COMPARATIVE STUDY OF MATERNAL AND FETAL OUTCOME IN SEVERE PIH 30-34 WEEKS - ACTIVE VS EXPECTANT MANAGEMENT

Dissertation submitted to

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

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In partial fulfillment of the regulations

for the award of

M.S DEGREE IN OBSTETRICS AND GYNAECOLOGY

GOVT. MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM

SEPTEMBER 2016

CERTIFICATE OF HOD

This is to certify that the dissertation titled "**COMPARATIVE STUDY OF MATERNAL AND FETAL OUTCOME IN SEVERE PIH 30-34 WEEKS - ACTIVE VS EXPECTANT MANAGEMENT"** is a bonafide work Done by **Dr.A.G.SUBHATHRA** in GOVT. MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM in partial fulfillment of the university rules and regulations for award of MS degree in Obstetrics and Gynaecology under my guidance and supervision during the academic year 2015 – 2016.

Dr B.JEYAMANI, M.D.,DGO
Professor & HOD,
Dept. of Obstetrics & Gynaecology
G.M.K.M. Kumaramangalam
Medical College Hospital,
SALEM-636 001.

Place: SALEM

Date: 07.10.16

Dr.B.Jeyamani, MD, DGO
Professor & HOD
Department of OG,
GMKMCH. Salem.

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Dr B.JEYAMANI, M.D., DGO.,
Professor & HOD,
Dept. of Obstetrics & Gynaecology,
Govt. Mohan Kumaramangalam
Medical College Hospital,
SALEM-636 001.

Dr.B.Jeyamani, MD, DGO
Guide
Professor & HOD
Department of OG,
GMKMCH. Salem.

Place: SALEM

Date: 07.10.16

CERTIFICATE BY THE DEAN

This is to Certify that, **Dr.A.G.SUBHATHRA**, Post Graduate student in the Department of Obstetrics and Gynaecology, Government Mohan Kumaramangalam Medical College, Salem has done the Dissertation,

"COMPARATIVE STUDY OF MATERNAL AND FETAL OUTCOME IN SEVERE PIH 30-34 WEEKS - ACTIVE VS EXPECTANT MANAGEMENT" With partial fulfillment of regulations laid down by the Tamilnadu DR.M.G.R.Medical University, Chennai for M.S., (Obstetrics and Gynaecology) Degree Examination to be held during April 2017.

Prof. DR.P.KANAGARAJ. MD

Dean,

Government Mohan Kumaramangalam

Medical college, Salem.

Date: 7/10/16

Place : Salem

DEAN
Govt. Mohan Kumaramangalam
Medical College Hospital,
Salem - 636 001.

DECLARATION

I solemnly declare that this dissertation titled "**COMPARATIVE STUDY OF MATERNAL AND FETAL OUTCOME IN SEVERE PIH 30-34 WEEKS - ACTIVE VS EXPECTANT MANAGEMENT** was done by me at GOVT. MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM during the year 2014 - 2017 under the guidance and supervision of **Dr.B.JEYAMANI, MD, DGO**. This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.S. Degree in Obstetrics and Gynaecology (Branch -II).

Signature of the candidate

Dr.A.G.SUBHATHRA

M.D OG Post Graduate

GMKMCH , Salem.

Place: Salem

Date: 7/10/16

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I thank all my professors, Assistant Professors and my colleagues for their support. Above all, I am thankful to all the patients in this study.

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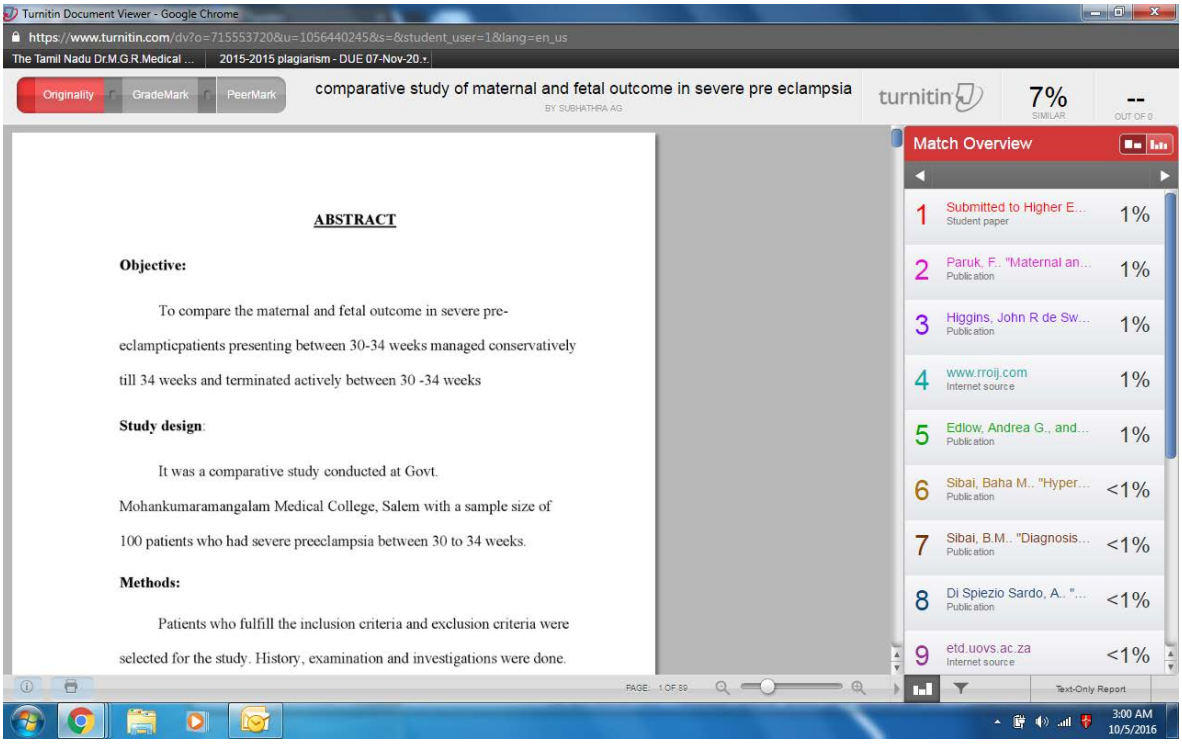
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ABSTRACT
Objective:
To compare the maternal and fetal outcome in severe pre-eclampticpatients presenting between 30-34 weeks managed conservatively till 34 weeks and terminated actively between 30 -34 weeks
Study design:
It was a comparative study conducted at Govt. Mohankumaramangalam Medical College, Salem with a sample size of 100 patients who had severe preeclampsia between 30 to 34 weeks.
Methods:
Patients who fulfill the inclusion criteria and exclusion criteria were selected for the study. History, examination and investigations were done.
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ABSTRACT

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Methods:

Patients who fulfill the inclusion criteria and exclusion criteria were selected for the study. History, examination and investigations were done. Details of the plan of treatment and the indication and mode of termination are noted. Maternal and fetal follow up done upto discharge / death.

Results:

- Mostly are in ages between 21-30 years
- Mostly primi (62%)
- Mean gestational age = 31 years
- 35% women had past history of preeclampsia
- 16% had family history
- 32 women had maternal morbidity
- There was no maternal death in our study
- Most women in active management delivered by vaginal delivery
- Termination of pregnancy in expectant management was due to maternal indication in 58% and fetal in 42%
- 29% perinatal death was recorded in our study
- Mean birthweight was 1.8 kg in expectant group and 1.6 kg in active group

Conclusion:

Severe preeclampsia is associated with significant maternal and fetal complications. Decision regarding pregnancy termination is to be taken on the grounds of both maternal and fetal factor. The expectant management of severe preclampsia results in a good fetal outcome for

1. Higher birth weight

2. Lower perinatal mortality

3. Lesser neonatal complications

But this must be weighed against the risk of maternal morbidity. Hence they should be carried out only in tertiary care centres

INTRODUCTION

Preeclampsia is a multisystem disorder involving placenta, blood, cardiovascular, liver, kidney and neurovascular system, occurring exclusively during pregnancy etiology of which is unknown. Approximately 5-7% of pregnancies are affected. It is an important cause of morbidity and mortality in both the mother and fetus.

Preeclampsia is described as a rise in blood pressure and proteinuria which is of new onset, occurring after 20^{th} week of gestation. It is described as severe preeclampsia if there is end organ damage and substantial increase of blood pressure and proteinuria or the occurrence of symptoms. Preeclampsia is considered early onset if elevation of blood pressure and proteinuria occur before 34^{th} week of pregnancy. Hypertension and its complications is ranked third as a leading cause of maternal mortality, causing over 17% of maternal deaths. Women still die from pre – eclampsia and eclampsia even in developed nations.

There is increased risk of abruptio placenta, acute renal failure, cardiovascular and cerebrovascular complications, DIC and even death. So, early diagnosis and close monitoring in preeclampsia is important for preventing its complications.

In early onset severe preeclampsia, there is progressive deterioration in the condition of the mother and also high morality in the fetus in perinatal period. Delivery of the fetus is considered to be the only way for reverting all these complications. Hence termination of pregnancy is needed if fetal distress occurs in case of multi organ dysfunction or if the gestation age reaches 34 weeks. But, early termination causes high perinatal morbidity and mortality in fetus due to prematurity. But expectant management to prolong pregnancy can be harmful to the mother. Hence, potential benefits of the fetus should be weighed against the potential dangers that may occur to the mother.

AIM OF THE STUDY

AIM:

To compare the maternal and fetal outcome in severe PIH patients presenting between 30-34 weeks managed conservatively till 34 weeks and terminated actively between 30 -34 weeks

OBJECTIVES

Primary objectives: to compare the maternal outcomes like abruptioplacenta, HELLP syndrome, eclampsia, PRES, Acute kidney injury, pulmonary edema in active and expectant group.

To compare the fetaloutcomes like Still birth, perinatal death, birth weight, apgar scores, admission to New born rate.

Secondary objectives include reason for termination ,duration of prolongation of pregnancy in expectant group, comparison of mode of delivery in both the groups.

REVIEW OF LITERATURE

HISTORICAL ASPECTS:

- Eclamptic convulsions dates back to 4000 years which was recognized in Indian, ancient Chinese, and Greek literature.
- Around 400 BC, Hippocrates stated that headache with convulsions during Pregnancy was to be considered bad.
- Bossier de saurages (1739)coined the term“Eclampsia”
- John Lever(1843) reported that Proteinuria was specific to the Pre-eclampsia condition.
- Eclamptic hypertension was discovered by Vasquez and Nobcourt (1897).
- Chesley (1984) said that Sensory stimuli were decreased by keepingpatients is quiet dark room.
- Horn (1906) was the first to use magnesiumsulphate on pre-eclampsia and Eclampsia.
- Several studies demonstrate that magnesium sulphate is superior over other anticonvulsants.
- In 1967, Robertson and Brosens described structural changes ofthe utero placental unit in pre-eclampsia.

- Sibai and Barten (2007) management of severe preeclampsia guidelines

MAGNITUDE:

50% of women with severe pre-eclampsia present before 34 weeks ofgestation. Delivery is the only way to relieve symptoms. It may be for maternal or fetal reasons.

CLASSIFICATION:

National High Pressure Education program classification

• Gestational Hypertension

• Pre eclampsia

• Eclampsia

• Superimposed Pre eclampsia

• Chronic Hypertension

DEFINITIONS

PRE-ECLAMPSIA

Pre-eclampsia is defined as rise in systolic blood pressure of 140mmHg or higher or diastolic blood pressure of 90 mmHg or higher at 2 different occasions atleast 6 hours apart thatdevelops forthe first time in pregnancy after 20 weeks of gestation accompanied by proteinuria.

Proteinuria is defined as urinary protein of 300mg or more per 24hours or persistent 30 mg/dL in a random sample.

SEVERE PRE-ECLAMPSIA:

Severe pre-eclampsia is defined as hypertension (diastolic blood

Pressure110 mm Hg or higher) with proteinuria (5gm or higher in 24 hours urinesample or more than 3+ on a dipstick).

EARLY ONSET SEVERE PRE- ECLAMPSIA:

When severe pre- eclampsia develops prior to 34 weeks of

Gestation[25] .

It complicates in 5-8% of pregnancy and is a major cause for maternal and perinatal morbidity and mortality[1,2].

ACOG CRITERIA FOR SEVERE PREECLAMPSIA

Preeclampsia is a clinical diagnosis encompassing three elements (committee on obstetric hypertension in pregnancy ACOG 1996)

1) **New onset hypertension (defined as a blood pressure consistently more than 140/90 mm of Hg in previously normotensive women) - according to the latest American college of obstetricians and gynecology bulletin**
2) **New onset proteinuria defined as more than 300mg /24hrs or > 2+ on a clean catch dipstick in the absence of urinary infection**
3) **New onset significant independent edema.**
4) **The diagnosis of PREECLAMPSIA should be made only after 20 weeks of gestation.**

Conventional mercury sphygmomanometry is still the gold standard device for blood pressure measurement. Blood pressure should be measured with the mother seated or reclined at 45º, with her feet on the ground or well supported, and her arm at the level of heart. The right arm should be used with the cuff of the appropriate size. Electronic blood pressure monitors may underestimate the true pressure.

POSITIONING OF PATIENT FOR BP RECORDING

Nowadays it has been recommended that korotkoff phase V be used as a measure of diastolic pressure[3]

1. K4/K5 difference is smaller in hypertensive than in normotensive pregnant women.

2. K5 which is close to the actual intra-arterial pressure, physiologically more accurate, is more reliably detected and is reproducible.
3. K4 has limited reproducibility [4]

Abnormality	Nonsevere[b]	Severe
Diastolic BP	< 110 mm Hg	≥ 110 mm Hg
Systolic BP	< 160 mm Hg	≥ 160 mm Hg
Proteinuria[c]	None to positive	None to positive
Headache	Absent	Present
Visual disturbances	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present
Convulsion (eclampsia)	Absent	Present
Serum creatinine	Normal	Elevated
Thrombocytopenia (< 100,000/μL)	Absent	Present
Serum transaminase elevation	Minimal	Marked
Fetal-growth restriction	Absent	Obvious
Pulmonary edema	Absent	Present

Pre eclampsiais classified as either “mild” or “severe”.

No classification of moderate Preeclampsia exists.

SEVERE PREECLAMPSIA (ACOG)

A diagnosis of severe Preeclampsia should be considered in women with new onset proteinuric hypertension and one or more of the following complications.

I. Blood pressure ≥ 160/110 mmHg on 2 different occasions at least 6 hours apart.

II. Proteinuria (5g or more in 24hr urine)

III. Oliguria <500ml in 24hrs

IV. Cerebral and visual symptoms like blurred vision, altered mental status and severe headache.

V. liver capsule distention

VI. Pulmonary Edema or cyanosis

VII. Thrombocytopenia (<1 lakh platelets / mm^3)

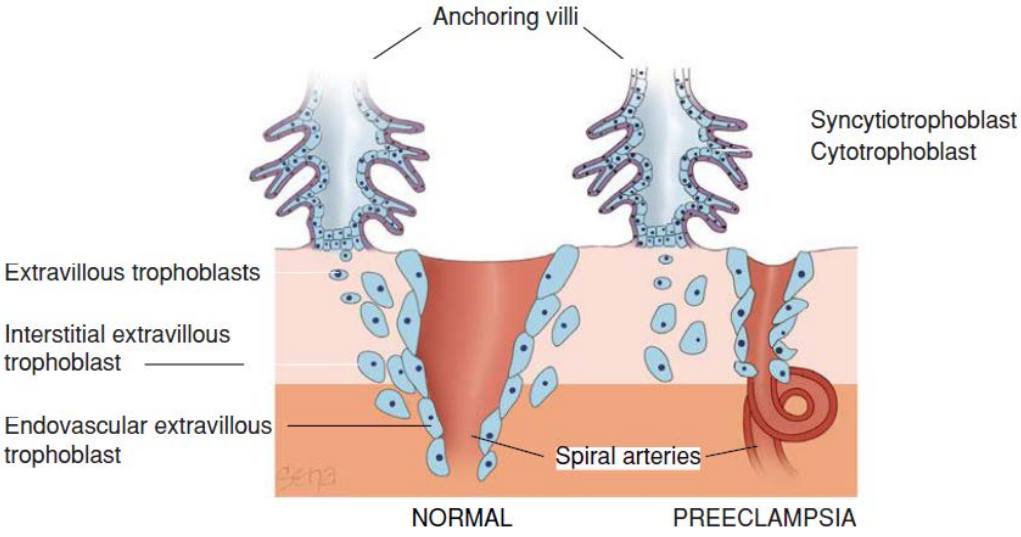
VIII. Hepatocellular injury

IX. Fetal growth restriction

THEORIES ABOUT CAUSES OF PREECLAMPSIA

1. Abnormal trophoblastic invasion

Normal implantation causes remodeling of spiral arterioles in decidua basalis. Endovascular trophoblasts normally replace the tunica intima and tunica muscularis layers and enlarge the diameter of vessel. But in pre eclampsia there is immunological resistance to invading trophoblast by maternal immune system and only the endometrial vessels get lined by trophoblasts and results in inadequate trophoblast invasion of myometrial spiral arterioles.this narrow lumen impairs the placental blood flow and lead to release of placental debris into circulation and induce a systemic inflammatory response[29]



Abnormal trophoblastic invasion of spiral arterioles – secondary invasion

2. Immunological mechanism – BARDEQUEZ[5]

Based on incompatibility at the feto-maternal interface. In pre eclamptic women , there is reduced level of immunosuppressant HLA G, increased Th1 and change in Th1/Th2 ratio. This enhances immunologically mediated inflammatory reaction.

