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Maternal and Neonatal Outcome in Severe Pre-Eclampsia in Relation to Platelet Count

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Abstract

Pre-eclampsia complicated 3-5% of first pregnancies and 1% of subsequent pregnancies with around 5-10% of cases being sever. Pre-eclampsia is progressive disorder of endothelial function that is unique to pregnancy and probably results from various micro vascular diseases, previous studies showed an association between platelets count from one side and maternal and neonatal outcome. Therefore, we conduct this study to assess compare the maternal and neonatal platelet count in severe pre- clampsia and normal pregnant women and to evaluate whether these parameters have a prognostic significance in determining maternal and neonatal outcome, a prospective comparative study was designed. Included 80 women in AL-Zahraa teaching hospital, Najaf, Iraq. Pre-eclampsia more frequent in late pregnancy and is a major cause of maternal, fetal and neonatal mortality and morbidity. Maternal and neonatal platelet count in severe pre-eclmapsia was less than in normotensive, no clinically significant change on trend in platelet count versus gestational age in severe pre-eclampsia. Low maternal platelet count in severe pre-eclampsia adversely affected maternal outcome but unrelated to fetal outcome. Further studies with larger sample size are highly suggested.

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Introduction

Pre-eclmpsia : the development of hypertension with proteinuria or oedema or both induced by pregnancy between 20 weeks of gestation and the end of 1st week postpartum (Hallak Mrhypertensive, 1999; Mendlowitz Mr Toxe, 1980)

Criteria for the diagnosis of Hypertension

Either a rise in blood pressure of ≥15 mmHg diastolic or ≥30 mmHg systolic from early pregnancy.

Or diastolic blood pressure of ≥90 mmHg on 2 occasions 4 hours apart 110 mmHg on 1 occasions.

Criteria for the diagnosis of proteinuria

Proteinuria is present when the urinary protein concentration is greater than 300 mg during a 24-hour period. The 24-hour urine collection is the definitive test to diagnose proteinuria; however, if it is not available, then a concentration of at least 30mg/dL (at least 1+ on dipstick testing) in at least 2 random urine samples collected at least 6 hours apart may be used.

Gestational hypertension (pregnancy induced hypertension)

Hypertension detected for the first time after 20 week's gestation, in the abscence.

Hypertension defined as systolic blood pressure 140 mmHg or diastolic blood pressure 90 mmHg.

Resolves within three months after the birth.

Pre-eclampsia and eclampsia

Hypertension and proteinuria detected for the first time after 20 week's gestation.

Hypertension defined as above proteinuria defined as 300 mg/day or 30 mg a single specimen or 1+ on dipstick.

Eclampsia is the occurrence of seizures superimposed on the syndrome of the eclampsia.

Chronic hypertension

Known hypertension before pregnancy; or a rise in blood pressure more than 140/90mmHg before 20 weeks.

"Essential" hypertension if there is no underlying cause.

"Secondary" hypertension if associated with underling disease.

Pre-eclampsia superimposed on chronic hypertension

Onset of new signs or symptoms of pre-eclampsia after 20 week's gestation woman with chronic hypertension.

Pre-eclampsia occurs in approximately 5% of all pregnancies, 10% of first pregnancies, and 20-25% of women with a history of chronic hypertension. Hypertensive disorders in pregnancy may cause maternal and fetal morbidity and remain a loading source of maternal mortality.

Pathophysiology

The etiology of pre-eclampsia is still unknown. However, placental delivery reverses the symptoms of preeclampsia, suggesting that the placenta has a central role in the condition. (Dekker and Sibai, 1998)

Additionally, women with increased placental tissue for gestational age, such as those with Hydatiform moles and twin pregnancies, have an increased prevalence of pre-eclampsia. In fact, the presence of proteinuric hypertension prior to 20 week's gestation should initiate a search for molar pregnancy because it raises the possibility of increased placental tissue for a given gestational age, which could cause the symptoms. Other causes include drug withdrawal or a chromosomal abnormally in the fetus (e.g., Trisomy) (Dekker and Sibai, 1998; Pridjian and Puschett, 2002)

Several theories, which are not mutually exclusive, have been proposed in an attempt to explain the pathophysiology of pre-eclampsia. One theory holds that an increase of a number of active circulating mediators during pregnancy causes the symptoms. For example, increased levels of Angiotensin II during pregnancy may lead to increased vasospasm. A second theory holds that improper placental development results in placental vascular endothelial dysfunction and a relative uteroplacental insufficiency. The vascular endothelial dysfunction results in increased permeability, hypercoagulability, and diffuse vasospasm. Finally, another model suggests that theincreased cardiac output observed during pregnancy causes pre-eclampsia.

