AGGRESSIVE VERSUS EXPECTANT MANAGEMENT OF SEVERE PREECLAMPSIA REMOTE FROM TERM 28-34 WEEKS.

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CONTENTS

INTRODUCTION

Preeclampsia is a multisystem disorder characterised by raised blood pressure and proteinuria. It complicates 5-8% of pregnancy and is a major cause of maternal and perinatal morbidity and mortality.

(ACOG 2002[2], Sibai et al 1997 (39)

Traditionally approach of balancing the interests of the mother with those of the fetus has been adopted in the management of preterm pregnancies with mild preeclampsia. Severe preeclampsia conversely has been delivered without delay regardless of fetal consideration.

With improved methods of monitoring maternal and fetal well being, several investigators begun to challenge the traditional view that women with severe preeclampsia need be delivered expeditiously.

Recent approach advocates conservative management in a selected group of women with severe preeclampsia remote from term with the aim of improving perinatal outcome without compromising maternal safety.

REVIEW OF LITERATURE

MINIMUM CRITERIA OF DEFINING PREECLAMPSIA

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

1) New onset hypertension (defined according to the latest American college of obstetricians and gynecology bulletin simply as a blood pressure consistently more than 140/90 mm of Hg in previously normotensive women).

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.

The diagnosis of PREECLAMPSIA should be made only after 20 weeks gestation

.In the past it has been recommended that an increment of 30 mmHg systole or 15 mmHg diastolic blood pressure be used as a diagnostic criterion, even when absolute values were below 140/90 mmHg. This criterion is no longer recommended because evidence shows that women in this group are not likely to

suffer **(Levine 2000, North & Colleagues 1999.(**24**))** but warrant close observation.

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

Nowadays it has been recommended that kortkoff phase V be used as a measure of diastolic pressure. **(Brown et al 1998).**

1. K4/K5 difference is smaller in hypertensive than in normotensive pregnant women.
2. K5 is closer to the actual intraarterial pressure, physiologically more accurate, is more reliably detected and is reproducible.
3. K4 has limited reproducibility **(shennam et al 1996)**

PREECLAMPSIA is classified as either “mild” or “severe”. There is no category of moderate Preeclampsia.

SEVERE PREECLAMPSIA (ACOG)

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

1) **Symptoms of central nervous system dysfunction, (blurred vision, scotomata, altered mental status, severe headache.**
2) **Eclampsia (seizures and /or unexplained coma)**
3) **Symptoms of liver capsule distention (right upper quadrant or epigastric pain)**
4) **Severe elevations of blood pressure ≥ 160/110 mmHg on 2 occasions at least 6hours apart.**
5) **Proteinuria (>5g/24h)**
6) **Oliguria or renal failure**
7) **Pulmonary edema**
8) **Cerebrovascular accident**
9) **Hepatocellular injury (serum transaminase levels > 2times normal)**
10) **Thrombocytopenia (<100000 platelets /mm^3)**
11) **Coagulopathy**

12) HELLP (hemolysis,elevated liver enzymes, low platelets).

THEORIES ABOUT CAUSES OF PREECLAMPSIA

1. **Immunological mechanism – BARDEQUEZ (**3**)**

There is immunological resistance to invading trophoblast by maternal immune system. Blocking antibodies and T helper cells, interleukins, interferons, growth factors play a major role. This results in inadequate trophoblast invasion of myometrial spiral arterioles.

2. Genetic predisposition – CHESLEY & COOPER 1986 (6)

Susceptibility is by both single gene and multifactorial inheritance.

a. Women with angiotensin gene variant T235 had increased incidence
b. There is higher incidence of factor V Leiden mutation in preeclamptic patients

3. Increased pressor response to angiotensinogen II – ABDUL KAREEM 1961 (1)

4. Altered vasoactive factors: **VOLHARDT 1918 (**45)

(A) Endothelin- 1. A potent vasoconstrictor produced by endothelium is increased.

(B) NITRIC OXIDE:- A potent vasodilator is decreased.**(Chang et al (**5**)**

(C) Reversal of PGI2 to TXA2 and vit.E ratio **(Wang et al 1991 Walsh 1985(**41**)**

(D) Vasoactive maternal factor (VMF) has been imposed to cause the endothelial changes involved in the patho physiology of PIH.

5. **Oxidants and Antioxidants**

Hubal et al (18**)** have confirmed that Preeclampsia may have its origin in a disturbed oxidation mechanism. Under normal conditions equilibrium is maintained by auto oxidants. With increase in severity of Preeclampsia there is increase in lipid peroxidase and reduction in auto oxidants. Vit E lipid peroxidase cause endothelial damage.

6. Endothelial dysfunction: **HAYMAN & ASS 2000 (**17**)**

Deficiency in trophoblastic invasion of placental blood spiral arteries leads to poorly perfused fetoplacental unit. This results in secretion of plasminogen activator inhibitor, into the maternal circulation leading to activation of endothelial cells to promote coagulation and increased sensitivity to vasopressor agents.

Baker and coll 1995 have shown that VEGF levels are increased in serum of PE which may activate endothelial cells and release of inflammatory substances.

(Dekar & Sibai 1998(8**)**

7. Placental proteins:

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

8. Dietary deficiency: DAWSON, KELLY & Coauthors

Mac Gillivray viewed the evidence for a role of dietary deficiency in pathology of PE. It was concluded that when concentration of calcium is low in extra cellular fluid, amount of ionic calcium entering cell wall increases making vascular smooth muscle more sensitive to excitation.

9. Hyperhomocystinemia: **COLLER ET AL**

Presence of infarcts retroplacentally is said to be cause of elevated levels of circulatory homocysteine. This is due to atherosis formed at placental site. Elevated levels damage endothelium by H2O2 generation depletes nitric oxide mediated detoxification of homocysteine. Elevated levels of factor V increase in prothrombin activation.

PATHOPHYSIOLOGY OF PREECLAMPSIA

PRIMARY LEVEL:

Changes that occur in placenta and placental vascular bed are the two lesions that involve spiral arterioles which are the end arteries supplying intravascular space **(REDEMAN 1991**(32)**)**

a. Relative lack of trophoblastic infiltration of arterial walls during placentation **(Bloen et al 1972)**

b. Acute atherosis **(Robertson et al 1963(33))**

c. Magnitude of defective trophoblast invasion of spiral arterioles correlated with severity of hypertensive disorder.

d. Lipid accumulates first in myometrial cells and then in macrophages (**Madzli & Collegues 2000**(20)**)**

SECONDARY LEVEL

I.Renal system

a) Decreased uric acid clearance, decreased glomerular function, decreased renal blood flow, proteinuria. Intrinsic renal changes caused by severe vasospasm

b) Renal pathology : Proteinuria reflects advanced disease associated with poor prognosis**(Naeye & Friedman 1979)** Glomerular endotheliosis **(Sargo et Al 1959)**

c) Urinary sediments reflects renal changes **(Leduc et Al**(19) **)**

d) Renal clearance of urate decreased **(Chesley & Williams 1945)**

e) Hypocalciuria **(Taufield et Al 1987**(40) **)** because of increased tubular reabsorbtion

f) Hyperuricemia **(Pollok et Al 1960)**

g) Proteinuria **(Meyer & Colleagues 1994)** Trace or negative proteinuria had negative predictive value only 34% in hypertensive women 3+ or 4+ proteinuria were positively predictive of severe preeclampsia in only 36% of cases