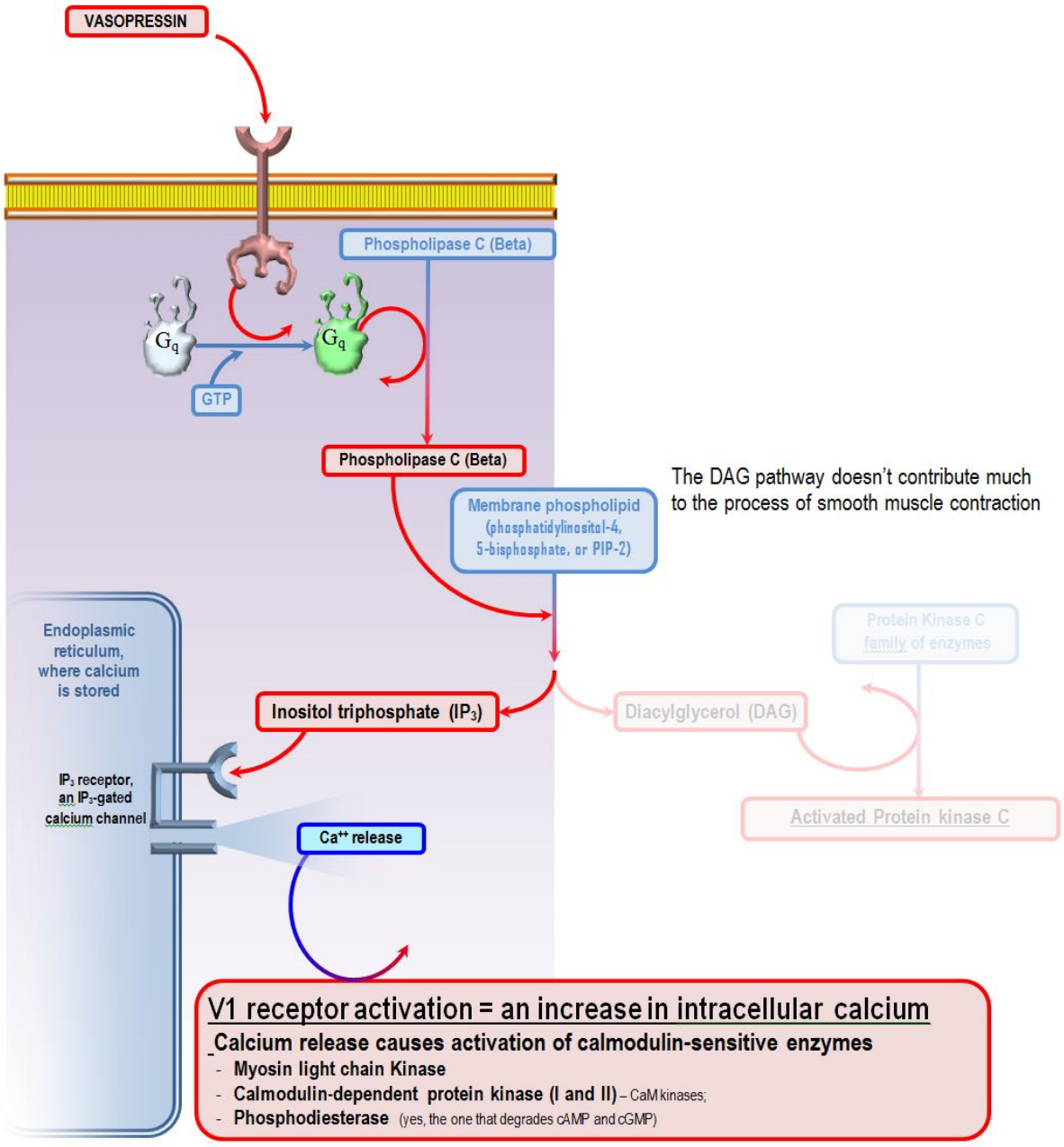
3. Genetic predisposition – CHESLEY & COOPER 1986[6]

Susceptibility is by both single gene and multifactorial inheritance. Angiotensin gene variant T235 is associated with increased incidence. inpreeclamptic patients there is higher incidence of mutation of factor V Leiden

Gene (Polymorphism)	Function Affected
MTHFR (C677T)	Methylene tetrahydrofolate reductase
F5 (Leiden)	Factor V_{Leiden}
AGT (M235T)	Angiotensinogen
HLA (Various)	Human leukocyte antigens
NOS3 (Glu 298 Asp)	Endothelial nitric oxide
F2 (G20210A)	Prothrombin (factor II)
ACE (I/D[at]Intron 16)	Angiotensin-converting enzyme
CTLA4	Cytotoxic T-lymphocyte-associated protein
LPL	Lipoprotein lipase
SERPINE1	Serine peptidase inhibitor

4. Increased pressor response to angiotensinogen II – ABDUL KAREEM 1961 [7]

Normally pregnant women , on vasopressor infusion, develop refractoriness. But pre eclamptic women are more reactive to noradrenaline and angiotensin II which results in gestational hypertension



5. Altered vasoactive factors: VOLHARDT 1918 [8]

(A) Endothelin- 1. A potent vasoconstrictor produced by endothelium. ET-1 is elevated in pregnancy. In preeclampsia it is elevated to an even higher level.[32]

(B) NITRIC OXIDE:- A potent vasodilator is decreased in preeclamptic women.[9]. This causes increased MAP, reduced heart rate.

(C) Reversal of PGI2 to TXA2 and vit.E ratio [10]

(D) Vasoactive maternal factor (VMF) is found to cause the endothelial changes in the pathophysiology of PIH.

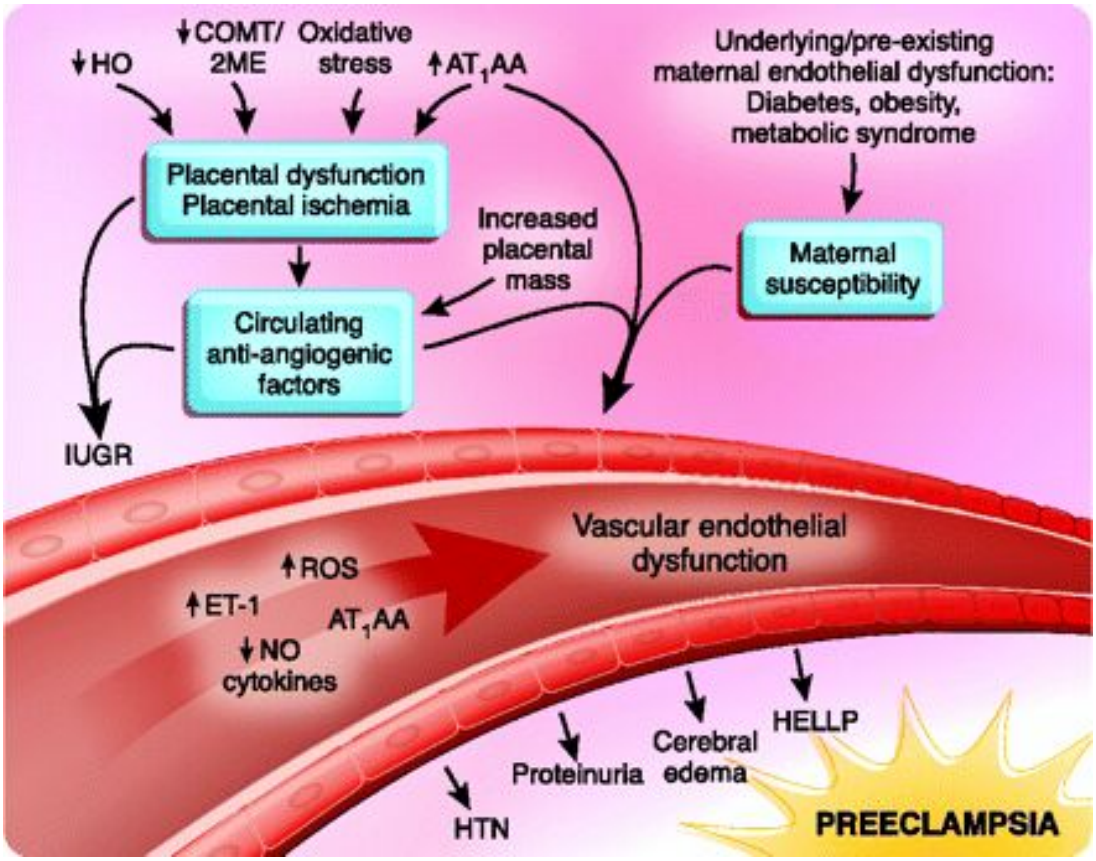
6. Oxidants and Antioxidants

Hubal et al [11] have confirmed that Preeclampsia may have its origin in a disturbed oxidation mechanism. Normally equilibrium is maintained by auto oxidants. With increase in severity of Preeclampsia, lipid peroxidase increases and auto oxidants decreases. Vit E lipid peroxidase is responsible for endothelial damage.

7. Endothelial dysfunction: HAYMAN & ASS 2000[12]

Activation of maternal leucocytes result in endothelial cell dysfunction. Endothelial cell dysfunction is a major factor in the pathogenesis of pre- eclampsia. TNF – α and interleukins release reactive

oxidation species and free radicals that lead to lipid peroxidase formation, which in turn generate toxic radicals and injure endothelial cells resulting in placental oxidative stress.Banker and Coll 1995 have shown that, in preeclampsia, BEGF levels are increased which activate endothelial cells and release inflammatory substances.[13]Recent studies show that raised level of fmstyrosine kinase , TGF beta in association with endothelial dysfunction.



8. Placental proteins:

HCG, Corticotropin releasing factor, Activin A, Inhibin A are said to play an important role.

9. Dietary deficiency:

DAWSON, KELLY & Coauthors Mac Gillivray viewed the evidence for a role of dietary deficiency in pathology of Pre eclampsia. It was found that when calcium is low in ECF, there is increase in amount of ionic calcium entering cell wall making vascular smooth muscle more sensitive to excitation.

10**. Hyperhomocystinemia: COLLER ET AL**

Retroplacental infarcts elevate the levels of circulatory homocysteine. This is due to atherosis formed at placental site. Elevated levels damage endothelium by H2O2 generation.

RISK FACTORS

Maternal and paternal genetic factors increase the risk of preeclampsia.

Pregnancy associated:

- Chromosomal abnormalities
- Hydrops fetalis
- Hydatidiform mole
- Multigravida

Maternal specific:

- Primi parity
- Age < 20 &> 35 Yrs.
- Family history of pre-eclampsia.
- pre-eclampsia in previous pregnancy
- BMI > 35
- Gestational diabetes.
- Chronic hypertension.
- Nephropathy.
- Thrombophilias.

Paternal Specific:

- New paternity
- Previously fathered a pre-eclampsia pregnancy in another woman.

PATHO PHYSIOLOGICAL CHANGES

PRIMARY LEVEL:

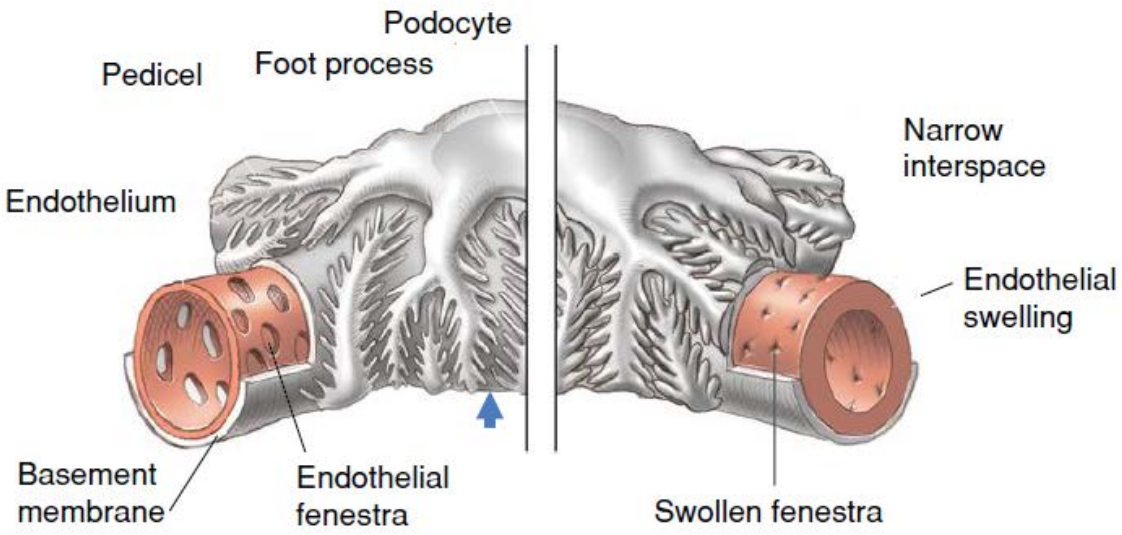
Pre eclamptic changes in placenta and its arterioles are due to:

- inadequate trophoblastic infiltration of arterial walls [33]
- Acute atherosis [34]
- Magnitude of defective trophoblast invasion of spiral arterioles correlated with severity of hypertensive disorder.
- Lipid accumulates first in myometrial cells and then in macrophages[35]

SECONDARY LEVEL

I.Renal system

- Decreased renal blood flow
- glomerular capillary endotheliosis causing glomerular function
- Proteinuria is a result of advanced disease with poor prognosis[36]
- Urinary sediments [37]
- Decreased uric acid clearance (Chesley& Williams 1945)
- Increased tubular reabsorption resulting in Hypocalciuria[38]



GLOMERULAR CAPILLARY ENDOTHELIOSIS

II. Cardiovascular system

- **Hemodynamic Changes:**

  a) decreased Cardiac preload due to generalized vasospasm[14]

  b) increased Cardiac after load due to increase in vascular resistance.

  c) reduced MAP due to increase is peripheral vascular resistance.

- **Hematological Changes**

  a) Thrombocytopenia – probably immunologically mediated or because of increase in platelet deposition at the endothelial damage site.

  b) Hyper coagulation is pre-eclampsia is due to

  • Increased platelet aggregation

•. Increased activity of intrinsic factors.

• Increased thrombin – Anti thrombin ratio.

• Decreased fibrinogens and anti thrombin III

C) HELLP SYNDROME:

Hemolysis- elevated LDH levels, fragmented RBC in peripheral smear, decreased serum haptoglobulin

Elevated AST or ALT more than 70 IU / L

Platetet count less than 1 Lakh

MISSISSIPPI CLASSIFICATION OF HELLP:

Class I - platelet count less than 50000

CLASS II - platelet count - 50,000 - 1,00,000

CLASS III- Platelet 100000- 150000 , elevated AST

III. Endocrine changes:

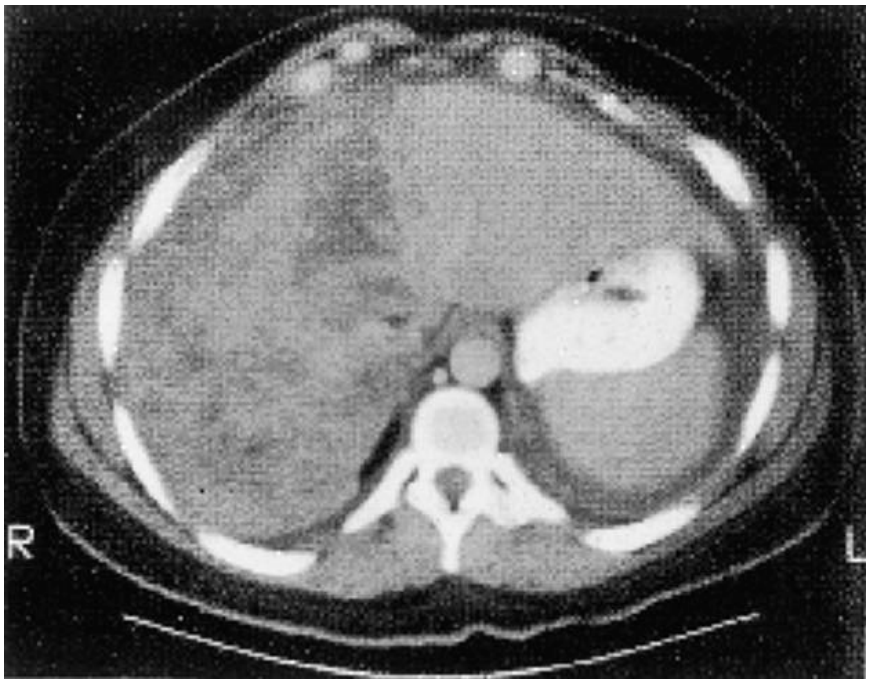
- Suppressed renin- angiotensin - aldosterone axis
- Increased renin levels
- Decreased angiotensin II and aldosterone
- Atrial natriuretic peptide is increased in women with preeclampsia increased sodium retention [39]

IV. Fluid Changes:

Endothelial damage leads to extra cellular fluid volume increases which results in proteinuria and edema

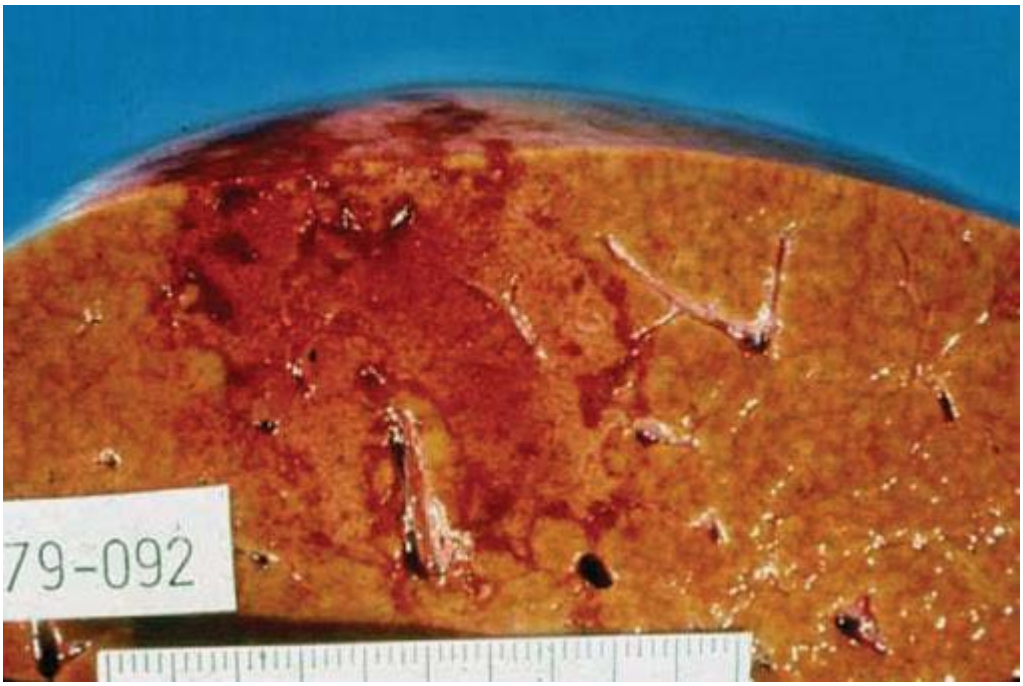
V. Hepatic changes:

- Elevated liver enzymes
- Raised hepatic artery resistance
- Periportal hemorrhagic necrosis
- HELLP syndrome



Subcapsular hemorrhage in liver

PERIPORTAL NECROSIS



VI. Coagulation changes

- Intravascular coagulation
- Prolonged thrombin time
- Decreased platelet aggregation
- Reduced antithrombin III
- Fragmentation haemolysis [40]
- Thrombophilias

TERTIARY CHANGES

Tertiary systemic effects of preeclampsia secondary to decompensationwhichpresents as one of the following features.

1. **Abruptio placenta:**

   Premature separation of placenta.incidence 1 in 80 deliveries, most commonly associated with severe preeclampsia.haemorrhage into decidua basalis of placenta.bleeding in abruption can be concealed, revealed or both.release of thrombolastin ,bleeding in myometrium - couvelarieuterus.severe abruption can resultin PPH, DIC, AKI, shock.fetal complication like IUD can occur in grade II / III

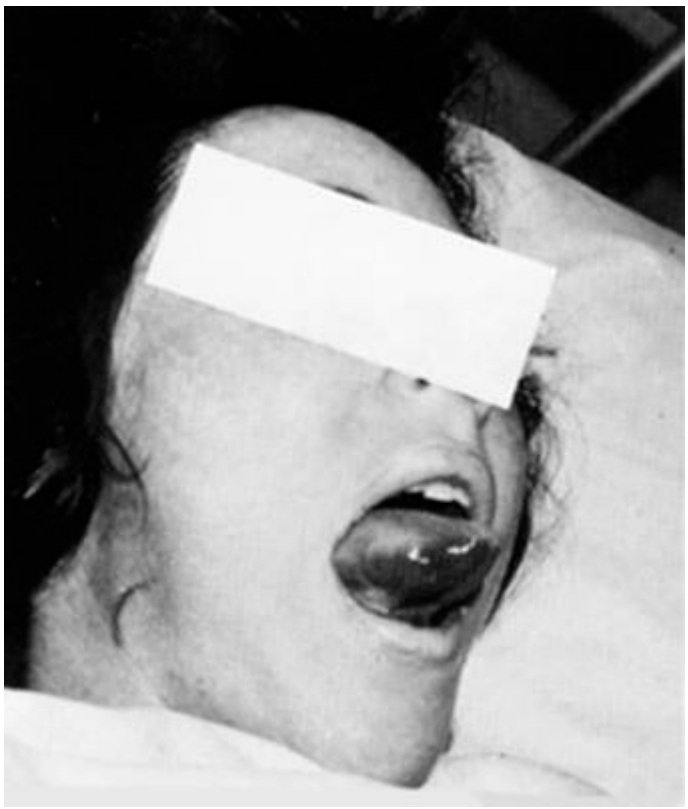
2. **Neurological** – Eclampsia, headache, scotoma, cerebral hemorrhage

   Eclampsia : extremely severe form of preeclampsia.characterised by sudden onset of GTCS or coma in pregnancy / postpartum period. most occur in the last trimester.occurence before 28 weeks carry worse prognosis. Frequency of eclampsia.

