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Original Research Article

Determining the Correlation between Elevated Levels of High-Sensitivity C-Reactive Protein and Dyslipidaemia in Pregnancy-Induced Hypertension: A Retrospective Study

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Abstract

Aim: To investigate the correlation between elevated levels of high-sensitivity C-reactive protein and dyslipidaemia with the occurrence of pregnancy-induced hypertension.

Material and Methods: This retrospective study was conducted in the Department of Obstetrics and Gynaecology, Patna Medical College and Hospital, Patna, Bihar, India from January 2020 to December 2020. The study population was 18-35 years old pregnant women after 20 weeks of gestation. Case: Women with pregnancy-induced hypertension with age 18-35 years after 20 weeks of gestation. Case includes-Gestational hypertension, Pre-eclampsia and Eclampsia. Control: Pregnant normotensive women with the same age group and gestation. As this study includes more variables so for better statistical results sample size is increased to 60 in each group. 60 patients with pregnancy-induced hypertension with age between 18-35 years after 20 weeks of gestation (case). 60 pregnant normotensive patients of the same age and gestation (Control).

Results: Mean serum total cholesterol (261.13±68.62), serum LDL (183.76±66.51), serum triglycerides (285.98± 125.61) and CRP (6.98±4.21) were higher in case compared to that in control (220.46±54.16), (121.90±33.36), (189.03±64.38) and (5.26±2.72) respectively. Mean serum HDL was lower in the case (45.86±15.25) as compared to that in control (54.06±12.03). All these findings were statistically significant ($p \leq 0.05$). Table 6 describes the comparison of lipid profile and C-reactive protein among groups-preeclampsia, eclampsia, gestational HTNand normotensive pregnancy, which were statistically significant ($p \leq 0.05$). Among the cases 58.3% and among controls 31.7% had serum cholesterol ≥ 299 mg/dl and respondents with serum cholesterol ≥ 299 mg/dl had a 3.02 times more chance to develop pregnancy-induced hypertension which was statistically significant ($p=0.003$). Among cases 15.0% and among controls 3.3% hadserum HDL < 52mg/dl and respondents with serum HDL < 52 mg/dl had 5.11 times more chance to develop pregnancy-inducedhypertension which was statistically significant ($p=0.027$). Among cases 61.7% and among controls 11.7% had serum LDL level ≥ 184 mg/dl and respondents with serum LDL level ≥ 184 mg/dl had 10.18 times more chance to develop pregnancy-induced hypertension which was statisticallysignificant ($p<0.001$). Among cases 68.3% and among control 38.3% had serum TG level ≥ 382 mg/dl and respondents with serum TG level ≥ 382 mg/dl had 3.47 times more chance to develop pregnancy-induced hypertension which was statisticallysignificant ($p=0.001$).

Conclusion: In conclusion, based on the results presented in this study we found that high CRP and dyslipidemia were associated with pregnancy-induced hypertension.

Keywords: C-reactive protein, high-sensitivity, dyslipidemia, pregnancy, hypertension

Introduction

Pregnancy-induced hypertension (PIH), including conditions like gestational hypertension and preeclampsia, remains a significant contributor to maternal and perinatal morbidity and mortality globally. The pathophysiology of PIH is complex, involving a myriad of factors including genetic predisposition, immune responses, and metabolic disturbances. Among these, inflammation and lipid metabolism have emerged as critical areas of interest. Recent research highlights the association of increased high-sensitivity C-reactive protein (hs-CRP), a marker of systemic inflammation, and dyslipidaemia with the development and severity of PIH. Understanding these associations is crucial for

improving prediction, prevention, and management strategies for PIH. [1-6] C-reactive protein (CRP) is an acute-phase protein produced by the liver in response to inflammation. High-sensitivity CRP (hs-CRP) assays can detect lower levels of CRP, making it a sensitive marker for chronic, low-grade inflammation. In the context of PIH, inflammation plays a pivotal role, with endothelial dysfunction and placental ischemia being key pathological features. Elevated hs-CRP levels have been consistently observed in pregnant women who develop PIH, suggesting that systemic inflammation may precede and contribute to the onset of hypertensive disorders in pregnancy. Dyslipidaemia, characterized by abnormal levels of lipids in the blood, is another critical factor implicated in the pathogenesis of PIH. Pregnancy naturally induces changes in lipid metabolism to support foetal development; however, exaggerated alterations can contribute to endothelial dysfunction and oxidative stress, both of which are central to PIH development. Elevated levels of triglycerides, low-density lipoprotein (LDL) cholesterol, and reduced high-density lipoprotein (HDL) cholesterol have been associated with an increased risk of PIH. The interplay between inflammation and lipid metabolism is evident in the pathophysiology of PIH. Both hs-CRP and dyslipidaemia independently and synergistically contribute to endothelial dysfunction, oxidative stress, and placental pathology, which are hallmarks of PIH. Elevated hs-CRP levels may exacerbate lipid peroxidation, leading to further endothelial damage, while dyslipidaemia can enhance inflammatory responses, creating a vicious cycle that exacerbates hypertensive disorders in pregnancy. [7-12]

