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The Analysis of Maternal and Fetal Rhesus Status in Cases of Preeclampsia

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Research Article

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Abstract

Introduction: Preeclampsia (PE), a severe pregnancy-related condition, affects various organ systems. PE poses significant risks to maternal and fetal health making careful monitoring and proactive management throughout pregnancy to improve prenatal care and outcomes.

Aim: The study aimed to prospectively investigate the relationship between maternal and fetal ABO and Rhesus status and PE Patients and Methods: A case control study was performed with 100 cases of PE (cases) and 200 cases of pregnancies not diagnosed with PE (controls). All cases and controls were women more than 18 years old, having their first baby. In all cases maternal and fetal (umbilical vein) blood samples were taken for ABO and Rhesus C, c, D, E and e status. Direct Coombs (DCT) and Indirect Coombs (ICT) tests were performed on umbilical blood.

Results: The total number of cases and controls were 100 and 200, respectively. There was no significant differences in the incidence of any blood group of the ABO and Rhesus systems in both the maternal and fetal specimens between the case and control groups. All Coombs test results (both ICT and DCT) fetal in both PE and control groups were negative.

Conclusion: This prospective study of maternal and fetal ABO and Rhesus blood groups indicates that there are no significant differences between cases of PE and controls that did not suffer from PE.

Keywords: Preeclampsia; ABO Blood Group System; Blood Pressure; Proteinuria

Abbreviations

PE: Preeclampsia; TLC: Total Leucocyte Count.

Introduction

Preeclampsia (PE), a severe pregnancy-related condition, affects various organ systems. It is characterized by elevated blood pressure and proteinuria occurring after the 20th week of gestation in previously normotensive and non proteinuric women [1].

The ABO blood group system is encoded by the ABO gene, which is located on chromosome 9 [2]. This system consists of three distinct carbohydrate antigens: A, B, and O. The single ABO gene locus has three allele types: A, B, and O.

The Rhesus (Rh) blood group locus includes two structurally related genes which code for red cell membrane proteins carrying the D, Cc, and Ee antigens [3]. It has been shown that the RhD-positive/RhD- negative polymorphism is determined by the presence or absence of the D gene [4]. Following the recent cloning of the RhD gene, these insights elucidate the molecular genetic foundation underlying the specificities of D, C, c, E, and e antigens [5].

PE poses a significant health risk globally, affecting approximately 2-10% of pregnancies worldwide and 8-10% in India.

Despite extensive research, the exact cause of PE remains unclear. It involves endothelial dysfunction and vasospasm, leading to cardiovascular risks and multiorgan involvement.

This disorder poses significant risks to maternal and fetal health, necessitating careful monitoring, especially in high-risk groups. Risk factors include a history of PE obesity, primiparity, multiple pregnancies, and pre-existing medical conditions such as renal disease or diabetes.

PE can be categorized as early onset (before 34 weeks) or late onset (after 34 weeks), with early onset often demonstrating more severe symptoms.

Despite ongoing research, no definitive predictive tests are available, underscoring the importance of vigilant monitoring and proactive management throughout pregnancy.

Given the role of Rh incompatibility in pregnancy complications, such as hemolytic disease of the newborn, it is plausible that Rh status could also influence the development or severity of PE. Understanding the relationship between maternal and fetal Rh status and PE could provide valuable insights into its pathogenesis and potentially inform clinical management strategies [6].

The study aimed to prospectively investigate the relationship between maternal and fetal Rhesus status and PE.

Patients and Methods

A prospective case-control study was performed with each case of PE controlled with 2 cases without PE who delivered in the same hospital as a control group (Department of Obstetrics and Gynecology, Institute of Medical Sciences, Banaras Hindu University, Varanasi). A case control study was planned with 100 cases of PE (cases) and 200 cases of pregnancies not diagnosed with PE (controls).

Inclusion Criteria

Cases - Women (aged over 18 years old) who have delivered a baby from a pregnancy diagnosed with PE in their first pregnancy after 22 weeks gestation.

Controls-Women (aged over 18 years old) delivering a baby in their first pregnancy who did not suffer from any hypertensive disorder of pregnancy.

Maternal and umbilical vein blood specimens were collected to determine ABO and Rhesus (C, c, D, E, e) status, along with performing Coombs' tests. Written informed consent was obtained from all participants. Consent was obtained from the institution's ethics committee.