   AP eclampsia - 38- 53%

   IP eclampsia - 15 - 20%

   PP eclampsia - 11 - 40%



Pathophysiology is abnormal cerebral autoregulatory response leading to vasoconstriction, ischaemic changes generating electrical discharges that generalise and cause convulsions.case fatality rate is approximately 1.8 %; morbidity - 35%.Perinatal mortality occurs in 5-12%

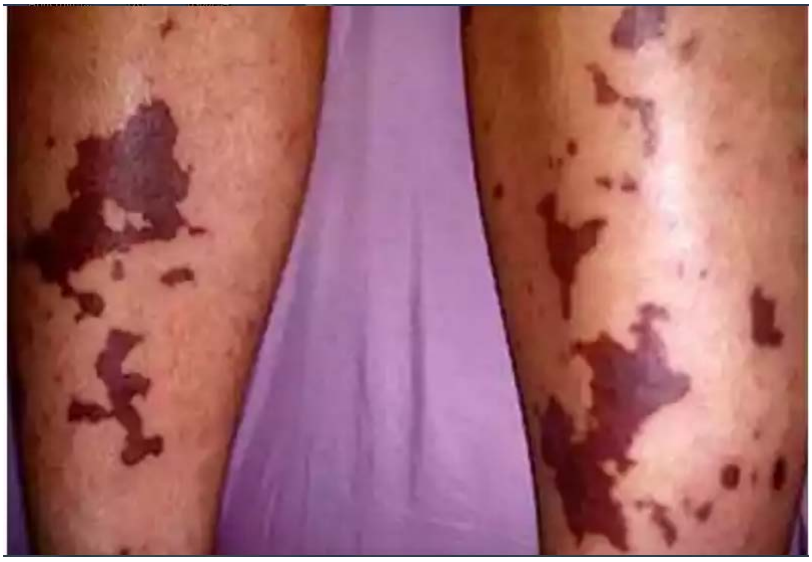
Management of eclampsia :

Control of convulsion : Mgso4 therapy

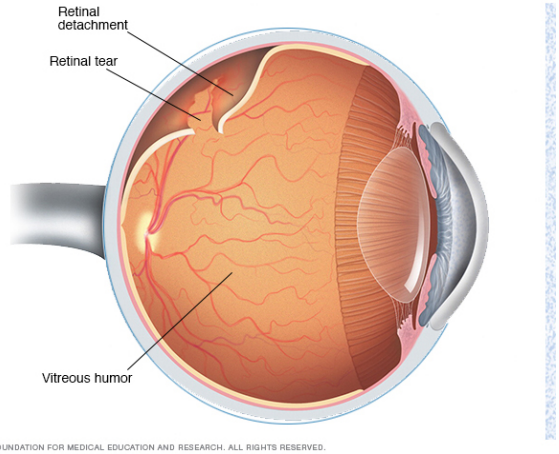
Control of hypertension

Delivery of the fetus

3. DIC
4. Ophthalmic- Corneal edema, retinal detachment
5. Pulmonary edema:
6. ARDS
7. Hepatic rupture



DISSEMINATED INTRAVASCULAR COAGULATION



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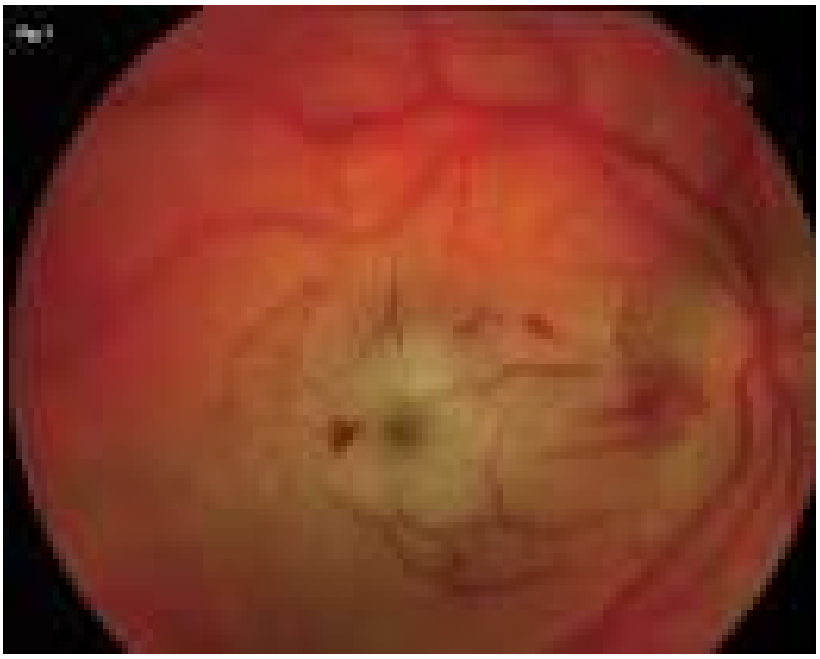
RETINAL DETACHMENT



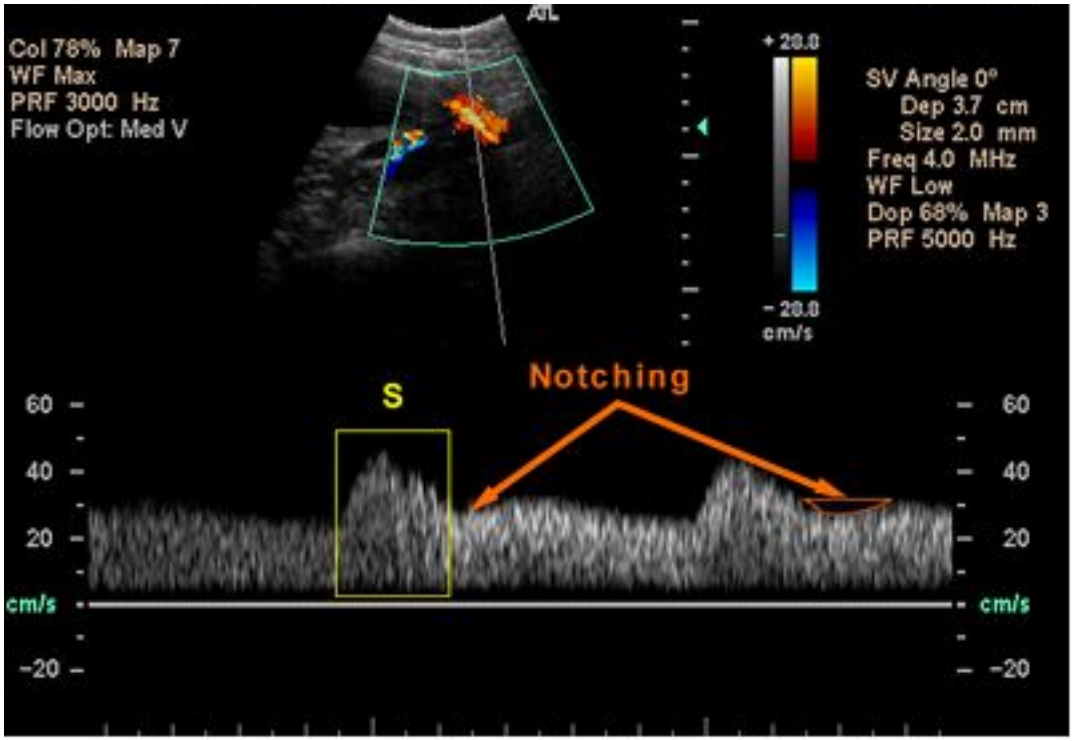
RETINAL DETACHMENT – FUNDAL CHANGES

Testing Related To:	Examples
Placental perfusion/ vascular resistance	Roll-over test, isometric handgrip or cold pressor test, pressor response to aerobic exercise, angiotensin-II infusion, midtrimester mean arterial pressure, platelet angiotensin-II binding, renin, 24-hour ambulatory blood pressure monitoring, uterine artery or fetal transcranial Doppler velocimetry
Fetal-placental unit endocrine dysfunction	Human chorionic gonadotropin (hCG), alpha-fetoprotein (AFP), estriol, pregnancy-associated protein A (PAPP A), inhibin A, activin A, placental protein 13, corticotropin-releasing hormone, A disintegrin, ADAM-12, kisspeptin
Renal dysfunction	Serum uric acid, microalbuminuria, urinary calcium or kallikrein, microtransferrinuria, *N*-acetyl-β-glucosaminidase, cystatin C, podocyturia
Endothelial dysfunction/ oxidant stress	Platelet count and activation, fibronectin, endothelial adhesion molecules, prostaglandins, prostacyclin, MMP-9, thromboxane, C-reactive protein, cytokines, endothelin, neurokinin B, homocysteine, lipids, insulin resistance, antiphospholipid antibodies, plasminogen activator-inhibitor (PAI), leptin, p-selectin, angiogenic factors such as placental growth factor (PlGF), vascular endothelial growth factor (VEGF), fms-like tyrosine kinase receptor-1 (sFlt-1), endoglin
Others	Antithrombin-III(AT-3), atrial natriuretic peptide (ANP), β_2-microglobulin, haptoglobin, transferrin, ferritin, 25-hydroxyvitamin D, genetic markers, cell-free fetal DNA, serum and urine proteomics and metabolomic markers, hepatic aminotransferases

ADAM12 = ADAM metallopeptidase domain 12; MMP = matrix metalloproteinase.

Uterine Artery Doppler Velocimetry:

Trophoblastic invasion of spiral artery is reduced in pre eclampsia. This is predicatble by using Doppler study. Increased uterine artery velocimetry in Doppler study act as indirect evidence for the decrease in trophoblastic invasion.increased artery flow resistance is depicted by an exaggerated diastolic notch in the abnormal wave form.



PREDISPOSING FACTORS OF PREECLAMPSIA

1. Age :

Patients<20 and >30 years showed increased incidence of preeclampsia. In patients > 35 years Pre-eclampsia accounts for over 40% of pre-mature deliveries with increased of risk of low birth weight and Small for gestational age babies

2. Parity :

Incidence of preeclampsia is 11.9% in primi and 4.7% in multigravida. (according to Clinical obs and gyn). The incidence is about 24% in new paternity multipara because they have shorter period of Sperm exposure preceding conception.

3. Race

Muslims, jews&arabs have higher incidence of preeclampsia [Davis et al 1970]

Africoamericans also have higher incidence.

4. Social status:

Women from low social economic status are said to have higher incidence of PIH, PE, Eclampsia. But Baird and colleagues

1969, in their study said that the five socioeconomic status does not show any difference in incidence.

5. Previous H/O preeclampsia

a. higher risk of preeclampsia is seen in subsequent pregnancies when there is history of severe pre eclampsia associated with low birth weight in the previous pregnancies

b. maternal age and interval between pregnancies is directly proportional to the risk of preeclampsia .

6. Family history

Family history of eclampsia and pre eclampsia have a impact on the current preganacy. Risk is three times in pre eclampsia and four times in severe pre eclampsia.

Risk	Over all	Mother with PE	Sisters with PE
Nullipara	5-6%	20-25%	35-40%
Multipara	0.25-5%	1-2%	2-4%

[41]

7. Urinary tract infection:

UTI results in increased production of inflammatory products such as

cytokines, free radicals and proteolytic enzymesthat causes endothelial dysfunction[42]

8. Pregnancy associated: Early onset

a. Twin gestation: Incidence of PE 25.3% due to hyperplacentosis with increased secretion of placental hormone and associated placental ischemia and immunological reaction.

b. Molar pregnancy: Incidence of PE is 70% in women with rapidly growing moles.there is no increased incidence of PE with slowly growing moles.

c. Congenital malformations: the risk of developing PE is 35% due to placentomegaly in triploidy

d. **Hydropsfetalis:**hyperplacentosisrises the risk.

9. Smoking:

Smoking causes a significant reduction in HCG andestradiol level due to direct effect on the placental function

OUTCOME

Maternal Outcome:

Women with Severe Pre-eclampsia are at increased risk for abruptio placenta, DIC, cerebral haemorrhage, acute renal failure, pulmonary edema and circulatory collapse[15].

Al –Mulhim et al reported that the commonest complication to be abruptio placenta.

Perinatal Outcome:

Perinatal outcome depends on:

1. Gestational age at the time of delivery.

2. Gestational age at the onset of pre eclampsia.

3. Present of multiple gestation.

4. Presence of other medical disorders.

5. Severity of the disease.

In a study, **Odegard et al**[16] compared 307 live singleton babies born to pre eclamptic women to 619 controls. Pre-eclampsia and severe preeclampsia were associated with 5% and 12% reduction in birth weight respectively. birth weight was 23% lower than the expected birth weight.

Magee et al[17] in a multi centric retrospective study found out that 16% of pre-eclampsia pregnancies being complicated by birth weight less than third percentile.

Very low birth weight (BW < 1500 grms) and extremely low birth weight (BW< 1000 grms) babies often require re-admission to hospital in the first two years of life for respiratory infections[18]

PULMONARY EDEMA

It Is A Common Complication of severe pre eclampsia. The clinical signs and symptoms include basal rashes over limb folds, respiratory distress, hypoxia and pulmonary edema.

The fluid overload in postpartum period which causes edema expansion and left ventricular failure results in pulmonary edema in preeclamptic patients. Typically the patients are with no prior history of heart disease, normal ecg, no cardiomyopathy on echo or chest xray

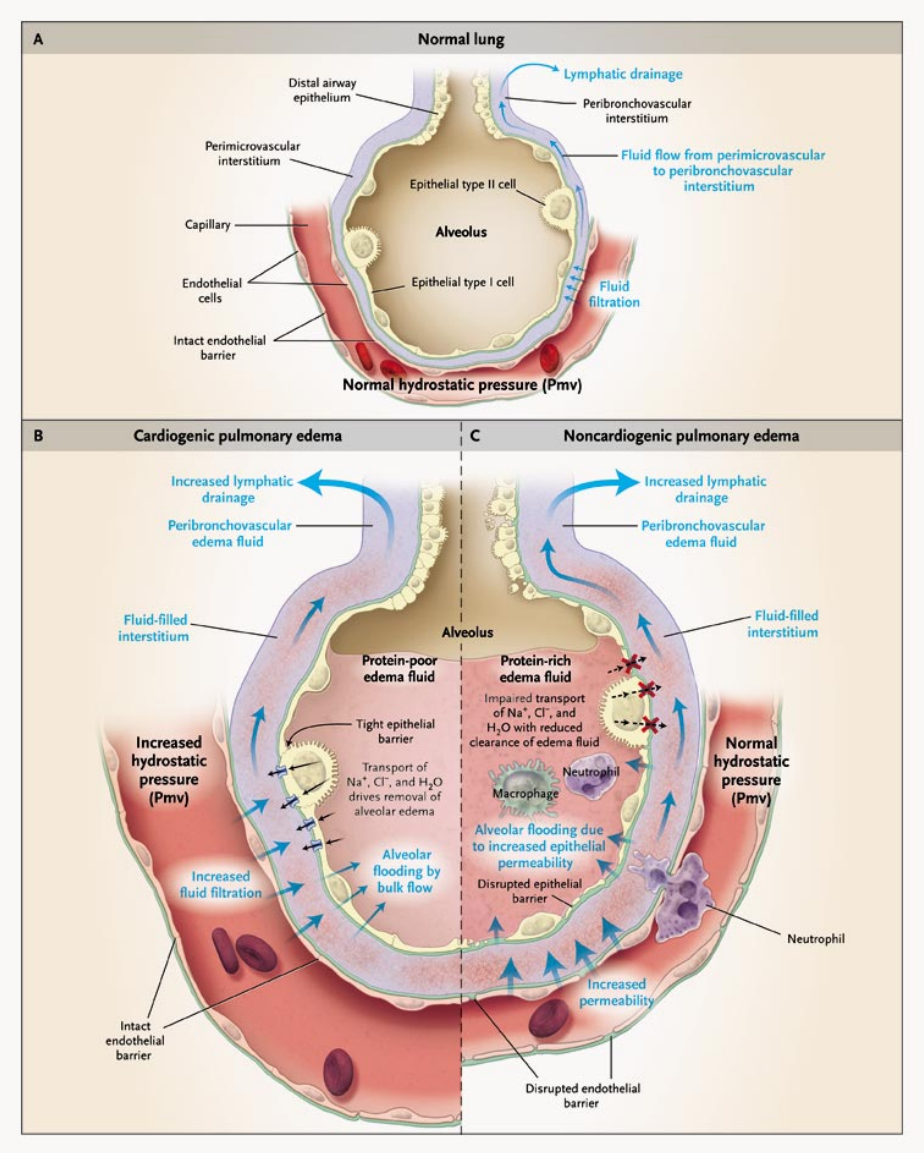
TREATMENT:

Propped up position, back rest, nasal o2 administration, fluid restriction

Diuretics: frusemide 40mg iv q 6hourly

CPAP may be used in severe condition of pulmonary edema with respiratory distress to sshift the fluid to interstitial compartment and thus into capillaries.

Frusemide act by profuse diuresis and thus reducing the intravascular fluid volume. Central venous pressure monitoring may be necessary in patients with co morbid illness.



ABRUPTIO PLACENTA

Abruption is one of the most leading cause of antepartum hemorrhage in patients with pre eclampsia. The premature separation of normally situated placenta prior to the delivery of the fetus occurs mostly in pre eclamptic patients.

It is indicated by antepartum hemorrhage typically associated with uterine tenderness.

Leading into the decidua basalis may be concealed or revealed externally concealed hemorrhage in mostly diagnosed after delivery as retroplacental clot.

The degrees of placental separation may be

1. Mild: concealed hemorrhage with retrospective diagnosis
2. Moderate: live fetus with signs and symptoms of abruption present
3. Severe: dead fetus with features of coagulopathy

Management mainly aims at preventing and reducing complications

- maternal : DIC, hypovolemic shock, AKI
- fetal: low birth weight, preterm labor, fetal hypoxia

conduct of normal labor is possible with continuous fetal heart rate monitoring. normal labor is preferably carried out in tertiary care centre

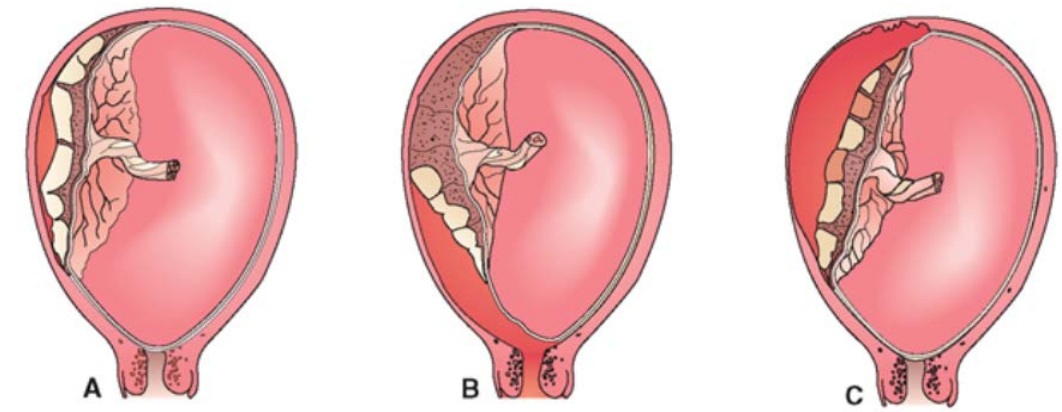
after securing 2large bore IV cannula, bed side clotting time assessment, drawing blood and reserving adequate blood .

