seen more in patients with preeclampsia followed by eclampsia and gestational hypertension. The result is significant at p < 0.01 (Tables 4,5).

The LDH levels were raised more in patients with eclampsia followed by preeclampsia and gestational hypertension. The p-value is < 0.00001 (Table 6).

224 (74.6%) patients had S. Uric acid < 7 mg/dl and only 76 (25.3%) patients had S. Uric acid ≥ 7 mg/dl. The s. uric acid was elevated more in patients with preeclampsia and eclampsia as compared with gestational hypertension. The p-value is < 0.00001 (Table 7).

The PT was elevated more in patients with eclampsia followed by preeclampsia followed by gestational hypertension. The p-value is < 0.00001 (Table 8).

179 (start number is not good) patients with gestational hypertension had urine for albumin in traces. Out of 100 patients

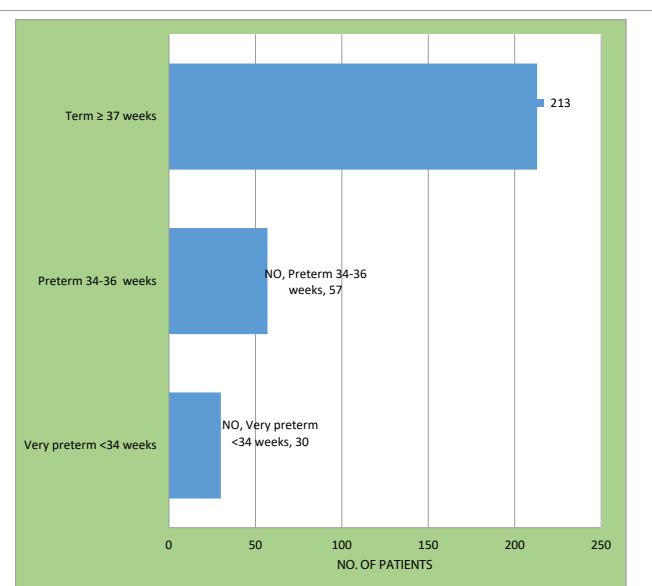


Figure 3: Bar chart showing no. of patients according to the period of gestation.



with preeclampsia, 30 patients had U/A 1+, 55 patients had U/A? 2+, 11 patients had U/A 3+ AND? 4 patients had U/A 4+. Out of 21 patients with eclampsia, 2 patients had U/A traces, 2 patients had U/A 1+, 10 patients had U/A? 2+ and 7 patients had U/A 3+. U/A 4+ was seen in preeclampsia in 4% cases, U/A? 3+ was seen more in eclampsia patients followed by preeclampsia. U/A? 2+ was seen more in patients with preeclampsia followed by eclampsia and early intervention was done. The Chi Square statistic is 315.8. The p -value is $< .0000001$ (Table 9).

Table 2: Frequency distribution of patients according to S. Creatinine and correlation of S. Creatinine with type of hypertension.

S. Creatinine (mg/dl)	G. HTN	Preeclampsia	Eclampsia	Total
< 0.8	165 (92.1%)	27(24%)	3 (14.2%)	195 (65%)
≥ 0.8	14 (7.8%)	73 (73%)	18 (85.7%)	105 (35%)
Total	179 (100%)	100 (100%)	21 (100%)	300 (100%)

The chi-square statistic is 145.33. The p -value is < 0.00001 (highly significant).

Table 3: Frequency distribution of patients according to S. bilirubin and relation between S. bilirubin and type of HTN.

S. Bilirubin (mg/dl)	G. HTN	Preeclampsia	Eclampsia	Total
0.1-1.1	178 (99.4%)	59 (59%)	7 (33.33%)	244 (81.3%)
> 1.1	1 (0.5%)	41 (41%)	14 (66.6%)	56 (18.6%)
Total	179 (100%)	100 (100%)	21 (100%)	300 (100%)

The chi-square statistic is 103.3814. The p -value is $< .00001$. The result is significant at $p < .05$ (even at $p < .01$).

Table 4: Frequency distribution of patients according to SGOT and relation between SGOT and type of hypertension.

