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HIGH RISK CASES IN OBSTETRICS

Shruti Bangale Daflapurkar



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Reena J Wani

JAYPEE

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CHAPTER

3 Pregnancy Induced Hypertension

Shruti Bangale Daflapurkar

ABSTRACT

Pre-eclampsia/eclampsia is an unpredictable, multiorgan disorder unique to human pregnancy. It is associated with significant maternal and fetal morbidity and mortality worldwide. Epidemiological studies, report that hypertension complicates 10% of all pregnancies. Treatment of this disorder is still remains a challenge to even the most experienced obstetricians, mainly because exact etiology is unknown. However, evidence accumulated in the past 20 years indicates that abnormal placentation is one of the initial events in this disease. The main causes of perinatal mortality and morbidity are due to placental insufficiency and complications of premature delivery. The majority of hypertensive women can be managed as outpatient with no intervention apart from monitoring of the mother and the fetus. However, around 25% of women are found to have persistent hypertension and are admitted to hospital for further management. Many of the severely pre-eclamptic patients will present acutely with no prodromal signs. It is important that these women are stabilized prior to any other management decision. Hence, it is important to have management protocols available in the labor ward of all hospitals aimed at the main sequelae of hypertension in pregnancy. Delay in or inadequate treatment of these women or a rushed delivery in an acute hypertensive pregnant woman can contribute to maternal mortality and morbidity. The primary aim should be to lower blood pressure. Early identification of women at risk for pregnancy-induced hypertension may help prevent some complications of the disease. Education about the warning symptoms is also important because early recognition may help women receive treatment and prevent worsening of the disease.

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Introduction

Pre-eclampsia/eclampsia is an unpredictable, multiorgan disorder unique to human pregnancy. It is associated with significant maternal and fetal morbidity and mortality worldwide. Treatment of this disorder is still remains a challenge to even the most experienced obstetricians, mainly because exact etiology is unknown. Many of the causal theories attributed

to pre-eclampsia describe pathological features of clinical presentation, which are the result rather than the cause of the disease process.[1] It has been thought to be neurological, renal, hepatic, hypertensive and most recently placental disorder. The truth is that it is probably the cause may be different in different people and is certainly more than just hypertension in pregnancy.[2]

Epidemiological studies, report that hypertension complicates 10% of all pregnancies. In USA 7–10% of all pregnancies are complicated by pregnancy-induced hypertension (PIH), while in South Africa, PIH is found in 18% of all pregnancies and eclampsia in 0.9% of cases.[3] In India, incidence of PIH among hospital patient is about 7–10% of all antenatal admissions and that of eclampsia is 0.94–1.8%.[4] Further hypertensive disorders of pregnancy are major factors in perinatal morbidity and mortality. Once the diagnosis of severe pre-eclampsia is established, the only cure is termination of pregnancy. Management is directed towards an attempt to achieve fetal maturity while preventing maternal complications. The main risks to the fetus are IUGR and iatrogenic interference resulting in preterm delivery.

Unfortunately, there is no one test that can be relied upon to confirm the diagnosis, but by accurately measuring the blood pressure, quantifying the proteinuria and serially monitoring the hematological and biochemical parameters appropriate evaluation is possible. Suitable early intervention is often required to prevent life-threatening complications.

History

References of PIH have been found from ancient times. The Greek prognosis XXXI says, "In pregnancy, the onset of drowsiness with headache and heaviness is bad, such cases are liable to some sort of fits at some time". Indian Atharvaveda and Sushruta both mention eclampsia. In 460–370 BC, Hippocrates mentioned convulsions of pregnancy, that they occurred mostly in woman who had headache and a tendency to sleep (coma). In 1886, when Sir Cliford Albuted discovered sphymomanometer in Great Britain, it came to be recognized as being associated with raised blood pressure. In 1916, Zweifel first called toxemia "the disease of theories".