The increased blood flow and pressure is felt to lead to capillary dilatation, which damages end organ sites, leading to hypertension, proteinuria, and edema.

Additional theories have arisen from epidemiologic research, suggesting the important role of genetic and immunologic factors. The increased prevalence observed in patients using barrier contraception, in multiparous women conceiving with a new partner, and in nulliparous women suggests an immunologic role. Also, inheritance pattern analysis supports the hypothesis of transmission of pre-eclampsia from mother to fetus by a recessive gene. (Pridjian and Puschett, 2002; Hubel, 1999)

New research suggests primparity plays a larger role than primigravidity as a risk factor for the development of pre-eclampsia. Moreover, the duration the woman is exposed to the male antigens prior to conception is inversely related to the risk of developing pre-eclampsia. (Duley, 2003)

The pathophysiology of eclamptic seizures is not understood. These events are believed to arise from the same pre-eclampsia effects observed in other areas of the body. In the brain, cerebral vasospasm, oedema, ischemia, and ionic shifts between intracellular and extracellular compartments are believed to incite eclamptic seizures.

Nearly 10% of women with severe pre-eclampsia and 30-50% of women with eclampsia are affected by the

heamolysis, elevated liver enzymes, and low platelet count HELP syndrome. The exact relationship between HELP syndrome and pre-eclampsia is unknown. Women with pre-eclampsia and HELP syndrome develop hepatocellular necrosis and liver dysfunction. They also have an increased mortality rate, and one third of women with pre-eclampsia develop disseminated intravascular coagulation. (Weinstein L Syndrome, 1982; Sibai, 1990)

Severe pre-eclampsia is characterized by

A systolic blood pressure greater than 160 mmHg or diastolic blood pressure greater than 110mmHg on 2 occasions at least 6 hours apart in a women on bed rest

The presence of significant proteinuria. Marked proteinuria is defined as 5g or more of protein in a 24-hour urine collection.

Severe pre-eclampsia at time may be associated with oliguria.

Cerebral or visual disturbances.

Pulmonary edema or cyanosis.

Epigastric or right upper quadrant abdominal pain.

Impaired liver function.

Thrombocytopenia.

Or intrauterine growth restriction.

In mild pre-eclampsia hypertension and proteinuria are present but not to these extreme levels and the patient has no evidence of other organ dysfunction. (Wanger, 2004)

Epidemiology

Pre-eclampsia complicated 3-5% of first pregnancies and 1% of subsequent pregnancies with around 5-10% of cases being sever (Robson, 1999). The increased incidence of pregnancy induced hypertension noted among patients over the age of 35 years probably reflects undiagnosed chronic hypertension with super imposed pregnancy induced hypertension

Risk factors

Epidemiological risk factors for pre-eclamosia

Race: - Black women have higher rates of pre-eclampsia complicating their pregnancies compared to other racial groups, mainly because they have a greater prevalence of underlying chronic hypertension.

Age: - pre-eclampsia is more common at the extremes of maternal age (<18y or>35y). The increased prevalence of chronic hypertension in women older than 35 years can explain the increased frequency of pre-eclampsia among older gravidas.

Maternal risk factors for pre-eclampsia

First pregnancy

New partner/paternity

Age younger than 18 years or older than 35 years

History of pre-eclampsia

Family history of pre-eclampsia in a first – degree relative

Black race

Medical risk factors for pre-eclampsia

Chronic hypertension

Secondary causes of chronic hypertension such as hypercorticolism, hyperaldosteronism, pheochromocytoma, or renal artery stenosis

Preexisting diabetes (type 1 or type 2), especially with microvasculer disease

Renal disease.
Systemic Lupus Erythromatosis
Obesity
Thrombophilia
Placental/fetal risk factors for pre-eclamosia
Multiple gestations
HydropsFetalis
Gestational trophoblastic disease
Triploidy

Lab studies

Laboratory testing to evaluate chronic hypertension includes testing for target organ damage potential secondary causes of hypertension, and other risk factors.

