British Journal of Medicine & Medical Research
10(3): 1-9, 2015, Article no.BJMMR.19217
ISSN: 2231-0614

SCIENCEDOMAIN *international*
www.sciencedomain.org

Early-Onset Severe Preeclampsia in Nigerian Women: Determining a Balance between Maternal Wellbeing and Fetal Survival in a Resource-Limited Setting

Nosakhare O. Enaruna[1*] and Jedidiah D. Sodje[1]

[1]Department of Obstetrics and Gynecology, University of Benin Teaching Hospital, P.M.B. 1111, Benin City, Nigeria.

Authors' contributions

This work was carried out in collaboration by both authors. Author NO designed the study, analyzed the data and wrote the first draft of the manuscript. Author JD was involved in data analysis and writing of the final version of the manuscript. Both authors read and approved the final manuscript.

Article Information

DOI: 10.9734/BJMMR/2015/19217
Editor(s):
(1) Yoshihiro Nishida, Department of Obstetrics and Gynecology, Faculty of Medicine, Oita University, Yuhu-City, Japan.
Reviewers:
(1) Anonymous, Yuzuncu Yıl University, Turkey.
(2) Anonymous, La Fe University Hospital, Spain.
(3) Anonymous, University Medical Centre Ljubljana, Slovenia.
(4) David Krantz, USA.
Complete Peer review History: http://sciencedomain.org/review-history/10413

Original Research Article

Received 30th May 2015
Accepted 13th July 2015
Published 5th August 2015

ABSTRACT

Background: Preeclampsia in Nigerian women is considered to have a rapidly progressive clinical course.
Aim: In the present study we sought to determine the most appropriate gestational age (GA) for delivery in severe preeclampsia occurring preterm to achieve optimal neonatal outcome, without causing undue maternal compromise.
Study Design: A retrospective cohort study.
Place and Duration of Study: Department of Obstetrics & Gynecology, University of Benin Teaching Hospital, Benin City, Nigeria between June 2012 and May 2014.
Methodology: Information on the sociodemographic characteristics, clinical management and outcome of women with severe preeclampsia was extracted from case records and analyzed using

**Corresponding author: Email: nosaenrus@gmail.com;*

SPSS 20.0 and GraphPad InStat 3 software.
Results: We included 312 women in the study. The median GA at delivery was 35 weeks. The incidence of eclampsia was 2.5% (102/4,106). Eclampsia occurred 1.5-fold, 1.6-fold and 1.6-fold more, respectively with proteinuria of ≥ 3+, severe anemia and mean arterial blood pressure ≥ 120 mmHg (*P*=0.00, *P*=0.00 and *P*=0.00, respectively). Eclampsia, severe anemia and proteinuria ≥ 3+ were associated with maternal mortality (14.7% vs 0, *P*=0.00; 28.5% vs 3.7, *P*=0.00; and 7.6% vs 0.8, *P*=0.00, respectively). Logistic regression analysis showed that delivery before 34 weeks gestation was associated with early neonatal death (*P*=0.00).
Conclusions: Severe preeclampsia occurring preterm is associated with significant perinatal mortality in our hospital. An approach of selective conservative management up to 34 weeks gestation could improve fetal salvage rate.

Keywords: Early-onset preeclampsia; pregnancy outcome; maternal mortality; perinatal mortality; timing of delivery.

ABBREVIATIONS

GA : Gestational age
UBTH : University of Benin Teaching Hospital
GPC : General practice clinic
DBP : Diastolic blood pressure
SBP : Systolic blood pressure
MABP : Mean arterial blood pressure
SCBU : Special care baby unit
PCV : Packed cell volume
ENND : Early neonatal death
BP : Blood pressure

1. INTRODUCTION

Maternal and perinatal complications due to preeclampsia tend to increase with progression to eclampsia [1], so that efforts directed at reducing the risk of disease progression constitute the focal point of management. Hence, the approach to managing severe disease emphasizes patient stabilization and immediate delivery in a bid to reduce risks of complications like eclampsia, pulmonary edema, abruptio placentae, acute renal failure, fetal death or maternal mortality [2-5]. Consequently, the survival of the fetus will depend, in addition to the severity of the disease, on the gestational age (GA) vis-à-vis lung maturation and other problems of prematurity. This concern over fetal salvage is compounded by the poor capacity of neonatal support facilities at our disposal. Recently, improved neonatal outcomes have been documented following a policy of selective expectant management to allow for marginal fetal maturation before delivery in patients with severe preeclampsia [6-8].

Early-onset preeclampsia has traditionally been described as occurring before 32 weeks gestation [9], but is now often defined as disease occurring before 34 weeks [10]. In settings where facilities are advanced, fetal salvage is nearly always assured in these cases. However, in less advanced places, with poorly developed neonatal support equipment and lack of trained personnel, babies delivered at less than 37 weeks may still be at high risk for perinatal mortality or morbidity. Moreover, most maternal and perinatal deaths attributable to preeclampsia occur when it is preterm though the toll appears to be most felt by the baby [11,12]. This observation has encouraged some clinicians to support conservative management in severe disease for the sake of the fetus while strictly monitoring the mother for worsening condition. However, to fully administer this plan, monitoring tools like cardiotocograph and ultrasound for biophysical profile and Doppler study are important to evaluate the state of the fetus, while maternal disease progression may become apparent from regular clinical, hematological and biochemical evaluations.

In advanced economies, where this recommendation for expectant management is more often advocated, early severe preeclampsia may be monitored till 34 weeks, but in our environment, maternal consideration appears to override that of the fetus such that immediate delivery is embarked on for both severe early and late preeclampsia. The corollary is that many neonates do not survive because of prematurity, low birth weight and overwhelming effects of the disease, especially in the absence of incubators and exogenous surfactant.

