AHMEDABAD OBSTETRICS & GYNAECOLOGICAL SOCIETY

AOGS TIMES

2024 - 25

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Motto : Knowledge is Power - Unity is Strength
Theme : Health & Happiness for Her

OCTOBER 2024 I VOLUME 07

HEALTH & HAPPINESS FOR HER

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TEAM AOGS MESSAGE

Dr. Sunil Shah
President

Dr. Akshay C. Shah
Hon. Secretary

Dear friends,

We have a very successful ICOG FOGSI conference at our karnavati club under the able leadership of Dr. Parul Kotdawala. We did four workshops and a conference. The workshops were at VS hospital. The committee is thankful to Dr. Shaswat Jani and VS hospital dean. It will be difficult to name a few and miss some as all the committee members have worked hard to make this conference successful.

Looking forward to more such academic extravaganzas.

Thank you,

9th ICOG FOGSI ANNUAL CONFERENCE 2024

Theme : Safe Practice in Ob.Gyn.

18 19 20 OCTOBER, 2024 VENUE : **Karnavati Club,** Sarkhej - Gandhinagar Highway, Ahmedabad.

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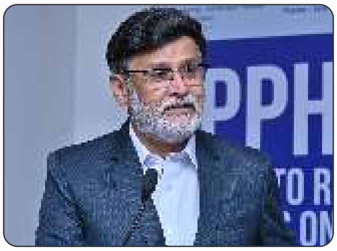
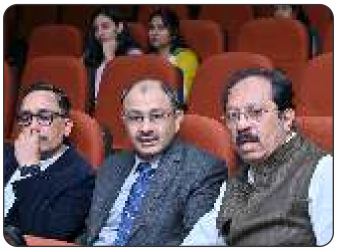












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DEFINITION, PATHOPHYSIOLOGY AND CLASSIFICATION OF PREGNANCY INDUCED HYPERTENSION

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Preeclampsia' (PE) as a disease refers to new onset hypertension with significant end-organ dysfunction in the presence or absence of proteinuria in a pre-gestational normotensive patient, typically after 20 weeks of gestation. PE accounts for almost 8% of all gestational-related complications and excess of greater than 50,000 maternal deaths and 500,000 fetal deaths globally.

PATHOPHYSIOLOGY

Studies during the past decade, however, have provided a better understanding of the potential mechanisms responsible for the pathogenesis of PIH. The initiating event in PIH appears to be reduced uteroplacental perfusion as a result of abnormal cytotrophoblast invasion of spiral arterioles. Placental ischemia is thought to lead to widespread activation/dysfunction of the maternal vascular endothelium that results in enhanced formation of endothelin and thromboxane, increased vascular sensitivity to angiotensin II, and decreased formation of vasodilators such as nitric oxide and prostacyclin. The quantitative importance of the various endothelial and humoral factors in mediating the reduction in renal hemodynamic and excretory function and elevation in arterial pressure during PIH is still unclear. Investigators are also attempting to elucidate the placental factors that are responsible for mediating activation/dysfunction of the maternal vascular endothelium. Microarray analysis of genes within the ischemic placenta should provide new insights into the link between placental ischemia and hypertension. More effective strategies for the prevention of preeclampsia should be forthcoming once the underlying pathophysiologic mechanisms that are involved in PIH are completely understood.

The recommended classification for hypertensive disorders of pregnancy is as follows:

Hypertension known before pregnancy or present in the first 20 weeks:

1. Chronic hypertension
a. Essential
b. Secondary
2. White-coat hypertension
3. Masked Hypertension

Hypertension arising de novo at or after 20 weeks:

1. Transient gestational hypertension
2. Gestational hypertension
3. Pre-eclampsia – de novo or superimposed on chronic hypertension
4. Preeclampsia with severe features
5. Eclampsia
6. Preeclampsia superimposed upon chronic hypertension
7. Chronic hypertension with superimposed preeclampsia with severe features 8.HELLP

1.Chronic Hypertension

Chronic hypertension refers to high blood pressure predating the pregnancy or recognised at < 20 weeks' gestation. In practice, this is often diagnosed for the first time at the first or early second trimester booking visit. Ideally, this 'office' or 'clinic' hypertension should be confirmed by 24 h. The majority of cases are due to essential hypertension. Secondary causes are uncommon. Hypertension diagnosed or present before pregnancy or on at least two occasions before 20 weeks of gestation. Hypertension that is first diagnosed during pregnancy and persists for at least 12 weeks postpartum is also considered chronic hypertension.