Studies include urinalysis; CBC count; and serum sodium, potassium, creatinine, a levels (the presence of high levels of progesterone, an aldosterone antagonist, during normal pregnancy may mask the hypokalemia from hyperaldosteronism)

Optional tests include creatinine clearance, microalbuminuria, 24-hour urinary protein calcium, uric acid, glycosylated hemoglobin, and thyroid-stimulating hormone (TSH).

Serum lipids (i.e., total cholesterol, high-density lipoprotein [HDL], low-density lipoprotein triglycerides) predictably increase during pregnancy, so defer measurement until the postpartum period.

The increase in endogenous corticosteroid levels during normal pregnancy makes diagnose secondary hypertension due to adrenal hormone excess.

Routine tests when evaluating a patient for pre-eclampsia include CBC count, electrolytes creatinine, liver enzymes and bilirubin, and a urine dip for protein.
CBC count

In cases in which the platelet count is less than 150,000/mL, 75% are secondary dilutional thrombocytopenia of pregnancy 24% are due to pre-eclampsia, and of cases are due to other platelet disorders not related to pregnancy. Counts 100,000/mm^3 Suggest pre-eclampsia.

Hemoglobin levels greater than 13g/dl suggest the presence of hemoconcentration low levels may be due to microangiopathic hemolysis.

Urinalysis may be used as a screen for proteinuria. Trace levels to +1 proteinuria a acceptable, but levels of +2 or greater are abnormal and should be quantified with urine collection.

Serum creatinine usually is less than o.8mg/dl during pregnancy; higher levels suggested intravascular volume contraction or renal involvement in pre-eclampsia.

A serum uric acid level greater than 5mg/dl is abnormal and is a sensitive, but not marker of tubular dysfunction in pre-eclampsia.

Elevated levels of hepatic transaminases may reflect hepatic involvement in pre-eclampsia and may occur in the absence of epigastric pain.

In a 24-hour urine collection, the reference range for protein excretion in pregnancy 300mg/dl. Higher levels are abnormal and may reflect renal involvement in pre-eclampsia. Creatinine clearance increase approximately 50% during pregnancy, and levels less 100Ml/min suggest renal dysfunction that is either chronic or due to pre-eclampsia.

Examine peripheral blood smear for evidence of microangiopathic hemolysis and thrombocytopenia. A clinician easily can detect the presence of red blood cell (RBC) fragments; thus, laboratory findings establish the diagnosis quickly. The presence of fragments confirms microangiopathic hemolysis. Also consider the diagnosis of hemolytic uremic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP), and HELP (heamolysis, elevated liver enzymes, and low platelet count). In these cases, consenat hematologist to evaluate the need for plasmapheresis.

Prothrombin time (PT) and/or international normalized ratio (INR) and/or activated prothrombin time (a PTT) results may be abnormal in consumptive coagulopathy are disseminated intravascular coagulopathy complicating severe pre-eclampsia. Check PT/INR/a PTT is not necessary in the absence of abnormal liver transaminases or thrombocytopenia.

Abnormal values of lactate dehydrogenase (LDH), bilirubin, haptoglobin, fibrinogrn dimmers may confirm the presence of hemolysis and disseminated intravasculer coagulation along with coagulation testing. Checking LDH, bilirubin, haptoglobin, fibrinogen, is unnecessary unless PT/INR/a PTT results are abnormal, thrombocytopenia is pre-eclampsia.

Platelet count

Platelet is formed primarily in the bone marrow. They are released into the blood stream where they normally live for about a week. Platelet serves to clotting coagulation and maintaining vascular integrity. Platelet are small unnucleated corpuscles derived from bone marrow megakaryocytic in both the pregnant and non pregnant states, mature platelets circulate for 8-9 days platelets act to repair defects in the vascular endothelium and reduce hemorrhage by promoting bloodclot formation when activated, typically by vessel wall trauma.Platelets become adherent to themselves and all surfaces including glass. Hence the need to collect blood for platelet counts in bottles containing anticoagulant such as sodium, heparin or citrate. Endothelial damage

promotes platelet aggregation and degranulation which is inturn stimulates the release of prostaglandin and thrombxane A2, amplifying the platelet clumping and vasoconstriction. The normal non-pregnant platelet count is 150000-400000/mm^3. During pregnancy the platelet count falls progressively but tends to remain in the normal range.