SGOT (U/L)	G. HTN	Preeclampsia	Eclampsia	Total
≤ 70	170 (94.9%)	24 (24%)	8 (38%)	202 (67.3%)
> 70	9(5%)	76 (76%)	13 (61.9%)	98 (32.6%)
Total	179 (100%)	100 (100%)	21 (100%)	300 (100%)

The chi-square statistic is 155.6987. The p -value is $< .00001$. The result is significant at $p < .0$.

Table 5: Frequency distribution of patients according to SGPT and relation between SGPT and type of hypertension.

SGPT (U/L)	G. HTN	Preeclampsia	Eclampsia	Total
≤ 70	164 (91.6%)	21 (21%)	8 (38%)	193 (64.3%)
> 70	15 (8.3%)	79 (79%)	13 (61.9%)	107 (35.6%)
Total	179 (100%)	100 (100%)	21 (100%)	300 (100%)

The chi-square statistic is 146.2212. The p -value is $< .00001$. The result is significant at $p < .01$.

Table 6: Frequency distribution of patients according to S.LDH and relation between LDH and type of hypertension.

LDH (U/L)	G. HTN	Preeclampsia	Eclampsia	Total
< 600	176 (98.3%)	34 (34%)	6 (28.5%)	216 (72%)
≥ 600	3 (1%)	66 (66%)	15 (71.4%)	84 (28%)
Total	179 (100%)	100 (100%)	21 (100%)	300 (100%)

The chi-square statistic is 152.8004. The p -value is < 0.00001 (highly significant).

Table 7: Frequency distribution of patients according to s. uric acid and relation between serum uric acid and type of hypertension.

Uric Acid (mg/dl)	G. HTN	Preeclampsia	Eclampsia	Total
< 7	178 (99%)	38 (38%)	8 (38%)	224 (74.6%)
≥ 7	1 (0.5%)	62 (62%)	13 (57.1%)	76 (25.3%)
Total	179 (100%)	100 (100%)	21 (100%)	300

The chi-square statistic is 144.0078. The p -value is < 0.00001 (highly significant).

Table 8: Frequency distribution of patients according to prothrombin time and relation between PT and type and hypertension.

PT (sec)	G.HTN	Preeclampsia	Eclampsia	Total
9.5-13.5	171 (95.5%)	36 (36%)	5 (23.8%)	212 (70.6%)
>13.5	8 (4.4%)	64 (64%)	16 (76.1%)	88 (29.3%)
Total	179 (100%)	100 (100%)	21 (100%)	300 (100%)

The chi-square statistic is 133.6043. The p -value is $< .00001$. The result is significant at $p < .01$.

Table 9: Frequency distribution of patients according to urinary albumin and correlation urine for albumin and type of hypertension (where is R?).

U/A	G. HTN n = 179	Preeclampsia n = 100	Eclampsia n = 21	Total n = 300
Traces	179 (100%)	0	2 (9.5%)	181 (60.3%)
1+	0	30 (30%)	2 (9.5%)	32 (10.7%)
2+	0	55 (55%)	10 (47.6%)	65 (21.7%)
3+	0	11 (11%)	7 (33.33%)	18 (6%)
4+	0	4 (4%)	0	4 (1.3%)
Total	179 (100%)	100 (100%)	21 (100%)	300 (100%)

The Chi-Square statistic is 315.8. The p -value is $< .0000001$. The result is significant at $p < 0.05$.

DISCUSSION

In a study conducted by Joshi P, et al. [6] in Dr. Vanast Rao Pawan Medical College Hospital and Research in 120 cases of HDP. The results seen were, majority of the patients were in the age group of 21-30 years. In another study conducted by Rajamma CK, et al. [7] in Kannur Medical College in Anjarakandy in 233 women, 73.4% were in the age group of 20-30 years with a mean age of 26 years with a standard deviation of 4.6 years. Gandhi MR, et al. [8] conducted a prospective study from February 2014 to January 2015 in the Department of obstetrics and gynecology of GMERS medical college and hospital, Dharpur-Patan, North Gujarat, India. In this study, the incidence of PIH was 63.15% in the age group of 20-30 years. This age of the study is consistent with many studies conducted in India (Figure 1).