II. Cardio vascular system:

a. Hemodynamic changes:

Increased arterial sensitivity to angiotensin II

Increased peripheral resistance

Decreased cardiac output

Increased BP

b. Blood volume

Hemo concentration

Blood volume decreased in women with homozygous for T 235 angiotensin genotype associated with preeclampsia **(Silver & Associates 2001**(35)**)**

III. Coagulation system

a. Intravascular coagulation and less often erythrocyte destruction commonly in PE and especially eclampsia **(Baker & Cunningham 1999)**

b. PT, aPTT, plasma fibrinogen level are unneccessary in management of hypertensive disorder of pregnancy **(Baker & Colleagues 1999)**

c. Thrombin time somewhat prolonged in a third of cases

d. Thrombocytopenia results from platelet activation, consumption and increased platelet production. Platelet aggregation is decreased compared with the normal increase seen in pregnancy **(Baker & Cunningham1999)**

e. Fragmentation hemolysis: **(Sanchel Ramos 1994**(34) **)** Increased RBC fluidity in women with HELLP

f. Antithrombin III lowered **(Chang & Coworkers 1992**(5) **)**

g. Fibronectin elevated **(Bru Baker & Colleagues 1992)**

h. Thrombophilias : Clotting factors deficiencies or mutation associated wuth early onset of preeclampsia

IV. Hepatic system:

a) With severe preeclampsia there is elevation of liver enzymes **(Combes & Adams 1972)**

b) Increased hepatic artery resistance by Doppler sonography **(Ooster Hof & Coworkers**(28) **)**

c) Periportal hemorrhagic necrosis in the periphery of liver **(Barton & Colleagues)**

d) HELLP syndrome : Hemolysis, elevated liver enzymes, low platelet **(Deboer & Coworker 1991**(9)**, Pritchard & Associates 1954**(30)**, Weinstein 1985**(43) **)**

V. Endocrine system

a. Increased plasma levels of renin

b. Angiotensin II and aldosterone are decreased **(Weir & Colleagues)**

c. Arial natriuretic peptide is increased in women with preeclampsia **(Cunningham & Lindheimer 1999**(7)**)**

VI. Fluid & Electrolyte system

a. Volume of extracellular fluid manifest as edema in women with severe preeclampsia has expanded beyond the normally increased volume that characterizes pregnancy
b. Electrolyte concentration do not differ appreciably in women with compared with those of normal pregnancy

TERTIARY LEVEL

Tertiary systemic effects of preeclampsia secondary to decompensation which presents as one of the following features

1. Eclampsia	2. Laryngeal edema
3. Cerebral hemorrhage	4. DIC
5. Corneal edema	6. HELLP syndrome
7. Retinal detachment	8. Renal cortical necrosis

9. Pulmonary edema 10. ARDS

11. Hepatic rupture

PREDISPOSING FACTORS OF PREECLAMPSIA

1. Age :

Young primi < 20 years

All patients > 30 years **Bobrowski & Bottoms 1995**

showed increased incidence of HT & PIH > 35 years

2. Parity :

Primi have incidence of 11.9% and multi have 4.7% **Clinical obs and gyn**.

The incidence is 24% in new paternity multipara because of shorter period of

Sperm exposure preceding conception

3. **Race**

Davis in 1970 found an increased incidence in muslims, jews &arabs Africoamerican ethinicity is quoted to have increased incidence by **Sibai 1997, Walker 2000**

4. **Social status:**

Women of low social economic status are reported to have greater incidence of PIH, PE, Eclampsia. But **Duffer & Mac Gillivray –1968** found that difference between social classes are small if allowance are made for age, parity and levels of antenatal and intrapartum care. **Baird and colleagues** 1969 said that incidence was not different among five socioeconomic status.

5. Previous H/O preeclampsia

a. The risk of preeclampsia in subsequent pregnancies is higher when it is severe earlier and associated with low birth weight

b. Risk increases on increasing maternal age and interval between pregnancies.

Sibai et al studied subsequent pregnancy outcome in women with severe PE in first pregnancy

Risk of developing eclampsia - 1.4%

Preeclampsia - 45.5 %

Abruption - 2.5 %

Perinatal mortality - 5.9%

Norwegian study showed risk of 13.1% of PE in second pregnancy.

6. Family history:

Severe preeclampsia, eclampsia have a familial tendency. There is three fold increase of preeclampsia and four fold increases severe PE.**Chesley** et al(6) found 26% incidence of PE in daughters. **Lie et al** found odds ratio 2.23 in sisters especially full sisters.

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

7. Pregnancy associated: Early onset

a. **Twin gestation**: Four fold increased risk Incidence of PE 25.3% this is due to hyperplacentosis with increased placental hormone secretion relative placental ischemia or immunological reaction to the large placental mass

b. **Molar pregnancy**: Confined to large rapidly growing moles in which the incidence of PE is 70% **(Page 1939**(29)**)** . With small slowly growing moles there is no increased incidence of PE.

c. **Hydrops fetalis** : Increased incidence due to hyperplacentosis.

d. **Congenital malformations**: In pregnancies complicated by triploidy, the risk of developing PE or hypertension in second trimester is 35% due to placentomegaly.

8. Urinary tract infection:

UTI resulting in an increased production of inflammatory products, cytokines, free radical species and proteolytic enzymes causes endothelial dysfunction **(Schieve et al)**

9. Underlying disorders:

a. **Chronic hypertension** is encountered in approximately 30 – 40 % of early onset severe PE .

b.Underlying **renal disorder** : 20% have superimposed PE

c. **Obesity, insulin resistance & diabetes Stone et al – BMI** is an independent risk factor of severe PE

i. Obesity correlated with hypertension by expanded blood volume, Cardiac output to meet the increased metabolic demands.

ii. Obesity- dyslipidemia- delivery of free fatty acids to tissue, higher cholesterol / TG ratio, insulin resistance & hyperinsulinemia.

iii. Adipocytes release TNF alpha thereby involved in aggravating cytokine mediated oxidative stress.

iv. Insulin resistance/ hyperinsulinemia associated with increased sympathetic activity and/or increased tubular sodium reabsorption thereby producing direct hemodynamic changes.

V. Overt DM – 30% PE especially when vascular changes present**. Khan & Daya** showed odds of having PE increased by 20% per nmol/L increase in plasma glucose level.

10. **Smoking:**

Decreases incidence of PE since it causes a significant reduction in HCG and estradiol level due to direct effect on the placental function **(Bernstein et al 1989)**

Management objectives in severe preeclampsia

There is no preventive therapy against preeclampsia available at present even though aspirin, calcium magnesium,fish oil have been tried severe preeclampsia is associated with maternal complications like HELLP, eclampsia, cerebral manifestations, abruption, fetal complications like IUGR, IUD, preterm delivery. As termination of pregnancy remains the only cure, the primary objective in the management of severe preeclampsia is to effect timely delivery in order to

- **Prevent maternal morbidity and mortality**
- **Deliver a baby in an optimal condition, thereby minimizing perinatal morbidity and mortality.**

In all circumstances, the well being of the mother is primary

In some circumstances, delays seriously jeopardize the well being of the mother, fetus or both.