Material and Methods

This retrospective study was conducted in the Department of Obstetrics and Gynaecology,

Patna Medical College and Hospital, Patna, Bihar, India from January 2020 December 2020. The study population was 18-35 years old pregnant women after 20 weeks of gestation. Women with pregnancy-induced hypertension with age 18-35 years after 20 weeks of gestation. Case includes-Gestational hypertension, Pre-eclampsia and Eclampsia. Pregnant normotensive women with the same age group and gestation. As this study includes more variables so for better statistical results sample size is increased to 60 in each group. 60 patients with pregnancy-induced hypertension with age between 18-35 years after 20 weeks of gestation (case). 60 pregnant normotensive patients of the same age and gestation (Control).

Inclusion Criteria

- Pregnant women after 20 weeks of gestation with age group 18-35 years diagnosed as pregnancy-induced hypertension.
- Pregnant normotensive women with the same above-mentioned age and gestation.

Exclusion Criteria for both group

- Women in labour.
- Women with ruptured membranes.
- Women with multiple pregnancies.
- Women with Molar pregnancy.
- Pre-existing hyperlipidemia before 20 weeks of pregnancy.
- Women receiving any drug that interferes with serum lipidprofile.
- Smoker and alcoholic women.
- History of receiving aspirin or any other medication known to interfere with inflammation.
- Autoimmune disease.
- Medical diseases like DM, chronic hypertension, renaldisease, and thyroid disease.
- Mistaken date.
- Obesity.

Methodology

All the pregnant mothers fulfilling the inclusion attending at obstetrics department were enrolled in the study by purposive sampling. After obtaining an informed written consent 120 subjects were grouped into 2 groups according to criteria. 60 patients with pregnancy-induced hypertension which includes pre-eclampsia (N=21), eclampsia (N=20), and gestational hypertension (N=19). 60 normotensive pregnant women with blood pressure < 140/90 mm of Hg without any signs of pre-eclampsia. Blood pressure was measured by standard procedure with a sphygmomanometer. Korotk off phase-1 (1st beat heard) and phase-v (disappearance of sound) were used for systolic and diastolic blood pressure. It was measured on the right arm, sitting comfortably, legs uncrossed with back and arm supported or lying on her back 45 degrees to horizontal. In both cases occluded brachial artery was kept at thelevel of the heart. Proteinuria is measured by the Dipstick method, when proteinuria is found ≥1+ in the collected urine sample then diagnosis of preeclampsia is established. After taking all aseptic precautions the blood samples were collected from a median antecubital vein into two separate red top vacutainer blood collection tubes. Then it was kept stand for about 45 minutes at room temperature to allow complete clotting and clot retraction, then sera were separated as quickly as possible and centrifugation was done for 5-10 minutes at 3000 rpm. The component of the lipid profile was

measured using an automated analyzer and hs-CRP was measured by Latex turbidimetry method with hs-CRP latex reagent by Atellica CH Analyzer. Normal reference values of serum total cholesterol, triglycerides, HDL, LDL and hs-CRP during pregnancy were taken from [10]. The following precautions were taken while sampling the blood as far as possible:

1. Preferably the patient was fasting overnight for at least12 hours.
2. It was made sure that the patient was not receiving any drug that interfered with serum lipid levels or their estimation.
3. The patient was afebrile for the last one week.
4. The patient was on a near-normal diet.

All the above precautions cannot be applied strictly in cases of eclampsia due to inherent difficulties associated with such cases.

Data Collection

Detailed Obstetric and medical history and clinical examinationswere done in all study subjects. Data was collected from the patient on variables of interest using the pre-formed structured questionnaire. For each and every subject separate data sheet was used. With all aseptic precaution blood samples were collected after overnight fasting from the median antecubital vein. Then the serum hs-C-reactive Protein and lipid profile were measured.