- Blood pressure criteria during pregnancy are:
- Systolic ≥140 mmHg and/or diastolic ≥90 mmHg
- Prepregnancy and 12 weeks postpartum blood pressure criteria are:
- Stage 1 – Systolic 130 to 139 mmHg or diastolic 80 to 89 mmHg • Stage 2 – Systolic ≥140 mmHg or diastolic ≥90 mmHg

2.Transient Gestational Hypertension

Transient gestational hypertension is de novo hypertension that develops at any gestation that resolves without treatment during the pregnancy. Transient gestational hypertension is not a benign disorder; it is associated with approximately 20% chance of developing preeclampsia and a further 20% chance of developing gestational hypertension. Therefore, such women should receive extra monitoring throughout their pregnancy, ideally including home BP measurements.

3.Gestational hypertension

Gestational hypertension is persistent de novo hypertension that develops at or after 20 weeks' gestation in the absence of features of preeclampsia. Gestational hypertension is not a uniformly benign condition. The risk of complications is dependent on the gestational age at which it develops. Gestational hypertension is important for two reasons: Firstly, Pre-eclampsia may develop in 25% of such women, this rate being higher the earlier the presentation [4]; to date, no tests have reliably predicted which women with gestational hypertension will later develop preeclampsia [5]; Secondly, Gestational hypertension, like pre-eclampsia, is also associate with cardiovascular disease in the long-term [6-9] New onset of systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg on at least 2 occasions 4 hours apart after 20 weeks of gestation in a previously normotensive individual And:

- No proteinuria
- No signs/symptoms of preeclampsia-related end-organ dysfunction (eg, thrombocytopenia, acute kidney injury, elevated liver transaminases, pulmonary edema, cerebral or visual symptoms

4.Pre-eclampsia

Pre-eclampsia is gestational hypertension accompanied by one or more of the following newonset conditions at or after 20 weeks' gestation: Proteinuria and/ or another maternal organ dysfunction New onset of systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg on at least 2 occasions at least 4 hours apart after 20 weeks of gestation in a previously normotensive individual. Patients with systolic blood pressure ≥160 mmHg and/or diastolic blood pressure ≥110 mmHg should have blood pressure confirmed within a short interval (minutes) to facilitate timely administration of antihypertensive therapy.

And:

- Proteinuria (≥300 mg per 24-hour urine collection [or this amount extrapolated from a timed collection], or protein:creatinine ratio ≥0.3, or urine dipstick reading ≥2+ [if other quantitative methods are not available]).

In a patient with new-onset hypertension without proteinuria, the diagnosis of preeclampsia can still be made

if any features of severe disease are present.

5. Preeclampsia with severe features

In a patient with preeclampsia, presence of any of the following findings are features of severe disease:

- Systolic blood pressure ≥160 mmHg and/or diastolic blood pressure ≥110 mmHg on 2 occasions at least 4 hours apart (unless antihypertensive therapy is initiated before this time)
- Thrombocytopenia (platelet count <100,000/microL)
- Impaired liver function as indicated by liver transaminase levels at least twice the normal concentration or severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by alternative diagnoses, or both
- Acute kidney injury (serum creatinine concentration >1.1 mg/dL [97 micromol/L] or doubling of the serum creatinine concentration in the absence of other kidney disease)
- Pulmonary edema
- Persistent cerebral or visual disturbances

6.Eclampsia

A generalized seizure in a patient with preeclampsia that cannot be attributed to other causes

7.Preeclampsia superimposed upon chronic hypertension

About 25% of women with chronic hypertension will develop superimposed preeclampsia. This diagnosis is made when a woman with chronic essential hypertension develops any of the above maternal organ dysfunction consistent with pre-eclampsia.