The incidence of thrombocytopenia is greatest in the third trimester and this has been attributed to haemodilution and platelet consumption in the placentae if the platelet count has fallen below 150000/mm^3 in the majority of cases there no accompanying disease process The cause of be related to increase in consumption or destruction, much less commonly thrombocytopenia in pregnancy may, to a decrease production as may occur in malignant disease. In the majority of cases, the platelet count return to normal 4-6 weeks postpartum. Very occasionally more commonly if there is associated prolonged bleeding time- the possibility of VonWillbrand disease or a qualitative platelet disorders be considered

Classification of thrombocytopenia in pregnancy

Spurious.

Gestational.

Autoimmune; Idiopathic, Drug induced or HIV

Pre-eclampsia and HELP syndrome

DIC

Haemolytic Uraemic syndrome

Folate deficiency

Marrow Aplasia, malignant infiltration

Platelet count in pathophysiology of severe pre-eclampsia

Because thrombocytopenia can be induced acutely by pre eclampsia – eclampsia, the platelet count is routinely measured in hypertensive pregnant women. The frequency and intensity of maternal thrombocytopenia varies and likely is dependent on the intensity of the disease process, duration of pre eclampsia, and the frequency with which platelet count are performed. Overt thrombocytopenia, defined by a platelet count less than 100,000/mm3 indicates severe disease. In most cases, delivery is indicated because the platelet count continues to decrease. After delivery, the platelet count increases progressively reach a normal level within 3 to 5 days.

Thrombocytopenia results from platelet activation, aggregation, and consumption that is accompanied by increased mean platelet volume and decreased life span. A drop in platelet often precedes clinical signs of the disease levels of platelet-activating factor are in-creased. Platelet production is increased and thromboprotien, a cytokine that promotes platelet proliferation from megakaryocytic, is increased in pre eclampsia with thrombocytopenia. Paradoxically, in most studies, platelet aggregation is decreased compared with the normal increase seen in pregnancy. This likely is due to platelet “exhaustion” following in vivo activation. Although the cause(s) is unknown, immunological processes or simply platelet deposition at sites of endothelial damage may be implicated. Platelet-bound and circulating platelet-bindable immunoglobulin are increased, which suggest platelet surface alterations.

Patients and Methods

This study was carried out at80 pregnant women presented in third trimester of pregnancy attending AL-Zahraa teaching hospital in AL-Najaf starting from Martch2006 to October 2006.

40 sever pre-eclamptic women were studied and 40 apparently healthy pregnant women as a control group. The following inclusion criteria were followed:

Severe pre-eclampsia was diagnosed by blood pressure elevation≥ 160/110 in combination proteinuria at least (+2) with or withoutoedema, developing after 20 weeks gestation in a previously normotensive nonproteinuric patient.

Age range from 16-40 years.
Parity less than 7.
Gestational age range from 28-40 weeks.

The exclusion criteria were

History of diabetes, chronic hypertension, renal disease or other medical diseases full HELLP syndrome.

Women included in this study were subdivided into two groups:-

Group A

40 pregnant women in labor or prepared for c/s with severe pre eclampsia which was evidenced by a systolic blood pressure of ≥ 160 mmHg and diastolic blood pressure of ≥ 110 mmHg with proteinuria at least (+2).

Group B

40 apparently healthy pregnant women of comparable age, parity and stage of pregnancy as a control group.

Blood pressure was recorded in the sitting position with a cuff that is large enough for the subjects arm on at least two occasions 6 hours apart. Korotkoff phase 5(ks) disappearance of the sound was used to detect the diastolic blood pressure.

After medical, surgical, and obstetric history was taken, all women were subjected to full physical and obstetric examination and there were followed during their labor, postnatal period and peurperium with follow up of their newborns.

Proteinaria was diagnosed by collecting clean catch midstream urine sample in a clean dry container, then proteinuria was determined using the reagent strips (Albustrix).

A reading of +2 (1g/L) or more was considered to be positive result for proteinuria in addition to the routine laboratory tests, we perform maternal and neonatal blood platelet count.