Data Analysis

Statistical analyses were carried out by using the Windows-based Statistical Package for Social Sciences (SPSS-26). For continuous variables distribution was expressed by mean and standard deviation. The mean comparison between the two groups was done by unpaired t-test. For qualitative variables distribution was expressed by frequency and their percentages. A Chi-square test was done to see the significance of the difference between the two groups. A scatterplot diagram was used to show the relationship between blood pressure and biochemical variables. ANOVA test was carried out to compare means between 4 groups and to see the relationship with pregnancy-induced hypertension. Cut-off values were used according to the literature. The p-value <0.05 was considered statistically significant.

Results

The hospital-based case-control study was carried out for the evaluation of High-Sensitivity C-reactive Protein and Serum Lipid profile in women with Pregnancy Induced Hypertension. Cases, where pregnant women (60 respondents) were diagnosed as PIH and Control were normotensive pregnant women (60 respondents). The findings of the study are presented by graphs and tables.

Table 1: Categorization of the respondents according to their socio-demographic characteristics (case=60, control=60)

Sociodemographic characteristics	Case		Control		P-Value
	N	%	N	%	
Age of the respondents					
Up to 20 years	7	11.7	8	13.3	0.926[a]
21-30 years	38	63.3	36	60.0	
More than 30 years	15	25.0	16	26.7	
Mean ± SD	27.07±5.23		26.73±5.01		0.722[b]
Educational Qualification					
Below SSC	36	60.0	46	76.7	0.077[a]
SSC and above	24	40.0	14	23.3	
Occupation					
Housewife	53	88.3	59	98.3	0.061[c]
Service	7	11.7	1	1.7	
Monthly family income					
Low income (<6,833)	8	13.3	18	30.0	0.064[a]
Lower middle income (6,833-26,900)	40	66.7	35	58.3	
Upper middle income (26,901-83,167)	12	20.0	7	11.7	
Area of residence					
Urban	10	16.7	2	3.3	0.002[a]
Semi-urban	22	36.7	40	66.7	
Rural	28	46.7	18	30.0	

Pregnant women with Pregnancy Induced Hypertension

Pregnant women without Pregnancy InducedHypertension

Table 1 shows the mean (±SD) age of the respondents of the case (27.07±5.23) years and control (26.73±5.01) years groups were almost similar. The majority of the respondents both in case (60.0%) and control (76.7%) groups were educated below the SSC level. Most of them were housewives (case vs control: 88.3% vs 98.3%)and belonged to lower-middle-class families. All these findingswere statistically non-significant (p≥0.05). A significant finding was observed in regards to the area of residence where the majority of the cases came from rural (46.7%) areas and among control more than half came from semi-urban (66.7%) areas (P=0.002)

: Categorization of the respondents according to past medical history (case=60, control=60)

Table 2 shows non-significant findings with regard to the past medical history of respondents among case and control.

Past medical history	Case		Control		P-Value
	N	%	N	%	
H/O GDM					
Yes	3	5.0	1	1.7	0.619[c]
No	57	95.0	59	98.3	
H/O preeclampsia					
Yes	6	10.0	4	6.7	0.496[a]
No	54	90.0	56	93.3	
H/O of IUD					
Yes	3	5.0	1	1.7	0.619[c]
No	57	95.0	59	98.3	
H/O birth weight > 4kg					
Yes	1	1.7	0	0.0	0.315[c]
No	59	98.3	60	100.0	

Table 3: Categorization of the respondents according to family history (Case=60, Control=60)

Family history	Case		Control		P-Value
	N	%	N	%	
Family H/O DM					
Yes	21	35.0	24	40.0	0.572[a]
No	39	65.0	36	60.0	
Family H/O HTN					
Yes	25	41.7	24	40.0	0.853[a]
No	35	58.3	36	60.0	
Family H/O renal disease					
Yes	1	1.7	0	0.0	0.315[c]
No	59	98.3	60	100.0	

Table 3 states that among cases 21 (35.0%) and among controls 24(40.0%) had a positive family history of DM. Among cases 25 (41.7%) and among controls 24 (40.0%) had a positive family history of HTN. Among cases (1.7%) and among controls none of the respondents (0.0%) had a positive family history of renal disease.

