MANAGEMENT OF ECLAMPSIA IN A TERTIARY CARE CENTRE-MATERNAL AND PERINATAL OUTCOME

V. Usha Rani[1], P. Vijaya[2]

[1]Assistant Professor, Department of Obstetrics & Gynaecology, Modern Government Maternity Hospital, Petlaburz, Hyderabad, Telangana.
[2]Assistant Professor, Department of Obstetrics & Gynaecology, Modern Government Maternity Hospital, Petlaburz, Hyderabad, Telangana.

ABSTRACT: AIM: Management of 240 cases of eclampsia during a period of 1yr 6 months.
MATERIALS AND METHODS : A study of 240 cases of eclampsia over a period of 1yr 6months at a tertiary level referral centre.They were analyzed regarding age, parity, socio economic status, period of gestation, antenatal care, No.of convulsions, condition at the time of admission.Management of eclampsia ,maternal and perinatal outcome analyzed.
RESULTS: Out of 240 cases of eclampsia most of them were primigravida belonging to low socio economic stata 73% had antenatal care but not regularly. 215 cases were given Mg So4 and the remaining patients Lorazepam and Phenytoin were added. The total perinatal mortality in our study was 28.3%.The perinatal mortality decreases with increasing gestational age and birth weight.Maternal Complications we encountered were Encephalopathy, Pyrexia, RTI, Retained Placenta. 6/240 Maternal deaths, in this two undelivered,CVA was the major cause of death.
CONCLUSIONS: Eclampsia is a life endangering obstetric emergency still prevails in developing countries due to inadequate antenatal care, low socio economic stata and lack of transport facility, more common in primis. Good antenatal care helps in preventing ecampsia. Attentive nursing and individualized treatment algorithms, include prompt fluid replacement, anticonvulsant therapy (Mg So4) aggressive antihypertensive therapy and prompt delivery, availability of CT scan with good neonatal unit will improve the maternal and fetal outcome.

KEYWORDS: APH–Antepartum Haemorrhage, APE-Ante Partum Eclampsia, IPE-Intra Partum Eclampsia, PPE- Post Partum Eclampsia, Mg So4-Magnesium Sulfate.

HOW TO CITE THIS ARTICLE: V. Usha Rani, P. Vijaya. "Management of Eclampsia In a Tertiary Care Centre-Maternal And Perinatal Outcome". Journal of Evidence based Medicine and Healthcare; Volume 2, Issue 49, November 19, 2015; Page: 8500-8504, DOI: 10.18410/jebmh/2015/1164

INTRODUCTION: Eclampsia occurs in 2 to 3 percent of women with severe features of pre-eclampsia not receiving antiseizure prophylaxis. Women at highest risk of developing eclampsia are primis, and from lower socioeconomic backgrounds. The peak incidence is in adolescence and the early twenties but is also increased in women over the age of 35.

CLINICAL PRESENTATION AND FINDINGS: Hypertension (75%), Headache (persistent frontal or occipital headaches or thunderclap headaches) (66%) Visual disturbances (scotomata, loss of vision [cortical blindness], blurred vision, diplopia, visual field defects [eg, homonymous hemianopia], photophobia) (27%)

Right upper quadrant or epigastric pain (25%) Asymptomatic (25 percent), Ankle clonus is also a common finding.[1]

Maternal mortality and severe morbidity rates are lowest among women receiving regular prenatal care who are managed by experienced physicians in tertiary centers (maternal mortality 0 to 1.8 percent).[2,3,4,5]

Perinatal morbidity is also increased in eclamptic pregnancies and closely related to gestational age. In addition, there is a two to threefold increased risk of delivery of a small for gestational age infant.

MATERIAL AND METHODS: Study of 240 cases of eclampsia over a period of one year six months at a tertiary care center. They were analyzed regarding age, parity and socio economic status, period of gestation, no. of convulsions, condition at the time of admission. Maternal and perinatal morbidity and mortality analyzed.At the time of admission detailed history taken, time of first convulsion, premonitory symptoms, condition of the patient noted.Neurological status, B.P, u/o, KJ, urine albumin. Investigations Bl grouping and typing, CBP platelets, RBS, LFT, RFT, CUE, USG. PRITCHARDS Regime followed.