Experienced neonatologist may be needed for resuscitation of the baby.

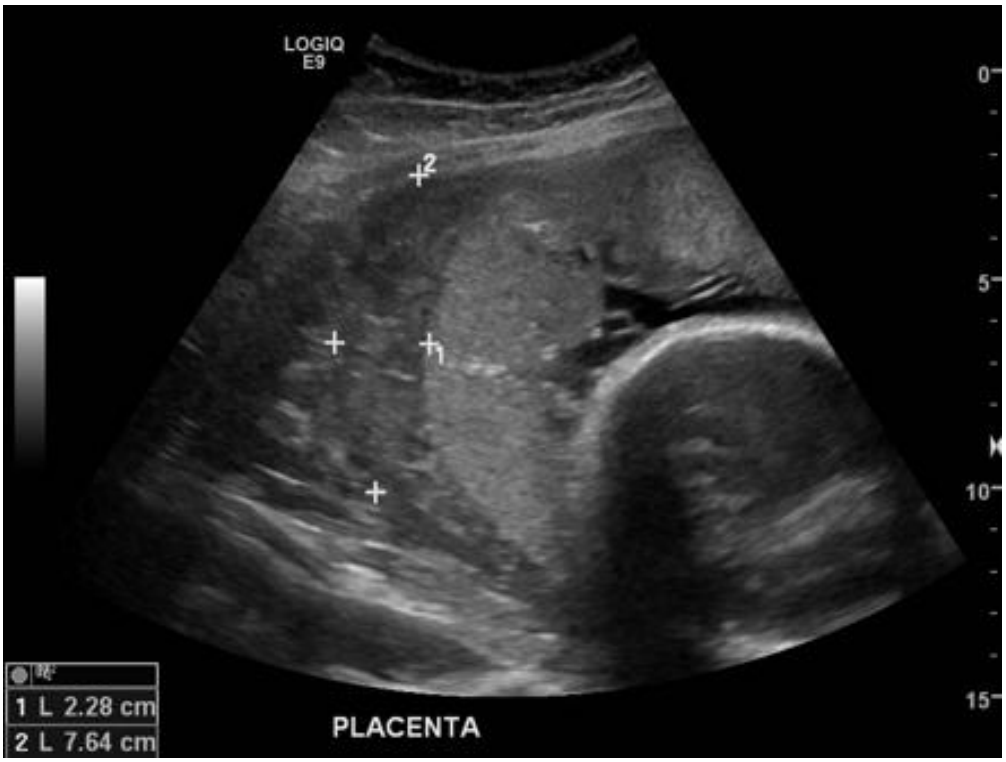
Strict monitoring of maternal vitals with special importance to pulse, urine output and BP.

Caesarean may be taken up in conditions like fetal distress, failure of progress of labor.

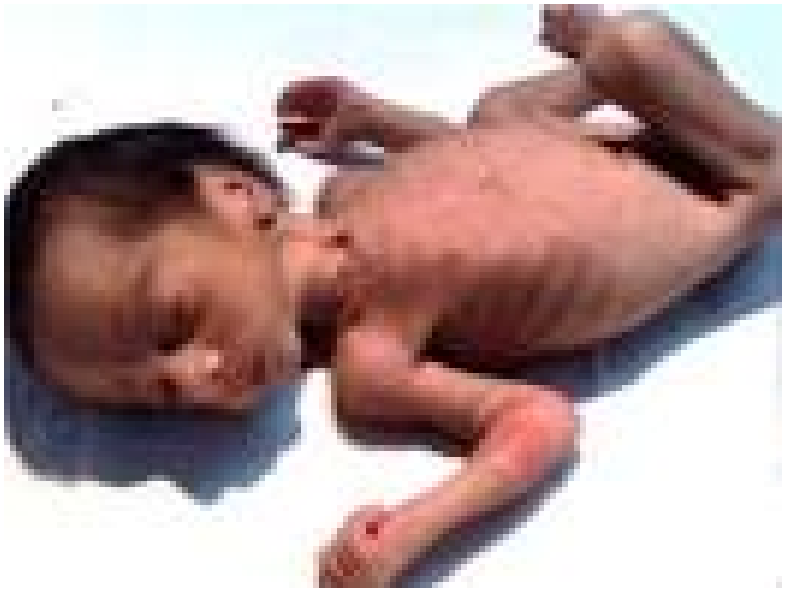
Couvalaire uterus is bleeding into the myometrial layers of the uterus which leads to release of thromboplastins causing DIC. Uterine laxicity leading to atony of uterus causing further bleeding is promptly interfered.



USG OF PLACENTA ABUPTION



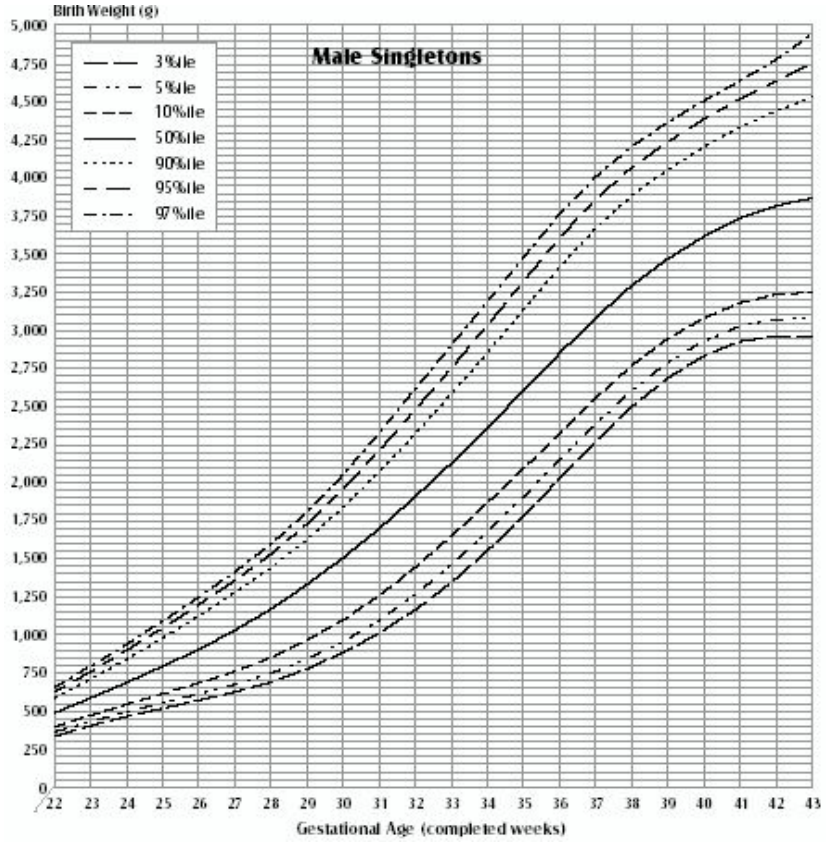
FETAL OUTCOMES



IUGR BABY

APGAR SCORING

INDICATOR	0	1	2
HR	Absent	<100	>100
RR	Absent	Slow, irregular weak cry	Good vigorous cry
MUSCLE TONE	Flaccid, limp	Some flexion of extremities	Good flexion, active motion
REFLEX IRRITABILITY	NR	Weak cry and grimace	Vigorous cry, cough, sneeze
SKIN COLOR	Blue	Acrocyanosis	Pink



GROWTH CHART

RANDOMIZED CONTROLLED TRIALS FOR EXPECTANT VS AGGRESSIVE MANAGEMENT OF SEVERE PREECLAMPSIA REMOTE FROM TERM

S.No	TRIAL	STUDY GROUP	FETAL OUTCOME	MATERNAL OUTCOME
1	Odendaal et al In 1990	38 patients 28-34 weeks GA	Babies in NICU 11% vs 35%) Neonatal omplications (33%vs75%)	No increase in complications
2	Sibai et al 1994	95 patients 28-34 weeks of GA	Lower No.of days in NICU (20.2days vs 36.6 days) Lower incidence of RDS (22.4 % vs 50%) Average birth weight being higher (1622gms Vs 1233gms).	No increase in complications

3.	HALL et al 2000	340 women 28 -34 weeks	------	One maternal death 8% major complications
4.	Visser et al 1994	50 patients 25 to 35 weeks	Perinatal mortality 7% in study group 14% in control group	
5.	Moodley et al 1993	50 patients GA < 32 weeks	Perinatal mortality 27% in 30-32 weeks	20% maternal complications
6.	Visser and wallenburg 1995	250 women < 34 weeks 50% severe preeclamptic 50% HELLP	Perinatal mortality 27%	5% placental abruption 3 women developed eclampsia
7.	Railton A,Allen DG 1987	56 Women 24 -32 weeks	24.5% perinatal mortality 19% small for gestational age	23.2 %major complications

EXPECTANT MANAGEMENT

RCOG AND ACOG RECOMMENDATIONS

AIM:-

To prolong pregnancy in severe preeclampsia women remote from term in order to improve perinatal outcome without increasing maternal morbidity or mortality.

PLACE:-

Management is best carried out in a tertiary care setting. Candidates depend on a number of factors like maternal and fetal conditions as well as gestational age[19]. Expectant management cannot be done in gestational age <26 weeks as there is increase in perinatal & maternal morbidity and mortality.

PROTOCOL FOR EXPECTANT MANAGEMENT

1. All patients are observed in Labour room for at least24 hours to determine their eligibility for expectant management.
2. Intravenous magnesium sulphate for seizure prophylaxis for selected patients.
3. Fetal outcome is improved by glucocorticoid administration.
4. Antihypertensive for BP control.

5. Complete Blood count, Platelet count, urine protein, serum uric acid, serum creatinine, AST, LDH.
6. Limited oral intake for the first 24 hours and intravenous fluids at the rate of 100 –125 ml/ hr within 24 hours.

MANAGEMENT OF SEVERE HYPERTENSION-ANTIHYPERTENSIVE THERAPY

RCOG guidelines- recommend ANTIHYPERTENSIVE if systolic BP more than 160, diastolic more than 110.

LABETOLOL:

First line drug in both acute severe hypertension, maintenance treatment.

Combined alpha and beta adregenic blocker

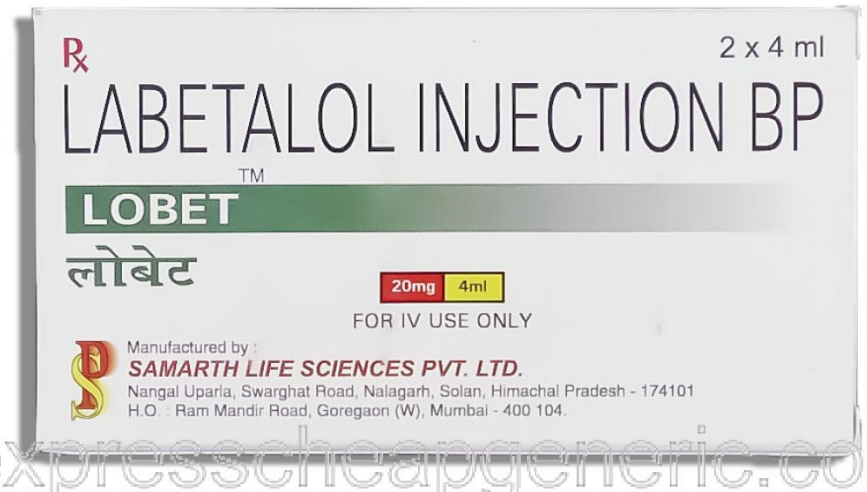
Available in both oral and parenteral forms.

Ratio of blockade is 1 : 3 in oral , 1: 7 in parenteral

Avoided in asthmatics, heart failure

Oral treatment- 100 to 400mgevery 6- 12 hours

For intermittent iv dose- 20 mg bolus followed by 40- 80 mg every 20 minutes. Maximum dosage 220 mg per cycle



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Continuous iv use, 500 mg Labetolol added to 400 NS, administered at 20 mg / hour.Dose doubled or halved according to BP. Maximum dose- 220 mg / hr

NIFEDIPINE:

Calcium channel blocker

Reduces peripheral vascular resistance, tocolytic agent by blocking influx of calcium ions in vascular smooth muscle cells. Initial dose of 10 mg repeated after 30 mins according to BP

Rapid onset of action, half life - 30 mins

Others:

- Alpha methyl dopa- not recommended in severe preeclampsia due to delayed onset of action.
- Diazoxide may result in sudden hypotensive response, not recommended
- Sodium nitroprusside to gradually decrease BP. Possibility of fetal toxicity with prolonged use because of cyanide content.
- Hydralizine - acts directly on the arteriolar smooth muscle thereby reducing PVR. Maternal tachycardia, late decelerations in CTG due to decrease in uteroplacentalflow are the disadvantages.

ROLE OF STEROIDS:

Dexamethasone 6mg im 4 doses, 12 hours apart is recommended

Betamethasone 12 mg im 2 doses, 24 hours apart.

Studies show that dexamethasone is better than betamethasone in Indian preparation.

Neonatal complications like RDS , death, IVH are reduced if STEROIDS administered prior to delivery

MGSO4 TREATMENT

Prevention of seizures by MgSo4 regimen is widely used. Strict monitoring of urinary output, patellar reflex, respiratory rate, and saturation is needed.

Disappearance of patellar reflex - First sign of impending MgSo4 toxicity is which is handled by administration of 10ml of 10% calcium gluconate, used as an antidote.

Pritchard regime:

- Most preferred regime
- Loading dose: 20 ml of 20% MgSo4 slow IV and IM 50% MgSo4 10grams – 5 grams each buttock deep IM.
- Followed by 10 ml of 50% MgSo4 deep IM (5ml on each buttock) every 4 hours until 24 hours after delivery or onset of last episode of last seizure whichever occurs later.

Other management protocols include:

- daily weight chart
- I/O chart
- Umblical and middle cerebral artery Doppler twice every week
- USG- for fetal growth every 2 weeks

BOX 13.9 Guidelines for Intramuscular Magnesium Sulfate (Pritchard's Regime)

Intravenous loading dose (only in patients with eclampsia):

- Give 20 mL of 20% magnesium sulfate (4 g) slow intravenous in 3–5 minutes at a rate not exceeding 1 g/min

Intramuscular loading dose:

- 10 mL of 50% magnesium sulfate (5 g) deep intramuscular in the upper outer quadrant of each buttock using a 3 inch, 20 gauge needle. The intramuscular injection should immediately follow the intravenous loading dose in patient with convulsions. Patients without convulsions may receive only the intramuscular loading dose.

Maintenance dose:

- Give 5 g magnesium sulfate (10 mL of 50% solution) deep IM injection in alternate buttock every 4 hours.

Monitoring for magnesium toxicity:

- Urine output should be at least 30 mL/hour or 100 mL in 4 hours
- Deep tendon reflexes should be present
- Respiration rate should be > 14 breaths/minute
- Pulse oximetry should be ≥ 96%
- Any change in these indices makes it necessary to reevaluate the rate of administration

Magnesium sulfate is discontinued 24 hours after delivery or after last convulsion.

Expectant Management in IUGR:

Studies have showed that in servere pre-eclampsia at 24 – 33 weeks IUGR is also associated with high risk of fetal mortality but does not result in maternal complications[20] and this may benefit from prolongation of pregnancy beyond 48 hours which is required for action of steroids.

Expectant management in HELLP Syndrome:

Studies show that women with HELLP syndrome should not be expectantly managed. Vaginal or caesarean delivery should be pursued. Concurrently antenatal steroids may be given.

GUIDELINES FOR AGGRESSIVE TREATMENT:

1. All patients should be observed in labour room.

2. IV magnesium sulphate given to selected patients for seizure prophylaxis.

3. Glucocorticoid therapy given for improving fetal outcome followed by delivery in 48 hrs.

4. Antihypertensives for BP control, blood investigations done - Complete Blood count, Platelet count, urine protein, serum uric acid, serum creatinine, AST, LDH

5. USG for fetal well-being .Then labour is induced either by misoprostol or cerviprime may or may not be augmented with syntocin infusion

GUIDELINES FOR MANAGEMENT

FETO – MATERNAL MONITORING:

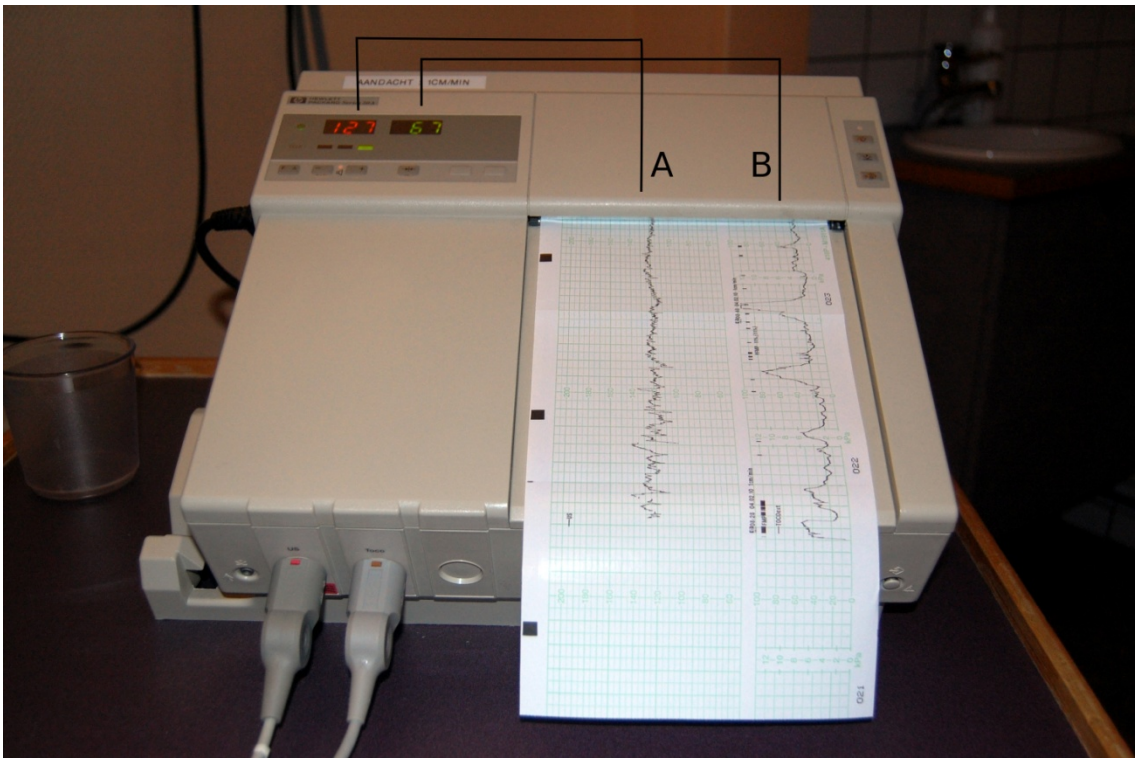
- Initially, blood pressure should be measured for every 15 minutes till stabilized and then to be checked every 30 minutes.
- Later, measure BP at least four times per day
- Daily weight chart, urine albumin chart
- Ophthalmic evaluation for retinal fundus changes
- CBC with platelet count,AST,ALT, LDH are repeated every other day
- If platelet count is above 1 Lakh cells/dL, clotting studies are not required.
- Input and output charting is essential in monitoring the fluid balance,especially in the immediate postpartum period
- To have vigilant watch for imminent symptoms and signs like headache, blurring of vision, vomiting, epigastric pain, decreased urinary output.
- Daily fetal movement count
- Antihypertensive treatment