Any of these findings in a patient with chronic hypertension:

- A sudden increase in blood pressure that was previously well-controlled or an escalation of antihypertensive therapy to control blood pressure
- New onset of proteinuria or a sudden increase in proteinuria in a patient with known proteinuria before or early in pregnancy
- Significant new end-organ dysfunction consistent with preeclampsia after 20 weeks of gestation or postpartum.

8.Chronic hypertension with superimposed preeclampsia with severe features Any of these findings in a patient with chronic hypertension and superimposed preeclampsia: Systolic blood pressure ≥160 mmHg and/or diastolic blood pressure ≥110 mmHg despite escalation of antihypertensive therapy

- Thrombocytopenia (platelet count <100,000/microL)
- Impaired liver function as indicated by liver transaminase levels at least twice the normal concentration or severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by alternative diagnoses, or both
- New-onset or worsening renal insufficiency
- Pulmonary edema
- Persistent cerebral or visual disturbances .

9.HELLP

- Hemolysis, Elevated Liver enzymes, and Low Platelets. Hypertension may be present (HELLP in such cases is often considered a variant of preeclampsia).

PREDICTION OF PREECLAMPSIA- INVESTIGATIONS AND ROLE OF DOPPLER

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Introduction:

Hypertensive disorders remain among the most significant and intriguing unsolved problems in obstetrics. These disorders complicate 5 to 10 percent of all pregnancies and together they are one of the deadly triad- along with hemorrhage and infection- that contributes greatly to maternal morbidity and mortality rates.Of hypertensive disorders, the preeclampsia (PE) syndrome, either alone or superimposed on chronic hypertension, is the most dangerous.PE is associated with reduced blood supply to the placenta with consequent impairment in fetal growth, oxygenation and increased risk of stillbirth. Additionally, a high proportion of women with PE require premature delivery for maternal and / or fetal indications and therefore the babies are subjected to the additional risks arising from prematurity. The risk of adverse outcome for the mother and fetus / baby is much higher with preterm PE than with term PE.

Prediction:

Prediction of PE may help in stratifying women into high risk group so that surveillance can be intensified and prophylactic therapies can be initiated. Biochemical markers that have been proposed for prediction of PE were chosen on the basis of specific pathophysiological abnormalities that have been found in association of PE. These biochemical markers include markers of placental dysfunction, endothelial cell activation and dysfunction, coagulation activation, angiogenesis and markers of systemic inflammation. However none of these tests are sufficiently reliable for use as a screening test in clinical practice, combination of them are being evaluated for their predictive accuracy.A global assessment of risk should encompass four broad areas, including personal risk profile (including age, ethnicity, parity, smoking, medical and obstetric history and conception method), metabolic risk profile (including body mass index (BMI) and history of diabetes), cardiovascular risk profile (including existing cardiovascular conditions and measurement of mean arterial blood pressure) and placental risk profile (including uterine artery Doppler and maternal serum biomarkers).

- **Personal Risk Profile:**

The variables from maternal characteristics and medical and obstetrical history that increase the risk of PE, include advancing maternal age, increasing weight, South Asian racial origin, medical history of chronic hypertension, diabetes mellitus and systemic lupus erythematosus or antiphospholipid syndrome, conception by in vitro fertilization and family history or personal history of PE. The risk of PE in women in their first pregnancy is three times higher than in women with previous pregnancies that were not complicated by PE. Women who had PE in a first pregnancy are up to 10 times more likely to develop PE in a second pregnancy. The risk for PE is lower in tall than in short women and is decreased in parous women with no previous PE. The protective effect against PE of a previous pregnancy without PE decreases with the time interval between the previous and the current pregnancy so that after 15 years the risk of PE is about the same as that in nulliparous women.

