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RISK FACTORS AND MODE OF DELIVERY IN SEVERE PRE-ECLAMPSIA PATIENTS ATTENDING LD HOSPITAL.

Obstetrics & Gynaecology

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KEYWORDS

INTRODUCTION:

Hypertensive disorders represent the most common medical complication of pregnancy affecting between 7% and 15% of all gestations and account for approximately a quarter of all antenatal admissions1. According to World Health Organization's (WHO) systemic review on maternal mortality worldwide, hypertensive disease remains a leading cause of direct maternal mortality. Together with haemorrhage and infection, hypertension forms the deadly triad that contributes to morbidity and mortality during pregnancy and childbirth2. Although maternal mortality is much lower in high-income countries than in developing countries, the incidence of pre-eclampsia has risen in the United States. This might be related to an increased prevalence of predisposing disorders such as chronic hypertension, diabetes and obesity3. About 16% of maternal deaths were attributed to hypertensive disorders in developed countries and over half of these hypertension-related deaths were preventable4. Recent confidential enquiry into maternal deaths in the United Kingdom found hypertensive disorders to be the second leading direct cause of maternal death5 Hypertensive disorders are responsible for not only maternal deaths but also substantial morbidity for the pregnant women. One-third of severe maternal morbidity was a consequence of hypertensive conditions in the United Kingdom. Five per cent of women (1 in 20) with severe pre-eclampsia or eclampsia were admitted to intensive care6. Longterm impact of hypertension in pregnancy in the form of chronichypertension and increased lifetime cardiovascular risk is also present. In India, the incidence of pre-eclampsia is reported to be 8-10% among the pregnant women. According to a study, the prevalence of hypertensive disorders of pregnancy was 7.8% with pre-eclampsia in 5.4% of the study population in India7. Hypertension in pregnancy is defined as: systolic pressure level of 140mmHg or higher and/or diastolic blood pressure of 90 mmHg or higher (Korotkoff V)8,9

These measurements have to be confirmed on at least two occasions 4 to 6 hours apart but within a maximum of a week period.

Risk Factors for Pre-eclampsia10.

COUPLE RELATED RISK FACTORS:

- Primipaternity
- Limited sperm exposure
- Pregnancy after donor insemination, donor egg, donor embryo.

Maternal or pregnancy related risk factors:

- Extremes of age
- Obesity and insulin resistance/gestational diabetes / Smoking
- Multifetal pregnancies
- Pre-eclampsia in previous pregnancy
- Maternal low birth weight
- Family history of pre-eclampsia
- Pre-existing medical disease:
- Pre-gestational diabetes
- Chronic hypertensive or renal disease
- Maternal immunological disease
- Preexisting thrombophilia, antiphospholipid antibody syndrome.

CLASSIFICATION OF HYPERTENSIVE DISORDERS IN PREGNANCY:

The clinical classification adopted by the International Society for the Study of Hypertension in Pregnancy (ISSHP) reflects the pathophysiology of the disorder as well as the potential maternal and perinatal risks and outcomes11.

Hypertensive disorders during pregnancy can be included into four well-defined groups:
Gestational hypertension

- Pre-eclampsia, eclampsia

Chronic hypertension(Essential & Secondary)

- Pre-eclampsia superimposed on chronic hypertension

Gestational hypertension:

New onset hypertension developing after 20 weeks of gestation, during labour, or in the first 24 hour postpartum, without proteinuria, or any other systemic features of pre-eclampsia, in a previously normotensive nonproteinuric woman and the blood pressure resolves within 3 months postpartum. Pre-eclampsia: Hypertension associated with proteinuria greater than 0.3g/L in a 24-hour urine collection or 1+ by qualitative urineexamination, after 20 weeks of gestation. Eclampsia: Convulsions occurring in a patient with pre-eclampsia are known as eclampsia. HELLP syndrome: Severe form of pre-eclampsia characterized by hemolysis (abnormal peripheral blood smear, bilirubin >= 1.2 mg/dL), thrombocytopenia (< 100,000/mm3) and elevated liver enzymes (AST > 70 U/L, LDH > 600 U/L). Chronic hypertension is defined as hypertension present before 20th week of pregnancy or that is diagnosed preconceptionally. Hypertension should be documented on at least two occasions, measured at least 4 hours apart. Blood pressure elevation that persists 12 weeks postpartum is also retrospectively considered as chronic hypertension. Essential hypertension is diagnosed when there is no apparent underlying cause for chronic hypertension. Secondary hypertension may be caused by renal parenchymal disease or scarring, renovascular disease, endocrine disorders or coarctation of aorta.