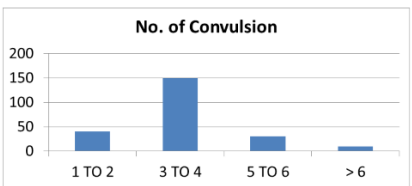
RESULTS: Out of the 240 cases of eclampsia majority belonged to the low socio economic status. 69% were primigravida, 48.75% are referred from district hospitals and private hospitals.

About 73% of cases were between 21-25 yrs, 93.75% cases were unbooked, 80.45% were antepartum eclampsia, 111 women were more than 34wks gestation. Severe B.P. recorded in 85 women. 130 women were conscious at the time of admission, only 15 women had

Submission 06-11-2015, Peer Review 07-11-2015, Acceptance 11-11-2015, Published 19-11-2015.
Corresponding Author:
Dr. V. Usharani, 3-5-1093/20, S. V. Colony, Narayanaguda, Hyderabad-500029, Telangana State.
E-mail: doctorvusharani@gmail.com, patnala_vijaya@rediffmail.com
DOI: 10.18410/jebmh/2015/1164

urine albumin 4+. Majority of the cases had 3-4 convulsions at admission. Greater the convulsions and delivery interval worse the maternal and perinatal mortality. 73% had antenatal care but not regularly. 215 cases were given Mg So4 and the remaining patients Lorazepam and Phenytoin were added. 165 women delivered vaginally, 73 delivered by LSCS, 2 cases died undelivered. 90% of the women delivered within 24 hrs of admission. Proteinuria was significantly present. 37 cases IUD confirmed at time of admission. The total perinatal mortality in our study was 28.3%. An early LSCS was done in unfavourable Cx cases. The neonatal salvage in LSCS is 86%, vaginal group 63.8%. The perinatal mortality decreases with increasing gestational age and birth weight. Complications we encountered were Encephalopathy, Pyrexia, RTI, Retained Placenta. 6/240 Maternal deaths, in this two undelivered, CVA was the major cause of death.

OBSERVATIONS



- MAJORITY OF CASES HAD-3-4 CONVULSIONS AT ADMISSION
- WE NOTED THAT MATERNAL MORTALITY WAS MAXIMUM IN THOSE PATIENTS WITH MORE NUMBER OF CONVULSIONS
- GREATER THE CONVULSION –DELIVERY INTERVAL, GREATER THE PERINATAL AND MATERNAL MORTALITY

OBSERVATIONS

AGE	NO. OF PT (n = 240)
< 20 Yrs	23 (9.5 %)
21 – 25	177 (73.75 %)
26 – 30	29 (12.08 %)
31 – 35	5 (2.08 %)
> 35	1 (0.41 %)

PARITY	NO. OF PT (n = 240)
Primi	166 (69.1 %)
G2	52 (21.6 %)
G3	12 (5 %)
G4	10 (4.16 %)
G5	7 (2.91 %)

		TOTAL
Booked	15 (6.25%)	240
Un Booked	225 (93.75 %)	240

Antepartum	193	(80.4%)
Intrapartum	18	(8.75%)
Post Partum	21	(7. 5 %)
Intercurrent	03	(1.25%)

OBSERVATIONS

Gest. Age	No. Of Pt (n = 240)
< 28 Weeks	30
29 – 34 Weeks	99
> 34 Weeks	111

CONDITION ON ADMISSION	NO. OF PT (n = 240)
Conscious	135
Drowsy	36
Unconscious	69