In our study, the majority of females were primigravida (154; 51.3%) followed by second gravid (93; 31%) (Figure 2). A study conducted by Rajamma CK, et al. [7] revealed that PIH was more common among primigravida and constituted 43.15 % of the total cases. Study by Bhattacharya S, et al. [5] reported that 65.6% cases were primigravidas. Villar J, et al. [9] and Duckitt, et al. [10] also reported that primigravida was a risk factor for preeclampsia and eclampsia.

In our study, the maximum number of patients were ≥ 37 Weeks gestational age (213; 71%) with mean \pm SD = 37.58 \pm 2.54 followed by 34-36 weeks (57; 19%) and ≤ 34 weeks (30; 1%) (Figure 3).



Donimath KV, et al. [11] conducted a Prospective observational study from November 2014 to June 2015 in the Department of Obstetrics & Gynaecology, Karnataka Institute of Medical Sciences, Hubli, Karnataka, India also concluded that 57% belonged to 37-40 weeks period of gestation. Our study is comparable to the study conducted by Nawaz F, et al. [12], conducted a descriptive-analytic study on 80 hypertensive primigravida women who were referred to Obs/Gynae Unit-C of Hayatabad Medical Complex, Peshawar from January 2012 to December 2012, to evaluate pregnancy outcomes in primigravida women complicated with pregnancy induced hypertension where the mean gestational age was 37.37 ± 2.25 weeks.

In our study, most of the patients had gestational hypertension (170; 59.7%) followed by preeclampsia (100; 33.3%) and only 21 (7%) patients had antepartum eclampsia (Figure 4). In the study by Kolluru V, et al. [13] total hypertensive cases accounted for 234 of the total deliveries; out of which gestational hypertension were 63 (27.3%) cases, preeclampsia 146 (61.6%) and eclampsia 25 (11.1%) cases which were not in accordance with our study because of more number of booked cases in our study, which allowed early detection and adequate treatment in our tertiary care center. Thrombocytopenia is reported frequently in severe preeclampsia. The association of platelet count with the type of hypertension is highly significant ($p < 0.01$) and also observed the decreasing trend of platelet count with increasing severity and the association is statistically significant. Similar association was shown by Poluri, et al. [14]. Thrombocytopenia was present in 5 out of the 100 PIH cases and in which 4 were eclampsia patients and only 1 was that of severe preeclampsia. Therefore, thrombocytopenia was mostly a feature of Eclampsia (Table 1). In our study, out of 100 patients with Preeclampsia, 73 (73%) patients had S. creatinine ≥ 0.8 mg/dl. Out of 21 patients with Eclampsia, 3(14.2%) patients had S. creatinine < 0.8 mg/dl and 7 (85.7%) patients had S. creatinine ≥ 0.8 mg/dl. The association of S. creatinine with hypertension is highly significant (p value < 0.00001) (Table 2). Out of 21 patients with eclampsia, 7 (33.33%) patients had S. Bilirubin 0.1-1.1 mg/dl and 14 (66.6%) patients had S. bilirubin > 1.1 mg/dl. Majority of patients (244; 81.3%) had S. bilirubin in range of 0.1 to 1.1 mg/dl and 56 (18.6%) patients had S. Bilirubin > 1.1 mg/dl. The p - value is $< .00001$. Study conducted by Pillai SS [15] showed that S. bilirubin was elevated in 14.54% cases with severe preeclampsia and eclampsia, which is in accordance to our study (Table 3). Out of 100 patients with Preeclampsia 24 (24%) patients had SGOT ≤ 70 U/L and 76 (76%) patients had SGOT > 70 U/L. Out of 21 patients with eclampsia, 8 (38%) patients had SGOT ≤ 70 U/L and 13 (61.9%) patients had SGOT > 70 U/L. About 202 (67.3%) patients had SGOT ≤ 70 U/L and 98 (32.6%) patients had SGOT > 70 U/L. The association of SGOT with the type of hypertension is significant with $p < .01$ (Table 4). Patil S [16] showed that the mean serum SGOT level in preeclamptic cases was found significantly higher ($p < 0.001$) than the normotensive counterparts. Pillai SS [15] found that SGOT was > 70 U/L in 19.09% cases which was in accordance with our study. In our study, Out of 100 patients with Preeclampsia 21 (21%) patients had SGPT ≤ 70 U/L and 79 (79%) patients had SGPT > 70 U/L. Out of 21 patients with eclampsia, 8 (38%) patients had SGPT ≤ 70 U/L and 13 (61.9%) patients had SGPT > 70 U/L. About 193 (64.3%) patients had SGPT ≤ 70 U/L and 107 (35.6%) patients had SGPT > 70 U/L. The results are significant at $p < .01$ (Table 5). Study by Pillai SS [15], found that SGPT was > 70 U/L in 19.09% cases which is almost in accordance to our study. Study by Patil S [16] showed that SGPT of severe preeclamptic and eclamptic women in this study was significantly ($p < 0.001$) elevated from their normotensive pregnant