- Steroids- InJ. Dexamethasone 6 mg , 4 doses over 48 hrs

ASSESSMENT OF FETUS:

Fetal Monitoring is done by fetal heart rate monitoring withcardiotocographythat gives information regarding fetal wellbeing. in women in labour, Continuous fetal monitoring should be done.furtherassessmentis done by

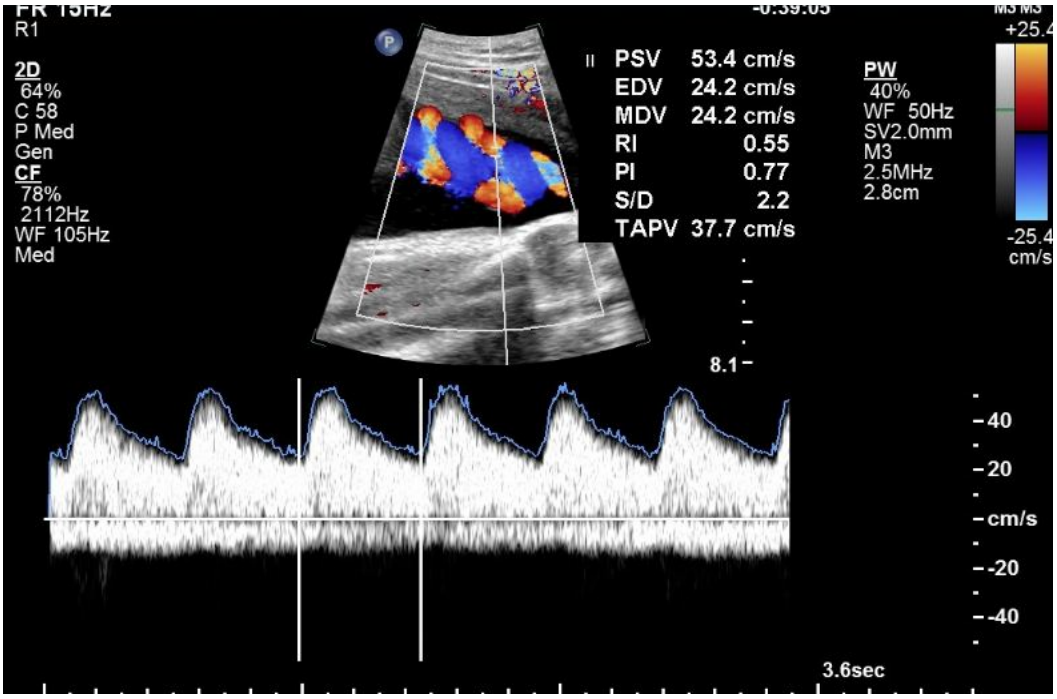
- NST weekly to daily depending on liquor status, fetal growth.
- Fetal biometry every 2 week
- liquor volume twice every week
- umbilical and cerebral Doppler once in 2 weeks



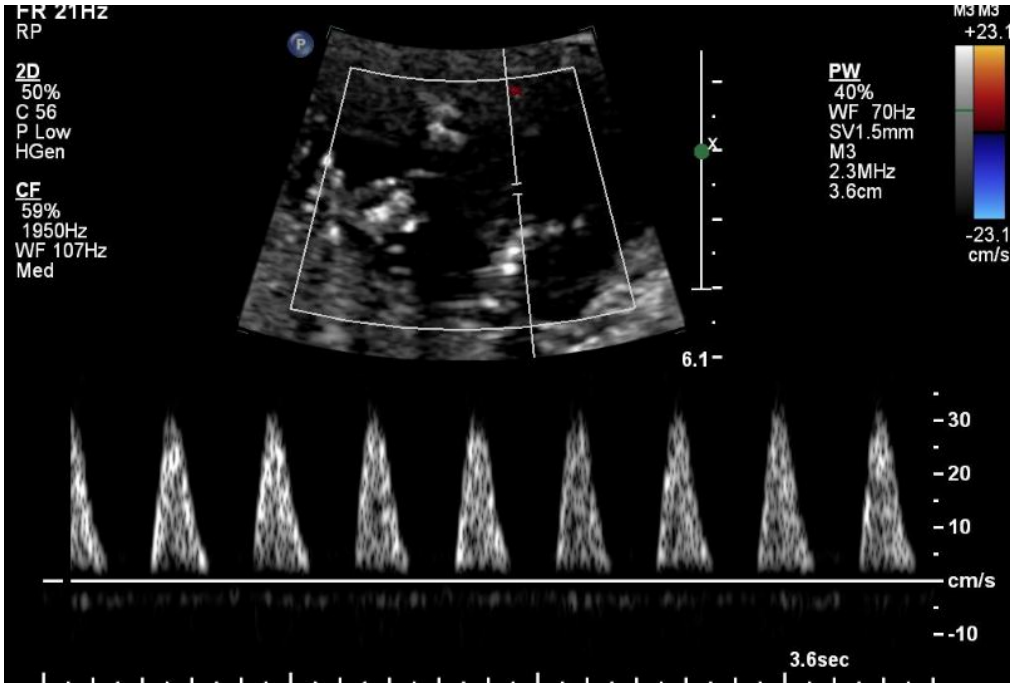
BIOPHYSICAL PROFILE

variables	normal score = 2	abnormal score = 0
fetal breathing movements	≥1 episodes in 30 min each lasting ≥30 sec	absent or no episode ≥30 sec in 30 min
gross body movements	three or more discrete body or limb movements in 30 min (episodes of active continuous movement = a single movement)	less than 3 episodes of body or limb movements in 30 min
fetal tone	≥1 episodes of active extension with return to flexion of fetal limb(s) or trunk; opening and closing of hand is considered normal tone	slow extension w/return to flexion, movement of limb in full extension, or fetal movement absent
reactive fetal heart rate	≥2 episodes of accelerations (≥ 15 beats/min) in 20 min, each lasting ≥ 15 sec and associated with fetal movement	< 2 episodes of accelerations or acceleration of < 15 beats/min in 20 min
qualitative amniotic fluid volume	≥1 pockets of fluid measuring > 1 cm in 2 perpendicular planes	pockets absent or pocket < 1 cm in 2 perpendicular planes

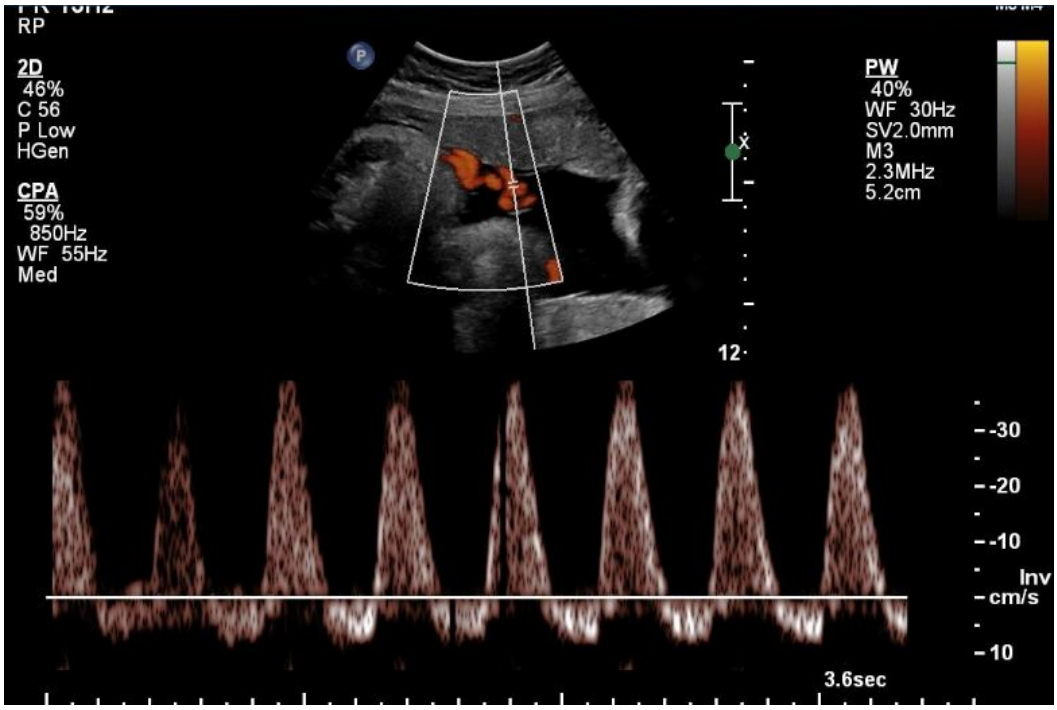
score		notes
normal	8 – 10 (if amniotic fluid index is adequate)	CNS is functional & fetus is not hypoxemic
equivocal	6	
abnormal	< 4	along w/oligohydramnio⟶ labor induction



Umbilical artery showing normal SD ratio



Absent diastolic flow Doppler



Reversal of diastolic flow doppler

Dopplerevaluation using absent or reversed umbilical artery diastolic flow improves fetal outcome[43]. Delivery time can be measured by continuous serial monitoring.Rate of perinatal death is about 33 % in reversal of diastolic flow. 10 % in absent diastolic flow.

TERMINATION OF PREGNANCY

Termination is needed is the following conditionsn(criteria to INTERRUPT EXPECTANT MANAGEMENT)

FETAL

- Non reassuring fetal surveillance
- Abnormal Doppler changes
- Severe IUGR
- Oligohydramnios
- Fetal death
- Biophysical profile less than or equal to 4

Maternal

- Abruptio placenta
- Progressive detoriation of liver and renal function
- Platelets < 1 lakh
- Signs and Symptoms of imminent eclampsia

MODE OF DELIVERY:

There are no randomized trials for comparing the ideal method for delivery in women with pre-eclampsia. Vaginal delivery should be attempted for all women with mild pre-eclampsia and for most women with severe pre-eclampsia, provided that there is no other indication for caesarean section[21]. **Pre-eclampsia per se is not an indication for caesarean section.**

INTRAPARTUM MANAGEMENT

Preeclamptic women are at higher risk of developing convulsions during labour. Highest being is seen in severe preeclampsia remote from term, and those with cerebral manifestations, HELLP syndrome.

1. Hence if not initiated with MgSO4, it should be started in labour in selected cases.

2. Once cervix becomes favourable for, oxytocin augmentation may be given.

3. If it is unripe, Caesarean section should be considered because of higher incidence of abruption, fetal distress and other complications.

4. ANALGESIA

- Provided by intermittent use of 25-50 mg of pethidine (parentral) or segmental epidural analgesia.
- Local infitration in vaginal delivery.
- Continuous epidural or balanced GA used for caesarean

5. Input and output monitoring

- Hourly urine output.
- restricted Fluid intake to 150 ml / hr.
- If oliguria (<100ml per 4 hrs), fluids & MgSO4 adjusted accordingly.

6. Antihypertensive therapy.

Goal is to maintain systolic BP 140 –150 and diastolic 90-100 mm Hg. mean arterial pressure should not be reduced by more than 20% from baseline.

POSTPARTUM MANAGEMENT

1. Intensive monitoring done for 2-4 days. Vitals, input output monitoring and reflexes are noted.

2. BP control

3. Prophylactic anticonvulsant are not given.

4. Patient is seen weekly until her BP returns to normal without medications.

5. If change does not occur by 6 weeks, hypertension work up is made

MATERIALS AND METHODS

TYPE OF STUDY: COMPARITIVE

SAMPLE SIZE: 100

PERIOD OF STUDY: JULY 2015-JUNE 2016

INCLUSION CRITERIA

- Patients Of age group- 18 to 35, diagnosed as severe PRE ECLAMPSIA with BP under control

EXCLUSION CRITERIA FOR EXPECTANT MANAGEMENT (AT THE TIME OF DIAGNOSIS)

- Uncontrolled hypertension with antihypertensives
- Signs of imminent eclampsia
- AP Eclampsia
- Abruptio placenta
- Deterioration of renal function, Liver function
- oliguria
- HELLP syndrome
- Abnormal fundus examination
- IUGR
- Abnormal Doppler study

A COMPARITIVE STUDY WILL BE CONDUCTED BETWEEN JULY 2015 to JUNE 2016

<u>GROUP 1:</u> 50 PATIENTS WITH SEVERE PRE ECLAMPSIA PRESENTING BETWEEN 30-34 WEEKS TERMINATED ACTIVELY

<u>GROUP 2</u>: 50 PATIENTS WITH SEVERE PRE ECLAMPSIA, MANAGED CONSERVATIVELY TILL 34 WEEKS PROVIDED BP IS UNDER CONTROL (with normal liver, renal function)

Procedure:

- All the pregnant women attending antenatal op are screened for hypertension.
- Patients with severe PIH, with BP- .≥160/110, urine albumin ≥2+ are hospitalised.
- On admission, thorough clinical examination including general examination, built, nourishment, height, weight, BP, pulse, along with pallor, pedal edema. Weight gain is noted.
- CVS, RS examination done.
- Abdominal examination done for height of uterus in weeks, lie of the fetus, presentation, position of the fetus, fetal heart rate.

- Blood investigations including CBC, RFT, LFT, SERUM URIC ACID done. 24 hr urine protein excretion evaluated, Fundus examination done. USG with Doppler done for fetal wellbeing.
- Treatment included rest, Anti hypertensives for BP control, Inj.dexamethasone for fetal lung maturiy.
- Maternal complications in both groups are compared.
- Fetal outcome in both groups including birth weight, incidence of NICU admissions are compared between both groups studied.

DATA ANALYSIS

Table 1: Maternal age

Age	EXPECTANT (n - 50)		ACTIVE (n-50)		TOTAL
	No.	%	No.	%	
<20	9	18%	7	14%	16
21-30	29	58%	25	50%	54
>30	12	24%	18	36%	30

Most no. of women are in age group 21-30 (54%)

Mean age= 26 years

Range = 18 to 36 years

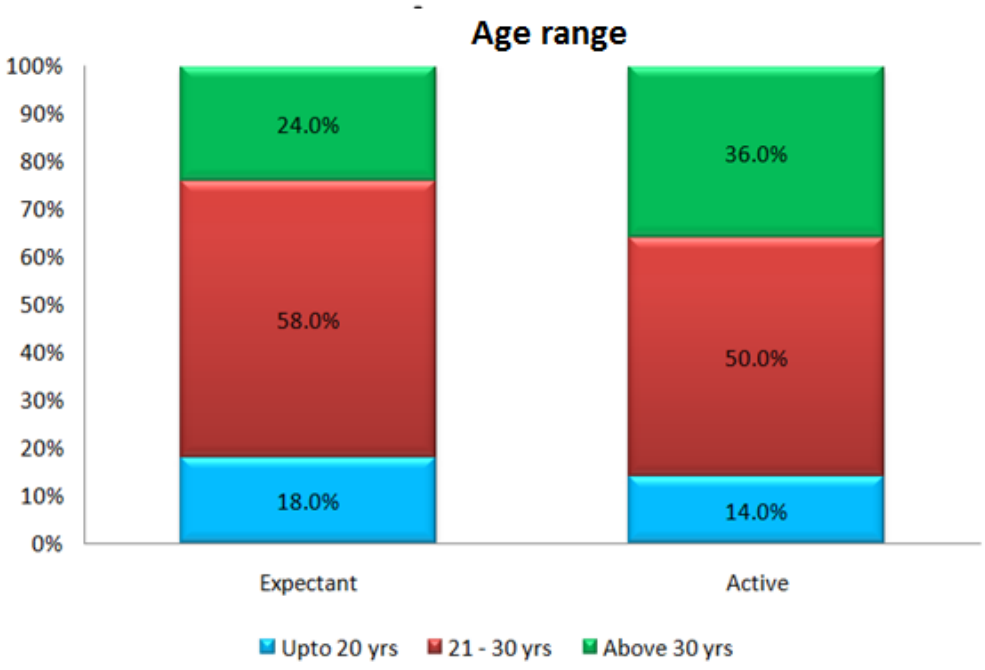


Table 2: Gravida

GRAVIDA	EXPECTANT (n-50)		ACTIVE (n-50)		TOTAL
	No.	%	No.	%	
Primi	30	60%	32	64%	62
multi	20	40%	18	36%	38

most women are Primi (62%)

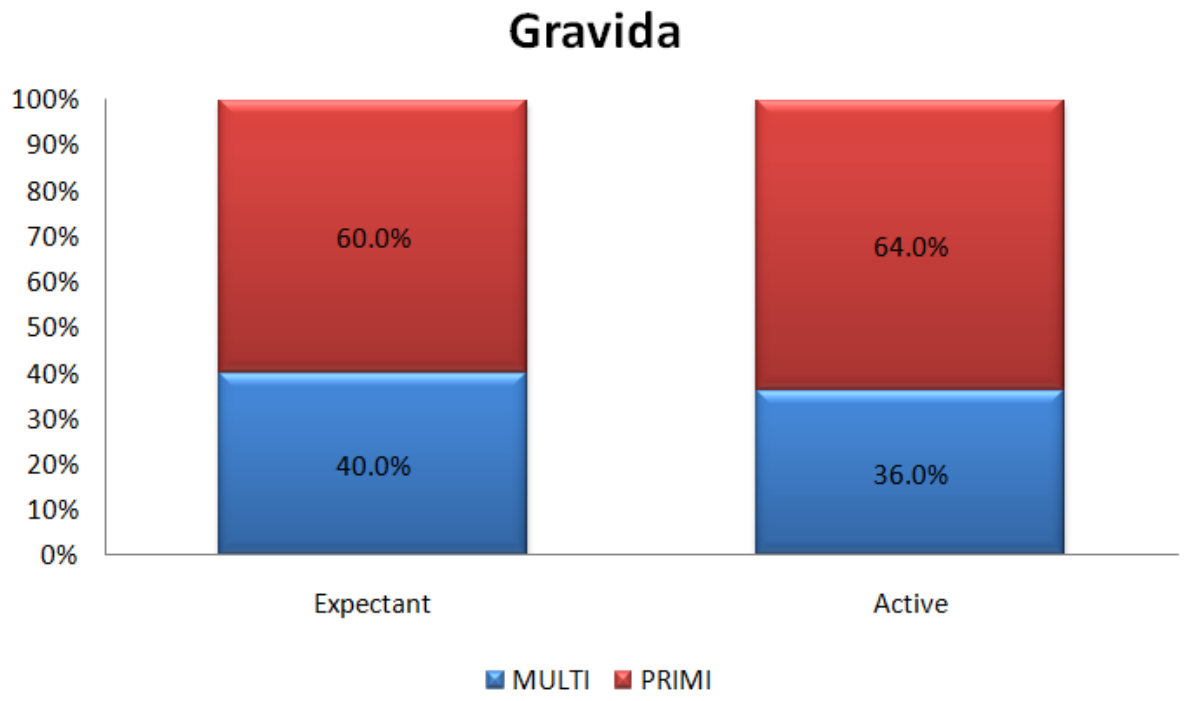


Table 3: Gestational age

GA	EXPECTANT (n-50)		ACTIVE (n-50)		TOTAL
	No.	%	No.	%	
30-32 weeks	35	70%	33	66%	68
32-34 weeks	15	30%	17	34%	32

Only women with gestational ages between 30 and 34 weeks were selected for the study. Mean gestational age = 31 weeks