BP	NO. OF PT (n = 240)
Mild	155
Severe	85

REFLEXES	NO. OF PT (n = 240)
Present	202
Absent	38

PEDAL EDEMA	NO. OF PT (n = 240)
Present	220
Absent	20

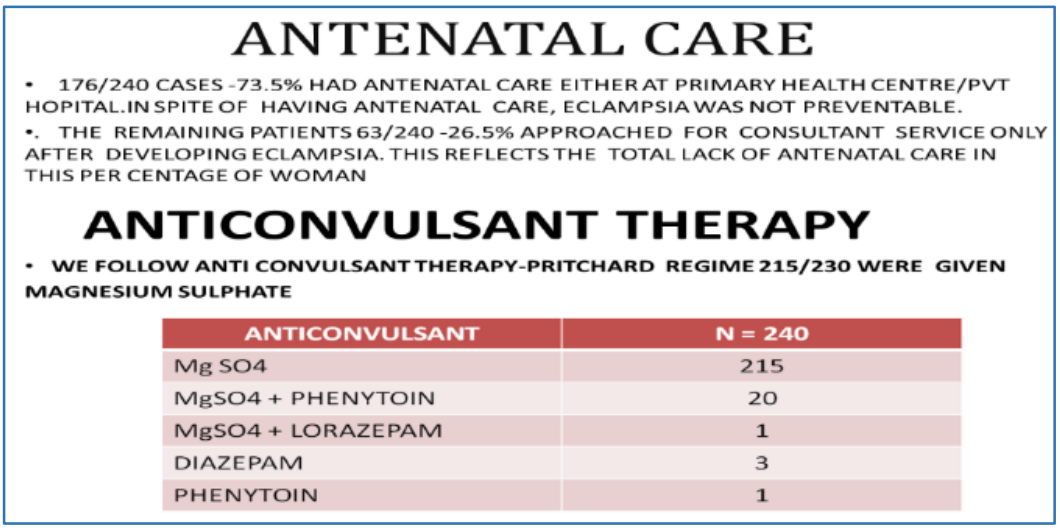
PROTEINURIA	NO. OF PT (n = 240)
+	50
++	80
+++	35
++++	15
Nil	60

VAGINAL DELIVERY IN ECLAMPSIA

MODE OF INDUCTION	n = 165
MISOPROSTOL	76
OXYTOCIN	58
EMERCREDYL – ETHACRIDANCE LACTATE	07
COMBINATION OF MISOPROSTAL + OXYTOCIN + EMECREDYL	04
ARM	20

MODE OF DELIVERY	n = 165
SPONTANEOUS	108
OUTLET FORCEPS	17
MIDLOW FORCEPS	01
VACUUM	29

▪ THE NUMBER OF MISOPROSTOL DOSES USED WAS PROPORTIONAL TO THE PERIOD OF GESTATION AND FAVOURABILITY OF CERVIX BASED ON BISHOP'S SCORE



ANTENATAL CARE

- 176/240 CASES -73.5% HAD ANTENATAL CARE EITHER AT PRIMARY HEALTH CENTRE/PVT HOPITAL.IN SPITE OF HAVING ANTENATAL CARE, ECLAMPSIA WAS NOT PREVENTABLE.
- . THE REMAINING PATIENTS 63/240 -26.5% APPROACHED FOR CONSULTANT SERVICE ONLY AFTER DEVELOPING ECLAMPSIA. THIS REFLECTS THE TOTAL LACK OF ANTENATAL CARE IN THIS PER CENTAGE OF WOMAN

ANTICONVULSANT THERAPY

- WE FOLLOW ANTI CONVULSANT THERAPY-PRITCHARD REGIME 215/230 WERE GIVEN MAGNESIUM SULPHATE

ANTICONVULSANT	N = 240
Mg SO4	215
MgSO4 + PHENYTOIN	20
MgSO4 + LORAZEPAM	1
DIAZEPAM	3
PHENYTOIN	1

DISCUSSION: The precise cause of seizures in preeclamptic women is not clearly understood. based on the central role of hypertension. Hypertension causes a breakdown of the autoregulatory system of the cerebral circulation, leading to hyperperfusion, endothelial dysfunction, and brain edema. Hypertension causes activation of the autoregulatory system, leading to vasoconstriction of cerebral vessels and resulting in hypoperfusion, localized ischemia, and subsequent fluid leakage. Cerebral inflammation may also play a role.

The prodromal symptoms include headache, shortness of breath, blurry vision, nausea or vomiting, edema, neurological deficit, and epigastric pain. Many women do not have hypertension during the antecedent pregnancy.

Neuroimaging—Neuroimaging findings consistent with reversible posterior leukoencephalopathy syndrome (RPLS) are the hallmark of eclampsia.

Eclampsia is a clinical diagnosis typically based upon the occurrence of new onset generalized tonicclonic seizures in a woman with preeclampsia.[6]

Reversible posterior leukoencephalopathy syndrome (RPLS; also called posterior reversible encephalopathy syndrome [PRES]) is a neurologic syndrome defined by clinical and radiologic features (headache, confusion, visual symptoms, seizures, vasogenic edema predominantly localized to the posterior cerebral hemispheres). Neuroimaging is essential to the diagnosis. In a pregnant woman with seizures, the typical clinical and neuroimaging findings of RPLS are indicative of eclampsia, even when features of preeclampsia (hypertension with or without proteinuria) are absent].