counterparts. Malvino E, et al. [17] observed that in preeclampsia the serum transaminase level was raised to > 70 U/L and can rise up to 210 U/L in eclampsia. These results are in accordance with our study. In preeclampsia hyper vascularization and vasoconstriction of liver leads to liver cell injury and alteration of cell membrane permeability and damage to the cells which allows intracellular enzyme to leak in to the blood, leading to elevated liver enzymes like SGOT, SGPT [16]. In our study, out of 179 patients with gestational hypertension, Out of 100 patients with preeclampsia, 34 (34%) patients had S.LDH < 600 U/L and 66 (66%) patients had S.LDH ≥ 600 U/L. Out of 21 patients with eclampsia, 6 (28.5%) patients had S.LDH < 600 U/L and 15 (71.4%) patients had S.LDH ≥ 600 U/L. The result is significant at $p < 0.01$. Qublan, et al. [18] and Geeta BK [19] concluded that LDH may prove useful as a selective predictive biochemical marker of preeclampsia and its severity. The latter authors found LDH levels > 600 IU/l in 54.8% with severe pre-eclamptic women. This could be due to cellular death and leakage of enzymes from cells. Andrews L, et al. [3] conducted a study to assess significance of the value of serum LDH as a marker of PIH and its severity on 110 cases. They concluded that there is significant rise in the LDH levels with the increasing severity of the disease (172.37 ± 28.09) normotensive, (356.33 ± 24.47) mild preeclampsia, (609.91 ± 136.92) severe preeclampsia and (854.05 ± 247.45) eclampsia ($p < 0.0001$). Study by Patil S [16] also concluded that in cases with severe preeclampsia and eclampsia, there is significant rise in the LFTs. This correlates to study by Paneri, et al. [20] LDH levels are significantly elevated ($p < 0.001$). LDH 5 is specific to liver pathology. Jaleel, et al. [21] found that there was a highly significant rise in serum lactate dehydrogenase and aspartate aminotranferase level in preeclamptic women compared to normotensive pregnant women (Table 6). Hyperuricemia is associated with higher maternal complication rates and fetal growth retardation. In our study, out of 179 patients with gestational hypertension, 178 (99%) patients have S. uric acid < 7 mg/dl and 1 (0.5%) patient had s. uric acid ≥ 7 mg/dl. Out of 100 patients with preeclampsia, 38 (38%) patients had S. uric acid < 7 mg/dl and 62 (62%) patients had s. uric acid ≥ 7 mg/dl. Out of 21 patients with eclampsia, 8 (38%) patients had s. uric acid < 7 mg/dl and 13(57.14%) patients had ≥ 7 mg/dl. 224 (74.6%) patients had S. Uric acid < 7 mg/dl and only 76 (25.3%) patients had S. uric acid ≥ 7 mg/dl. The p -value is $< .00001$ (Table 7). In study by Patil S, et al. [16] there is a significantly raised uric acid level and the extent of the elevation in serum uric acid level in preeclamptics was indicator for the degree of severity of this disorder. Elevated serum uric acid levels have also been interpreted to act as an important cofactor involved in the pathogenesis and manifestation of pre-eclamptic disorder. This is in accordance to our study. Similar results were seen with study by Andrews L, et al. [3] and concluded that the s. uric acid showed statistically higher levels in eclamptic women (7.86 ± 2.38) in comparison to mild PIH (5.82 ± 2.25), severe PIH (6.31 ± 1.88) and normal pregnant women (3.66 ± 1.36) ($p < 0.0001$). In our study, out of 100 patients with preeclampsia, 36 (36%) patients had PT < 13.5 seconds and 64 (64%) patients had PT > 13.5 seconds. Out of 21 patients with eclampsia, 5 (23.8%) patients had PT < 13.5 seconds and 16 (76.1%) patients had PT > 13.5 seconds. 212 (70.6%) patients had PT in normal range i.e. 9.5-13.5 seconds and 88 (29.3%) patients had PT > 13.5 seconds. The p -value is $< .00001$. In our study, Out of 179 patients with gestational hypertension, 4 (2.2%) patients had PTI $\leq 85\%$ and 175 (97.7%) patients had PTI 86-100%. Out of 100 patients with preeclampsia, 18 (18%) patients had PTI $\leq 85\%$ and 82 (82%) patients had PT 86-100%. Out of 21 patients with eclampsia, 3 (28.5%)