Williams obstetrics says "we are reluctant to advice clinician that is safe to expectantly manage …… such women are not managed expectantly at Parkland hospital".

In some situations, delay of delivery will benefit the mother fetus and both. Such an approach has been advocated by research workers in various parts of the world.

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(20)	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(39)	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**(14)	**340 women 28 -34 weeks**	**------**	**One maternal death 8% major complications**
4.	**Visser et al 1994**(38)	**50 patients 25 to 35 weeks**	**Perinatal mortality 7% in study group 14% in control group**	
5.	**Moodley et al 1993**(21)	**50 patients GA < 32 weeks**	**Perinatal mortality 27% in 30-32 weeks**	**20% maternal complications**
6.	**Visser and wallenburg 1995**(38)	**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**(31)	**56 Women 24 -32 weeks**	**24.5% perinatal mortality 19% small for gestational age**	**23.2 %major complications**

Labour induction

Labour is induced by either misoprostol or cerviprime in both the groups.

Misoprostol is prescribed as a intravaginal dose of 50 mcg at 4 hrs interval which can be repeated for 4 times.

Sahin 2000 has concluded that intravaginal Misoprostol in an equally effective and safe method of induction of labour in patients with preeclampsia and normal pregnant women. Patients went in labour earlier within 12 hours, median time from induction to delivery being 14 hours. Rate of vaginal delivery was significantly higher in the Misoprostol group 82% than that of inducing with oxytocin.

As there was no significant difference between Misoprostol and oxytocin with regards to APGAR scores and NICU admissions we advocated Misoprostol mostly.

AIM OF THE STUDY

1. To compare the merits and demerits of aggressive and expectant management of women with severe Preeclampsia remote from term 28 – 34 weeks.
2. To determine which is more beneficial by comparing perinatal and maternal outcome by statistical analysis by Chi square test

MATERIAL AND METHODS

Study design **:** Prospective

Study period **:** Sep.2003 – Aug 2005

Sample:

Group A: - Patients of severe PE remote from term 28 34 weeks treated aggressively that is glucocorticoid therapy followed by delivery in 48 hrs.

All patients who delivered within 96 hrs of admission were noted.

Group B: - Patients of the same group treated expectantly i.e glucocorticoid therapy, intensive maternal and fetal monitoring followed by delivery only for specific maternal or fetal indication beyond 96hours.

Sample size :

97 patients of group A who delivered after 48hours of glucocorticoid therapy and 92 patients of groupB were compared.

SELECTION CRITERIA

Inclusion Criteria :

All subjects had

1) GA 28 - 34 weeks.

2) Severe pre eclampsia defined as

i) Blood pressure ≥ 160/110 with proteinuria≥ 2+

ii. Blood pressure ≥ 150/100 with proteinuria ≥3+

iii.Blood pressure > 140/90 with proteinuria with c/o Headache or oliguria

Exclusive Criteria :

1. Women with medical complications

2. Rupture of membrane

3. Preterm labour

4. Multifetal gestation

5. Fetal compromise/ fetal death

6. Platelet count < 1, 00,000/ Mic.L or HELLP

7.Eclampsia

8.Fetal congenital mal formation

Babies born without completing steroids are not compared with exp. Group

GUIDELINES FOR EXPECTANT MANAGEMENT

1. All patients are observed in Labour room atleast for 24 hours to determine their eligibility for expectant management.
2. Intravenous magnesium sulphates for seizure prophylaxis for selected patients.Glucocorticoid are given to improve fetal outcome.
3. Antihypertensive for BP control.
4. Complete Blood count, Platelet count, urine protein, serum creatinine, serum uric acid, AST, LDH.
5. Limited oral intake for the first 24 hours and IV fluids at the rate of 100 – 125 ml/ hr within 24 hours.

After 24 hours

MATERNAL	**FETAL**
1. BP measured every 4 to 6 hours	1. We do NST daily
2. Platelet everyday	2. USG for fetal weight

3. SerumAST, creatinineeveryotherday and IUGR weekly

4. Serum Uric Acid biweekly

5. Oral antihypertensive drugs to control BP systole in the range of 130 – 150 mmHg, diastole in the range of 80 – 100 mmHg

6. Daily Weight, Daily Urine albumin, Gravidogram.

7. Retinal changes.

Then later

1. Headache in preeclamptic women are treated with analgesic and bed rest.

2. If there is no resolution within 6 hours, head ache is severe, BP is controlled meticulously and IV magnesium sulphate is started.

3. If the headache persists then decision is made for delivery.

What to expect of expectant management?

At any time during the concerned period of prolonging pregnancy, contraindications to expectant management appear, we terminate the pregnancy may be by vaginally or abdominally.

MATERNAL INDICATIONS

1. Uncontrolled severe BP persistently $\geq$ 160 mmHg systole & $\geq$110 mmHg diastole despite maximum dose of the antihypertensive medication for four

days

2. Eclampsia

3. Platelet count <1, 00,000/ mic ml

4. AST or ALT >2 times of upper limit of normal value with epigatric pain or right upper quadrant tenderness

5 Pulmonary edema

6. Compromised renal functions- rise in serum creatinine of 1 mg/dl over baseline levels.

7. Abruptio placenta

8. Persistent severe head ache or visual changes

Guidelines as adopted as in University of Tennessee, Memphis

FETAL INDICATIONS

If any one or more present like

1. Repetitive late or severe variable decelerations

2. AFI $\leq$2cm

3. USG estimated fetal weight $\leq$ 5 th percentile & reverse umbilical artery diastolic flow.

TERMINATION

a.LABOUR INDUCTION:

1. **MISOPROSTOL**
2. **Cerviprime gel to improve bishops score before induction of labour in the absence of fetal distress. May or maynot need augementation**

b.ABDOMINALLY:

By LSCS

GUIDELINES FOR AGGRESSIVE TREATMENT:

1. All patients are observed in labour room.
2. Intravenous 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 as like that of the expectant management.

5. Ultrasonogram for fetal well being .Then labour is induced either by misoprostol, cerviprime may or may not be augmented with syntocinon infusion

INTRAPARTUM MANAGEMENT

Preeclamptic women are at higher risk of developing convulsions during labour as compared to normotensive women, highest being in severe preeclampsia remote from term, 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, oxytocin augmentation be given.
3. If it is unripe, Caesarean section is to be considered because of higher incidence of complications like abruption, fetal distress.

4. ANALGESIA

    1. Provided by intermittent use of small dose of 25-50 mg of parentral pethidine or segmental epidural analgesia.

    2. Local infitration or epidural in vaginal delivery.

3. Continuous epidural or balanced general anaesthesia used for caesarean patients.

5. Input and output monitoring

   Hourly urine output.

   Fluid restricted to 150 ml / hr.

   If oliguria say <100ml per 4 hrs, fluids & MgSO4 reduced accordingly.

6. Antihypertensive therapy.

   Goal is to maintain systole 140 –150 systole and diastole 90-100 mm Hg. And not to reduce the mean arterial pressure by more than 20% from baseline value.

