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Original Research Article

Determination of oxidative stress and some haematological coagulation parameters for assessment of severity of preeclamsia in gravidas.

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Abstract

Background & Methods: The aim of the study is to determine of oxidative stress and some haematological coagulation parameters for assessment of severity of preeclamsia in gravidas. Plasma samples for assay of coagulation parameters were stored at -800C & serum samples were stored at -200C in deep freezer until analysis. Random urine samples for albumin /creatinine ratio (UACR) were also collected.

Results: The number of primigravidas were (66%) whereas the number of multigravidas were along (34 %.). In Severe, the primigravidas were 88% & 12% respectively. The number of primigravidas were (66%) whereas the number of multigravidas were along (34 %.). In Severe, the primigravidas were 88% & 12% respectively.

Conclusion: Coagulation parameters (APTT & PT) are the most important markers to detect preeclampsia & suggest how much the disease could be complicated. APTT & PT markers are only helpful when the placental abruption occurs or there is an abnormal elevation of liver function tests. Thus they should be estimated only if these complications are observed thus decreasing hospital charges without compromising patient safety.

Keywords: gravidas, coagulation, hematological & preeclampsia.

Study Design: Observational Study.

Introduction

Preeclampsia, also called as toxemia of pregnancy, is a medical condition, characterized by new onset of hypertension (SBP >140 mm of Hg or DBP >90mm of Hg) & proteinuria (>0.3g or 300mg protein in a 24 hour urine specimen or 1+ on dipstick) & also may be associated along myriad other signs & symptoms, such as pitting edema, visual disturbances, headache, & epigastric pain after 20th weeks gestation in a previously normotensive & non proteinuric patients[1]. The clinical features usually develop in the latter part of the third trimester i.e. close to term (near 38-40th weeks gestation) & progress until delivery. Preeclampsia may also occur

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in the immediate post-partum period. This is referred to as “postpartum preeclampsia”. It may occur up to a week following delivery of the baby or six weeks postpartum[2]. It is extremely rare prior to 20 weeks gestation as in cases of hyditiform moles, molar pregnancy, antiphospholipid antibody syndrome & acute polyhydramnios. Delivery of fetus & placenta remains the only curative treatment & should be timely. Preeclampsia can severely affect the health of both the mother & the unborn child & contribute significantly to prematurity[3].

It is characterized by abnormal vascular response to placentation that is associated along increased systemic vascular resistance, enhanced platelet aggregation, activation of coagulation system, DIC, endothelial cell dysfunction or endothelial cell injury, generalized vasospasm, poor blood perfusion, intravascular volume depletion, acute renal failure, cerebrovascular & cardiovascular complications, circulatory collapse & maternal death[4].

Preeclampsia has been variably called as PET (preeclamptic toxemia), GPH (gestational proteinuric hypertension), toxemia, hypertension gestosis, & HOP (hypertension, oedema & proteinuria). Preeclampsia & PIH are interchangeable terms. The term PIH is defined as the hypertension that develops as direct result of the gravid state. Thus PIH is the isolated hypertension occurring during pregnancy[5]. It is considered as very serious condition & requires careful monitoring of mother & fetus. PIH includes gestational hypertension, preeclampsia & eclampsia. Gestational hypertension is hypertension without proteinuria or other findings of preeclampsia. It first occurs at >20 weeks of gestation along no proteinuria before pregnancy & resolves by 12 weeks (usually by 6wk) postpartum. If gestational hypertension is present along proteinuria, the condition is then referred to as preeclampsia. One fourth of women along gestational hypertension develop proteinuria & thus progress to preeclampsia[6].

Material & Methods

Present Study was conducted at Index Medical College Hospital & Research Centre, Indore, M.P for 01 Year. Blood samples were collected in citrate/ plain/ EDTA vials for analysis of Coagulation/ Biochemical/ Hematological parameters respectively. Sample collection during febrile duration was avoided. 5 ml of venous blood was drawn from the antecubital aseptically & was allowed to clot. Centrifugation of these specimens was done for ten minutes at room temperature & at 2500 x g.

Group-C: Age matched healthy normotensive non-pregnant women (N=50).

Group-D: Healthy normotensive pregnant women in third trimester of pregnancy (28-40 weeks) (N=50).

INCLUSION CRITERIA

1. Healthy individuals.

2. Child bearing age between 20-40 years.

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EXCLUSION CRITERIA

1. Severe anemic pregnant women were excluded. (Hb <6 gm/dl)

2. Past history of Diabetes, chronic infection, hypertension, chronic renal disease, active urinary tract infection, absence of thromboembolic disease, viral hepatitis.