patients had PTI \leq 85% and 18 (85.7%) patients had PT 86-100% Majority of the patients (275; 91.6%) had PTI 85-100% and 25 (8.3%) patients had PTI \leq 85%. The p-value is .000017. The result is significant. Naaz S, et al. [22] showed an increase in plasma Prothrombin Time in patient with PIH when compared with normal pregnant women the levels are within normal range and the difference is statistically significant. Fitzgerald, et al. [23] who did not observe any prolongation of PT in subgroup of gestational hypertension of 5 cases but observed prolonged PT in 1 out of 28 cases of preeclampsia and 11 out of 40 cases of severe preeclampsia. One case of eclampsia showed no prolongation of PT (Table 8). The results are in agreement to our study. In our study, 179 patients with gestational hypertension had urine for albumin in traces. Out of 100 patients with preeclampsia, 30 patients had U/A 1+, 55 patients had U/A 2+, 11 patients had U/A 3+ AND 4 patients had U/A4+. Out of 21 patients with eclampsia, 2 patients had U/A traces, 2 patients had U/A 1+, 10 patients had U/A 2+ and 7 patients had U/A 3+. Most patients (181; 60.3%) had urine for albumin in traces, followed by 65 (21.7%) patients had 2+ urine for albumin. 32 (10.7% patients had urine for albumin 1+. 18 (6%) patients had 3+ urine for albumin and only 4 (1.3%) patients had urine for albumin 4+. The p-value is $<$.0000001. In study by Pillai SS [13], showed that the proteinuria \leq 1+ was seen in 13.63%, \geq 2+ was seen in 28.18% and \geq 3+ was seen in 58.18% in women with severe preeclampsia and eclampsia. In the study by Saxena N, et al. [24] showed that proteinuria \leq 1+ was seen in 17.3%, \geq 2+ was seen in 18.67%, and \geq 3+ was seen in 64% in women with severe preeclampsia and eclampsia. The results are almost similar to our study except for the proteinuria 3+. This could be explained with the fact as majority of the patients in our study had gestational hypertension and proteinuria was seen in traces in majority of the patient (Table 9). The visual system may be affected in 30% to 100% of patients with hypertensive disorders of pregnancy; the most common abnormality seen in the fundus is narrowing of retinal arterioles. Richard RO [25]. The total number of patients with fundoscopic changes were 28% and grade 2 and grade 3 changes were seen more with preeclampsia and eclampsia patients. The p-value is 0.00001. The result is significant at $p <$ 0.05. Bharathi RN, et al. [4] showed that Fundus findings were seen in 35 cases (23.33%). 26 (17.33%) had Grade I changes, 1 (0.66%) had grade II changes, 6 (3.9%) had grade III changes 2 (1.3%) had retinal detachment/ grade -IV. They concluded that degree of retinopathy was correlating with the severity of the disease and levels of hypertension. Tadin, et al. [26] from Croatia have reported 45% of retinal changes in their study of 40 patients with PIH. They found a statistical correlation between proteinuria, blood pressure and hypertensive retinopathy. The degree of retinopathy was directly proportional to severity of preeclampsia. They stated that hypertensive retinopathy is a valid and reliable prognostic factor in determining the severity of preeclampsia; examination of the fundus is a valuable and necessary diagnostic procedure in pregnant women with preeclampsia. The results are consistent our study.