- **Measurement of mean arterial pressure (MAP):**

MAP (systolic+2(diastolic)/3) >= 90 mmHg in second trimester is a predictor of PE. Blood pressure should be taken by validated automated devices, which are calibrated at regular intervals. Mercury sphygmomanometers should not be used because of concerns for both the clinical performance and safety of these instruments. The women should be in a sitting position and their legs should not be crossed. Crossing of the legs may raise the MAP. The arms of the patient should be supported at the level of her heart. If the upper arm is below the level of the right atrium the MAP is over-estimated and if the arm is above the level of the heart the MAP is under-estimated. If the arm is held up by the patient

without support the MAP is overestimated. A normal (22-32 cm) or large (33-42 cm) adult cuff should be used depending on the mid-arm circumference of the patient. If the cuff is too large the MAP is under-estimated and if the cuff is too small the MAP is over-estimated. After rest for five minutes, two measurements of MAP should be taken from each arm simultaneously and the average of the four should be considered in the assessment of risk.In isolation it has low sensitivity and low positive predictive value.

- **Maternal Urinary calcium:**

There are suggestions that hypocalciurea occurs early and persists throughout gestation in women with PE.May increase sensitivity of screening when used in combination.

- **Maternal Serum Biomarkers:**

1. Placental growth factor (PLGF): PlGF is synthesized by the placenta and has potent angiogenic functions. In pregnancies that develop PE serum PLGF is lower than in normal pregnancies and this decrease is thought to be the consequence of placental hypoxia.
2. Serum soluble fms-like tyrosine kinase-1 (sFLT-1): sFLT-1 is an anti-angiogenic factor that is thought to play a central role in the pathogenesis of PE.In pregnancies with established PE serum sFLT-1 is increased and this increase precedes the development of the disease by about five weeks. Measurement of sFLT-1 at 11-13 weeks does not improve the prediction of PE achieved by maternal factors alone. Measurement at 22 weeks is useful in the prediction of PE at <32 weeks.
3. Pregnancy associated plasma protein-A (PAPP-A): PAPP-A is produced by the placenta and is thought to play an important role in placental growth and development.In pregnancies that develop PE, compared to unaffected pregnancies, maternal serum PAPP-A is decreased during the first-trimester, not significantly different in the second-trimester and increased in the early third-trimester.

- **Screening for pre-eclampsia using ultrasound:**

The use of ultrasound as a tool for screening/prediction of PE is based on the fact that defective placentation results in incomplete transformation of the spiral arteries. Placental villous and vascular histopathological lesions are four-to-seven times more common in PE than in non-PE pregnancies and are associated with increased resistance to uterine artery blood flow. The pulsatility index (PI) should be used for examination of uterine artery resistance in the context of PE screening.

Doppler examination of the uterine arteries at 11 + 0 to 13 + 6 weeks can be performed either transabdominally or transvaginally. For the first-trimester transabdominal assessment of uterine artery resistance, a midsagittal section of the uterus and cervix is obtained initially. The pulsed-wave Doppler sampling gate should be narrow (set at approximately 2 mm) and positioned on either the ascending or descending branch of the uterine artery at the point closest to the internal cervical os, with an insonation angle < 30^0. In order to verify that the uterine artery is being examined, the peak systolic velocity should be > 60 cm/s. The PI is measured when at least three identical waveforms are obtained. Transvaginal assessment of uterine artery resistance follows the same principles. The woman is placed in the lithotomy position, with her bladder empty, and a transvaginal probe is used to obtain a sagittal view of the cervix. The probe is then moved laterally until the paracervical vascular plexus is seen, and the uterine artery is identified at the level of the internal cervical os. Measurements are taken with an angle of insonation< 30^0.

-The 95th centile for mean uterine artery PI obtained using a transabdominalapproachbetween 11 + 0 and 13 + 6 weeks is 2.35. Uterine artery resistance is higher on transvaginal compared with transabdominal measurement; the 95th centile for mean uterine artery PI obtained using a transvaginal approach is approximately 3.10 for crown–rump lengths (CRL) up to 65 mm, gradually declining with increased CRL thereafter. -----The 95th centile for mean uterine artery PI is 1.44 for the transabdominal approach and 1.58 for the transvaginalapproach at 23 weeks.