POSTPARTUM MANAGEMENT

1. Intensive monitoring done for 2-4 days. Vitals, reflexes, input output monitored.
2. BP control

3. Prophylactic anticonvulsives not given.
4. Patient is seen at weekly intervals until her BP is normal without medications.
5. If this change does not occur by 6 weeks work up for hypertension made

OUT COME

Parameters like prolongation of pregnancy, perinatal outcome, and maternal morbidity were evaluated.

DATA ANALYSIS

From September 2003 to august 2005, women with severe preeclampsia were noted. Among 259 patients of severe preeclampsia remote from term 28to 34 weeks ,167 patients got terminated with in 96 hours of admission .

Among them 97 patients who got full dose of steroids were compared with the remaining 92 patients who got terminated after 96 hours of admission. Participants with similar characteristics were assigned.

Maternal age, parity, gestational age, initial systole and diastole, dose of antihypertensives were comparable in both the groups.

Principal measures like

a. **Maternal outcome**
b. **Mode of delivery and their indication**
c. **Perinatal out come were analysed in both the groups**

INCIDENCE IN STUDY PERIOD

Total Number of Deliveries 26,500

Mild Preeclampsia 10%

Severe Preeclampsia 1%

Eclampsia0.04%

TABLE 1

MATERNAL AGE

AGE(YRS)	AGGRESSIVE	EXPECTANT	TOTAL
16-19	10 (10.3%)	5 (5.4%)	15 (7.9%)
20- 24	23 (23.7%)	22 (23.9%)	55 (29.1%)
25- 29	28 (28.8 %)	32 (34.7%)	60 (31.7 %)
30-34	32 (39.1%)	37 (33.6%)	63(33.3%)
35-40	4 (4.1%)	2 (2.1 %)	6 (3.1%)
TOTAL	97	92	189

>60% of patients were around 25-34 years of age

TABLE 2

GRAVIDITY

GRAVIDITY	AGG.(97)	EXP.(92)	TOTAL(189)
PRIMI	60 (61.8%)	62 (67.3%)	122(64. 5%)

SECOND	12 (12.3%)	16 (17.3 %)	28 (14.8%)
THIRD	13 (13.4%)	12 (13 %)	25 (13.2%)
FOURTH	8 (8.2%)	2 (2.17%)	10 (5.2%)
FIFTH	4 (4.1%)	-	4 (2.1%)

Around 64.5% of Patients were primis

TABLE 3

GESTATIONAL AGE

GA – WEEKS	**AGG.(97)**	**EXP.(92)**	**TOTAL(189)**
28-30	34(35.0%)	27(29.3%)	61(32.3%)
31-32	39(42.3%)	39(42.3%)	78(41.3%)
33-34	24(24.7%)	26(28.2%)	50(26.4%)

Mean GA on admission was 31 weeks

TABLE 4

ANALYSIS OF PAST OBSTETRIC HISTORY

PREECLAMPSIA	AGGRESSIVE (37)	EXPECTANT (32)	TOTAL (69)
Past History	15 (40.5%)	10 (31.2%)	25(36.2%)
Mild	10 (27%)	9 (28.1%)	19 (27.5%)
Severe	3 (3%)	1 (3.1%)	4 (5.8%)
Eclampsia	1 (2.7%)	-	1 (0.01%)
Abruption	1 (2.7%)	-	1 (0.01%)
Not known / No Previous history	22	22	44 (63.7%)

36% of patients had recurrent Preeclampsia

TABLE 5

ANALYSIS OF BODY MASS INDEX

BMI	AGG	EXP	TOTAL
<25	20(20.5%)	35(38%)	55(29.1%)
25-30	28(28.9%)	25(27%)	53(28%)
31-35	25(25.7%)	22(24%)	47(24.8%)
36-40	18(18.56%)	8(8.75%)	26(13.75%)
>40	6(6.18%)	2(2.1%)	8(4.2%)

Around 43% of patients had BMI >30

18% of patients had BMI >35

Mean BMI 27.4

TABLE 6

ANALYSIS OF FAMILY HISTORY

F/H/O	Aggressive			Expectant		
	P 50	M37	T 97	P 62	M 30	T 92
Not known /no history	50	29	79 (81.4%)	54	27	81(88%)
Mother PE	4	2	6(6.18%)	2	-	2(2.17%)
Sister PE	6	6	12(12.37%)	6	3	9(9.78%)

4.23% of patients had mother withpreeclampsia

11%of patients had sister with preeclampsia

TABLE 7

ANALYSIS OF BLOOD PRESSURE SYSTOLE

Initialsystole mmHg	Agg.	Exp.	Total
140-149	30(30.9%)	29(31.5%)	59(31.2%)
150-170	48(49.4%)	55(59.7%)	103(54.5%)
>170	19(20.1%)	8(8.69%)	27(14.3%)

Around 54.5% of patients had systolic BP 150-170 mmHg

Mean being 160mmHg

TABLE 8

ANALYSIS OF BLOOD PRESSURE DIASTOLY

Initial Diastole mmHg	AGG.	EXP.	TOTAL
90-100	25(25.8%)	30(32.6%)	55(29.1%)
101-110	37(38.2%)	39(42.4%)	76(40.2%)
111-120	20(20.6%)	16(17.4%)	36(19.0%)
>120	15(15.4%)	7(7.6%)	22(11.7%)

40% of patients had diastole 101-110 mmHg

Mean being 106mmHg

TABLE 9

ANALYSIS OF ANTIHHPERTENSIVE DRUGS

ALPHA METHYL DOPA

Dose(mg)	Agg. %	Exp . %	Total %
500-750	17 (17.5)	15 (16.3)	32 (16.9)
750-1000	25 (25.8)	38 (41.3)	63 (33.3)
1000-1500	45 (46.4)	32 (34.8)	77 (40.7)
1500-2000	10 (10.3)	7 (7.6)	17(9.0)

About 40.7% patients were given 1000-1500 mg of alpha methyl dopa per day

TABLE 10

NIFIDEPINE DOSAGE

Dose (Mg)	Agg (%)	Exp (%)	Total
10 –15	20 (20.6)	25 (27.2)	45 (23.8)
15-20	42(43.4)	40(43.5)	82(43.4)
20-25	15(15.5)	24(26.1)	39(20.6)
25-30	20(20.6)	3(3.2)	23(12.2)

About 43.4% patients were given 15-20 mg per day of nifedipine

TABLE 11

INITITATION OF MAGNESIUM SULPHATE

Intial Diastole	Aggressive(97)	Expectant (92)	Total
>120 (22)	15 (15.4%)	7(7.6%)	22(11.6%)
111-120 (36)	20(20.6%)	16(17.4%)	36(19%)
101-110(76)	27(27.8%)	22(24%)	49(25.9%)
90-100 (55)	10(10.3%)	11(12.9%)	21(11.1%)
Not given	25(25.8%)	36(39.1%)	61(32.2%)

128 patients were given. **Magnesium Sulphate**

All 58 patients whose diastolic BP > 110 got **Magnesium Sulphate**

TABLE 12

RETINAL CHANGES

FUNDUS	AGG.(97)	EXP.(92)	TOTAL(189)
Normal	78(80.4)	84(91.3)	162(85.7)
GR I HTR	16(16.5)	8(8.7)	24(12.7)
GR II HTR	3(3.1)	-	3(1.6)