Determination of maternal and neonatal platelet count.

Venous blood sample was taken by antecubital venipuncture without the use of tourniquet from the mothers.

While neonatal blood sample taken from umbilical vein immediately after delivery.

The blood sample putted into vacutainer tubes containing tri potassium EDTA. All samples were analyzed within 1 hr after collection, to minimize change in platelet.

Statistical analysis

Statistical analysis of the data was done to observe maternal and neonatal platelet count in the two groups and its relation to the maternal and neonate outcome. The statistical analysis was performed using the appropriate statistical tests in SPSS.

Results and Discussion

This table shows demographic characteristic of two study groups between severe pre-eclampsia and control group regarding maternal age, parity gestational age and blood pressure (systolic and diastolic).

There is a significant difference between two groups in systolic and diastolic blood preassure.

t-test analysis showed a significant differences in the both maternal and neonatal PL count for control and sever PE mother groups.

This table shows most of patients in severe pre eclampsia have maternal platelet counts in range of 100000-1500000/ mm3 while in control group most of them have maternal platelet count more than 1500000/mm3 and this is statistically significant between two groups.

This table shows most patients in severe pre-elampsia have neonatal platelet count in range of 100000-1500000/mm3 while in control group most of them have neonatal more than 1500000/mm3 and this is statistically significant.

This table shows maternal complications include imminent eclampsia, eclampsia and placental abruption in severe pre-eclambtic and finds that imminent eclampsia more common in maternal platelet between 100000-1500000/mm3 while eclampsia and placental abruption more common in maternal platelet count 100000/mm3

This table shows that most of patients have platelet count less than 100000/mm3 delivered by caesarian section although this is statically. This table shows comparison fetal complications (preterm birth, IUGR, and perinatal death) according to maternal platelet count shows most of preterm birth in maternal platelet count more than 1500000 (62.5%) while IUGR in maternal platelet count less than 100000 (66.67%) and perinatal death more in maternal platelet count between 100000-1500000/mm3 (11.54%)

This table shows comparison fetal complications (preterm birth, IUGR, perinatal death) according to neonatal platelet count shows most of patients in platelet count more than 1500000/mm3 (26%) while IUGR more

common in neonatal platelet less than 100000/mm3 (31.1%) and perinatal birth more common in neonatal platelet count 100000-1500000/mm3 (4%) no significant associations between the groups. Two-way ANOVA showed a non-significant differences in the platelets count according to the Gestational age [F=0.976, *P* (0.326)>0.05] also there is no significant differences between mean maternal plateletes count and Mean neonatal platelet count [F= 3.033, *P* (0.086) >0.05]. Tests demonstrated non-significant interaction between Gestational age and platelet count [F=0.525, *P* (0.471)>0.05].

Table.1 Mean (±SD) of Age,Parity, Gestational age Diastolic & Systolic Blood Pressure of Patients in Control and Severe PE Groups

Parameters	Control (Mean±SD)	Sever PE (Mean±SD)	P value
Age (year)	24.75±7.813	24.59±7.573	0.940
Parity	1.75±1.943	1.31±2.092	0.435
GestationalAge (week)	38.45±1.317	36.00±2.259	0.940
Diastolic BloodPressure (mmHg)	73.00±8.013	114.87±7.208	0.0001 *
SystolicBloodPressure (mmHg)	117.00±9.234	173.00±15.473	0.0001 *

* Significant differences at α≤0.05.

Table.2 Relationship between maternal and neonatal platelet count in severe pre-eclampsia (PE) and control group

Group	Maternal platelet count($x10^3$)/ mm^3 Mean (±SD)	Neonatal platelet count/ $mm3(x10^3)$ Mean (±SD)
Severe pre eclampsia (n = 40)	124.4 (±33.53)	113.78 (±37.20)
Control (n = 40)	185.5 (±21.631)	176.5 (±39.20)
P. value and t test	0.0001 * (t=9.102)	0.0001* (t=7.124)

* Significant differences at α≤0.05.

