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Retrospective Analysis of Coagulation Profile in Pregnancy Induced Hypertension Cases in a Tertiary Care Hospital

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Abstract

Background: It is a physiological process in women and is associated with risk to health of both mother and child. To prevent maternal and fetal morbidity and mortality by early assessment of severity of pre eclampsia and eclampsia. *Objective:* to compare the coagulation parameters between the test group and control group. *Materials and Methods:* A total number of 30 normal antenatal women without Pregnancy induced hypertension are included in control group and a total number of cases of Pregnancy induced hypertension are taken as test group. Comparision of coagulation parameters like Activated partial thromboplastin time, Prothrombin time and platelet count were studied in these patients. This study was undertaken for a period of one year of January 2016 to January 2017 in department of pathology, central laboratory, narayana medical college & hospital, Nellore. *Results:* Coagulation parameters such as Activated partial thromboplastin time, Prothrombin time and platelet count were studied in both 30 control and cases. Study showed significant alteration of coagulation profile depending on severity of disease. All results were statistically analysed. *Conclusion:* Estimation of different parameters like Activated partial thromboplastin time, Prothrombin time and platelet count play s a role in the diagnosis of Pregnancy induced hypertension and evaluation of risk factors, early detection of which will decrease maternal morbidity and mortality.

Keyords: Pregnancy Induced Hypertension; Preeclampsia; Activated Partial Thromboplastin Time; Prothrombin Time; Platelet Count.

Introduction

Pregnancy induced hypertension is one of the common disorders seen in pregnancies[1]. It is defined as as hypertension that occurs in pregnancy for the first time after 20 weeks of gestation and disappear following delivery. In both underdeveloped and developed countries, hypertensive disorders during pregnancy are associated with high maternal fetal mortality and morbidity. Approximately 70% of hypertensive disorders are due to gestational hypertension this condition is called pre eclampsia [2]. Preeclampsia is associated with 17% of all maternal deaths and reported 8-10% of the pregnancy in India. PIH is classified in to i) Gestational hypertension ii) pre eclampsia iii) Eclampsia. According to the criteria of the International society of the study of hypertension in pregnancy, preeclampsia is defined as the blood pressure > 140/90 mm Hg occurring after 20week gestation with proteniuria 300 mg/day or urinary protein/creatine ratio=30mg/ml. Eclampsia is occurrence of convulsion or coma with signs and symptoms of pre eclampsia[3]. Based on the classification of American college of Obstetrician & Gynaecologist (ACOG), the preeclampsia can be categorized in to mild (140-159/90-109mm Hg) and severe (>160/110)[3,4]. In PIH cases to prevent complications like HELP(Hemolysis, elevated liver enzymes, low platelet count) syndrome, Disseminated intravascular coagulation and maternal and fetal morbidity and mortality by early assessment of severity of pre eclampsia and eclampsia.

The main aim and objectives of this study were

1. To study the following parameters in antenatal women after 20 weeks of pregnancy.

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a. Activated partial thromboplastin time
b. Prothrombin time
c. Platelet count

2 . To assess the correlation of coagulation parameters of normal pregnancy with PIH cases.

Materials and Method

The data has been taken for period of 1 year from Jan 2016 to Jan 2017 from ,Dept of pathology, central laboratory, Narayana general hospital&medical college, Nellore, Andhrapradesh, India. Clinical data were collected from antenatal ward and coagulation parameters values from central laboratory .Total of 30 normal antenatal women without PIH are included in control group and total number of 30 diagnosed cases of PIH are taken as test group. The coagulation parameters of test group is estimated and compared with those of control group. Clinical data like blood pressure, gravid and fetal complications and delivery route were taken from record book of antenatal ward. The coagulation parameters such as partial thromboplastin time Prothrombin time and platelet count of control and test group were carried out in the central laboratory by using Elite pro coagulation analyzer and Beckman coulter LH 780 and 750 hematology analyzers. Dip stick was used to test for proteinuria and if this test was positive, infection was excuded by doing microscopy of the same specimen.