Results

Overall there were 100 cases of PE and 200 controls analysed. There were no differences between maternal age (Table 1). There was a significant difference in gestational age at delivery with 62% of the PE cases late onset and 7% of the control group delivered after 34 weeks (Table 2).

Table 3 outlines the results of the maternal and fetal blood groups in the PE and control groups. There was no significant difference between the maternal ABO and Rhesus groups between the PE and control groups. Similarly, in the fetal specimens. there was no difference between the ABO and Rhesus groups between the PE and control groups.

All DCT and ICT results were negative. The uniformity of negative results across all categories suggests that the presence of antibodies against red blood cells, as detected by both ICT and DCT, does not differ between PE cases and controls in this dataset.

There were no differences in Haemoglobin or platelet count and that total leucocyte count (TLC) was significantly lower in PE cases. Table 4 also demonstrates that there was no differences in renal function tests. There were significant elevations in UA, LDH and proteinuria in the PE group.

Table 4 demonstrates that there were significant differences in systolic and diastolic blood pressure indicating that the selection of case and control groups were valid.

Age (in Years)	Preeclampsia Frequency (N)	Preeclampsia %of total	Control Frequency (N)	Control %of Total
17-20	8	8.00%	4	2.00%
21-30	67	67.00%	158	79.00%
31-40	24	24.00%	38	17.00%
>40	1	7.00%	0	0.00%
Total	100	100.00%	200	100.00%

Table 1: The Age Range of Cases of PE (N=100) and Controls (N=200) in the Study.

Gestational age (Weeks)	Preeclampsia Frequency (N)	Preeclampsia % of Total	Control Frequency (N)	Control % of Total
26-28	5	5.00%	110	55.00%
29-31	11	11.00%	15	7.50%
32-34	22	22.00%	61	30.50%
35-37	31	31.00%	8	4.00%
38-40	30	30.00%	5	2.50%
41-42	1	1%	1	0.50%
Total	100	100%	200	100%

Table 2: Gestational Age of Cases of PE (N=100) and Controls (N=200).

	PE MOTHER (n=100)	PE BABY (n=100)	CONTROL MOTHER (n=200)	CONTROL BABY (n=200)	CHI-SQ MOTHERS	CHI-SQ BABIES
ABO					4.6, p=0.2	1.0, p=0.8
A	17 (17%)	20 (20%)	35 (17.5%)	37 (18.5%)		
B	35(17%)	48(48%)	76(38%)	98(49%)		
AB	28(28%)	9(9%)	36(18%)	13(6.5%)		
O	20(20%)	23(23%)	53(26.5%)	52(26%)		
Rh D					0, p=0.9	0.8, p=0.4
Pos	91(91%)	81(81%)	182(91%)	170(85%)		
Neg	9(9%)	19(19%)	18(9%)	30(15%)		
Rh C					0, p=0.95	0.01, p=0.91
Pos	67(67%)	74(74%)	134(67%)	146(73%)		
Neg	33(33%)	26(26%)	66(33%)	54(27%)		
Rh c					0.08, p=0.8	0.38, p=0.54
Pos	55(55%)	42(42%)	113(56.5%)	93(46.5%)		
Neg	45(45%)	58(58%)	87(43.5%)	107(53.5%)		
Rh E					1.5, p=0.22	0.01, p=0.91
Pos	36(36%)	38(38%)	86(43%)	74(37%)		
Neg	64(64%)	62(62%)	114(57%)	126(63%)		
Rh e					0.08, p=0.80	0.15, p=0.7
Pos	42(42%)	41(41%)	80(40%)	79(39.5%)		
Neg	58(58%)	59(59%)	120(60%)	121(60.5%)		

Table 3: Distribution of Study Population on the Basis of ABO and Rhesus Grouping.