Atypical cases, such as women who do not meet criteria for diagnosis of preeclampsia or who have persistent neurologic deficits, prolonged loss of consciousness, onset of seizures >48 hours after delivery, onset of seizures before 20 weeks of gestation, or seizures despite adequate magnesium sulfate therapy should be evaluated for other causes of seizures.

Differential diagnosis—brain tumor, ruptured aneurysm, thrombotic thrombocytopenic purpura [TTP], hemolytic uremic syndrome [HUS], cerebral venous thrombosis.

Key principles in the Management—If the seizure is witnessed, maintaining airway patency and preventing aspiration are the initial priority. The woman should be rolled onto her left side. The immediate issues include: Prevention of Maternal hypoxia and trauma, Treatment of severe hypertension, if present, Prevention of recurrent seizures, Evaluation for prompt delivery.

Women who do not improve promptly following control of hypertension and seizures and those who develop localizing neurologic signs should be evaluated by a neurologist.

Maternal oxygenation and protection from trauma—The patient is placed in a lateral position, if possible. Supplemental oxygen (8 to 10 L/min) is administered via a nonrebreather face mask to treat hypoxemia from hypoventilation during the seizure.[2] Raised, padded bedrails provide protection from trauma.

Treatment of hypertension—Antihypertensive therapy is administered when diastolic pressures greater than 100 mmHg or systolic blood pressures ≥160 mmHg. As in our study we have given Labetolol 100mg BID and added Nefidipine 10mgTID depending on Hypertension.

Magnesium sulfate is the drug of choice based on randomized trials demonstrating that it reduces the rate of recurrent seizures by onehalf to twothirds (relative risk [RR] 0.44, 95% CI 0.320.51) and the rate of maternal death by onethird (RR 0.62, 95% CI 0.390.99).

A series of systematic reviews reported magnesium sulfate was safer and more effective than phenytoin, diazepam, or lytic cocktail (ie, chlorpromazine, promethazine and pethidine) for prevention of recurrent seizures in eclampsia.

Additional advantages of magnesium sulfate therapy were its low cost, ease of administration. (eg, cardiac monitoring is not required), and lack of sedation. In utero exposure to magnesium sulfate therapy decreases the risk

of cerebral palsy and severe motor dysfunction in offspring born prematurely.

Administration of magnesium sulfate-Loading dose–of 4 g intravenously 20% solution, 5 g intramuscularly into each buttock of 50% solution for a total of 10 g; however, the onset of a therapeutic effect will be slower and intramuscular injection is painful. These loading doses may be given safely to patients with renal insufficiency. Maintenance dose–We administer a maintenance dose of magnesium sulfate 2 g/hour as a continuous intravenous infusion to women with good renal function. Maintenance doses of 1 to 3 g/hour are commonly used. Alternatively, magnesium sulfate 5 g can be given intramuscularly every four hours. The maintenance phase is given only if a patellar reflex is present (loss of deep tendon reflexes is the first manifestation of symptomatic hypermagnesemia), respirations are greater than 12 per minute, and urine output is over 100 mL in four hours. This is the protocol we followed in our study.

Calcium gluconate (1g intravenously) may be administered to counteract magnesium toxicity, if necessary. Concurrent use of magnesium sulfate with calcium channel blockers may result in hypotension, but the risk appears to be minimal. Magnesium sulfate is contraindicated in women with myasthenia gravis since it can precipitate a severe myasthenic crisis.

There is no role for mannitol in the routine care of women with eclampsia.[7] It can be harmful because it can enter the brain through a damaged bloodbrain barrier and reverse the osmotic gradient, thus increasing intracranial pressure.

Stabilizing the mother by administering anticonvulsant drugs and oxygen and treating severe hypertension (if present) can help the fetus recover in utero from the effects of maternal hypoxia, hypercarbia, and uterine tachysystole. However, if the fetal heart rate tracing does not improve within 10 to 15 minutes despite maternal and fetal resuscitative interventions, then the possibility of an occult abruption should be considered and emergent delivery may be Indicated.[2]

The definitive treatment for eclampsia is prompt delivery; however, this does not necessarily preclude induction and a trial of labor.[6,7] After maternal stabilization, factors to consider in determining the mode of delivery are gestational age, cervical status, whether the patient is in labor, and fetal condition and position.