Table 4: Categorization of the respondents according to present medical condition (case=60, control=60)

Present medical condition	Case		Control		**P-value**
	n	%	n	%	
Oedema					
Yes	46	76.7	40	66.7	0.224[a]
No	14	23.3	20	33.3	
Proteinuria					
Yes	40	33.3	0	0.0	<0.001[a]
No	20	66.7	60	100.0	
IUGR					
Present	9	15.0	4	6.7	0.142[a]
Absent	51	85.0	56	93.3	

Table 4 states that among the cases 40 (33.3%) had a positivehistory of proteinuria and none (0.0%) of the control had a positive history of proteinuria. This finding was statisticallysignificant (<0.001).

Table 5: Distribution of the respondents according to lipid profile and C reactive protein (Case=Mean ± SD, Control=Mean ± SD)

Lipid profile and C reactive protein	Case (Mean ± SD)	Control (Mean ± SD)	P-value
Serum Total Cholesterol	261.13±68.62	220.46±54.16	<0.001 [b]
Serum HDL	45.86±15.25	54.06±12.03	<0.001 [b]
Serum LDL	183.76±66.51	121.90±33.36	<0.001 [b]
Serum Triglycerides	285.98±125.61	189.03±64.38	<0.001 [b]
hs-CRP	6.98±4.21	5.26±2.72	0.009 [b]

Table-5 denotes mean serum total cholesterol (261.13±68.62), serum LDL (183.76±66.51), serum triglycerides (285.98± 125.61) and CRP (6.98±4.21) were higher in case compared to that in control (220.46±54.16), (121.90±33.36), (189.03±64.38) and (5.26±2.72) respectively. Mean serum HDL was lower in the case (45.86±15.25) compared to that in control (54.06±12.03). All these findings were statistically significant (p≤0.05). Table 6 describes the comparison of lipid profile and C-reactive protein among groups-preeclampsia, eclampsia, gestational HTN and normotensive pregnancy, which were statistically significant (p≤0.05).

Table 6: Comparison of lipid profile and hs-CRP among the different categories of PIH (Preeclampsia=21, Eclampsia=20, Gestational HTN=19)and control=60

Lipid profile and CRP	Preeclampsia (Mean ± SD)	Eclampsia (Mean ± SD)	estational HTN (Mean ± SD)	Control (Mean ± SD)	P-value
Cholesterol	252.4±59.6	292.3±72.1	237.8±65.1	220.4±54.1	<0.001 [d]
HDL	48.9±14.9	35.9±11.6	52.5±14.0	54.0±12.0	<0.001 [d]
LDL	183.6±72.1	193.5±75.8	173.5±49.2	121.9±33.3	<0.001 [d]
S. TG	189.0±64.3	286.2±116.0	353.0±123.9	215.1±101.3	<0.001 [d]
hs-CRP	6.7±3.9	8.2±4.6	5.9±3.9	5.2±2.7	0.011 [d]

Table 7: Odds ratios (OR) and 95% confidence interval of hypercholesterolemia in PIH.

Serum Cholesterol	Case		Control		χ2 value	p-value	OR (95% CI)
	N	%	N	%			
≥ 299 mg/dl	35	58.3	19	31.7	8.62	0.003	3.02 (1.43-6.38)
<299 mg/dl	25	1.7	41	68.3			

Table 7 shows among the cases 58.3% and among controls 31.7% had serum cholesterol ≥ 299 mg/dl and respondents with serum cholesterol ≥ 299 mg/dl had 3.02 times more chance to develop pregnancy-induced hypertension which was statisticallysignificant (p=0.003).

Table 8: Odds ratios (OR) and 95% confidence intervals (CI) of decreased HDL in Pregnancy Induced Hypertension

Serum HDL level	Case		Control		χ2 value	P-VALUE	OR (95% CI)
	N	%	N	%			
< 52 mg/dl	9	15.0	2	3.3	4.90	0.027	5.11 (1.05-9.78)
≥ 52 mg/dl	51	85.0	58	96.7			

Table 8 shows among cases 15.0% and among controls 3.3% hadserum HDL < 52mg/dl and respondents with serum HDL < 52 mg/dl had 5.11 times more chance to develop pregnancy-inducedhypertension which was statistically significant (p=0.027).

Table 9: Odds ratios (OR) and 95% confidence intervals (CI) of increased serum LDL level in Pregnancy Induced Hypertension

Serum LDL level	Case		Control		χ2 value	P-Value	OR (95% CI)
	N	%	N	%			
≥ 184 mg/dl	37	61.7	7	11.7	32.29	<0.001	10.18 (4.73-

< 184 mg/dl	23 38.3	53 88.3			16.32)

Table 9 states that among cases 61.7% and among controls 11.7% had serum LDL level ≥ 184 mg/dl and respondents with serum LDL level ≥ 184 mg/dl had 10.18 times more chance of developing pregnancy-induced hypertension which was statisticallysignificant (p<0.001).