Table.3 The relationship between maternal and neonatal platelet count in seven pre eclampsia according to gravidity

Gravidity	No.	Maternal platelet count x 10^3 / mm^3 Mean (±SD)	Neonatal platelet count x 10^3/ mm^3 Mean (±SD)
G1	21	125.4 (±38.2)	112.4 (±35.5)
G2-G4	12	122.0 (±30.6)	110.0 (±26.1)
G5 and more	7	125.0(±22.5)	115.7 (±25.7)

No significant interaction between the effects of gravidity on maternal and neonatal platelet count.

Table.4 Maternal platelet count in severe pre eclampsia and control groups

Mean maternal platelet count / mm3	Platelet Count range			
	<100000	100000-150000	>150000	X^2 P-value
SEVERE PE GROUP A	6	24	8	38.1 0.0001*
CONTROL GROUP B	0	4	36	

Significant differences at α≤0.05.

Table.5 Neonatal platelets count in severe preeclampsia and control group

Mean neonatal platelet count / mm3	Platelet Count			
	<100000	100000-150000	>150000	X^2 P-value
SEVERE PE GROUP A	14	20	6	33.333 0.0001*
CONTROL GROUP B	0	10	30	

* Significant differences at α≤0.05.

Table.6 Maternal complications in severe preeclampsia according to maternal platelets count

Maternal platelet count /mm3($X10^3$)	No.	Maternal complications			X^2 P-value
		Imminanteclampsia %	Eclampsia %	Placental abruption %	
<100	6	33.33	33.33	33.33	**0.000 *23.453**
100-150	26	34.6	19.2	11.5	
>150	8	12.5	0	12.5	

* Significant differences at α≤0.05.

Table.7 Mode delivery in patients with severe preeclampsia according to maternal platelet count

Maternal plalelet count /mm3($X10^3$)	No.	Mode of Delivery (%)		X^2 P-value
		C.S	V.D	
<100	6	83.33	16.67	0.867 0.648
100-150	26	65.38	34.62	
>150	8	75.00	25.00	

Table.8 Neonatal complications in severe preeclampsia according to maternal platelet count

Maternal plalelet count /mm3($X10^3$)	No.	Fetal complications(%)		
		Preterm birth	IUGR	Perinatal death
<100	6	33.33%	66.67%	0%
100-150	26	30.77%	53.85%	11.54%
>150	8	62.5%	62.5%	0%
X^2 = 2.76 **P-value = 0.599**				

Table.9 Neonatal complications in severe preeclampsia according to neonatal platelet count

Neonatal platelet count $X10^3/mm^3$	No.	Neonatal Complications		
		PTL	LUGR	Perinatal death
<100	14	5 (14.2%)	11 (31.1)	1 (2.8%)
100-150	20	8 (16%)	15 (30%)	2 (4%)
>150	6	4 (26%)	1 (26.6%)	0
X^2 = 4.547 **P-value = 0.603**				

Table.10 The relationship between gestational age and mean maternal and neonatal platelet count in severe pre eclampsia

Gestational age (Weeks)	No.	Mean maternal platent count X10^3 /mm^3	Mean neonatal platelet count X 10^3 /mm^3
28-36	19	123.5±40.03	105.5±28.32
≥37	20	125.42±25.86	118.05±33.34

Pre-eclampsia is progressive disorder of endothelial function that is unique to pregnancy and probably results from various micro vascular diseases: - it arises in the placenta but may affect all major organ systems of the mother, including the brain, kidney, liver, and lung.

Although most cases of pre-eclampsia are first detected at routine screening of symptom free women attending routine antenatal clinics, the mode of presentation is highly variable and a small proportion of women present with the symptoms of systemic complications such as headache, visual disturbance, and eclampsia.

Pre-eclampsia complicated by raised liver enzyme concentrations and a low platelet count with or without haemolysis is at least as common eclampsia and often more difficult to manage, but it is less well recognized.

Several authors have reported high rates of serious maternal and perinatal problems such as hepatic failure, renal failure, pulmonary oedema, placental abruption, preterm birth, IUGR, and perinatal asphyxia. Early diagnosis and prompt action are essential, therefore, to help avert life threatening complications. So pre-eclampsia is importance cause of maternal and neonatal mortality and morbidity it accounts for around 16% of maternal death in the UK and match higher in developing countries. Of maternal death 40% are associated with eclampsia. Cerebral hemorrhage in the principle cause of death although pulmonary complications have now superseded cerebral causes. Over all perinatal mortality in pre-eclampsia is around 35/1000 total birth but may reach 160/1000 in severe disease.