In 1958, Browne stated that there are developmental resemblance between normal pregnancy and Cushing's syndrome. This is usually due to hyperplasia of adrenal cortex and is in 90% cases associated with raised blood pressure (BP). Every pregnant woman would become hypertensive if it were not for the protective placenta that furnishes an O_2-sensitive oxidase that inactivates the pressor hormones from adrenal cortex.

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If the placenta is made ischemic and its O_2 tension lowered, the protective enzyme is made ineffective and hypertension results.

Richard Worley, 1984 said pre-eclampsia remains a disease of theories. At least two or three major concepts however have substantial investigative support. Pre-eclamptic women have accentuated vascular sensitivity to vasoactive agents and exhibit decreased prostanoids especially PGI_2 derivatives while evidence of immune mechanism of PIH is less substantial, it seems probably that immune factors are involved.

❍ Definition, Classification and Pathophysiology

The diagnosis of hypertensive disorders complicating pregnancy is outlined by the working group (2000).[5] According to this, there are five types of hypertensive diseases.

That include:
1. Gestational hypertension
2. Pre-eclampsia
3. Eclampsia
4. Pre-eclampsia superimposed on chronic hypertension
5. Chronic hypertension.

Gestational Hypertension

- BP ≥ 140/90 mm Hg for the first time during pregnancy
- No proteinuria
- BP return to normal <12 weeks postpartum
- Final diagnosis made only postpartum
- May have other signs of pre-eclampsia, for example, epigastric discomfort or thrombocytopenia.

Pre-eclampsia

Minimum Criteria

- BP ≥140/90 mm Hg after 20 weeks of gestation
- Proteinuria ≥ 300 mg/24 hours or ≥+1 on dipstick.

Increased Certainty of Pre-eclampsia

- BP ≥160/110 mm Hg
- Proteinuria 2.0 g/24 hours or ≥2+ on dipstick
- Serum creatinine >1.2 mg/dL unless known to be previously elevated
- Platelets <1,00,000/mm^3

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- Microangiopathic hemolysis (increased LDH)
- Elevated ALT or AST
- Persistent headache or other cerebral or visual disturbance
- Persistent epigastric pain.

Eclampsia

Seizures, which cannot be attributed to other causes in a woman with pre-eclampsia.

Superimposed Pre-eclampsia (On Chronic Hypertension)

- New-onset proteinuria ≥ 300 mg/24 hours in hypertensive women but no proteinuria before 20 weeks of gestation
- A sudden increase in proteinuria or blood pressure or platelet count < 1, 00,000/mm^3 in women with hypertension and proteinuria before 20 weeks of gestation.

Chronic Hypertension

Blood pressure ≥140/90 mm Hg before pregnancy or diagnosed before 20 weeks of gestation.

Or

Hypertension first diagnosed after 20 weeks of gestation and persistent after 12 weeks postpartum.

Definition: (American College of Obstetricians and Gynecologists)[6]

Hypertension associated with proteinuria, greater than 0.3 g/L in 24 hours urine collection or greater than 1 g/L in a random sample, generalized edema, greater than 1$^+$ pitting edema after 12 hours of rest in bed or a weight gain of 5 lb or more in 1 week or both after 20 weeks of gestation.

According to American College of Obstetricians and Gynecologists (ACOG), the diagnosis of hypertension in pregnancy is made by any one of the following criteria:

1. A rise of 30 mm Hg or more in systolic blood pressure
2. A rise of 15 mm Hg or more in diastolic blood pressure
3. A systolic blood pressure of 140 mm Hg or more
4. A diastolic blood pressure of 90 mm Hg or more.

These alterations in blood pressure should be observed on at least two different occasions at least 6 hours apart.

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International Society for the Study of Hypertension in Pregnancy (ISSHP)

The diagnosis of hypertension in pregnancy according to ISSHP is made by either of the following criteria.[7]

1. One measurement of diastolic blood pressure equal to or greater than 110 mm Hg.
2. Two consecutive measurement of diastolic blood pressure equal to or greater than 90 mm Hg, 4 or more hours apart.