Inclusion Criteria

1. Age-18-35 years
2. 30 Pregnant females with preeclampsia in 3 rd trimester, were taken as cases
3. 30 Pregnant females with preeclampsia in 3 rd trimester without any disease were taken as controls
4. Controls were Age and parity matched with cases
5. Preeclampsia Cases were categorized in to mild(140-159/90-109) and severe(>160) based upon classification of American college of obstetrician and Gynaecologist (ACOG) [4].

Exclusion Criteria

1. History of chronic hypertension before completion of 20 weeks of pregnancy
2. History of Diabetes, Receiving drugs like aspirin, anticoagulants, renal disorders, Epilepsy, sickling, Hydatid form mole and abruption placentae, idiopathic thrombocytopenic purpura and auto immune disorder were excluded from the study.

Statistical Analysis

Data were analyzed using MS excel 2007. All data were expressed as mean with standard deviation. Statistical significance among groups were assessed using SPSS version 21.0. A level of p value <0.01 was used to indicate statistical significance in all analyses.

Results

In our study the coagulation Parameters like Activated Partial thromboplastin time, prothombin time, platelet count were estimated in 30 controls and 30 cases in normotensive and hypertensive pregnant women respectively. Both in controls and cases subjects predominant age group was 24-29 years (Table 1). The mean and standard deviation of age distribution in controls is 25.54±4.67 as compared to 25. 65±4.72 in test group as shown in Table1. The parity of the two groups almost similar. The percentage composition of primigravidae and multigravidae with in the group and between the two groups were similar (Table 2). The out come of pregnancy in the present study among 30 controls subjects, 17 delivered normally and in remaining 13 subjects Elective lower segment caesarian section, where as in 30 cases, 9 subjects delivered normally, 19 cases Elective lower segment caesarian section was done, 2 subjects has fetus with IUD (Table 2). The mean and standard deviation of systolic blood pressure in control is 106.33± 9.99 as compared to 158.0±14.23 in test group. The difference is statistically significant (p-value-<0.01(0.000) as shown in Table 3. The standard error of mean of systolic blood pressure in control is 1.82 as compared to 2.59 in test group (Table 3). The mean and standard deviation of diastolic blood pressure in control is 76.66±5.46 as compared to 104.33± 5.68 in test group. The difference is statistically significant (p-value-<0.01(0.000) as shown in Table 3. The standard error of mean of diastolic blood pressure in control is 0.99 as compared to 1.03 in test group (Table 3). The mean and standard deviation of activated partial thromboplastin time in controls is 22.80±1.75 as compared to 33.53± 4.25 in test group. The difference is statistically significant (p-value-<0.01(0.000) as shown in Table 3. The standard error of mean of activated partial thromboplastin time in control is 0.32 as compared to 0.72 in test group (Table 3). The difference is statistically significant as shown in table-

3 The mean and standard deviation of prothombin time in controls is 11.78±0.62 as compared to 15.35± 1.5 in test group. The difference is statistically significant as shown Table 3. The difference is statistically significant (p-value-<0.01(0.000) as shown in Table 3. The standard error of mean of prothrombin time in control is 0.62 as compared to 1.5 in test group (Table 3). The mean and standard deviation of platelet count in controls is 3.1±0.51 as compared to 1.49±0.33 in test group. The difference is statistically significant (p-value-<0.01(0.000) as shown in Table 3. The standard error of mean of platelet count in control is 0.09 as compared to 0.06 in test group (Table 3). In our study mild preeclampsia 17 cases (56.66%) showed the mean systolic blood pressure (146.47), diastolic blood pressure (100.0) and severe preeclampsia 13cases (43.33%) showed the mean systolic blood pressure(173.07),diastolic blood pressure(110.0) (Table 4). Urine protein +1 finding was noted in 13 cases of mild preeclampsia and 10 cases of severe eclampsia (Table 4). Urine protein +3 finding was noted in 4 cases of mild preeclampsia and 3 cases of severe eclampsia (Table 4). Symptoms are like blurring of vision, headache noted in all (13) severe preeclampsia cases (Table 4). The platelet count was significantly reduced in severe pre eclampsia as compared to normal pregnancy (Table 5).