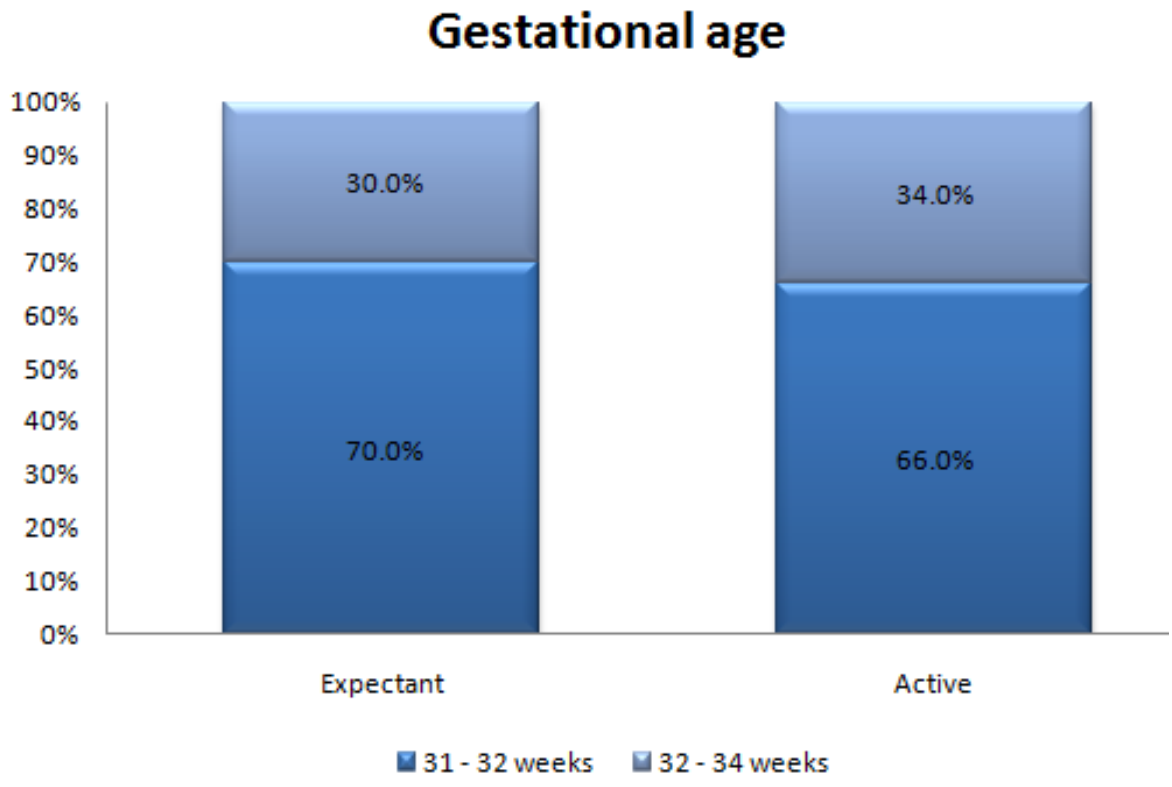


TABLE 3: BOOKED OR UNBOOKED

GA	EXPECTANT (n-50)		ACTIVE (n-50)	
	No.	%	No.	%
Booked	50	100%	48	96%
Unbooked	0		2	4%

On the whole, 98 patients were booked. Only 2 were unbooked

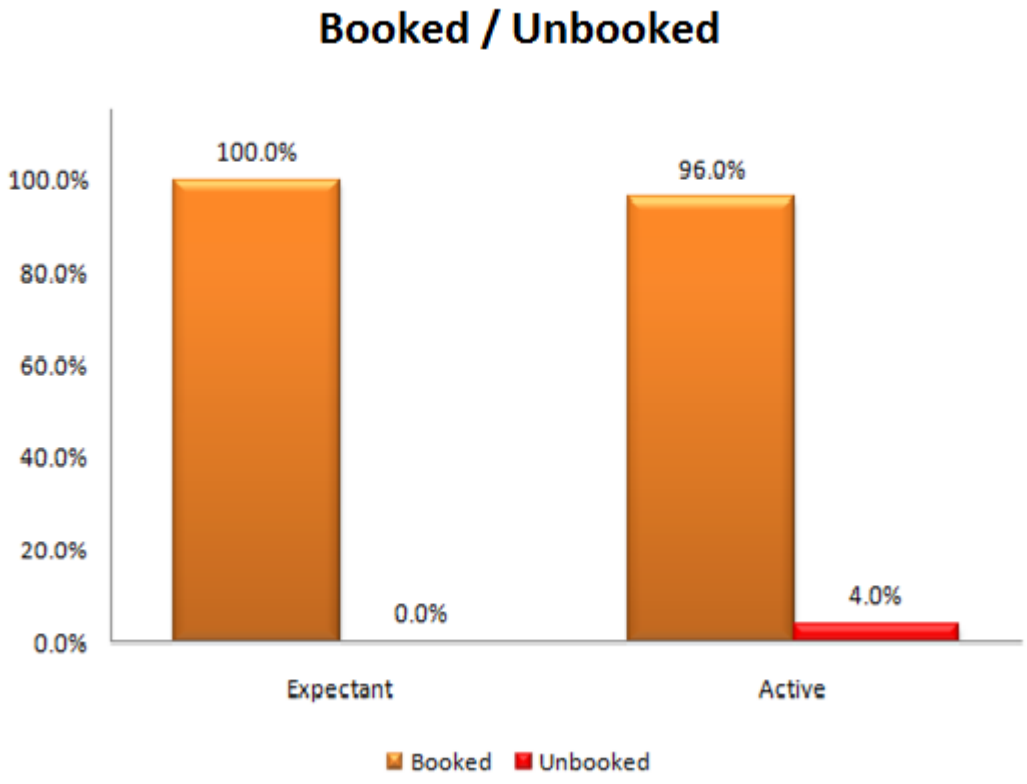


TABLE 4: GESTATIONAL AGE AT DELIVERY

GA	EXPECTANT (n-50)		ACTIVE (n-50)		TOTAL
	No.	%	No.	%	
30-32 weeks	17	34%	29	58%	46
32-34 weeks	33	66%	21	42%	54

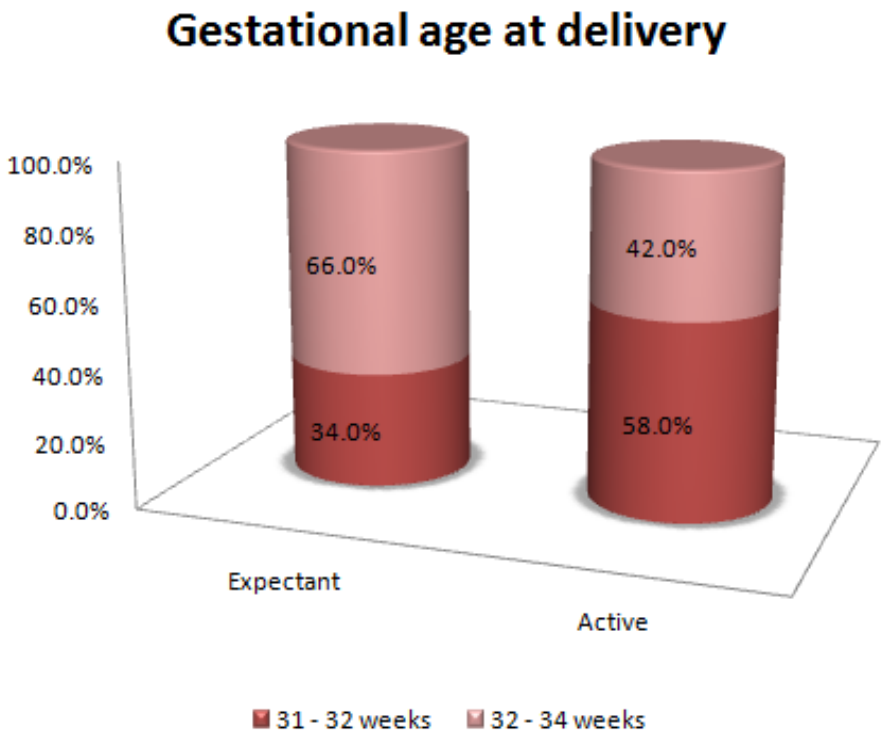


Table 5: STEROID INJECTION GIVEN/NOT

Steroid given	EXPECTANT (n-50)		ACTIVE (n-50)		TOTAL
	no	%	No	%	
yes	50	100%	33	66%	83
no	0		17	34%	17

All women managed expectantly were given steroid injection or fetal maturity. Whereas only 66% required steroid in active group

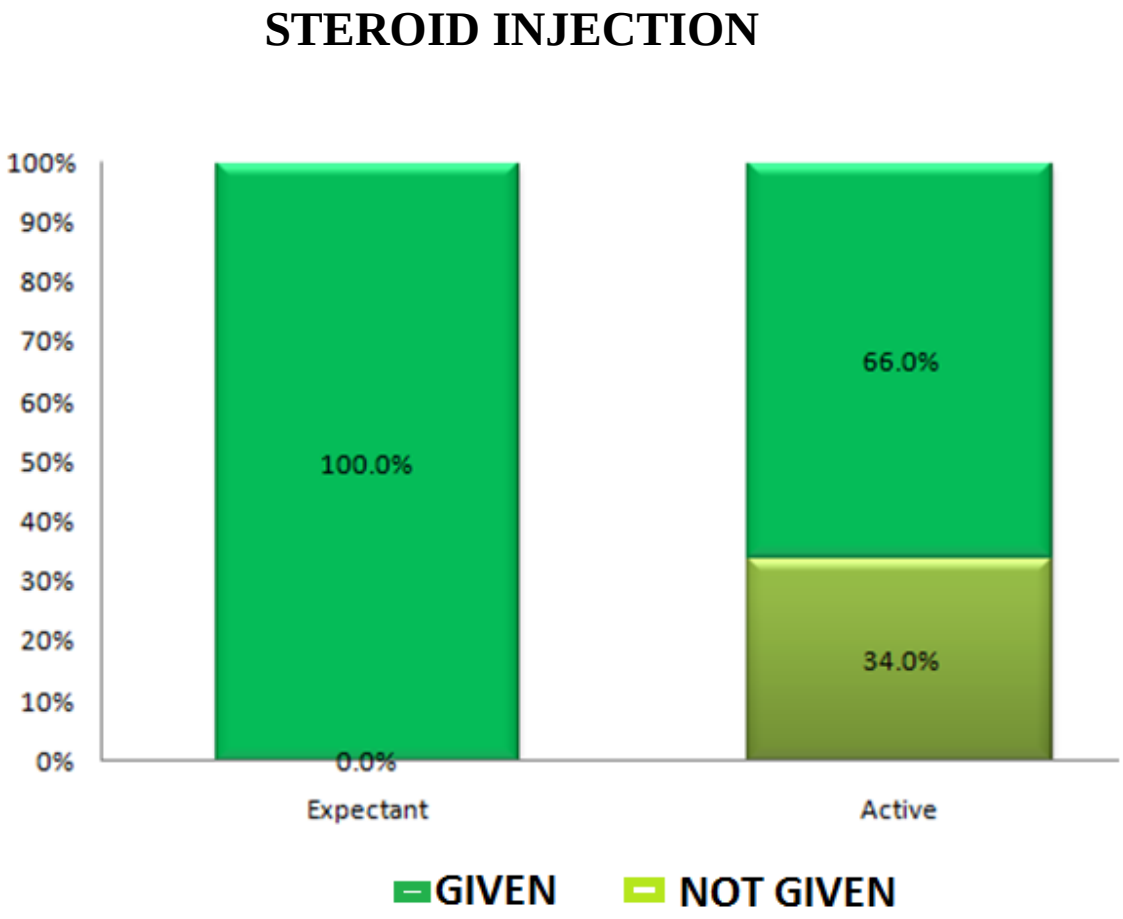


TABLE 7: INDICATION FOR TERMINATION

GA	EXPECTANT (n-50)	
	No.	%
FETAL	21	42%
MATERNAL	29	58%

Maternal indication accounts for 58% of the cases while Fetal indication for 42%

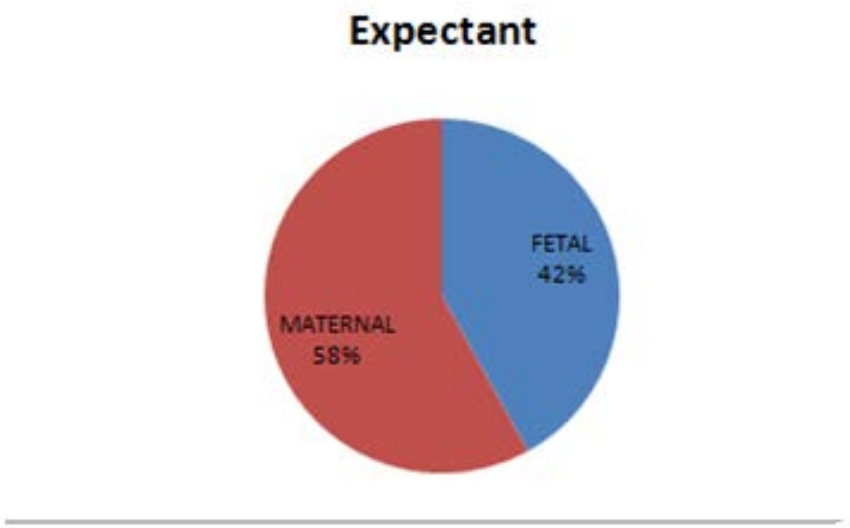


Table 8: INDICATION FOR TERMINATION

Complication	Expectant
Abnormal coagulation profile	1
Abnormal CTG	4
Abnormal Doppler	0
Abnormal platelet count	1
Abruption	4
Acute kidney injury	2
Eclampsia	1
Fetal distress	0
Fundus changes	2
HELLP	0
Imminent eclampsia	10
IUD	1
IUGR	5
Oligohydramnios	10
Pulmonary edema	1
Uncontrolled BP	8
Total	50

Table 9: MODE OF DELIVERY:

Mode	Expectant (n-50)		Active (n-50)		total
	no	%	No	%	
vaginal	31	62%	34	68%	65
caesarean	19	38%	16	32%	35

Chi square= 0.396

Degree of freedom= 1

P=0.53 (insignificant)

Total LSCS= 35

Mode of delivery has no significant influence over the fetal / maternal outcome.

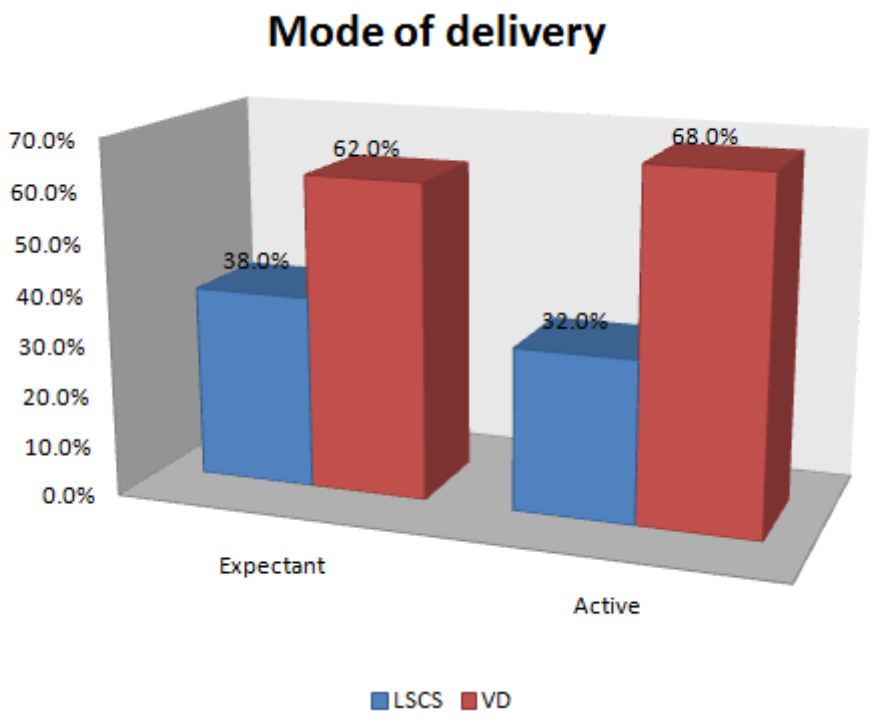

Mode of delivery
70.0%
60.0%
50.0%
40.0%
30.0%
20.0%
10.0%
0.0%
38.0%
62.0%
32.0%
68.0%
Expectant
Active
LSCS
VD

Table 10: MATERNAL OUTCOME

			Expectant	Active	Total
	ABRUPTION	Count	4	5	9
		%	23.5%	38.5%	30.0%
	ACUTE KIDNEY INJURY	Count	2	1	3
		%	11.8%	7.7%	10.0%
	ANTEPARTUM ECLAMPSIA	Count	3	2	5
		%	17.6%	15.4%	16.7%
	DIC	Count	1	0	1
		%	5.9%	0.0%	3.3%
	HELLP SYNDROME	Count	2	2	4
		%	11.8%	15.4%	13.3%
	INTRAPARTUM ECLAMPSIA	Count	1	0	1
		%	5.9%	0.0%	3.3%
	POSTPARTUM ECLAMPSIA	Count	1	0	1
		%	5.9%	0.0%	3.3%
	PRES	Count	1	1	2
		%	5.9%	7.7%	6.7%
	PULMONARY EDEMA	Count	2	2	4
		%	11.8%	15.4%	13.3%
Total		Count	17	13	30
		%	100.0%	100.0%	100.0%

In our study there was no maternal mortality. Maternal morbidity was seen in 30%. Abruption was the highest accounting to 9 patients, followed by eclampsia in 7. Other complications were HELLP, pulmonary edema, PRES, DIC and acute kidney injury