Table 10: Odds ratios (OR) and 95% confidence intervals (CI) of hypertriglyceridemia in pregnancy-induced hypertension

Serum TG level	Case	Control	χ2 value	P-Value	OR (95% CI)
≥ 382 mg/dl	41 (68.3)	23 (38.3)	10.84	0.001	3.47 (1.63-7.37)
< 382 mg/dl	19 (31.7)	37 (61.7)			

Table 10 shows that among cases 68.3% and among controls 38.3% had serum TG level ≥ 382 mg/dl and respondents with serum TG level ≥ 382 mg/dl had 3.47 times more chance to develop pregnancy-induced hypertension which was statisticallysignificant (p=0.001).

Table 11: Odds ratios (OR) and 95% confidence intervals (CI) of increased hs-CRP in Pregnancy Induced Hypertension.

Serum hs-CRP	Case	Control	χ2 value	p-value	OR (95% CI)
≥ 9.66 mg/l	18 (30.0)	7 (11.7)	6.11	0.013	3.24 (1.24-6.49)
< 9.66 mg/l	42 (70.0)	53 (88.3)			

Table 11 states that among the case 30.0% and among control 11.7% had serum hs-CRP level ≥ 9.66 mg/l and respondentswith serum hs-CRP level ≥ 9.66 mg/l had 3.24 times morechance to develop pregnancy-induced hypertension which was statistically significant (p<0.013).

Discussion

This case-control study was carried out to determine the association of high sensitivity C-reactive protein and Lipid profile in women with Pregnancy-induced Hypertension (PIH) and normotensive pregnant women. A total of 120 pregnantwomen in their 2nd half of pregnancy between the ages of 18-35 years, attending the Department of Obstetrics and Gynecology, who fulfilled the inclusion criteria were enrolled in this study. They were divided into two groups, case: Pregnant women (60 respondents) diagnosed with PIH and control: normotensive pregnant women (60 respondents). In the present study, the mean (±SD)age of the respondents of the case (27.07±5.23) years and control (26.73±5.01) years, were almost similar. The age difference was statistically not significant (p=>0.05) between the two groups. In a study by Rout and Mahalik *et al*. [11], the mean age in the study group with gestational hypertension was 27.41 compared to 26.19 years in the control group with pregnant normotensive women. In another study, Mandal *et al*. [12] where the mean age in the study group was 29.42 years and the mean age in the control group was 25.96 years. A study found that the mean age was 24.10±4.70 years in preeclampsia and 24.80±4.02 years in normal pregnancy there was no difference between the two groups Das *et al*. [13] All these studies are similar to the present study. The majority of the respondents both in case (60.0%) and control (76.7%) were educated below the SSC level. Most of them were housewives (case vs control: 88.3% vs 98.3%) and belonged to the lower-middle-class family. All these findings were statistically non-significant (p ≥ 0.05). A significant finding was observed in regard to the area of residence where a majority of the cases came from rural (46.7%) areas and among control more than half came from semi-urban (66.7%) areas (p=0.002). A study conducted by Koppula and Sawant, demonstrated that 97% of the cases belonged to the rural background, and most of them were literate (71.33%). 52% belonged to Class I status. Sociodemographic parameters and PIH showed no association in another study conducted by Koppula and Sawant *et al*. [14] which did not fully support the present study and these dissimilarities might be due to geographical variation and small sample size. In the present study among the cases (33.3%) had a positive history of proteinuria andin control where none (0.0%) of the respondents had a positive history of proteinuria. This finding was statistically significant (*p*<0.001). In a similar study, Stepan *et al*. demonstrated patients with proteinuria showed the highest blood pressure value. [15] In this study mean serum total cholesterol (261.13±68.62), serum LDL (183.76±66.51), serum triglycerides (285.98±125.61) and CRP (6.98±4.21) were higher in cases compared to that in control group (220.46±54.16), (121.90±33.36), (189.03±64.38) and (5.26±2.72) respectively. Mean serum HDL was lower in the case (45.86±15.25) compared to that in control (54.06±12.03). All these findings were statistically significant (p≤0.05). The significantly high levels of TG and cholesterol with significantly low HDL-c could reflect a compromised vascular function. These explanations agree with a study by