Result

Table No. 1: Comparison of Mean values of Anthropometric factors

	Group C		Group D		P Value
	Mean	**SD**	**Mean**	**SD**	
Age (yrs)	21.96	6.47	21.03	4.49	0.37
BMI (Kg/m2)	23.78	1.59	23.21	1.95	0.61
Gestational age (wks)	-	-	36.37	6.37	0.04
Systolic blood pressure (mm of Hg)	112.11	3.09	114.27	11.25	0.04
Diastolic blood pressure (mm of Hg)	74.24	2.67	75.33	8.37	0.02

The age & BMI between Group C & Group D was not significant ($p>0.05$) statistically. The mean values of gestational age, systolic & diastolic blood pressure when compared between Group C & Group D were significant ($p<0.05$) statistically.

Table No. 2: Distribution of cases as per gravidas in Mild Preeclamptic & Severe Preeclamptic.

	Mild Preeclamptic		Severe Preeclamptic	
	No.	**%**	**No.**	**%**
Primigravidas	**33**	**66**	**44**	**88**
Multigravidas	**17**	**34**	**06**	**12**

The number of primigravidas were (66%) whereas the number of multigravidas were along (34 %.). In Severe, the primigravidas were 88% & 12% respectively.

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Table No. 3: Comparison of Mean values of Coagulation Factors in Mild Preeclamptic & Severe Preeclamptic

	Group C		Group D		P Value
	Mean	**SD**	**Mean**	**SD**	
APTT (sec	29.21	6.47	30.37	4.49	0.78
PT (sec)	12.56	1.59	13.64	1.95	0.09
Fibrinogen (mg/dl)	613.41	-	689.47	6.37	0.03
FDP (µg/ml)	9.71	3.09	10.66	11.25	0.04
AT III (%)	77.83	2.67	74.29	8.37	<0.001

The mean values of APTT & PT were not significant ($p>0.05$) statistically whereas the mean values of Fibrinogen & FDP were significant ($p<0.05$). On comparing the mean value of AT III between Subgroup C & Subgroup D was highly significant ($p<0.001$) statistically.

Discussion

Hypertensive disorders of pregnancy are one of the leading causes of maternal mortality & severe maternal morbidity. Preeclampsia is one of several blood pressure disorders that are common complications of pregnancy. Typically developing after the 20th week of pregnancy, it can lead to many complications for the expectant mother & her fetus. If untreated, the condition may escalate to a syndrome characterized by HELLP or eclampsia (preeclampsia along seizures), two potentially fatal conditions[7]. Complications of preeclampsia for the fetus include premature birth & intrauterine growth retardation. Thus preeclampsia contributes to the death of one pregnant woman every 3 min & records one of the five causes of maternal death in the world. Although the pathogenesis of preeclampsia is not well understood, preeclampsia seems to start along a placenta that doesn't grow the usual network of blood vessels deep into the uterine wall. This leads to poor blood circulation through the placenta.

In addition to causing mild to severe high blood pressure, preeclampsia can also cause problems along blood supply to the fetus & sometimes along the mother's liver, kidney, & brain functions.

The possible reason for reduction of AT III in preeclampsia firstly may be because it inactivates thrombin by forming thrombin–antithrombin complexes. AT is an important regulator of thrombin in blood coagulation[8]. A decrease in plasma AT level indicates increased thrombin binding, secondary to increased thrombin generation. Secondly due to reduced hepatic synthesis. Thirdly the extrinsic loss is observed in nephrotic patients. Another major cause may be enhanced consumption due to generalized fibrin deposition in maternal vascular tree. The glomerular endotheliosis of preeclampsia is associated along fibrin deposition & AT III is consumed in this process. In a similar manner AT III may be consumed in the liver, since fibrin is deposited in the periportal areas. Hypertension occurring in preeclampsia might be responsible, at least in part, for endothelial dysfunction that could lead to low AT III activity due to intravascular coagulation[9-10].

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Conclusion

The gestational age was found to be lower in preeclamptic pregnant women & it further decreased along the severity of the disease. Blood pressure is an indicator for the severity of preeclampsia. An increase in blood pressure is one of the essential criteria in the diagnosis of preeclampsia. We therefore conducted an estimation of maternal levels of BP to predict maternal & fetal complications in preeclampsia.

Coagulation parameters (APTT & PT) are the most important markers to detect preeclampsia & suggest how much the disease could be complicated. APTT & PT markers are only helpful when the placental abruption occurs or there is an abnormal elevation of liver function tests. Thus they should be estimated only if these complications are observed thus decreasing hospital charges without compromising patient safety.

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