Pre-eclampsia superimposed on chronic hypertension: It is diagnosed when one or more features of preeclampsia (e.g. elevated liver enzymes, low platelets, proteinuria) develop for first time during pregnancy after 20 weeks, in a woman with pre-existing chronic hypertension. As per the NICE guidelines, for the purpose of management, hypertension is further divided as per the severity into mild, moderate and severe hypertension12.Mild hypertension: Systolic blood pressure 140-149 mmHg, diastolic blood pressure 90–99 mmHg. Moderate hypertension: Systolic blood pressure 150-159 mmHg, diastolic blood pressure 100–109 mmHg. Severe hypertension: Systolic blood pressure 160 mmHg or greater, diastolic blood pressure 110 mmHg or greater. DIAGNOSTIC CRITERIA FOR PREECLAMPSIA14 BLOOD PRESSURE Systolic BP of 140 mm Hg or more or diastolic BP of 90 mm Hg or more on two occasions at least 4 hours apart after 20 weeks gestation in a woman with previous normal BP.

Systolic BP of 160 mm Hg or more or diastolic BP of 110 mm Hg or more (severe hypertension can be confirmed within a short interval [minutes] to facilitate timely antihypertensive therapy) And Proteinuria 1. 300 mg or more per 24-hour urine collection (or this amount extrapolated from a timed collection) or 2. Protein/creatinine ratio of 0.3 mg/dL or more or 3. Dipstick reading of 2+ (used only if other quantitative methods not available)

Or in the absence of proteinuria, new-onset hypertension with the new onset of any of the following: Thrombocytopenia: Platelet count less than 100,000 × 109/L. Renal insufficiency: Serum creatinine concentrations greater than 1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease.

Impaired liver function: Elevated blood concentrations of liver transaminases to twice normal concentration.

Pulmonary oedema. New-onset headache unresponsive to medication and not accounted for by alternative diagnoses.

Visual symptoms. CRITERIA FOR SEVERE PRE-ECLAMPSIA14 BP of ≥160 mm Hg systolic or ≥110 mm Hg diastolic, recorded on at least two occasions at least 4 hours apart (unless antihypertensive therapy is initiated before this time)

Thrombocytopenia (platelet count less than 100,000 × 109/L) Impaired liver function as indicated by abnormally elevated liver enzymes (to twice the upper limit normal concentration), and severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by alternative diagnoses.

Renal insufficiency (serum creatinine concentration more than 1.1 mo/dL or a doubling of the serum creatinine concentration in the absence of other renal disease)

Pulmonary oedema New-onset headache unresponsive to medication and not accounted for by alternative diagnoses

Visual disturbances.Clinical features of preeclampsia are15 Severe headache Visual disturbances Epigastric pain and/or vomiting Signs of clonus Papilloedema Liver tenderness Platelet count below 100 x 106/l Abnormal liver enzyme (ALT or AST rising to above 70 IU/L) HELLP syndrome.

THEORIES ABOUT ETIOPATHOGENESIS OF PREECLAMPSIA
1. ABNORMAL PLACENTATION Failure of secondary wave of trophoblastic invasion into myometrial spiral arterioles results in reduced uteroplacental blood flow. The ensuing ischemia and hypoxia leads to aberrant expression of genes which encode for proinflammatory cytokines capable of eliciting endothelial dysfunction16.