According to the ISSHP, Korotkoff's IV sound, the point at which the sound becomes muffled, should be used when measuring the diastolic blood pressure.

It also recommends obtaining blood pressure measurements with the patient in the lateral decubitus position with the cuff placed on her right arm at the level of the heart.

The ISSHP has proposed the following classification for hypertension disorders in pregnancy.

Gestational Hypertension and/or Proteinuria

Hypertension and/or proteinuria developing during pregnancy, labor or the puerperium in a previously normotensive nonproteinuric woman. This group of patients is subdivided into:

1. Gestational hypertension (without proteinuria)
   a. Developing antenatally
   b. Developing for the first time in labor
   c. Developing for the first time in the puerperium.
2. Gestational proteinuria (without hypertension)
   a. Developing antenatally
   b. Developing for the first time in labor
   c. Developing for the first time in the puerperium.
3. Gestational proteinuric hypertension (pre-eclampsia)
   a. Developing antenatally
   b. Developing for the first time in labor
   c. Developing for the first time in the puerperium.

Chronic Hypertension and Chronic Renal Disease

Hypertension and/or proteinuria in pregnancy in a woman with chronic hypertension or chronic renal disease diagnosed before, during or after pregnancy. This group is subdivided into:

1. Chronic hypertension (without proteinuria)
2. Chronic renal disease (proteinuria with or without hypertension)

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3. Chronic hypertension with superimposed pre-eclampsia. Proteinuria developing for the first time during pregnancy in a woman with known chronic hypertension.

Unclassified Hypertension and/or Proteinuria

Hypertension and/or proteinuria found either:

1. At first examination after twentieth week of pregnancy (140 days) in a woman with known chronic hypertension or chronic renal disease.
2. During pregnancy, labor or the puerperium in a case in which information is insufficient to permit classification.
   i. Unclassified hypertension (without proteinuria)
   ii. Unclassified proteinuria (without hypertension)
   iii. Unclassified proteinuria hypertension.

The definition of gestational proteinuria is more than 300 mg in 24-hour urine sample. However, in practice proteinuria is detected by the use of reagent strips. A diluted (<1010 specific gravity) or concentrated (>1030 specific gravity) urine or an alkaline specimen (pH > 8.0) may produce false results when tested with the reagent strips. Also, the frequency of false positive may be as high as 25% in trace reactions and as high as 6% when the result it 1 +. If one considers a value of 2 + as the lower limit of significance, false positive results will be eliminated.

The ISSHP does not consider degrees of severity for hypertension in pregnancy. However, it has been suggested that severe hypertension occurs when.

1. A diastolic blood pressure equal to or greater than 120 mm Hg is present on any one occasion.
2. A diastolic blood pressure equal to or greater than 100 mm Hg is present on two or more consecutive occasions 4 hours apart.

Severity of Pre-eclampsia

Importantly the differentiation between mild and severe pre-eclampsia can be misleading because apparently mild disease may progress rapidly to severe disease (Table 3.1).

Table 3.1: Comparison between mild and severe pre-eclampsia

Abnormality	Mild	Severe
1. Diastolic blood pressure	< 100 mm Hg	110 mm Hg or higher
2. Proteinuria	Trace to 1+	Persistent 2+ or more
3. Headache	Absent	Present
4. Visual disturbances	Absent	Present

Contd...

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Contd...

Abnormality	Mild	Severe
5. Upper abdominal pain	Absent	Present
6. Oliguria	Absent	Present
7. Convulsion	Absent	Present
8. Serum creatinine	Normal	Elevated
9. Thrombocytopenia	Absent	Present
10. Liver enzyme elevation	Minimal	Marked
11. Fetal growth retardation	Absent	Obvious
12. Pulmonary edema	Absent	Present

Risk Factors

There is an increased association of pre-eclampsia in the following categories of patients.[8]

- Elderly and young primigravida (Fig. 3.1)
- Poor and under privileged
- Pregnancy complicated by hydatidiform mole, multiple gestation, hydramnios, etc.
- Medical disorders
- Hereditary.