Induction is a reasonable option for pregnancies at least 32 to 34 weeks of gestation and for earlier gestations with a favorable Bishop score. Cervical ripening agents can be used to improve the Bishop score; however, in our opinion, long inductions should be avoided and a clear endpoint for delivery planned (eg, within 24 hours).

The optimal duration of magnesium sulfate therapy has not been determined. When begun before delivery, we continue magnesium sulfate for 24 to 48 hours postpartum

PREGNANCY OUTCOME: Maternal—Maternal complications occur in up to 70 percent of women with eclampsia. Brain damage from hemorrhage or ischemia may result in permanent neurologic sequelae and is the most common cause of death in eclamptic women.[8,9]

Maternal mortality rates of 0 to 14 percent have been reported over the past few decades.[6,7] Maternal mortality and severe morbidity rates are lowest among women receiving regular prenatal care who are managed by experienced physicians in tertiary centers (maternal mortality 0 to 1.8 percent).[2,3,4,5] The highest mortality rates are in low income countries where prenatal, intrapartum, and neonatal care are compromised by limited resources.[10]

Fetal and neonatal—Premature delivery, abruptio placenta, and intrauterine asphyxia are the primary causes of perinatal death in eclamptic pregnancies.

Perinatal morbidity is also increased in eclamptic pregnancies and closely related to gestational age. In addition, there is a two to threefold increased risk of delivery of a small for gestational age infant.

Recurrence risk—Recurrent eclampsia occurs in 2 percent of subsequent pregnancies. The risk appears to be reduced by close maternal monitoring and timely intervention if preeclampsia develops.[11] Preeclampsia, however, cannot be prevented in most cases.

Both preeclamptic and eclamptic women are at increased risk of developing cardiovascular and cerebrovascular disease and diabetes later in life.

CONCLUSIONS: Eclampsia is a life endangering obstetric emergency still prevails in developing countries due to inadequate antenatal care, low socio economic stata and lack of transport facility, more common in primis. Good antenatal care helps in preventing ecampsia. Attentive nursing and individualized treatment algorithms, include prompt fluid replacement, anticonvulsant therapy (Mg So4) aggressive antihypertensive therapy and prompt delivery, availability of CT scan with good neonatal unit will improve the maternal and fetal outcome.

REFERENCES:

1. Chuan FS, Charles BG, Boyle RK, Rasiah RL. Population pharmacokinetics of magnesium in preeclampsia. Am J Obstet Gynecol 2001; 185: 593.
2. Sibai BM. Diagnosis, prevention, and management of eclampsia. Obstet Gynecol 2005; 105: 402.
3. Pritchard JA, Cunningham FG, Pritchard SA. The Parkland Memorial Hospital protocol for treatment of eclampsia: evaluation of 245 cases. Am J Obstet Gynecol 1984; 148: 951.
4. Sibai BM. Eclampsia. VI. Maternalperinatal outcome in 254 consecutive cases. Am J Obstet Gynecol 1990; 163: 1049.
5. MacKay AP, Berg CJ, Atrash HK. Pregnancyrelated mortality from preeclampsia and eclampsia. Obstet Gynecol 2001; 97: 533.

6. American College of Obstetricians and Gynecologists, Task Force on Hypertension in Pregnancy. Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Forceon Hypertension in Pregnancy. Obstet Gynecol 2013; 122: 1122.
7. Demir BC, Ozerkan K, Ozbek SE, et al. Comparison of magnesium sulfate and mannitol in treatment of eclamptic women with posterior reversible encephalopathy syndrome. Arch Gynecol Obstet 2012; 286: 287.
8. Sibai BM, Spinnato JA, Watson DL, et al. Eclampsia. IV. Neurological findings and future outcome. Am JObstet Gynecol 1985; 152: 184.
9. Okanloma KA, Moodley J. Neurological complications associated with the preeclampsia/Eclampsia syndrome. Int J Gynaecol Obstet 2000; 71: 223.
10. Moodley J. Maternal deaths due to hypertensive disorders in pregnancy: Saving Mothers report 20022004. Cardiovasc J Afr 2007; 18: 358.
11. Gilstrap LC 3rd, Cunningham FG, Whalley PJ. Management of pregnancyinduced hypertension in the nulliparous patient remote from term. Semin Perinatol 1978; 2: 73.