ACKNOWLEDGMENT

It is my proud privilege to acknowledge with reverence and gratitude, the keen personal and ever available guidance rendered to me by my esteemed guide and mentor, Dr. Swarn Kanta, Associate Professor, Department of Obstetrics & Gynaecology. Dr. Rita Thaku for their encouraging attitude, cooperation and never ending support. My heartfelt thanks to Mr. Shivam Chopra for always being there for me.

REFERENCES

- Adu-Bonsaffoh K, Ntumu MY, Obed SA, Seffah JD. Perinatal outcomes of hypertensive disorders in pregnancy at a tertiary hospital in Ghana. *BMC Pregnancy Childbirth*. 2017 Nov 21;17(1):388. doi: 10.1186/s12884-017-1575-2. PMID: 29157196; PMCID: PMC5696910.
- Ajah LO, Ozonu NC, Ezeonu PO, Lawani LO, Obuna JA, Onwe EO. The Feto-Maternal Outcome of Preeclampsia with Severe Features and Eclampsia in Abakaliki, South-East Nigeria. *J Clin Diagn Res*. 2016 Sep;10(9):QC18-QC21. doi: 10.7860/JCDR/2016/21078.8499. Epub 2016 Sep 1. PMID: 27790527; PMCID: PMC5072027.
- Andrews L, Patel N. Correlation of serum lactate dehydrogenase and pregnancy induced hypertension with its adverse outcomes. *Int J Res Med Sci*. 2016;4(5):1347-1350. doi: 10.18203/2320-6012.ijrms20161112.
- Bharathi RN, Raju RSN, Prasad KP, Raju RSN, Premalatha, Mayee K. Fundus changes in pregnancy induced hypertension: A clinical study. *Journal of Evolution of Medical and Dental Sciences*. 2015;4(9):1552-1562.
- Bhattacharya S. Pregnancy induced hypertension and prior trophoblastic exposure. *J Obstet Gynecol India*. 2004; 54(6):568-570.
- Joshi P, Kathaley M, Borada S, Dashrathi R. Maternal and perinatal outcome in HDP: A retrospective study. *MVP JMS*. 2018;5(1):87-91.
- Rajamma CK, Sridevi P. Maternal and perinatal morbidity and mortality in hypertensive disorder complicating pregnancy. *Int J Sci Stud*. 2016;3(11):206-209. doi: 10.17354/ijss/2016/86.
- Gandhi MR, Jani PS, Patel UM, et al. Perinatal outcome in pregnancy-induced hypertension cases at GMERS Medical College, Dharpur-Patan, North Gujarat region, India: A prospective study. *Int J Adv Med*. 2015;2(2):152-155.
- Villar J, Carroli G, Wojdyla D, Abalos E, Giordano D, Ba'aqel H, Farnot U, Bergsjø P, Bakketeig L, Lumbiganon P, Campodónico L, Al-Mazrou Y, Lindheimer M, Kramer M; World Health Organization Antenatal Care Trial Research Group. Preeclampsia, gestational hypertension and intrauterine growth restriction, related or independent conditions? *Am J Obstet Gynecol*. 2006 Apr;194(4):921-31. doi: 10.1016/j.ajog.2005.10.813. PMID: 16580277.
- Duckitt K, Harrington D. Risk factors for pre-eclampsia at antenatal booking: systematic review of controlled studies. *BMJ*. 2005 Mar 12;330(7491):565. doi: 10.1136/bmj.38380.674340.E0. Epub 2005 Mar 2. PMID: 15743856; PMCID: PMC554027.
- Donimath KV, Sambrani AM, Rathod PM. A study of association of thrombocytopenia with pregnancy induced hypertension. *Int J Reprod Contracept Obstet Gynecol*. 2016;5(3):808-812. doi: 10.18203/2320-1770.ijrcog20160589.
- Nawaz F, Sultan S, Siddiqi L. Pregnancy outcome in primigravida complicated with pregnancy induced hypertension. *J Med Sci*. 2014;22(1):46-48.
- Kolluru V, Harika RY, Kaul R. Maternal and perinatal outcome associated with pregnancy induced hypertension. *J Reprod Contracept Obstet Gynecol*. 2016;5(10):3367-3371. doi: 10.18203/2320-1770.ijrcog20163113.
- Poluri SL, Ramakrishna S. Predictive value of platelet count as a prognostic marker of PIH. *Int J Sci Res*. 2016;5(10):724-726.
- Pillai SS. Fetomaternal outcome in severe preeclampsia and eclampsia: A Retrospective study in tertiary care centre: *Int J Reprod Contracept Obstet Gynecol*. 2017;6(9):3937-3941. doi: 10.18203/2320-1770.ijrcog20174039.
- Patil S, Jyothi A, Babu A, Veerabhadra GSK. A study of liver function tests and Renal function tests in Preeclampsia. *IJBR* 2016; 7(10):713-717. doi: 10.7439/ijbr.v7i10.3671.
- Malvino E, Muñoz M, Ceccotti C, Janello G, Mc Loughlin D, Pawlak A, Desmery P, Lopez Gaston O. Complicaciones maternas y mortalidad perinatal en el síndrome HELLP. Registro multicéntrico en unidades de cuidados intensivos del área Buenos Aires [Maternal morbidity and perinatal mortality in HELLP syndrome. Multicentric studies in intensive care units in Buenos Aires area]. *Medicina (B Aires)*. 2005;65(1):17-23. Spanish. PMID: 15830788.
- Qublan HS, Ammarin V, Bataineh O, Al-Shraideh Z, Tahat Y, Awamleh I, Khreisat B, Nussair B, Amarin ZO. Lactic dehydrogenase as a biochemical marker of adverse pregnancy outcome in severe pre-eclampsia. *Med Sci Monit*. 2005 Aug;11(8):CR393-7. Epub 2005 Jul 25. PMID: 16049382.