Median (range)	Control (n=200)	PE (n=100)	Mann-Whitney U statistic (significance)
Systolic BP	116(100-140)	151(136-190)	14.1 (p<0.001)
Diastolic BP	74(60-90)	92(80-130)	12.6 (p<0.001)
Haemoglobin concentration	11(2.5-14)	11(6-15)	1.2 (p=0.23, NS)
Total leucocyte count	10890 (1154-20540	10132 (6790-18776)	14.1(p<0.001)
Platelet count	1.4(0.2-3.1)	1.5(0.32-4.4)	1.1 (p=0.2, NS)
Urea	19.8((8.4-90)	20(8.4-90)	1.8 (p=0.075, NS)
Creatinine	0.7(0.2-4.9)	0.7(0.3-4.9)	1 (p=0.306)
Uric acid	5.7(2.8-11.0)	7.25(3.2-14.5)	5.6(p<0.001)
Lactate dehydrogenase	515(225-1653	731(326-1869)	4.7 (p<0.001)
International normalized ratio	1.0(0.8-2.4)	1.0(1-4)	1.3 (p=0.196, NS)
Proteinuria (estimated protein content 0-4+)	0 (0-1)	2 (1-4)	15.4 (p<0.001)

Table 4: The Distribution of Measured Parameters in the PE Group (N=100) and Control Groups (N=200).

Discussion

This is the first detailed study of the relationship between the Rh genotype in mothers with PE and their babies. One hundred PE women were compared to 200 women who delivered in pregnancies that were not diagnosed with PE. The significant elevation in systolic, diastolic blood pressure (median 150/92 versus 116/74) and proteinuria indicate that the 2 groups demonstrated significantly different parameters. PE was found to be most prevalent among women aged 21-30, accounting for 67% of cases, while controls in this age group make up 79% of the total control population. Advanced maternal age has been found to be associated with an increase in the risk of poorer maternal and neonatal outcomes in PE patients. The incidence of PE was higher in later stages of gestation, particularly between 35-37 weeks (31%) and 38-40 weeks (30%) in the current study.

Overall, when PE women were compared with controls there were no significant differences in ABO, Rh D, C, c, E, or e blood groups. There was also no significant differences between the ABO and Rhesus genotype in the fetal blood specimens.

Li T, et al. [7], conducted a systematic review and meta-analysis exploring the association between ABO blood group and the risk of preeclampsia. Their study analyzed data from 12 articles involving 714,153 patients. They found that individuals with blood type O had a lower risk of preeclampsia compared to the control group, while those with blood type AB had a higher risk. However, no significant association was found for blood types A and B. This suggests that ABO blood group may play a role in the risk of PE development, with AB blood group individuals being more susceptible.

Manjunatha S, et al. [8], examined 100 PE cases and 100 controls. Their findings suggesting an increased risk of PE among women with blood group AB shed light on a potential factor that may contribute to the development of this condition. When compared with women with blood group O, women of blood group AB have an an increased risk of preeclampsia. AB blood group was associated with an increased risk of thrombotic events this may be related to the increased incidence of PIH in this group. Thus attention should be given to the AB blood group pregnant women in order to achieve an early diagnosis of PE.

Avci D, et al. [9], In this study, we found that the patients with blood group AB have a higher risk for PE. The patients with PE of blood group O are at high risk of developing HT, and Rh factor was identified as another risk at this point and these patients should be closely followed postpartum.

In contrast, Cordero-Franco HF, et al. [10] performed a case-control study that included patients with PE (n = 253) and without PE (n = 457) in Northeastern Mexico and found that ABO blood groups were not associated with the incidence of PE in Mexican women.

Sinha M, et al. [11] performed a 4-year retrospective study from 2010 to 2014, of all admitted primigravidas with singleton deliveries diagnosed with a hypertensive disorder. It was observed that pregnancy -induced hypertensive disorders were significantly associated with blood groups (p = 0.017). Primigravidae with AB blood group had about five times higher risk of developing eclampsia and blood group A had 50% greater chance of developing PE when compared

to women with blood group O. However, no association was found between PIH and RhD blood group.

The above studies looked at the maternal blood groups retrospectively and did not analyse the fetal blood group. The present study was a prospective analysis of the maternal and fetal blood group of ABO and Rhesus C, c D, E and e status and their respective interactions. It was found that there were no significant differences in the maternal ABO and Rh blood groups between cases of PE and controls. Similarly there were no significant differences in fetal ABO and Rhesus blood groups between cases of PE and controls.

The search for any underlying cause of PE remains elusive.

Conclusion

This study aimed to investigate the relationship between maternal and fetal Rh status and PE. The distribution of blood groups in maternal and fetal specimens in cases of PE and controls showed no difference in either ABO or Rhesus C, c, D, E or e status in either maternal of fetal (cord blood) specimens. This study highlights the multifaceted nature of PE, with age, gestational stage, and various biochemical markers playing significant roles in its development and progression.

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