Chi Square= 3.163

Degree of Freedom= 8

Significance- 0.923

The mode of management has no influence over maternal outcome

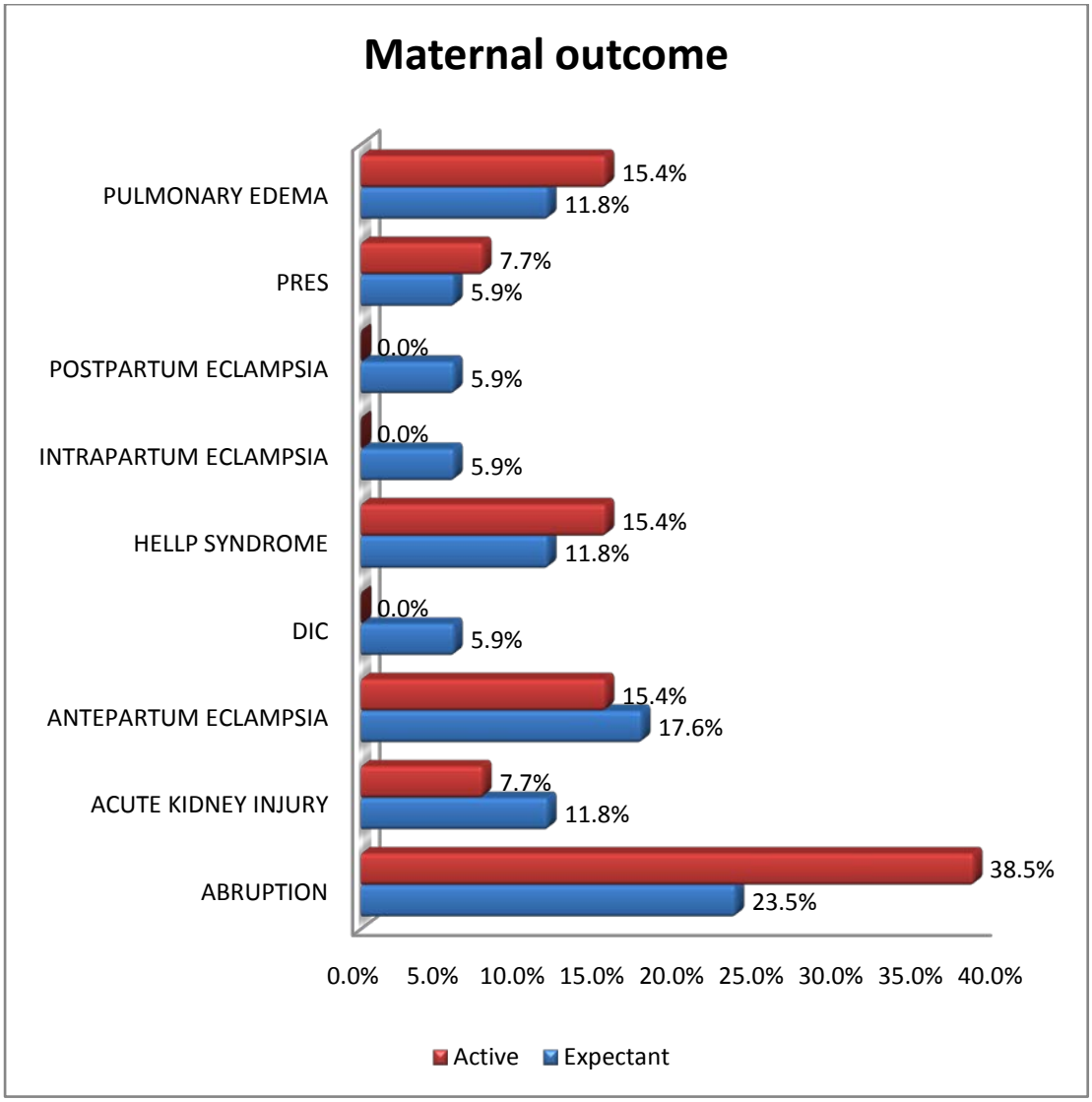


Table 11: FETAL OUTCOMES

Outcome	expectant		Active		**total**
	no	%	no	%	
Total birth	50		50		**100**
Live	45	90%	44	88%	**89**
Stillbirth	2	4%	4	8%	**6**
IUD	3	6%	2	4%	**5**
PND	5	10%	13	26%	**18**

Perinatal loss= 10% in expectant and 26% in active

Total stillborn=6

Total PND= 18

Chi square	4.336
Degree of freedom	1
Significance	0.037

Expectant management has a significant effect on the Fetal outcome with p=0.037

Babies born of expectant management have better survival than those managed actively

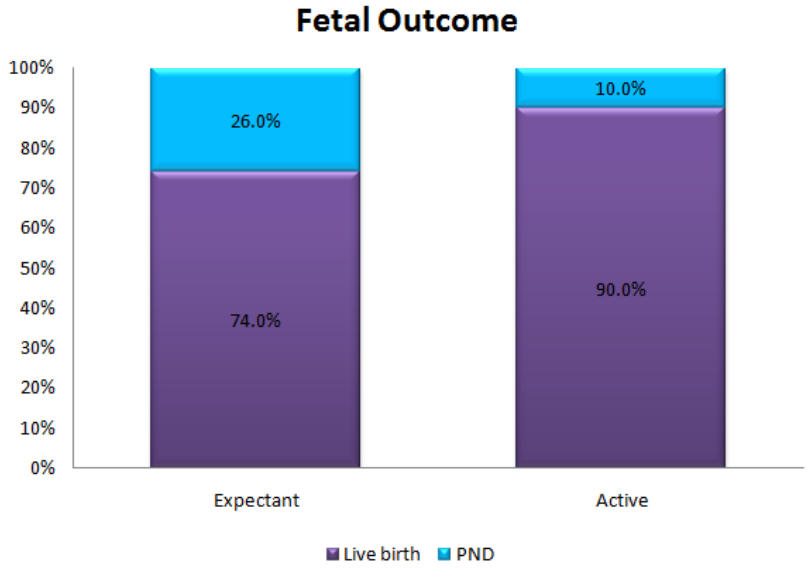


Table 12: NICU Admission:

NICU admission	Expectant (n-50)		Active (n-50)		Total
	No.	%	No.	%	
Yes	33	66%	40	80%	73
No	17	34%	10	20%	27

Chi square= 2.486

Degree of feedom = 1

P=0.115

80% of babies born to actively managed mothers required NICU admission

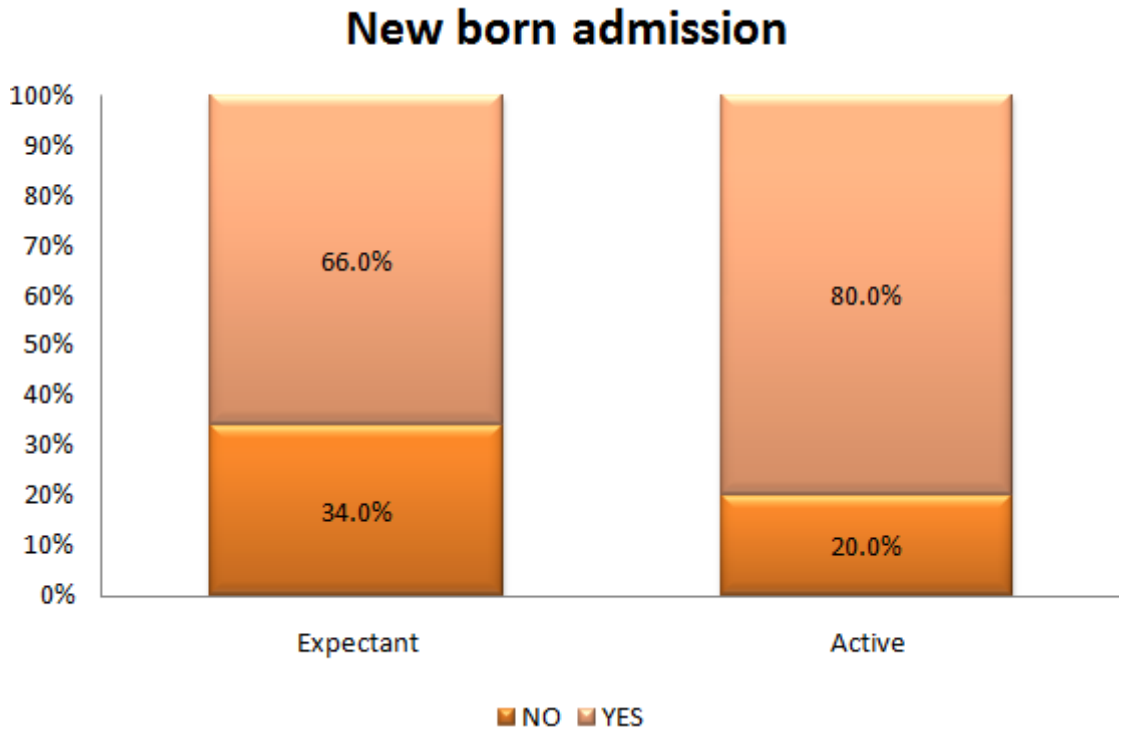


TABLE 13: BIRTH WEIGHT

	EXPECTANT (n-50)		ACTIVE (n-50)		TOTAL
Kg	No.	%	N0.	%	
1-1.5	9	18%	18	36%	27
1.5-2	24	48%	23	46%	47
2-2.5	17	34%	9	18%	26

Max. birth weight = 2.4 kg

Mean BW= ACTIVE- 1.66 KG

EXPECTANT – 1.89 KG

Chi square=5.483

Degree of freedom=2

Significance=0.064

Babies delivered by expectant management have higher mean birth weight (1.89kg) than those actively delivered (1.66kg)

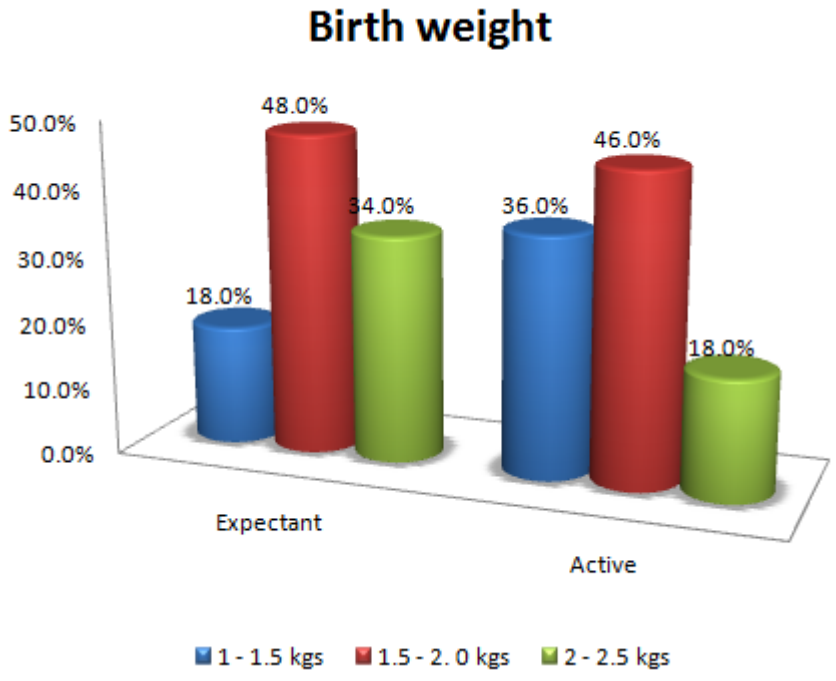


TABLE 14: NO OF DAYS GAINED IN EXPECTANT MANAGEMENT

Days	Cases (n-50)	%
<5	22	44%
5-8	13	26%
9-12	12	24%
13-20	2	4%
>20	1	2%

Max no of days gained = 24

Mean= 6.8 days

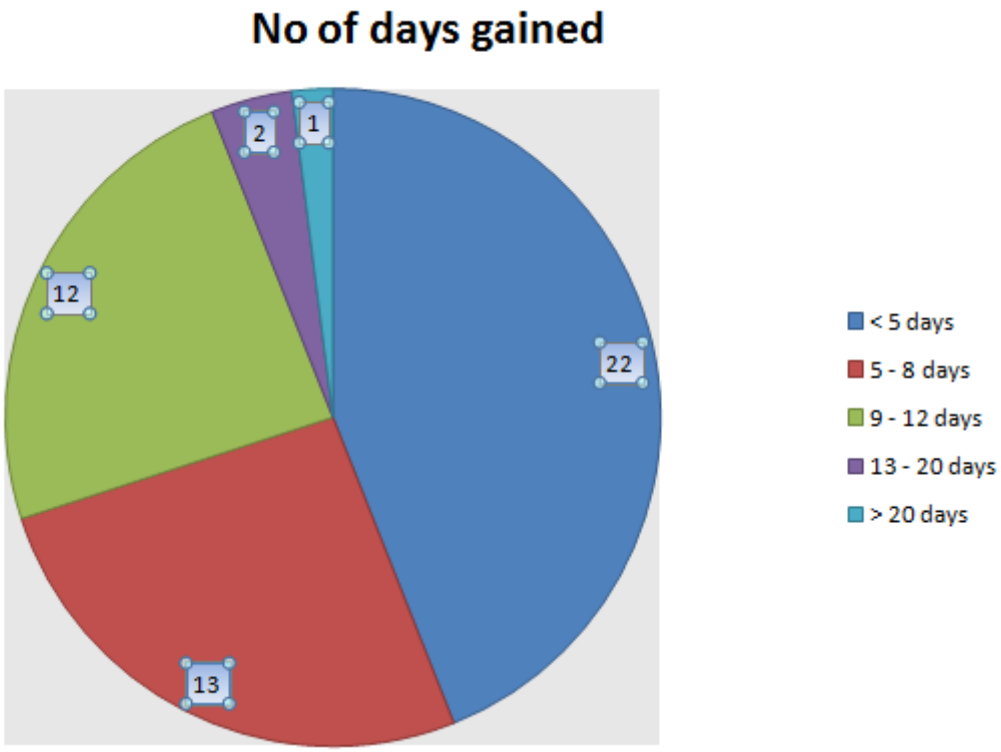


TABLE 16: APGAR SCORE AT 5 MIN

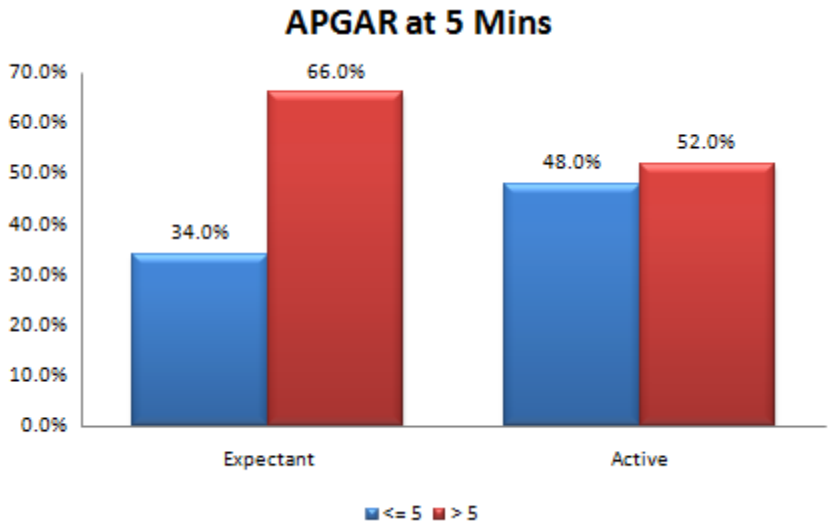
	EXPECTANT (n-50)		ACTIVE (n-50)		TOTAL
APGAR	No.	%	No.	%	
≤ 5	17	34%	24	48%	41
>5	33	66%	26	52%	59

Active and expectant management did not have much impact on the contribution to the APGAR score

Chi square = 2.06

Degree of freedom = 1

P= 0.155



The collected data were analysed with IBM.SPSS statistics software 23.0 Version.To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To find the significant difference between the bivariate samples in Independent

groups the Unpaired sample t-test was used.To find the significance in categorical data Chi-Square test was used. In both the above statistical tool the probability value .05 is considered as significant level.

DISCUSSION

Age:

54% of the women in the study were of ages between 21 and 30 years with mean age of 26 years. This result correlates with the studies of Moodleyand studies done by Brown MA and Buddle ML, D.R.Hall (mean age was 26 years).

Parity:

Preeclampsia is common in primi. 62% of our study group was primi. Brown MA and Buddle ML said preeclampsia is predominant in nulliparous.

MATERNAL OUTCOMES:

In our study there was no maternal mortality. Maternal morbidity was seen in 30%. abuption was the highest accounting to 9 patients, followed by eclampsia in 7. Other complications were HELLP, pulmonary edema, PRES, DIC and acute kidney injury

Study	Abruption	Pulmonary edema	HELLP	eclampsia	Renal failure
DR Hall [25]	20%	2%	5%	1.2%	0.3%
Murphy DJ [23]	1.5%	-	21%	1.4%	1.3%
Vissur and Wallenberg [22]	5%	-	-	2%	-
Our study	9%	4%	4%	7%	3%

TERMINATION

Indication of Termination is distributed as follows

Study	Maternal indication	Fetal indication
Blackwell Sc[24]	80%	20%
Hall DR, Odendaal	55%	45%
Our study	58%	42%

Maternal indication was the most common cause for termination of pregnancy in 58% and the fetal cause in 42%.

Mode of delivery

Delivery is the ultimate cure for preeclampsia. LSCS was done in 35 women and vaginal in 65 women in our study. Mode of delivery had no influence over fetal outcome

Study	Vaginal	caesarean
Hall et al	18.5%	81.5%
Murphy DJ	20%	80%
Nasser et al[26]	48.3%	51.7%
Railton and Allen[27]	25%	75%
Our study	65%	35%

PERINATAL OUTCOME

Prolongation of pregnancy

Study	Mean prolongation
Odendall et al	7.1 days
Vissur w, wallberg	14 days
Railton and allen	11.2 days
Olah KS	9.5 days
Murphy DJ	14 days
Our study	6.8 days

Our study correlates with Odendaal et al.

mean no of days prolonged is 6.8 days with range from 1 to 24 days

Perinatal Mortality

In expectantly managed group

Study	% loss
Railton and allen	24.5%
Hall Dr	24%
Murphy DJ	30%
Odendaal and pattison	22.3%
Our study	10%

In actively managed group perinatal mortality was 13 (26%). Overall perinatal mortality was 29%

Birth weight

Study	Active	expectant
Sibai et al[28]	1.2kg	1.62 kg
Our study	1.6 kg	1.8 kg

Survival rate

Study	Active	expectant
Sibai et al	24%	65%
HALL DR	70%	94%
Our study	70%	84%

In Our study there was higher survival rate in babies born of expectantly managed

Neonatal Hospitalisation

Study	Active	expectant
Olah KS	64.3%	28.6%
Sibai et al	100%	76%
Our study	80%	66%

Babies born from mothers in expectant management have higher survival rate and required lesser hospitalization than those from actively managed cases.

SUMMARY

- Total no of patients = 100
- Actively managed = 50
- Expectantly managed = 50
- Mostly are in ages between 21-30 years
- Mostly primi (62%)
- Mean gestational age = 31 weeks
- There was no maternal death and 7 cases of eclampsia (5 in expectant & 2 in active)
- Most women in active management delivered by vaginal delivery(65%)
- Termination of pregnancy was due to maternal indication in 58% and fetal in 42%
- 29% perinatal death was recorded
- Mean birthweight was 1.6kg in active and 1.8 kg in expectant group
- Maximum birth weight was 2.4kg
- Maximum prolongation was 24 days

CONCLUSION

Severe preeclampsia is associated with significant maternal and fetal complications. Decision regarding pregnancy termination is to be taken on the grounds of both maternal and fetal factor. The expectant management of severe preclampsia results in a good fetal outcome for

1. Higher birth weight

2. Lesser neonatal complications

But this must be weighed against the risk of maternal morbidity.Hence they should be carried out only in tertiary care centres where there is experienced obstetrician and neonatologist are available

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PROFORMA

Name :
Age:
Gravida:
ht :
weight:

IP No:
Occupation:
LMP:
EDD:
Blood group:
Booked / Not:

Complaints:

- Present H/o:
- Period Of Amenorrhea
- Edema feet
- Headache
- Oliguria
- Pain abdomen
- Vomiting
- Blurring of vision
- Palpitation

Past H/o:

- H/o Preeclampsia in previous pregnancy

Menstrual h/o

Marital H/o:

Medical / Surgical H/o:

- H/o Epilepsy, Head injury, Neurologicaldisorder.

Family H/o: H/o PE in Mother/Sister

Personal H/o:

General Examination:

- Temp
- CVS
- PR
- RS
- BP
- Cns
- Anaemia
- Pupils
- Edema
- P/A
- P/V

Investigations:

CHG:

- Hb
- Blood Urea
- Blood glucose
- PCV
- TC
- Fibrinogen
- Platelets
- Uric Acid
- Electrolytes
- Creatinine

Urine :

- Alb
- Deposits
- Sugar

LFT:

USG:

ECG:

Gravidogram: Date urine alb wt. SFH AC BP . Imminent symptoms

1. Mode of induction

2. Indication of termination : Maternal / Fetal

3. Vaginal delivery / LSCS

Indication of LSCS:

4. Latency Interval

5. Baby alive/dead

- Term/preterm
- Cried/not
- Birth weight
- Distress/Not
- Admitted / not

6. Intrapartum / Postpartum complications

7. Follow up:

- NICU stay
- Neonatal complications
- Discharged alive / not.