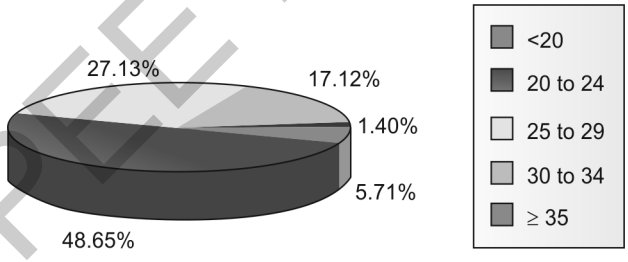

Figure 3.1: Incidence of PIH according to age

Pathological Changes in Pre-eclampsia

Pathogenesis

The exact nature of primary event causing pre-eclampsia is not known. However, evidence accumulated in the past 20 years indicates that abnormal placentation is one of the initial events in this disease. The main feature of abnormal placentation is inadequate trophoblastic invasion of the maternal spiral arterioles. In normal pregnancy, the wall of the

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spiral arteries is invaded by trophoblastic cells and transformed into large, tortuous channels that carry a large amount of blood to intervillous space and are resistant to effects of vasomotor agents. These physiologic changes are restricted in patients with pre-eclampsia,[9] with resulting decreased uteroplacental perfusion. The anatomic and physiologic disruption of normal placentation is thought to lead to altered endothelial cell function,[10] multiple organ damage by a mechanism that is unknown at present time.[11]

Pathological Changes in Pre-eclampsia

Pathological deterioration of function in a number of organs and systems, presumably as a consequence of vasospasm and ischemia, has been identified in severe pre-eclampsia and eclampsia.

1. *Cardiovascular changes:*
   - Decreased cardiac output[12]
   - Increased peripheral resistance
   - Hyperdynamic left ventricular function
   - Increased pulmonary wedge pressure
   - Hemoconcentration is the main feature.
2. *Hematological changes:*
   - Thrombocytopenia
   - Increased fibrin degeneration products
   - Decreased plasma fibrinogen, antithrombin III
   - Fragmentation hemolysis.
3. *Endocrine and metabolic changes:*
   - Inadequate rise in levels of renin, angiotensin II, aldosterone
   - Increased levels of deoxycorticosterone, which is a potent mineralocorticoid. This may explain Na^+ retention[13] in such patients despite decreased renin levels.
4. *Renal changes:*
   - Renal perfusion and glomerular filtration are significantly reduced due to intrinsic renal changes caused by severe vasospasm
   - This results in elevated serum uric acid[14] and creatinine levels.
5. *Hepatic changes:* Liver involvement in PIH is serious and is frequently accompanied by evidence of other organ involvement, especially kidney and brain along with hemolysis and thrombocytopenia, commonly referred to as HELLP syndrome.[15] It is associated with increased incidence of complications like stroke, cardiac arrest, disseminated intravascular coagulation (DIC), acute respiratory distress syndrome (ARDS), acute renal failure (ARF), hepatic failure, etc. It can complicate[16] up to 20% pregnancies with severe PIH.

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6. *Changes in brain:* Eclampsia and visual disturbances are the commonest manifestations.
7. *Uteroplacental perfusion:* Decreased placental perfusion from vasospasm is almost certainly a major factor for fetal and maternal mortality and morbidity.

Signs and Symptoms

Symptoms (Fig. 3.2)

- *Headache:* Usually present in moderate to severe form of pre-eclampsia. Pain is frontal, occipital and may occur with visual symptoms. It becomes intense before the onset of convulsions.
- *Edema:* Though it is no longer considered the only symptom of pre-eclampsia but it is frequently found to be associated with it.
- *Epigastric or right upper quadrant pain:* It is common in patients with severe pre-eclampsia. Often associated with raised serum glutamic oxaloacetic transaminase (SGOT), serum glutamic-pyruvic transaminase (SGPT) and lactate dehydrogenase (LDH).
- *Blurring of vision:* The most common visual symptom is scotoma (transient perception of bright or black spot in visual field). In severe cases, complete blindness can occur. On fundoscopy, vasospasm of retinal blood vessels is seen without any other structural defect. This suggests that the reason of visual defect is at level of occipital cortex. This cortical blindness quickly recovers after delivery.