Table 1: Age distribution of control and cases subjects

Age in years	No of control subjects	%	No of cases subjects	%
18-23	9	30	8	26.66
24-29	17	56.66	19	63.33
30-35	4	13.33	3	10.00
Total	30	100	30	100
The mean age was 25.65years with a standard deviation of 3.82 year			The mean age was 26.20years with a standard deviation of 3.90 years	

Table 2: Parity distribution and pregnancy out come in control and cases subjects

Parity Distribution		Control		Cases	
		No	%	No	%
Pregnancy	primi	14	46.66	13	43.33
	Multi	16	53.33	17	56.66
Out come	Normal delivery	17	56.66	9	30
	Elective lower segmentcaseriansection	13	43.33	19	63.33
	Intra uterine death	0	0	2	6.33

Table 3: Statistical analysis of PIH cases & controls

S. No	Investigation	Statistical Parameter	Controls		Cases
1	Systolic blood pressure(mm Hg)	Mean	106.33		158.00
		Standard deviation	9.99		14.23
		Standard error of mean	1.82		2.59
		P-value		<0.01(0.000)	
2	Diastolic Blood pressure(mm Hg)	Mean	76.66		104.33
		Standard deviation	5.46		5.68
		Standard error of mean	0.99		1.03
		p-value		<0.01(0.000)	
3	Activated partial Thromboplastin time(APTT) Seconds	Mean	22.80		33.53
		Standard deviation	1.75		4.25
		Standard error of mean	0.32		0.77
		p-value		<0.01(0.000)	
4	Prothrombin time(PT) Seconds	Mean	11.78		15.35
		Standard deviation	0.62		1.5
		p-value	<0.01(0.000)		
5	Platelet count(Lacs/cumm)	Mean	3.1		1.49
		Standard deviation	0.51		033
		Standard error of mean	0.09		0.06
		p-value		<0.01(0.000)	

Table 4: Clinical profile of cases of mild and severe preeclampsia

Parameter	Mild preeclampsia n=17	Severe preeclampsia n=13
Systolic BP mm Hg Mean	146.47	173.07
Diastolic BP mmHg Mean	100	110
Urine Protein Nil	-	-
Urine Protein +1	13	10
Urine protein+3	4	3
Symptoms -Headache,Blurring of vision	-	13

Table 5: Comparision of platelets in various studies

Authors	Control (x10 ^{3}/cmm)	Mild (x10 ^{3}/cmm)	Severe (x10 ^{3}/cmm)
Srivastava(1995)[14]	1.94	1.79	1.64
Jambhulkar et al(2001) [15]	2.38	2.3	1.7
Joshi et al(2004)[16]	2.2	2	1.64
J.Davies et al(2007)[17]	2.57	2.3	1.77
Ellora Devi et al(2012)[18]	2.44	1.82	1.42
Present study	3.1	1.58	1.16

Discussion

Pregnancy induced hypertension is one of the most common medical complications of pregnancy and it affects 2-10% of all pregnancies. It remains a disease of theories as its exact cause is not yet fully established [5]. Women with PIH are at a greater risk of abruption placentae, cerebrovascular events, organ failure and disseminated intravascular coagulation. Fetuses of their mothers are at greater risk of intrauterine growth retardation, prematurity and intrauterine death. Preeclampsia is a triad of hypertension, edema and proteinuria. The basic pathology of preeclampsia is endothelial dysfunction, poor placentaion and vasospasm of vessels along with thrombocytopenia of hematological profile alteration. It is also termed toxemia of pregnancy by at least 150 years of ago because of the toxins that were believed to be in the pregnant women's body but not used in present nomenclature [6]. The clinical syndrome of preeclampsia is due to the changes initiated by endothelial injury set in motion a dysfunctional cascade of coagulation, vasoconstriction and intravascular fluid redistribution [7, 8]. First note of defective coagulation in pregnancy was made by Delee in 1901, the subject has receive extensive focus in the literature of obstetrics [9]. Schmorl demonstrated in 1983, the occurrence of coagulation defect in eclampsia is better viewed than in preeclampsia and he noted the finding of thrombosis of capillaries in a dying eclampsia patients. The changes in coagulation and fibrinolytic system in severe preeclampsia with that intravascular coagulation which being first demonstrated by Bonnar [10]. It is a pregnancy specific disorders which rates among one of the major cases of maternal and fetal morbidity and mortality. More number of cases are seen in underdeveloped and developing countries due to late diagnosis and inadequate antenatal services.