2. ENDOTHELIAL DYSFUNCTION Endothelial dysfunction resulting in a. Loss of vascular integrity is evidenced by elevated biomarkers of endothelial dysfunction such as plasma fibronectin and thrombomodulin.17
b. Increased vascular reactivity results in generalised and intense vasospasm leads to reduced perfusion. This is due to increase in vasoconstrictors thromboxane and endothelin and increased sensitivity to angitensin II and decrease in vasodilators nitric oxide and prostacyclin.18
c. Activation of coagulation cascade.

3. IMMUNOLOGICAL FACTOR Immune maladaptation in pre-eclampsia. a) Pathological lesions in placenta similar to acute graft rejection. b) Lower level messenger RNA for HLA-G.19 c) Th1/Th2 response with Th2 dominance. Cytokines like tumour necrosis factor-α, interleukin-2 and 6 mediate immune maladaptation20. d) Impaired production of blocking antibodies.

4. GENETIC PREDISPOSITION Explained by both single gene model and polygenic inheritance. a) Tendency for preeclampsia is inherited21 b) Women heterozygous for the angiotensin gene variant T235 had a higher incidence of preeclampsia.22
c) Association between HLA DR4 and preeclampsia.23 d) Inherited thrombophilia predispose women to preeclampsia

5. NUTRITIONAL FACTOR Mac Gillivray viewed the evidence for the role of dietary deficiency in the pathology of preeclampsia a) Obesity: C reactive protein is increased associated in turn with preeclampsia.24
b) Ascorbic acid: Incidence of preeclampsia was doubled in women whose dietary intake of ascorbic acid was less than 85 mg. 25
c) Calcium deficiency: When concentration of calcium is low in extra cellular fluid amount of calcium entering the cell increases making vascular smooth muscle more sensitive to excitation.

6. OXIDATIVE STRESS
a) Preeclampsia may have its origin in a disturbed oxidative mechanism.
b) Abnormal levels of lipid peroxidise in preeclamptic women inhibits prostaglandin synthetase.
c) Risk of preeclampsia increased in women with increased oxidised low density lipoprotein and triglyceride26.

Mode of delivery in patients with severe pre-eclampsia:-
1-The only treatment of severe pre-eclampsia is delivery of the fetus. However, sometimes this is delayed for monitoring of patient after increasing the dosage of anti hypertensives and providing the fetus with betamethasone exposure for lung maturity. This should be carried out at a tertiary care center with facilities for proper monitoring and maternal interest should not be neglected any time during the course .Indications for termination included uncontrolled BP in 33 %, eclampsia in 13%, colour Doppler changes in 13%, intrauterine death in 12%, plumonary oedema in 6 %, Abruption in 5%, anhydramnios in 5 %, preterm labour in 4 %, HELLP in 4 %, AFD in 4% and renal dysfunction as seen by rising creatinine in 1%.

2-The mode of delivery was lscs in 77% and 23% had vaginal deliveries. Out of the 77 LSCS, the following indications were seen: Previous LSCS (23.3%), Color doppler changes (13%), Severe FGR (11.6%), Anhydramnios (11.6%), NRCTG (10.3%), FOI (9.1%), NPOL (7.8%), AFD (5.2%), Breech (4%), Precious pregnancy (4%). In most cases of severe pre-eclampsia before 34 weeks, induction of labour is unsuccesstul and approximately 80% these women will end up having a caesarean delivery53. Similar results were concluded by A. Nankali et al,82 who in their study found a rate of vaginal delivery 34.4% and lscs65.6%.

CONCLUSION
In this study of Risk Factors and Mode of delivery in patients with severe pre-eclampsia detected before 34 weeks of gestation while maternal outcome suggests that severe pre-eclampsia can be managed under monitoring and proper antihypertensive treatment, till corticosteroids are given to the mother for fetal lung maturity, the fetal outcome however for gestational age 28 to 32 weeks came out poor and that for gestational age 32 to 34 weeks is still better and worth giving a chance for. At any given time with onset of signs and symptoms suggesting worseing of maternal or fetal condition pregnancy was terminated in view of maternal interest or when worsening of fetal condition was suggested where prolongation of pregnancy would not provide any benefit to mother or fetus.

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