Signs

- *Increased BP:* It is most important sign.
  – Common errors:
  a. Erroneously high BP reading may be observed with obese patients when regular sized cuff is used.

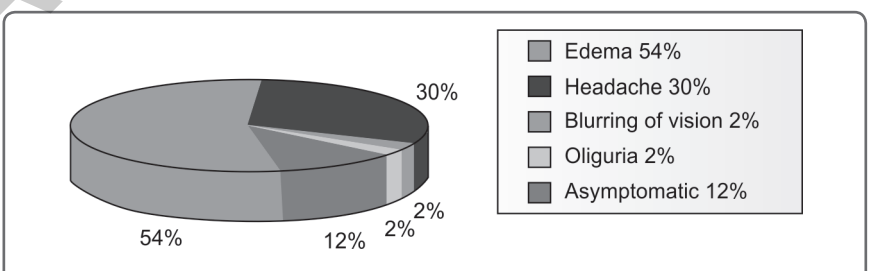

Figure 3.2: Symptoms in PIH

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b. Different maternal positions while taking repeated measurements. (BP should always be taken in sitting posture).
c. Different end points to measure diastolic pressure. Korotkoff IV sound, i.e. point of muffling is best marker of diastolic pressure.

- *Proteinuria:* It most often appears with hypertension. It is of nonselective type.
  Proteinuria ≥ 300 mg/24 hours or ≥+1 dipstick is one of the important criteria to diagnose PIH.
- *Excessive weight gain:* However, it is no longer considered the only symptom of pre-eclampsia but it is frequently found to be associated with it.
- *Brisk deep tendon reflexes:* It results from central nervous system irritability resulting from severe arterial vasospasm causing rupture of vascular endothelium and pericapillary hemorrhage.
- Fundoscopy shows following changes:
  Grades 0—Normal.
  Grade 1—Angiospasm, mild arteriovenous narrowing.
  Grade 2—Moderate narrowing.
  Grade 3—Angiospasm and edema, hemorrhage, exudates, "pre-eclamptic retinopathy".
  Grade 4—Papilledema, retinal detachment.

Laboratory Investigations

1. *Altered renal function:*
   - Serum creatinine: Upper limit of normal during pregnancy is 0.8 mg/dL. Raised levels are due to involvement of glomerulus, which is affected last; it is an indicator of renal shutdown.
   - Blood urea nitrogen (BUN): Upper limit of normal during pregnancy is 15 mg/dL.
   - Serum uric acid: Upper limit of normal during pregnancy is 7 mg/dL. Raised levels are due to abnormality in distal convoluted tubule which is affected first in PIH so it is an important marker to detect early PIH.
2. *Changes in liver function:*
   - Increased SGPT, SGOT, LDH
   - After delivery, SGOT and SGPT levels rapidly decrease and reach normal levels by fifth postpartum day
   - LDH falls more slowly and normal values are reached by postpartum day 8/10.

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3. *Hematologic abnormalities:*
   - Elevation of hemoglobin and hematocrit caused by characteristic decrease in plasma volume
   - Thrombocytopenia[21]
   - Platelet count below 1,50,000. Below 50,000 is critical value.
   - Plasma fibrinogen—below 200 mg/dL.

Management

The patients who require urgent termination of pregnancy:

Maternal Causes

- Eclampsia
- DIC
- HELLP syndrome
- ARF
- Abruptio placenta
- Pulmonary edema.

Fetal Causes

- Abnormal color Doppler findings
- Poor BPP
- Non-reactive NST/IPM
- Intrauterine fetal death (IUFD).