-Mean uterine artery PI should be used for prediction of PE. In case of a unilateral placenta, a unilaterally increased PI does not appear to increase the risk for PE if the mean PI is within normal limits.

The objective of screening for PE at 11-13 weeks' gestation is to identify the cases that would benefit from prophylactic use of aspirin that reduces the risk of preterm PE by more than 60%.Combined screening by maternal factors, MAP, UTPI and PLGF predicts about 90% of early PE (<34 weeks), 75% of preterm PE (<37 weeks) and 45% of term PE (≥37 weeks), at screen positive rate of 10% and should be preferred over Doppler-based screening only. Inclusion of PAPP-A and sFLT-1 does not improve the performance of screening significantly.

Management of PIH - Practical Approach

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A. Anti-hypertensive Agents

The existing guidelines for the management of hypertension in pregnancy recommend one of the four pharmacological agents- Methyldopa, Labetalol, slow-release Nifedipineand hydralazine in single or combinative manner. Second-line treatment options comprise clonidine (0.1 to 0.6 mg per day in divided doses), hydrochlorothiazide (12.5 to 25 mg per day orally), nicardipine (3 to 9 mg per hour intravenously), and sodium nitroprusside (0.24 to 5 µg/kg/min). Other potential alternatives include verapamil, diazoxide, prazosin, and oxprenolol.

Drug	Initial Dose	Maximum Suggested dose
Labetalol (Combined alpha and beta blocker)	100 mg twice daily- increase by 100 mg twice daily every 2 to 3 days as needed	2400 mg
Nifedipine ER (Calcium Channel Blocker)	30 to 60 mg once daily as extended-release tablet, increase at 7-to-14-day intervals	120 mg
Methyldopa (Centrally acting alpha agonist)	250 mg 2 to 3 times daily, increase every 2 days as needed	3000 mg
Hydralazine (Peripheral Vasodilator)	Begin with 10 mg 4 times a day, increase by 10-25 mg/ dose every 2 to 5 days	200 mg

Table 1: Drug doses for oral treatment of hypertension in pregnancy

B. Magnesium Sulfate

Eclampsia, characterized by seizures, frequently arises as a complication of preeclampsia (PE), and the administration of antihypertensive medications alone is insufficient to avert its onset. Consequently, magnesium sulfate is employed due to its neuroprotective properties. Although magnesium sulfate is not conventionally utilized as an antihypertensive agent, it is routinely administered to mitigate the risk of seizures in pregnant women exhibiting severe manifestations of PE. The American College of Obstetricians and Gynecologists (ACOG) advises that magnesium sulfate should be reserved for cases of PE with severe features. These features include a systolic blood pressure (SBP) of 160 mm Hg or greater, or a diastolic blood pressure (DBP) of 110 mm Hg or greater, measured on at least two occasions four hours apart, along with thrombocytopenia, impaired liver function tests (LFTs), pulmonary edema, new onset headaches unresponsive to treatment, and visual disturbances. Magnesium sulfate exerts its effects by interacting with acetylcholine receptors, N-methyl-D-aspartate (NMDA) receptors, and calcium channels within the central nervous system (CNS). Additionally, it plays a protective role for the blood-brain barrier and diminishes the formation of cerebral edema. Moreover, magnesium sulfate functions as a vasodilator in both cerebral and peripheral blood vessels, and it is generally regarded as more effective than other anticonvulsants such as phenytoin, diazepam, and nimodipine.

Current clinical guidelines recommend an initial loading dose of 4-6 grams of magnesium sulfate, administered through an infusion pump over a period of 20-30 minutes, followed by a maintenance dose of 1-2 grams per hour as a continuous intravenous infusion, which should be sustained for 24 hours post-delivery. Close monitoring is essential during magnesium sulfate administration due to the potential for toxicity and adverse effects. Hypermagnesemia, or magnesium toxicity, can result in areflexia (loss of reflexes, particularly the patellar deep tendon reflex) at blood magnesium levels of 8-10 mEq/L, and respiratory paralysis at levels exceeding 13 mEq/L. Significantly elevated magnesium levels may ultimately lead to cardiac arrest.