85.7% them had normal fundus

TABLE 13

MATERNAL OUTCOME

Complication	**Aggressive(97)**				**Expectant(92)**			
	AP	**IP**	**PP**	**T**	**AP**	**IP**	**PP**	**T**
Abruption	2	3	-	5(5.15%)	5	3	-	8(8.7%)
HELLP/DIC	2	-	1	3(3.1%)	1	2	1	4(4.3%)
Eclampsia	_	-	-	-	-	-	-	-
Renal failure(dialysis)	_	-	-	-	-	-	1	1(1.1%)
Pul.edema	-	1	-	1(1%)	1	1	1	3(3.3%)
ARDS	-	-	1	1(1%)	-	-	-	-
Cerebral edema	-	-	-	-	-	-	1	1(1.1)
Maternal death	-	-	-	-	-	-	-	-
Total	4	4	3	10(10.3%)	7	6	4	17(18.4%)

18.4% of major maternal complications occurred in expectant while 10.3% in aggressive. Though slightly higher, tackled well by anesthetist & ICU, indicating institutional supervision of expectant management.

Chi square value = 2.57

Degree of freedom = 1

It was found to be statistically not significant.

TABLE 14

LABOUR INDUCTION

Mode	**Aggressive (97)**			**Expectant(92)**		
	Primi	**Multi**	**Total**	**Primi**	**Multi**	**Total**
Misoprostol(M)	39	25	64(66%)	25	10	35(38%)
Cerviprime(C)	6	3	9(9.28%)	10	2	12(13%)
M/C+synto aug.	10	5	15(15.5%)	11	2	13(14.1%)
Not induced due to mat ,fetal cause	5	4	9(9.3%)	16	16	32(34.8%)

Labour was indued in 52% of our study by misoprostol.

16 multies of expectant group were not induced due to post caesarean pregnancy

TABLE 15

DURATION OF LABOUR

Duration in hrs	**Aggressive(97)**		**Expectant(92)**	
	Primi(55)	**Multi(33)**	**Primi(46)**	**Multi(14)**
4-7	11	9	10	4

8-11	28	20	25	7
12-15	10	3	9	3
16-19	6	1	2	-

Most of them delivered in 8-11 hours after induction

TABLE 16

MODE OF DELIVERY

Mode	**Aggressive(97)**				**Expectant(92)**			
	Primi	**Multi**	**Total**	**PN Loss**	**Primi**	**Multi**	**Total**	**Pnloss**
Vaginal	50(83.3%)	32(86.5%)	82(84.5%)	52	38(61.2%)	10(33.3%)	48(52.17%)	16(33.3%)
LSCS	10(16.7%)	5(13.5%)	15(15.5%)	2	24(38.7%)	20(66.6%)	44(47.82%)	12(27%)
Total	60	37	97	54	62	30	92	28

As salvagability and fetal weight were lower in aggressive group, vaginal delivery preferred.

Increase in LSCS in exp. was due to post caesarean pregnancy in them.

27% of patients delivered by LSCS lost their babies.

Chi square value is 20.16

DF is 1. Significant at 0.001 (p< 0.001)

PN Loss indicates perinatal loss

TABLE 17

INDICATIONS OF LSCS IN EXP.GROUP

Indication	Primi (24)	Multi (20)	Total (44)	Perinatal loss (28)
Post LSCS	-	16 (80%)	16(36.4%)	5(31.2%)
Nonreassuring CTG	5 (20.8%)	2(10%)	7(15.9%)	4(57.1%)
Malpresentation	5 (20.8%)	---	5(11.3%)	1(20%)
Fail to progress /unfavourable Cx	14(58.3%)	2(10%)	16(36.4%)	2(12.5%)

80% of multis were post caesarean pregnancies.

42.8% of perinatal loss occurred after LSCS

INDICATIONS OF LSCS IN AGG. GROUP

Indication	P(10)	M(5)	T(15)	PN loss
Nonreassuring CTG	5	4	9	1

Fail to progress	3	1	4	-
Abruption	2	-	2	1

13.33%of patients lost their babies by LSCS

P –primi, M-multi,T-Total

TABLE 18

INDICATION OF TERMINATION IN EXP. GROUP

MATERNAL INDICATION

Indication	**P (60)**	**M (32)**	**T (92)**	**Pn loss**
Abruption	3	2	5(5.4%)	4
Imminent sym.	28	10	38(41.3%)	8
Uncontrolled BP	5	6	11(12%)	3
Compromised renal function	1	3	4(4.3%)	-
Pul edema	1	-	1(0.01%)	1
Eclampsia	-	-	-	-
Total	39	20	59(64.1%)	16(27.1%)

64.1% of patients terminated by LSCS for maternal indication most common being imminent sym.

27.1% of patients lost their babies.

There was 57.2% of perinatal loss in patients who were terminated for maternal indication

TABLE 19

FETAL INDICATIONS FOR TERMINATION

Fetal cause	P	M	Total	Pnloss
Oligohydramnio	10	6	16(17.3%)	3
Persistent Late deceleration	5	2	7(17.6%)	4
IUGR<5th percentile	6	4	10(10.9%)	5
Total	21	12	33(35.9%)	12(36.3%)

35.9% of patients got terminated due to fetal indication, most common among being oligohydramnios.

Among them 36.3% of pts lost their babies.

42.8% of perinatal death occurred in patience who were terminated for fetal indications.

P = primi

M = multi

Pn loss = perinatal loss

TABLE 20

FETAL OUTCOME

Born	**Aggressive**			**Expectant**		
	P	**M**	**T**	**P**	**M**	**T**
Totalbirth	60	30	97	62	30	92
Live born	46	31	77(79.3%)	58	28	86(93.5%)
Still birth	14	6	20(20.6%)	4	2	6(6.5%)
Neonatal D	24	10	34(35.1%)	14	8	22(24%)
Perinatal D	38	16	54(55.7%)	18	10	28(30.4%)

Perinatal loss in agg. 55.7%

Inexp. 30.4%

These perinatal loss include still birth and early neonatal death

For live born,

Chisquare value -7.91, Degree of freedom-1, significant at 0.005% P (<0.005)

For perinatal loss,

Chisquare value -2.24, Degree of freedom-1, significant at 0.001% P (<0.001)

TABLE 21

BIRTH WEIGHT SPECIFIC DEATH

Birth wt(kg)	Aggressive		Expectant	
	Born	Death	Born	Death
< 1	10	10(100%)	2	2(100%)
1.0-1.25	27	21(78.4%)	16	12(75%)
1.26-1.5	35	16(46%)	27	10(37%)
1.51-1.75	18	6(33%)	30	3(10%)
1.76-2	4	1(25%)	7	1(14%)
2.1-2.25	3	-	7	-
2.25-2.4	1	-	3	-

Maximum birth wt born was 2.4kg

51% of expectant group and 27% of aggressive group had babies birth weight more than 1.5 kg. Average birth weight was 1.58 kg

Chisquare value -13.69 Degree of freedom 2

Significant at 0.005 %(P<0.005)

TABLE 22

GESTATIONAL AGE SPECIFIC PERINATAL MORTALITY

GA(wks)	Aggressive		Expectant	
	Born	**Death**	**Born**	**Death**
28-30	34	26(76.5%)	29	18(62%)
31&32	38	18(47.4%)	24	8(33.3%)
33&34	25	10(40%)	38	2(5.3%)
Total	97	54(55.7%)	92	28(30.4%)

As gestational age gets prolonged, perinatal death is lesser both agg.& exp. giving 5.3% death in 32-34wks in exp.