Management

- Detailed history of symptoms of current illness, their onset, duration and progress, past medical history, obstetric history is taken
- General, systemic and obstetric examination is done
- IV line put in, blood collection is done for blood grouping and cross-matching along with emergency investigations like Hb, PCV, BUN, electrolytes and DIC profile
- NST/IPM done for fetal wellbeing
- USG with BPP done
- Ophthalmology reference is done for fundoscopy
- If patient is in active labor then low artificial rupture of membranes (ARM) done and labor is augmented with oxytocin
- If patient is not in labor then irrespective of fetal lung maturity, emergency LSCS is done usually in fetal indications and abruption placentae and induction of labor is done with Cerviprime (Prostaglandin) gel for the other maternal indications

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- If induction of labor fails after two successive instillations of prostaglandin gel vaginally then patient is taken up for emergency LSCS
- Maternal complications like eclampsia is managed with general care, $MgSO_4$ by Pritchard's regime, antihypertensive agents, adequate plasma substitutes for the management of DIC and HELLP syndrome, blood replacement for anemia.

Management of Eclampsia

- Patient is kept nil by mouth
- TPR, BP, FHS checked every ½ hourly
- Propped up position is given to the patient
- Patient is shifted to silent and dark room
- Nasal oxygen is given at 3 L/min
- After convulsions, oropharyngeal secretions suction to be done
- Mouth gag inserted to avoid tongue fall
- Injection $MgSO_4$ is given by Pritchard's regime. 4 g diluted in 10 CC saline IV with 5 g IM on each buttock is given stat
- Capsule Nifedipine (10 mg) is given stat
- Tablet Alpha Methyldopa (500 mg) is given stat
- Broad spectrum antibiotic cover is given IV
- Injection vitamin K (10 mg) IM given OD for 3 days
- Injection Eptoin (200 mg) IM is given if there are repeated seizures.
- Blood products, i.e. fresh frozen plasma, platelets, cryo, whole blood are given as per the blood investigation reports
- Patient is hydrated well with IV fluids round the clock
- Watch for knee jerks, respiratory rate and urine output
- Watch for progress of labor.

Patients who could be Managed Conservatively

- These patients are shifted to antenatal care ward and kept under closed monitoring
- At any time if there is worsening of maternal or fetal condition then pregnancy is terminated
- In the ward, patient is monitored for:
  i. BP (6 hourly)
  ii. Proteinuria (12 hourly)
  iii. Daily weight gain
  iv. Strict input/output charting
  v. Daily fetal kick count
  vi. Fundal height in cm (weekly)

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Shruti Bangale Daflapurkar DGO DNB FCPS MNAMS FICMCH MICOG is currently working as Consultant Gynecologist and Infertility Specialist at Aster Medical Center, Dubai, United Arab Emirates. She has been in clinical practice for last 5 years. She was always good in academics and secured FIRST merit rank in Maharashtra State Merit List in HSC in 1996. She was awarded Gold Medal by Shankarrao Chavan Pratishthan for securing highest marks in HSC in 1996. She also received Manjara Shetkari Sahakari Sakhar Karkhana first prize by Mr Vilasrao Deshmukh for securing highest marks in HSC in July 1996. She was awarded National Talent Scholarship by Maharashtra state for a period of 1996–2001 and Dhirubhai Ambani scholarship by Dhirubhai Ambani Foundation for a period of 1996–2001. She completed her graduation from prestigious Seth GS Medical College and KEM Hospital, and completed postgraduation from prestigious TN Medical College and BYL Nair Charitable Hospital. She secured highest marks in Mumbai University DGO examination in January 2006. She was awarded Dr Dina Patel prize with Gold Medal for having passed the FCPS examination with highest number of marks in March 2006. She got prestigious Fellowship in Infertility and ART at Asan Medical Center, Seoul, South Korea, in July 2011.

Dr Shruti has presented 9 papers in national and international conferences, published 7 papers in index journal, contributed chapters in textbook as Author and Co-author. She has written many articles in newspapers and magazines. Her special interest is Infertility Management and ART. She wishes to achieve much more in this field.

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