The results of the present study the coagulation parameters like activated partial thromboplastin time, prothrombin time and platelet count are studied in normotensive and PIH pregnant women. In control subjects, the results of estimation of activated partial thromboplastin time, prothombin time and platelet count are within normal limits. In PIH cases all subjects showed an increased in plasma Activated partial thromboplastin time levels when compared with normal pregnant women, the levels are within normal range and the difference is statistically significant. This finding was correlated with study done by Asia Naaz[11]. All subjects showed an increased Plasma prothrombin time when compared with normal pregnant women the levels are within normal range and the difference is statistically significant. This finding was correlated with study carried out by Asia Naaz [11]. There is significant decrease in platelet in cases when compared with normal pregnant women the levels are within normal range and difference is statistically significant. This finding was correlated with study carried out by Asia Naaz [11]. In our study mild preeclampsia 17 cases (56.66%) showed the mean systolic blood pressure (146.47mm Hg), diastolic blood pressure(100mmHg) and severe preeclampsia 13cases (43.33%) showed the mean systolic blood pressure (173.07mmHg), diastolic blood pressure(110mmHg). Urine protein +1 finding was noted in 13 cases of mild preeclampsia and 10 cases of severe eclampsia. Urine protein +3 finding was noted in 4 cases of mild preeclampsia and 3 cases of severe eclampsia. Symptoms are like blurring of vision, headache noted in all severe preeclampsia

cases. All these findings were correlated with study carried out by Chaware S A [12]. The platelet count was significantly reduced in severe pre eclampsia as compared to normal pregnancy (Table 5). The Platelet count was low in 61% of severe preeclampsia. This finding was correlated with study carried out by Mirza Asif Baig [13]. The mean platelet count in mild preeclampsia (1.58) and severe preeclampsia (1.16) and controls (3.1). This findings were correlated with srinivastava [14], Jambhulkar[15], Joshi [16], J. Davies[17], Ellora Devi [18].

The hematological changes that occur in response to pregnancy especially in coagulation profile. The levels of several coagulation factors are increased during pregnancies. Activated partial thromboplastin time is a measure of classic intrinsic pathway and is prolonged by deficiencies of prekalikrein, high molecular weight kininogen, factors xii, xi, ix, viii, x, v, ii and fibrinogen. Shortening of activated partial thromboplastin time may be seen in patients with compensated low grade consumptive coagulapathy. Prothombin time measures the integrity and adequacy of classic extrinsic pathway especially the factors vii, x, v, ii and fibrinogen. Deficiency of coagulation factors like factors II, V, VII, X and fibrinogen may be associated with prolongation of Prolongation of prothrombin time.

Platelet aggregation is increased in preeclampsia women and induced maternal thrombocytopenia. Maternal thrombocytopenia mainly due to immunological process or platelet deposition at sites of endothelial injury can be induced by preeclampsia, eclampsia. Platelet aggregation is increased in preeclampsia women. The degree of thrombocytopenia increases with severity of the disease like lower the platelet count, the greater are maternal and foetal mortality and morbidity. Serum fibrinogen and Di-dimer tests were not done in our study due to financial constraints. Limitations of this study were small sample size and only one time assessment of coagulation profile was done before child birth.

Pregnancy Outcome

The derangement in coagulation profile has potential to identify individuals at risk of developing life threatening complications and poor fetal outcome such as Disseminated intravascular coagulation and Intrauterine growth retardation, intrauterine death, still birth, and lower birth weight. The outcome of pregnancy in the present study among 30 controls subjects, 17 delivered normally and in remaining 13 subjects Elective lower segment caesarian section where as in 30 cases, 9 subjects delivered normally, 19 cases Elective lower segment caesarian section was done, 2 subjects has fetus with Intra uterine death.

Conclusion

Timely measurement of these coagulation parameters plays an important role in the diagnosis of PIH and the evaluation of risk factors, early detection and effective antenatal services prompt and proper management will reduce systemic complications and maternal death. The detection of coagulation will be helpful to obstetrician in treating the patients who probably may go in to disseminate intravascular coagulation.

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