Catarino *et al* [16] who reported that preeclampsia is associated with enhanced hyperlipidemia which seems to have a negative impact on fetal lipid profile as reflected by an atherogenic LDL-c/HDL-c ratio and higher TG level. A study conducted by Das *et al* [13] showed that in the preeclamptic group mean TG 212.75±50.29 mg/dl was increased significantly than other parameters compared to mormotensive pregnant 185.60±40.67mg/dl. Among the cases, 58.3% and among controls 31.7% had serum cholesterol ≥ 299 mg/dl and respondents with serum total cholesterol ≥ 299 mg/dl had 3.02 times more chance to develop pregnancy-induced hypertension which was statistically significant (p=0.003).Among the case 15.0% and among control 3.3% had serum HDL < 52 mg/dl and respondents with serum HDL <52 mg/dl had 5.11 times more chance to develop pregnancy-induced hypertension which was statistically significant (p=0.027).Among the cases 61.7% and among controls 11.7% had serum LDL level ≥ 184 mg/dl and respondents with serum LDL level ≥ 184 mg/dl had 10.18 times more chance of developing pregnancy-induced hypertension which was statistically significant (p<0.001). Among the cases 68.3% and among controls 38.3% hadserum TG level ≥ 382 mg/dl and respondents with serum TG level ≥ 382 mg/dl had 3.47 times more chance to develop pregnancy-induced hypertension which was statistically significant (p=0.001). A study conducted by Farag *et al* [17] showed a significant increase of CRP, TG, Cholesterol and LDLc in women who developed PIH compared with normotensive a pregnant woman which was similar to the present study. Another study also supports the findings of the present study where mean values of triglyceride (TG), total cholesterol (TC) and low-density lipoprotein-cholesterol (LDL-c) were significantly higher in pre-eclamptic women as compared with normotensive pregnant women (TG = 229.61±88.27 and 147.00±40.47, TC = 221.46±45.90 and 189.67±39.18, LDL = 133.92±38.77 and 112.41±36.08, VLDL = 41.44±19.68 and 26.64±7.87), respectively. The serum high-density lipoprotein cholesterol (HDL-c) level was lower, but it is not statistically significant (HDL-c = 51.02±16.01 and 61.80±25.63) in pre-eclamptic women as compared with controls Tesfa *et al*. [18] Present study showed positive correlation of triglyceride (TG), totalcholesterol (TC) and low-density lipoprotein-cholesterol (LDL- c) level with systolic blood pressure and diastolic blood pressure which was statistically significant (p=0.001). Previous studies demonstrated that high triglyceride (TG), total cholesterol (TC) and low-density lipoprotein-cholesterol (LDL-c) `levels strengthened the association between high blood pressure (BP) Satoh *et al*. [19] In the present study among the cases 30.0% and among controls, 11.7% had high sensitivity CRP ≥ 9.66 mg/l and respondents with high sensitivity CRP ≥ 9.66 mg/l had 3.24 times more chance to develop pregnancy-induced hypertension which was statistically significant (p=0.013). A similar finding was observed in a study conducted by Ertas *et al* [7] where high sensitivity CRP ≥ 9.66 mg/l was found associated with pregnancy-induced hypertension. Wolf *et al*., (2001) in a prospective case-control study reported that women with CRP conc.≥4.1mg/l experience a 3.5-fold increased risk of preeclampsia as compared with women whose CRP conc. were less than 1.1 mg/l. All these studies support the present study. The present study demonstrates a positive correlation between hs-CRP and systolic and diastolic blood pressure which is statistically significant (p=0.001). Mirzaie *et al* [20] also found that there was a positive correlation between serum CRP level and systolic and diastolic blood pressure. In another study, serum CRP concentration was found to be significantly higher (p=0.001) in pre-eclamptic patients (2.10±1.36 mg/l) than in normal pregnant women (0.39±0.09 mg/l) Das *et al*. [21] Fatemeh M *et al*. and Ahmed k *et al* [22, 23] conducted their studies in Iran and Egypt respectively and both found a positive correlation between serum CRP and systolic (p=<0.005) and diastolic blood pressure (p=<0.005). Thus, considering the result of the present study correlating with another study of various countries of the world, this can be easily concluded that raised CRP and components of lipid profile significantly play an important role in the pathogenesis of Pregnancy Induced Hypertension, but to find out causative factors behind this change, further studies need to be carried out.

Conclusion

In conclusion, based on the results presented in this study we found that high hs-CRP and dyslipidemia were associated with pregnancy-induced hypertension

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