C. Delivery: Timing and Methods

The sole definitive treatment for preeclampsia (PE) is the termination of pregnancy, which can be achieved through either labor induction or cesarean delivery. Induction is typically recommended no earlier than 37 weeks of gestation. The American College of Obstetricians and Gynecologists (ACOG) Preeclampsia Guidelines suggest two management strategies for pregnant individuals diagnosed with PE, contingent upon the gestational age, specifically at 34 weeks and 0 days. If the gestational age is below 34 weeks, expectant management is advised. Nevertheless, in cases where severe features are present, while expectant management may benefit the fetus or newborn, it also poses certain risks to the mother. Factors that determine the appropriateness of delivery for a pregnant individual with PE are detailed in Table 2.

Maternal Factors	Fetal Factors
Severe hypertension, which is unresponsive to an antihypertensive agent	Abnormal antenatal testing
Complaints of persistent headache or persistent RUQ/epigastric pain, unresponsive to the treatment	Fetal demise
Complaints or findings of visual disturbance or altered sensorium or motor deficit	Fetal lethal anomaly or extreme prematurity
Diagnosis of Stroke or MI	Doppler: UA - REDF
Diagnosis of HELLP syndrome	-
Worsening RFTs (Serum Cr. > 1.1)	-
Pulmonary edema	-
Eclampsia	-
Placental abruption or bleeding in the absence of placenta previa	-

Table 2: Candidate for Delivery in Preeclampsia (HELLP: Hemolysis, Elevated Liver enzyme levels, and Low Platelet levels; RUQ: Right upper quadrant; MI: myocardial infarction; RFTS: renal function tests; UA - REDF: umbilical artery - reversed end-diastolic flow; CR.: creatinine)

The determination of the delivery method is a critical consideration following the decision to proceed with delivery. Among the various factors influencing the choice between labor induction and cesarean section, the cervical score plays a pivotal role. Research indicates that inducing labor in patients with suboptimal cervical scores is linked to a higher likelihood of failed induction, extended labor duration, and an increased incidence of cesarean delivery. Labor induction or augmentation is deemed advantageous when the patient presents with a clinically stable condition, a favorable cervical status, and normal fetal growth. For patients experiencing severe preeclampsia at or before 34 weeks of gestation, labor induction is a viable option, as it enhances the likelihood of achieving a successful vaginal delivery. The Bishop score assessed at the time of admission is recognized as the most reliable predictor of successful delivery outcomes. Notably, the chances of successful induction improve as gestational age advances . Substantial evidence indicates that scheduling deliveries can mitigate the risk of maternal complications and severe hypertension compared to a more conservative management approach. Although planned deliveries may lead to an increase in neonatal unit admissions due to preterm births, there is no conclusive evidence indicating a heightened incidence of neonatal health complications in these scenarios.

CONCLUSIONS

In summary, preeclampsia (PE) represents a multifaceted hypertensive disorder associated with pregnancy, marked by irregularities in placental and systemic vascular function. Current guidelines for antihypertensive treatment identify methyldopa as the preferred medication, while the only definitive treatment option remains the termination of pregnancy. Existing protocols face significant challenges, particularly the absence of reliable predictive biomarkers that could indicate the risk of PE prior to the manifestation of clinical symptoms. The management of PE stands at a pivotal juncture, necessitating innovative approaches and dedicated research efforts aimed at developing advanced biomarkers, targeted pharmacological therapies, and sophisticated diagnostic technologies for timely detection, alongside effective treatment strategies to enhance outcomes for both mothers and their infants. A multidisciplinary strategy that includes preconception counseling for women at high risk, psychoeducational support for expectant mothers, and comprehensive training for healthcare professionals is crucial in mitigating maternal mortality and improving overall outcomes.

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