Mean Gestational age 32 weeks

TABLE 23

PROLONGATION OF PREGNANCY IN EXP. GROUP

Latency interval(days)	Cases	Pnloss
<5	24(26%)	14(58.3%)
5-8	32(34.8%)	8(25%)
9-12	27(29.3%)	4(14.8%)
12-20	7(7.6%)	2*(28.5%)
>20	2(2.17%)	-
Total	92(100%)	-

* two cases were abruption

Maximum prolongation of pregnancy was 24 days& median prolongation was 10.5days.Mean being 8.6 days

PERINATAL OUTCOME IN PREGNANCIES LATENCY <96 HOURS

Latency interval (hrs)	**Cases**	**Pnloss**
<24	48	39(81.25%)
24-48	32	24(75%)
49-72	75	44(58.6%)
73-96	22	10(45.4%)

97 patients who were terminated more than 48 hour of steroids were compared in our study.

TABLE 24

NEONATAL HOSPITALISATION

Live babies		**Aggressive**	**Expectant**
Admission to NICU		77(100%)	79(92%)
Neonatal deaths		34(44.2%)	22(27.9%)
Survival hospital stay(days)	<5	2(2.6%)	25(31.6%)
	5-10	18(23.4%)	17(21.5%)
	11-15	10(13%)	9(11.4%)

	>15	13(16.9%)	6(7.6%)
Total survival		43	57+7=64
Survival rate		55.8%	74.4%

Mean stay of hospitalization is eight days in expectant group

Whereas in aggressive group it is 14 days.

DISCUSSION

Incidence of Preeclampsia is 5 to 8% according to ACOG 2002.

- Incidence of Preeclampsia in IOG is 10%

Severe Preeclampsia contributes 1% of all deliveries. There were 259 cases of severe Preeclampsia remote from term 28 to 34 weeks in our study period. 189 patients got full dose of steroids and they were assigned as aggressive

and expectant group in their management.

- 64.5% of patients were Primi…

Majority of cases say 75 to 80% occur during the first pregnancy.(clinical obstetrics and gynecology journal volume 42)

- 60% patients were in age group 25 to 34 yrs.

Young patients <20 & patients > 30 yrs said to have increased incidence of Hypertension and Preeclampsia.(AMJ of obstet gynecol 1993)

- 18% patients had BMI > 35

36% patients had Recurrent Preeclampsia

4.23% patients had mother with Preeclampsia

11% patients had sister with Preeclampsia.

Likelihood of developing Preeclampsia is increased in according to

BJ OG October 2003.

1. **Primi**
2. **Age .> 30**
3. **With family history**
4. **BMI > 35**
5. **Preexisting Hypertension**

Sibai et al(37) **showed risk of developing Preeclampsia in next pregnancy being 45.5%. Out of them 21% go for Severe Preeclampsia**.

MATERNAL OUTCOME

Comparisons of major complications in both the groups

Odendaal et al 1990(26)	No increase in complications
Sibai et al 1994 (39)	No increase in complications
Hall et al 2000 (340 women)	No maternal death, 3 required ICU 1 required dialysis in expectant.
Haddad B, Deis S 2004(11)	No maternal death or eclampsia.Morbidities similar among both groups.
Railton A, Allen DG 1987(31)	23.2% had increase in major complications
Our Study, 2005	10.3% in aggressive group 13.6% in expectant group. Not statistically significant

MATERNAL COMPLICATIONS VISE

Study	Abruption	Pulmonary Edema	HELLP/ ELLP	Eclampsia	Renal Failure
Hall and colleagues 2000	20%	2%	5%	1.2 %	0.3%

Vissur and Wallenburg 1995	5 %	---	NA	1.9 %	---
Murphy DJ and Stirrat GM 2000(22)	1.5 %	----	21 %	1.4%	1.3%
Olah KS, RedmanCW,Gee H 1993	----	----	14.2 %	----	3.5 %
Our study 2005	8.7%	3.3%	4.3%	---	1.1%

Our study showed relatively low incidence of abruption and HELLP than Hall's study and renal failure correlating with Murphy's.

There was no maternal death or eclampsia in our study.

TERMINATION

Indication of Termination shows following distribution in studies.

Study	Maternal indications	Fetal indications
Blackwell Sc and others 2002	80%	20%
Hall DR,Odendaal HJ 2000(12)	55%	45%
Our study	64.1%	35.9%

Most common maternal indication being imminent eclampsia. Our study's value was in between the both studies available.

MODE OF DELIVERY

Though mode of delivery had no influence on fetal outcome

LSCS rate was higher in expectant management in all.

STUDY	V AGINAL	LSCS
Hall BR and Odendaal HJ, 2000(12)	18.5%	81.5%
Nassar et al 306 patients(23)	48.3%	51.7%
Murphy DJ, Stirrat M 2000	20%	80%
Railton A and Allen OG,1987	25%	75%
Our Study, 2005	52.2% (group B)	47.8% (group B)

Our study is similar somewhat to Nassar et al.

Among 4 patients, complicated by HELLP Syndrome or DIVC 2 patients had coagulopathy with increased clotting time and bleeding from the surgical wounds on table, and they were treated with platelet transfusion, fresh frozen plasma and steroids.

Third patient who developed symptoms in postpartum period was given 6 units of fresh frozen plasma.

One patient developed signs and symptoms of Renal failure in immediate postpartum period and she was transferred to GH, taken over by Nephrologists, she underwent Peritoneal Dialysis twice and she was discharged alive after 22 days in a stable condition. Three patients who had compromised renal function got settled conservatively.

This situation explains necessity of the intensive care facilities in the management of severe preeclampsia.

Hemodynamic monitoring plays a major role in the treatment and trained persons and anaesthetists were available all the time for Central vein cathetarisation and monitoring.

PERINATAL OUTCOME

PROLONGATION OF PREGNANCY

Study	Mean Prolongation Of Pregnancy
Odendaal et al, 1990	7.1 days
Sibai et al	15.4 days
Vissur W, Wallenburg , 1995	14 days
Yang 2, Li R and others , Beijing(46)	11 days (28 to 31 weeks) 8 days (32 to 33 weeks)
Railton and Allen DG, 1987	11.4 days
Olah KS, Gee H 1993	9.5 days
Murphy DJ, Stirrat GM, 2000	14 days
Mithagen MI , Vissur W 2001	14 Days
This study ,2005	8.6 days

Median prolongation of pregnancy in this study was 10.5 days.Our study correlates with three studies among eight.

PERINATAL MORTALITY

Study	percentage loss
Railton A; Allen DG 1987	24.5%
Odendaal HJ, Pattinson RCL 1987(20)	22.3%
Hall DR, 2000	24%
Beneditti TJ, Beneditti JK	14.3%
Murphy DJ, Stirrat GM 2000	30%
Haddad B, Deis S, 2004	10.7%
This Study 2005	30.4%

In aggressively managed patients, the perinatal mortality was 55.7% giving

overall perinatal mortality 43.3%.Our study correlates with Murphy's and is closer to three other studies.