The most important features determining outcome is gestational age add delivery. Morbidity increase two folds if the fetus small for gestation. Our study shows no significant difference in demographic characteristic of two study groups between severe pre-eclmpsia and control group apart from systolic and diastolic blood pressure were show significant difference between them also shows there is significant difference in both maternal and neonatal platelet count in severe pre-eclaampsia and control group.

Thrombocytopenia is a charecteristic of worsening pre-eclampssia, and it is probably caused by platelet activation and aggregation as well as microangiopathic haemolysis induced by severe vasospasm evidence of gross haemolysis suchas. Haemoglobinemia, haemoglobinuren or hyper bilirubinemia is indication of sever disease.

There is no significant interaction between effects of gravidity on maternal and neonatal platelets counts. This is considered with study Baker and Cunningham, 1999 also our result similar to study curried out at department of obstetric and gynecology, Gulhane military medical academy Ankara Turkey by Geyhan T *et al.*, may 2006.

Our study shows most of severe pre-eclamptic patient have platelet count in range of 100,000-150,000/mm^3 while in control normotensive women most of them have platelet count more than 150,000/mm^3 and this is agreement with study done by Rowland *et al.*, 2000.

The present study also shows most of neonate severe pre-eclamptic women have platelet count range from 100,000 -150,000/mm^3 while most of neonate of normotensive women have platelet count more than 150,000 and this is agreement with Thiagarjah *et al.*, (1984) and Weinstein 1985. So a neonatal platelet count usually follows that of maternal platelet count.

Also maternal complication include imminant eclampsia and placental a braption in severe pre eclampsia and finds that imminant eclampsia more common in maternal platelet between 100,000-150,000/mm^3 while eclampsia and placental abruption more common in maternal platelet count 100,000/mm^3 no case of DIC, Renal failure, pulmonary oedema recorded this may be due to small sample size and we need further large study to see the relationship between platelet count and these complications. And this agreement with Gulhane military medical academy, Ankara, Turkey BY Geyhan T and *et al.*, may 2006. Also most of patient whose platelet count less than 100,000/mm^3 delivered by caesarean section although this is statistically not significant high percentage of caesarean section in platelet count less

than 100,000 may be due to maternal complications that more common in this group which need immediate delivery maternal thrombocytenia in severally preeclampsia women is not indication of caesrean section for fetal sake.

Also our study shows comparison between fetal complications (preterm birth, intrauterine growth restrication, perinatal death) according to maternal platelet count shows most of preterm birth in platelet count >150,000/mm^3 while intrauterine growth restricted more common in platelet count less than 100,000 and perinatal mortality in is more in platelet count 100,000-150,000/mm^3 so maternal platelet count cannot used as predictor for neonatal complications while it is helpful in predction maternal complications and this agreement with baker and cuningham, 1999 also our result similar to study, carried out at department of obstetric and Gynaecology, Guhane military academy, Ankara, Turkey by Geyhn T *et al.,*

Also our study shows comparison of fetal complications (pretermbirth, intrauterine growth restricted and perinatal death) in relation to neonatal platelet count shows most of preterm birth in neonatal platelet count more than 150,000/mm^3 while perinatal death) in relation to neonatal platelet count shows most of preterm birth in neonatal platelet count more than 150,000/mm^3

While intrauterine growth restricted in neonatal platelet count 100,000-150,000/mm^3 while perinatal death in neonatal platelet count 100,000-150,000/mm^3 so neonatal platelet count also can notused as a prediction of neonatal outcome. No case of cphalohaematoma was reported and this is consistant study baker and ccunningham, 1999 also study of Gulhane military medical according Gynaecology, Guhane military academy, Ankara, Turkey by Geyhn T *et al.,*

Maternal platelet counts is a prediction of maternal complications in preeclampsia. Maternal and neonatal platelet counts can notused as prediction of neonatal complications. Termination of pregnancy for maternal sake guided by maternal platelet count in severe pre-eclmpsia is a advisable while for fetal sake unadvisable.

Recommendations

Platelet count are used as predictor of maternal prognosis in patient with severe preeclampsia. Further study are needed which include a large sample size to observe the relationship of platelet count to development of other maternal and fetal complications.

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