19. Geeta BK. Ind medica. Cyber lectures. 2006.
20. Paneri S, Panchonia A, Varma M, Yadav S. Evaluation of LFTS and RFTS and ascorbic acid in preeclampsia among of Indore women. Indian Journal of Fundamental and Applied Life Sciences. 2011;1(4):312-315.
21. Jaleel A, Baseer A, Aamir S. Biochemical parameters for detection of hemolysis in pregnancy induced hypertensive women. J Coll Physicians Surg Pak. 1999;9(1):41-42.
22. Naaz A, Padugupati S, Sharma DVHS, Sushma P. A Study on coagulation profile in pregnancy induced hypertension cases. IOSR-JBB. 2015;1(6):82-88.
23. FitzGerald MP, Floro C, Siegel J, Hernandez E. Laboratory findings in hypertensive disorders of pregnancy. J Natl Med Assoc. 1996 Dec;88(12):794-8. PMID: 8990805; PMCID: PMC2608132.
24. Saxena N, Bava AK, Nandanwar Y. Maetranl and perinatal outcome in severe preeclampsia and eclampsia. Int J Reprod Contracept Obstet Gynecol 2016;5(7):2171-2176. doi: 10.18203/2320-1770.ijrcog20162086.
25. Richard RO. Pregnancy induced hypertension (preeclampsia- eclampsia) In: Schachat AP, Murphy RB, editors. Retina. 2nd ed. St Louis: Mosby; 1994. p.1405-1412.
26. Tadin I, Bojić L, Mimica M, Karelović D, Dogas Z. Hypertensive retinopathy and pre-eclampsia. Coll Antropol. 2001;25 Suppl:77-81. PMID: 11817020.