BIRTH WEIGHT

Study	Aggressive	Expectant
Sibai et al , 1994	1.2 kg	1.62 kg
Our Study, 2005	1.05 kg	1.58 kg

Our study correlates with Sibai et al and

all other studies showed higher birth weight by expectant management

SURVIVAL RATE

Study	Aggressive	Expectant
Sibai 1997	24%	65%
Hall DR 2000	70%	94%
Our study 2005	55.8%	74.4%

Railton A, Allen DG, 1987 showed 100% survival rate of babies born > 30 weeks in either the groups.

Our Study showed higher survival rate in babies of higher Gestational Age and with higher Birth weight and it was in between both studies.

NEONATAL HOSPITALISATION

Study	AGGRESSIVE	EXPECTANT	AGGRESSIVE	EXPECTANT
	Admission to NICU		Hospital stay	
Sibai et al 1974	100%	76%	30.6 days	20 days
Olah KS, 1983	64.3%	28.6%	>15 days	7.4 days
Our study 2005	100%	92%	14 days	8 days

Our study showed expectant management babies had highest survival rate and lower neonatal complications.In our study admission wwas similar to that of Sibai et al study because of the lower birth weight but hospital stay as that of Olah due to improvisation of neonatal facilities for the past two decade.

SUMMARY

- **Severe preeclampsia contributes 1% of all deliveries.**

  In our study there were 259 patients of severe preeclampsia remote from term. Among them 189 patients who had full dose of steroids were compared.

- **Most of them were primis**
- **Most of them were in age group 25 to 34 years**
- **Mean gestational age on admission was 31 weeks.**
- **36% had recurrent preeclampsia.**
- **15.8% (29) patients had family history of preeclampsia.**
- **Mean body mass index was 27.4.**
- **Mean systolic BP 160 mmHg and mean diastolic BP being 106 mmHg.**
- **Most of them needed 1000 – 1500 mg of alpha methyl dopa and 15-20mg of nifedipine for BP control.**
- **Most patient had normal fundus.**
- **Major maternal complications were higher in the expectant group, proved to be statistically non significant.**

  4 Patients had HELLP

  8 Patients had ABRUPTION

- **1 Needed renal dialysis in expectant group. There was no Maternal death and no eclampsia in both the groups.**

- **Most of them delivered by 8-11hrs by inducing labor.**
- **65% terminated for maternal indication 35% for fetal indication.**
- **LSCS rate was higher in expectant group (47.8% vs 15.5%) But this was mainly contributed by post cesarean pregnancy in expectant group.**
- **Perinatal loss was significantly lower in expectant group 30.4% vs 55.7% proved to the statistically significant P (<0.001).**
- **Perinatal loss is not influenced by cesarean section in expectant group as 42.8% perinatal death was contributed by LSCS and 57.2% by vaginal delivery statistically not significant.**
- **Expectant group had higher birth weight than aggressive significant. 51% had birth weight > 1.5kg maximum being 2.4kg**
- **Mean birth weight being 1.58kg.**
- **Mean gestational age at delivery 32weeks.**
- **Mean prologation of pregnancy is 8.6 days.**
- **Perinatal mortality was higher in the patients delivered <48hrs of steroids.**

- **Expectant group had relativety low admission of babies to NICU (92% vs 100%), lower mean stay of hospitalization (8 days vs 14 days).**
- **Babies in expectant group had higher survival rate (74.4% vs 55.8%).**

CONCLUSION

The conservative approach to the management of severe preclampsia remote from term results in a good obstetric outcome for most fetuses, in view of

1. **Higher birth weight**
2. **Lower perinatal mortality**
3. **Lesser neonatal complications**

but this must be balanced against the significant risk of morbidity to the mothers.

The success rate of expectant management will depend on both fetal gestational age and maternal and fetal conditions at the time of hospitalization. Since maternal and perinatal complications are significantly increased in these patients, expectant management should be carried out in well selected patients only at tertiary centers with adequate maternal and neonatal intensive care facilities.

Finally patients with preeclampsia are at increased risk for recurrence of preeclampsia in subsequent pregnancies.

PROFORMA

Name :

Age:

IP No:

Occupation:

Gravida:

ht : weight:

BMI:

LMP:

EDD:

Blood group:

Booked / Not:

Complaints:

Present H/o:

Period Of Amenorrhea

Edema feet

Headache

Oliguria

Pain abdomen

Vomiting

Present Pregnancy

I trimester

II trimester

III trimester

Blurring of vision

Palpitation **Treatment of Preeclampsia**

Past H/o: H/o Preeclampsia in previous pregnancy

Menstrual / Marital H/o:

Medical / Surgical H/o: H/o Epilepsy, Head injury, Neurological disorder.

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

Personal H/o:

General Examination:

Temp	**CVS**
PR	**RS**
BP	**PUPILS**
EDEMA	**CNS**
ANAEMIA	**P/A**
	P/V

INVESTIGATIONS:

Urine Alb	**CHG: Hb**	**Blood Urea**
Sugar	**PCV**	**Sugar**
Deposits	**RBC**	**Creatinine**
	TC	**Fibrinogen**
	Platelets	**Uric Acid**
		Electrolytes

LFT: **USG:** **ECG:**

Antihypertensive **Drug:** **Dose:**

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

Magnesium sulphate: Time Dose Temp RR I/O knee jerk.

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.

1. Abdul Kareem 1961.
The Effect of Pressor response in Preeclampsia.
By J. Obstet Gynaecol 82:246, 1961

2.American Journal of Obstet & Gynaecology 2002

3.Bardeguez AD, Mc Nerncy R, Frieri M, Verma UL, Tejani N.
Cellular immunity in preeclampsia. Alterations in T-lynphocyte subpopulations during early pregnancyObstet – Gynaecol 773859, 1991

4.Blackwell SC, Redman ME, Tomlinson M, Berry SM, Sorokin Y, Cotton DB.
Severe pre-eclampsia remote from term: what to expect of expectant management.
J Matern Fetal Neonatal Med. 2002 May; 11(5):321-4.

5.Chang et al. Effect of endothelium – derived relaxing factor inhibition on the umbilical – placental circulation in fetal lambs in utero.
AM J.Obstet – Gynacol 166:727, 1992

6.Chesley and Cooper DW 1986.
Genetics of Hypertension in Pregnancy.
By J. Obstet Gynecol 93:898-908

7.Cunningham FGI,Lindheimer MD:hypertension in pregnancy. Current concepts.N England. J.Med 326:972, 1992.

8.Dekker GA, Sibai BM:
Etiology and pathogenesis of preeclampsia current concepts.
AM J.Obstet – Gynacol 179:1359-1998.

9.De Boer K, Buller HR, Ten cate JW, Treffers PE.
Coagulation studies in the syndrome of HELLP
BR. J.Obstet Gynacol 98:42,1991.

10.Haddad B, Louis-Sylvestre C, Doridot V, Touboul C, Abirached F, Paniel BJ.
[Criteria of pregnancy termination in women with preeclampsia]
Gynecol Obstet Fertil. 2002 Jun;30(6):467-73. French.