EXPECTANT MANAGEMENT

NAME	AGE	GRAVIDA	BOOKED/ UNBOOKED	GAESTATIONAL AGE	STEROIDS GIVEN	NO.OF DAYS GAINED	GA AT DELIVERY	REASON FOR TERMINATION	INDICATION FOR TERMINATION	MODE OF DELIVERY	MATERNAL OUTCOME	BIRTH WEIGHT	APGAR AT 5 MIN	NEW BORN ADMISSION	FETAL OUTCOME
LAKSHMI	20	PRIMI	BOOKED	30W2	YES	4	30W6D	MATERNAL	ABRUPTION	LSCS	ABRUPTION	1.2	4	YES	PND
NANDHINI	32	MULTI	BOOKED	31W	YES	13	32W6D	MATERNAL	IMMINENT ECLAMPSIA	VD	NORMAL	1.5	7	YES	NORMAL
REKA	21	PRIMI	BOOKED	31W2D	YES	9	32W2D	FETAL	IUGR	VD	NORMAL	1.6	6	YES	NORMAL
PRIYA	24	PRIMI	BOOKED	30W2	YES	1	30W1D	MATERNAL	ABRUPTION	LSCS	ABRUPTION	1.3	7	YES	NORMAL
OVIYA	18	MULTI	BOOKED	32W	YES	10	32W3D	MATERNAL	ECLAMPSIA	LSCS	M ECLAMPSIA	2	4	YES	PND
JOTHI	33	MULTI	BOOKED	31W	YES	7	32W	FETAL	ABNORMAL CTG	LSCS	NORMAL	2.1	6	YES	NORMAL
DEEPA	25	PRIMI	BOOKED	33W	YES	2	33W2D	FETAL	OLIGOHYDRAMNIOS	LSCS	NORMAL	2.2	7	NO	NORMAL
MANJU	16	PRIMI	BOOKED	31W5D	YES	5	32W4D	MATERNAL	IMMINENT ECLAMPSIA	VD	NORMAL	2	8	YES	NORMAL
AMUTHA	20	MULTI	BOOKED	33W	YES	1	33W1D	MATERNAL	ABNORMAL PLATELET COUNT	VD	DIC	1.9	8	YES	NORMAL
KAMALI	28	MULTI	BOOKED	30W	YES	22	33W1D	FETAL	IUGR	VD	NORMAL	1.5	6	YES	NORMAL
POONGODI	21	PRIMI	BOOKED	32W	YES	7	33W	MATERNAL	IMMINENT ECLAMPSIA	LSCS	POSTPARTUM ECLAMPSIA	1.9	8	YES	NORMAL
SELVI	18	PRIMI	BOOKED	31W2D	YES	3	31W5D	MATERNAL	FUNDUS CHANGES	VD	NORMAL	1.7	7	YES	NORMAL

NAME	AGE	GRAVIDA	BOOKED/ UNBOOKED	GAESTATIONAL AGE	STEROIDS GIVEN	NO.OF DAYS GAINED	GA AT DELIVERY	REASON FOR TERMINATION	INDICATION FOR TERMINATION	MODE OF DELIVERY	MATERNAL OUTCOME	BIRTH WEIGHT	APGAR AT 5 MIN	NEW BORN ADMISSION	FETAL OUTCOME
SATHYA	29	MULTI	BOOKED	31W2D	YES	2	31W4D	FETAL	IUGR	VD	NORMAL	1.5	0	NO	STILL BORN
NITHYA	22	PRIMI	BOOKED	32W4D	YES	9	33W6D	FETAL	OLIGOHYDRAMNIOS	LSCS	NORMAL	2.2	6	NO	NORMAL
SRI	24	PRIMI	BOOKED	33W	YES	4	33W4D	FETAL	ABNORMAL CTG	LSCS	NORMAL	2.2	6	NO	NORMAL
SEVVANTHI	24	MULTI	BOOKED	33W	YES	2	33W2D	MATERNAL	COAGULATION PROFILE	VD	HELLP SYNDROME	2.1	7	NO	NORMAL
REVATHI	26	MULTI	BOOKED	31W2D	YES	5	32W	FETAL	IUGR	LSCS	NORMAL	1.8	6	YES	NORMAL
SASI	28	MULTI	BOOKED	32W	YES	3	32W3D	MATERNAL	IMMINENT ECLAMPSIA	VD	NORMAL	2	8	YES	NORMAL
BUVANA	35	PRIMI	BOOKED	30W2D	YES	12	32W	MATERNAL	ABRUPTION	LSCS	ABRUPTION	2.1	6	NO	NORMAL
BARATHI	20	PRIMI	BOOKED	33W	YES	1	33W1D	FETAL	IUD	LSCS	NORMAL	2.1	0	NO	IUD
GOKILA	22	MULTI	BOOKED	30W1D	YES	18	32W5D	MATERNAL	IMMINENT ECLAMPSIA	VD	NORMAL	2	8	YES	NORMAL
MANI	36	PRIMI	BOOKED	32W2D	YES	2	32W4D	MATERNAL	PULMONARY EDEMA	LSCS	PULMONARY EDEMA	2	7	YES	NORMAL
ABIRAMI	24	MULTI	BOOKED	31W	YES	9	32W2D	MATERNAL	FUNDAL CHANGES	VD	NORMAL	1.9	5	YES	NORMAL
ANITHA	31	PRIMI	BOOKED	33W	YES	5	33W5D	FETAL	IUGR	LSCS	NORMAL	1.9	5	YES	NORMAL
PADMA	26	PRIMI	BOOKED	31W2D	YES	12	33W1D	MATERNAL	IMMINENT ECLAMPSIA	VD	M ECLAMPSIA	2.2	4	NO	NORMAL
SINDHU	30	PRIMI	BOOKED	31W4D	YES	1	31W5D	MATERNAL	ACUTE KIDNEY INJURY	LSCS	KIDNEY INJURY	1.9	7	YES	NORMAL

NAME	AGE	GRAVIDA	BOOKED/ UNBOOKED	GAESTATIONAL AGE	STEROIDS GIVEN	NO.OF DAYS GAINED	GA AT DELIVERY	REASON FOR TERMINATION	INDICATION FOR TERMINATION	MODE OF DELIVERY	MATERNAL OUTCOME	BIRTH WEIGHT	APGAR AT 5 MIN	NEW BORN ADMISSION	FETAL OUTCOME
SRIDIVYA	18	MULTI	BOOKED	32W1D	YES	6	33W	MATERNAL	UNCONTROLLED BP	VD	HELLP SYNDROME	2.2	8	NO	NORMAL
NANDHINI	36	PRIMI	BOOKED	32W	YES	7	33W	FETAL	OLIGOHYDRAMNIOS	VD	NORMAL	2	8	YES	NORMAL
DEVI	29	PRIMI	BOOKED	32W	YES	5	32W5D	FETAL	OLIGOHYDRAMNIOS	VD	NORMAL	2.1	4	NO	PND
KANAG	32	MULTI	BOOKED	30W	YES	9	31W2D	MATERNAL	IMMINENT ECLAMPSIA	VD	NORMAL	1.8	7	YES	NORMAL
RAMYA	22	PRIMI	BOOKED	30W2D	YES	12	32W	FETAL	OLIGOHYDRAMNIOS	VD	NORMAL	1.5	0	YES	IUD
VIDYA	18	PRIMI	BOOKED	32W	YES	6	32W6D	MATERNAL	UNCONTROLLED BP	LSCS	PRES	1.9	0	YES	NORMAL
KALA	19	MULTI	BOOKED	33W1D	YES	2	33W3D	FETAL	OLIGOHYDRAMNIOS	VD	NORMAL	2.5	8	YES	NORMAL
ALMELU	21	PRIMI	BOOKED	30W	YES	7	31W	FETAL	OLIGOHYDRAMNIOS	VD	NORMAL	1.6	6	NO	NORMAL
GETHA	35	PRIMI	BOOKED	31W	YES	5	31W5D	FETAL	OLIGOHYDRAMNIOS	VD	NORMAL	1.9	2	YES	NORMAL
REVATHY	30	PRIMI	BOOKED	31W	YES	10	33W3D	MATERNAL	UNCONTROLLED BP	VD	PULMONARY EDEMA	2	6	YES	NORMAL
MAHA	36	MULTI	BOOKED	33W	YES	6	33W6D	MATERNAL	IMMINENT ECLAMPSIA	VD	NORMAL	2.2	7	NO	NORMAL
VENI	22	PRIMI	BOOKED	31W3D	YES	11	34W	FETAL	ABNORMAL CTG	LSCS	NORMAL	2.5	1	YES	PND
TAMILSELVI	33	MULTI	BOOKED	30W	YES	1	30W1D	FETAL	ACUTE KIDNEY INJURY	LSCS	KIDNEY INJURY	1.3	3	YES	NORMAL
MLLIGA	24	PRIMI	BOOKED	30W3D	YES	12	32W1D	MATERNAL	IMMINENT ECLAMPSIA	LSCS	NORMAL	1	8	YES	NORMAL

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PAVITHRA	31	PRIMI	BOOKED	30W2D	YES	7	31W2D	FETAL	OLIGOHYDRAMNIOS	VD	NORMAL	1.5	2	YES	PND
KRIPA	21	PRIMI	BOOKED	32W1D	YES	5	32W5D	MATERNAL	UNCONTROLLED BP	VD	NORMAL	1.6	8	YES	NORMAL
VIJAYA	30	MULTI	BOOKED	31W	YES	7	32W	MATERNAL	UNCONTROLLED BP	VD	M ECLAMPSIA	1.75	7	YES	NORMAL
DEEPA	21	PRIMI	BOOKED	32W2D	YES	8	33W3D	FETAL	ABNORMAL CTG	LSCS	NORMAL	2.1	7	YES	NORMAL
JOTHI	30	PRIMI	BOOKED	30W1D	YES	12	32W6D	FETAL	OLIGOHYDRAMNIOS	VD	NORMAL	2	0	NO	STILL BORN
JESI	22	MULTI	BOOKED	32W1D	YES	5	32W6D	MATERNAL	IMMINENT ECLAMPSIA	VD	NORMAL	2.2	7	NO	NORMAL
SUMATHI	30	PRIMI	BOOKED	31W	YES	7	32W	MATERNAL	UNCONTROLLED BP	VD	NORMAL	2	6	NO	NORMAL
AYESHA	23	MULTI	BOOKED	33W	YES	5	33W5D	MATERNAL	ABRUPTION	VD	ABRUPTION	2.4	0	NO	IUD
VENNILA	34	PRIMI	BOOKED	31W5D	YES	8	32W6D	MATERNAL	UNCONTROLLED BP	VD	NORMAL	2	0	YES	NORMAL
POOJA	24	MULTI	BOOKED	31W	YES	7	32W	MATERNAL	UNCONTROLLED BP	VD	M ECLAMPSIA	2.1	7	NO	NORMAL

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ACTIVE MANAGEMENT															
ANU	21	PRIMI	BOOKED	30W	YES		30W2D	MATERNAL	_	VD	NORMAL	1	4	YES	PND
SOUNDARYA	21	PRIMI	BOOKED	31W3D	NO		31W3D	MATERNAL	ABRUPTION	VD	ABRUPTION	1.2	0	NO	STILL BORN
RAMYA	31	PRIMI	UNBOOKED	31W	YES		31W2D	MATERNAL	_	VD	NORMAL	1.2	4	YES	NORMAL
VEENA	22	MULTI	BOOKED	31W3D	YES		31W5D	MATERNAL	_	VD	NORMAL	1.2	4	YES	NORMAL
SUJATAH	23	PRIMI	BOOKED	32W	YES		32W1D	MATERNAL	_	VD	NORMAL	1.8	4	YES	NORMAL
ARYA	17	PRIMI	BOOKED	32W2D	NO		32W2D	MATERNAL	ABRUPTION	LSCS	ABRUPTION	1.8	6	NO	PND
ANJALI	20	PRIMI	BOOKED	32W	YES		32W	MATERNAL	_	VD	NORMAL	1.7	6	YES	NORMAL
RAJI	32	MULTI	BOOKED	31W5D	YES		31W6D	MATERNAL	_	VD	NORMAL	1.8	6	YES	NORMAL
ARUNA	24	PRIMI	BOOKED	30W2D	YES		30W4D	FETAL	OLIGOHYDRAMNIOS	VD	NORMAL	1.1	3	YES	PND
ARCHANA	25	MULTI	BOOKED	33W4D	YES		33W6D	MATERNAL	_	VD	NORMAL	2	7	YES	NORMAL
VIDHYA	32	PRIMI	BOOKED	31W2D	NO		31W2D	MATERNAL	HELLP SYNDROME	VD	HELLP SYNDROME	1.3	3	YES	NORMAL
SAROJA	26	MULTI	BOOKED	33W5D	YES		34W	MATERNAL	_	VD	NORMAL	2.1	6	YES	NORMAL
MANISHA	33	PRIMI	UNBOOKED	31W5D	YES		32W	MATERNAL	_	VD	NORMAL	1.8	6	YES	NORMAL

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SOUMIYA	27	MULTI	BOOKED	30W3D	YES		30W5D	FETAL	OLIGOHYDRAMNIOS	VD	NORMAL	1.3	0	NO	STILL BORN
SURYA	34	PRIMI	BOOKED	3W5D	YES		32W	MATERNAL	_	VD	NORMAL	1.7	6	YES	NORMAL
DEEPIKA	28	MULTI	BOOKED	33W2D	NO		33W2D	MATERNAL	ANTEPARTUM ECLAMPSIA	LSCS	M ECLAMPSIA	2.1	6	YES	NORMAL
ANBU	20	PRIMI	BOOKED	31W2D	YES		31W3D	MATERNAL	_	VD	NORMAL	1.8	0	NO	STILL BORN
TAMILSELVI	22	MULTI	BOOKED	31W5D	YES		32W	MATERNAL	_	VD	NORMAL	1.9	8	YES	NORMAL
SELVI	16	PRIMI	BOOKED	32W2D	NO		32W2D	MATERNAL	ACUTE KIDNEY INJURY	LSCS	KIDNEY INJURY	2	8	YES	NORMAL
THIRU	36	PRIMI	BOOKED	30WD	NO		30W3D	MATERNAL	ABRUPTION	LSCS	ABRUPTION	1.1	3	NO	PND
ABIRAMI	24	PRIMI	BOOKED	32W	YES		32W2D	MATERNAL	_	LSCS	NORMAL	1.8	6	YES	NORMAL
APSARA	37	PRIMI	BOOKED	33W	YES		33W1D	MATERNAL	_	VD	NORMAL	2	6	YES	NORMAL
RAMELA	26	PRIMI	BOOKED	30W	YES		30W2D	FETAL	ABNORMAL DOPPLER	VD	NORMAL	1	2	NO	PND
SINDU	33	MULTI	BOOKED	32W1D	YES		32W3D	MATERNAL	_	LSCS	NORMAL	1.8	6	YES	NORMAL
NITHYA	28	PRIMI	BOOKED	30W5D	NO		31W	MATERNAL	PULMONARY EDEMA	LSCS	PULMONARY EDEMA	1.2	0	NO	STILL BORN
KAVITHA	31	PRIMI	BOOKED	31W3D	YES		31W5D	MATERNAL	_	VD	NORMAL	1.8	6	YES	NORMAL
SUGANYA	22	PRIMI	BOOKED	33W2D	YES		33W4D	MATERNAL	_	VD	NORMAL	2.1	6	YES	NORMAL

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AGALYA	21	PRIMI	BOOKED	30W2D	NO		30W2D	FETAL	OLIGOHYDRAMNIOS	VD	NORMAL	1.1	3	YES	PND
VIJAYA	17	MULTI	BOOKED	30W5D	YES		31W	MATERNAL	_	LSCS	NORMAL	1.3	4	YES	PND
PAPPATHI	30	PRIMI	BOOKED	31W6D	NO		31W6D	MATERNAL	ANTEPARTUM ECLAMPSIA	LSCS	M ECLAMPSIA	1.5	6	YES	NORMAL
VELLACHI	31	MULTI	BOOKED	35W5D	YES		34W	MATERNAL	_	VD	NORMAL	2.4	8	YES	NORMAL
THANGAM	21	PRIMI	BOOKED	31W6D	YES		32W1D	MATERNAL	-	VD	NORMAL	2	8	YES	NORMAL
ARUNA	31	MULTI	BOOKED	32W2D	NO		32W2D	MATERNAL	PULMONARY EDEMA	LSCS	PULMONARY EDEMA	1	4	YES	PND
ARIVU	22	PRIMI	BOOKED	30W6D	YES		31W1D	MATERNAL	_	VD	NORMAL	1.8	6	YES	NORMAL
NIVIRA	31	MULTI	BOOKED	32W1D	YES		32W3D	MATERNAL	_	VD	NORMAL	2	6	YES	NORMAL
SALMA	22	PRIMI	BOOKED	31W	NO		31W	FETAL	ABNORMAL DOPPLER	LSCS	NORMAL	1.3	4	YES	PND
KUMARI	30	MULTI	BOOKED	33W2D	YES		33W4D	MATERNAL	_	VD	NORMAL	2.1	8	NO	NORMAL
GEETHA	23	PRIMI	BOOKED	31W3D	YES		31W5D	MATERNAL	_	VD	NORMAL	1.9	6	YES	NORMAL
PREMA	31	MULTI	BOOKED	31W3D	NO		31W3D	MATERNAL	HELLP SYNDROME	LSCS	HELLP SYNDROME	1.7	4	YES	NORMAL
SUGNYA	22	PRIMI	BOOKED	31W5D	YES		32W	MATERNAL	_	VD	NORMAL	1.8	6	YES	NORMAL
SARMAN	32	MULTI	BOOKED	33W	NO		33W	FETAL	IUD	VD	NORMAL	2.1	0	NO	IUD

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FATHMA	21	PRIMI	BOOKED	30W3D	NO		30W3D	MATERNAL	ABRUPTION	LSCS	ABRUPTION	1.1	3	YES	PND
PARVATHY	20	PRIMI	BOOKED	31W6D	NO		31W6D	MATERNAL	UNCONTROLLED BP	LSCS	PRES	1.6	4	YES	NORMAL
SUJITHA	20	PRIMI	BOOKED	31W3D	YES		33W	MATERNAL	_	VD	NORMAL	2.1	4	YES	PND
THARANI	31	MULTI	BOOKED	33W	YES		33W2D	MATERNAL	_	VD	NORMAL	2.1	6	YES	NORMAL
NITHYA	21	PRIMI	BOOKED	32W3D	NO		32W5D	MATERNAL	ABRUPTION	LSCS	ABRUPTION	1.8	0	NO	IUD
SELVI	33	MULTI	BOOKED	30W5D	NO		30W5D	FETAL	FETAL DISTRESS	LSCS	NORMAL	1.2	4	YES	PND
MANI	22	PRIMI	BOOKED	31W5D	YES		32W1D	MATERNAL	_	VD	NORMAL	2	8	YES	NORMAL
RAJI	34	MULTI	BOOKED	33W2D	YES		33W4D	MATERNAL	_	VD	NORMAL	2.1	8	YES	NORMAL
SUGANYA	31	PRIMI	BOOKED	31W3D	YES		31W5D	MATERNAL	_	VD	NORMAL	1.4	3	YES	PND