11.Haddad B, Sibai BM.
Expectant management of severe preeclampsia: proper candidates and pregnancy outcome.
Clin Obstet Gynecol. 2005 Jun; 48(2):430-40. Review.

12.Hall DR, Odendaal HJ, Steyn DW, Grove D.
Urinary protein excretion and expectant management of early onset, severe pre-eclampsia. Int J Gynaecol Obstet. 2002 Apr; 77(1):1-6.

13.Hall DR, Odendaal HJ, Steyn DW.
Expectant management of severe pre-eclampsia in the mid-trimester.
Eur J Obstet Gynecol Reprod Biol. 2001 Jun; 96(2):168-72.

14.Hall DR, Odendaal HJ, Kirsten GF, Smith J, Grove D.
Expectant management of early onset, severe pre-eclampsia: perinatal outcome.
BJOG. 2000 Oct;107(10):1258-64.

15.Hall DR, Odendaal HJ, Steyn DW, Grove D.
Expectant management of early onset, severe pre-eclampsia: maternal outcome.
BJOG. 2000 Oct;107(10):1252-7.

16.Hall R Swart D Grove H J Odendaal D.
The influence of maternal age on pregnancy outcome in patients with early onset, severe pre-eclampsia. J Obstet Gynaecol. 2001; 21(3):246-249.

17.Hayman R,Warren A,Brockleysby J, Johnson I, Baker P.Endothelial dysfunction in preeclampia.Br J obstet Gyneol 107:108,2000.

18.Hubel CA,R oberts JM, Taylor RN, Mclaughlin MK:Lipid peroxidation in pregnancy. New perpectives in preeclampia Am J obstet Gynaecol 161:1025, 1989.

19.Leduc L, Wheeler JM, Krishon B, Mitchell P, Cotton DB
Coagulation profile in severe preeclampsia
Obstet – Gynacol 79:14,1992.

20.Madazli R, Budak E, Calay K Z, Aksu MF
Correlation between placental bed biopsy findings, vascular cell adhesion molecule and fibronectin levels in preeclampsia

21.Moodley J, Koranteng SA, Rout C.
Expectant management of early onset of severe pre-eclampsia in Durban.
S Afr Med J. 1993 Aug;83(8):584-7. Erratum in: S Afr Med J 1993 Oct;83(10):811

22.Murphy DJ, Stirrat GM St Michaels hospital Briston,United kingdom,
Mortality and mortbidity associated with early onset preeclampia
Hypertens pregnancy 200:19(2) 221-31.

23.Nassar AH, Adra AM, Chakhtoura N, Beyodouns.
Sever preeclampsia remote from term. Labour induction or elective cesarean delivery?
AM.J.Obstet gynaecol 1979:1210,1998.

24.North RA, Taylor RS ,Schellenburg J.C Evaluation of a definition of preeclampia.
Br J obstet Gynaecol 106:767, 1999

25.Odendaal HJ, Hall DR, Grove D.
Risk factors for and perinatal mortality of abruptio placentae in patients hospitalised for early onset severe pre-eclampsia - a case controlled study. J Obstet Gynaecol. 2000 Jul;20(4):358-64.

26.Odendaal HJ, Steyn DW, Norman K, Kirsten GF, Smith J, Theron GB.
Improved perinatal mortality rates in 1001 patients with severe pre-eclampsia.
S Afr Med J. 1995 Oct;85(10 Suppl):1071-6.

27.Olah KS, Redman CW, Gee H.
Management of severe, early pre-eclampsia: is conservative management justified?
Eur J Obstet Gynecol Reprod Biol. 1993 Oct 29;51(3):175-80.

28.Oosterhof H, Voorhoeve PG, Aarnoudse JG
Enhancement of hepatic artery resistance to blood flow in preeclampsia and in HELLP.
AM J.Obstet – Gynacol 171:526,1994.

29.Page EW. On the pathogenesis of preeclampsia & eclampsia
BR J.Obstet Gynaecol 79:883,1972

30.Pritchard JA, Cunningham FG, Mason RA
Coagulation changes in preeclampsia.
AM J. Obstet – Gynacol 124:855,1976.

31.Railton A, Allen DG-Management and outcome of pregnancy complicated by severe preeclampia by severe preeclampia of early onset
S Afrs Med J1987 Nov7: 72(9):608-10.

32.Redman CWG, Sacks GP, Sargent K
Preeclampsia an excessive maternal inflammatory response to pregnancy.
AM J.Obstet Gynacol 180:499-1999.

33.Roberts JM:Preeclampia:what we know and what we do not know. seminar perinatoll 24:24,2000.

34.Sanchez-Ramos L, Jones DC, Cullen MT,
Urinary calcium as an early marker of preeclampsia.
Obstet Gynacol 77:685, 1991

35.Silver H, Morgan T, Ward K.
Blood volume stratified by angiotensinogen genotypes in normal and hypertensive pregnancies. Presented at 21st annual meeting of society of maternal – fetal medicine, February 5-10,2001 held in Reno, Nerada.

36.Sibai BM.
Diagnosis and management of gestational hypertension and preeclampsia.
Obstet Gynecol. 2003 Jul;102(1):181-92. Review.

37.Sibai BM.
Friedman SA, Schiff E, Lubarsky SL,
Expectant management of severe preeclampsia remote from term.
Clin Obstet Gynecol. 1999 Sep;42(3):470-8. Review.

38.Visser W, Wallenburg HC.
Maternal and perinatal outcome of temporizing management in 254 consecutive patients with severe pre-eclampsia remote from term.
Eur J Obstet Gynecol Reprod Biol. 1995 Dec;63(2):147-54.

39.Sibai BM, Mercer BM, Schiff E, Friedman SA.
Aggressive versus expectant management of severe preeclampsia at 28 to 32 weeks' gestation: a randomized controlled trial.
Am J Obstet Gynecol. 1994 Sep; 171(3):818-22.

40.Taufield P. Alas KL, Resuick LM: ENG J Med 316:715, 1987.

41.Walsh SW et al 1986.
Low dose aspirin prevents preeclampsia by inhibiting lipid peroxide and thromboxane and not on prostacyclin.
By AM j. Obstet Gynacol 167:926-930

42.Weiner CP, Brandt J. Plasma antithrombin III activity.
An aid in the diagnosis of preeclampsia – eclampsia
AM J.Obstet Gynacol 142:275, 1982.

43.Weinstein L.
Preeclampsia – eclampsia with hencolysis, elevated liver enzymes and thrombocytoperia.
Obstet Gynacol 66:657, 1985.

44.Withagen MI, Visser N,Wallenburg the Erasmus university school of medicine and healthsciences, institute O&G-Rotterdam, Netherlands Eur J Obstet Gyneol reprod jol.2001(feb) in94(2):211-15.

45.Volhard F.
Die doppel settigen haematogenon nierenerkrankungen
Berlin, Springer, 1917.

46.Yang Z, Li R, Shi LY, Wang LN, Ye RH, Wang R, Huang P.
[Clinical delimitation and expectant management of early onset of severe pre-eclampsia.]
Zhonghua Fu Chan Ke Za Zhi. 2005 May; 40(5):302-5. Chinese.