At the University of Benin Teaching Hospital (UBTH), the policy of managing severe early-onset preeclampsia (defined as severe preeclampsia occurring before 32 weeks gestation) is stabilization and immediate delivery

by the most expedient route. Afore-mentioned is partly executed because preeclampsia in our environment is described as rapidly progressive. Perinatal mortality due to preeclampsia in Nigeria is between 4.1% and 12.3% [13,14]. It is currently not certain whether these perinatal figures could be improved by a policy of selective expectant management when certain clinical or laboratory indices are present to warrant need for fetal maturation, perhaps up till 34 weeks, but at no excess risk to the mother. Furthermore, this approach will foster an easier international data comparison in the light of changing definitions and practice. Therefore, the present study is aimed at documenting the role of GA at delivery on maternal and perinatal outcome in severe preeclampsia occurring before 34 weeks gestation in UBTH.

2. MATERIALS AND METHODS

A retrospective study of all patients managed for severe preeclampsia at the Department of Obstetrics and Gynecology, UBTH, Benin City, between June 2012 and May 2014 was conducted with Institutional Review Board's approval. Multi-fetal pregnancies and postpartum cases of severe preeclampsia were excluded.

2.1 Setting

The UBTH is a multi-center facility located in Edo State, South-south region of Nigeria, which serves as a major referral point, attracting patients from at least the three neighboring states of Kogi, Ondo and Delta. Patients are also referred from both public and private hospitals within Edo State. Within the hospital, the major portal of entry for pregnant women is the general practice clinic (GPC) from where they are referred to the antenatal clinic for booking, but in emergency situations, patients are admitted via the emergency unit of the hospital to the labor ward. On the average, between 100 and 150 patients are booked for antenatal care every week in the hospital, while follow up attendance rate is between 250 and 500 patients per week. The delivery rate in the hospital in the last five years has been about 2,700 per year, which gives an average monthly delivery rate of 225. The hospital has a total antenatal and postnatal bed capacity of 82 spaces, and 8 functioning delivery rooms in the labor ward. There are 2 operating theatres attached to the labor ward.

2.2 Clinical and Laboratory Management

In this study, patients who had antenatal care in UBTH were referred to as 'booked' and those not registered in UBTH as 'unbooked'. Severe preeclampsia was defined as diastolic blood pressure (DBP) of ≥110 mmHg and/or systolic blood pressure (SBP) of ≥160 mmHg associated with proteinuria of at least 2+ on dipstick examination, on at least 2 occasions, at least 6h apart, and/or presence of end-organ dysfunction [1]. Severe hypertension was defined as mean arterial blood pressure (MABP) ≥ 130 mmHg or SBP ≥ 170 mmHg or DBP ≥ 110 mmHg [8], and severe proteinuria was defined as 3+ or more from a dipstick examination on at least 2 occasions, at least 6 hours apart. All patients were managed using the same standardized departmental (labor ward) protocol [15]. At admission, all the patients had seizure prophylaxis, blood pressure control, and fluid management was instituted. Venous blood was drawn for full blood count and platelets, electrolytes and urea estimation, liver function test and bedside clotting time, and abnormal results were corrected. Thereafter, preparation was made for delivery by the most expedient route. Caesarean section was done for obstetric indications.

Magnesium sulfate was the anticonvulsant used to control and prevent seizures. It was delivered intravenously as a loading dose of 4g over 10 minutes, with maintenance dose of 1g per hour given as continuous infusion according to the Zuspan regimen [16]. Monitoring for toxicity was by clinical assessment of respiratory rate, deep tendon reflexes and urinary output. Intravenous labetalol was used for control of acute rise in blood pressure at an initial dose of 25mg bolus given slowly intravenously, up to a cumulative dose of 300 mg in 24 hours.

2.3 Data Management

Early-onset preeclampsia has been considered as disease occurring before 32 weeks gestation in our hospital over several years. Immediate delivery in those with severe condition has been promoted through these years. This is usually done to obviate disease progression and occurrence of severe morbidity or mortality, especially in the face of what is frequently described as "a rapidly progressive disease course" in Nigerian women. In this study, we reviewed the presentation and outcome of our

patients introducing 34 weeks as the reference GA for defining early-onset preeclampsia.

The primary outcome measure was the occurrence of maternal death, while secondary outcome measures included the rates of eclampsia, stillbirth and early neonatal death. The medical records of these patients were retrieved from departmental obstetric data sheets, case notes as well as records of the laboratories, theatres, intensive care unit, renal unit and special care baby unit (SCBU). Socio-demographic, clinical and laboratory information retrieved was used to generate a database for analysis. The data obtained was subjected to statistical analysis with a personal computer using SPSS version 20.0 (SPSS IBM Corp, Armonk, NY) and GraphPad InStat 3 (GraphPad Software Inc., San Diego, CA). Univariate analysis was conducted using Chi-square test or Fisher's Exact Test as appropriate. Binary logistic regression was done to determine the contributions of confounders. P value < 0.05 was considered significant.

3. RESULTS

There were a total of 4,106 deliveries during the two-year study period. Out of 346 women managed for severe preeclampsia, 312 (96%) met the inclusion criteria. About a third (102) of them had eclampsia, giving an incidence of 2.5% (102/4,106) (Table 1). The unbooked women were in majority (190/312; 60.9%) and they accounted for 86% (88/102) of the eclamptics. In 70% (218/312) of them, Caesarean delivery was required, and only 5.1% (16/312) had assisted vaginal delivery (Information not in Table). There were 15 maternal deaths and 31 perinatal deaths (Table 1).

The median age of the women was 30 years (range: 19 to 41). The risk of eclampsia was 2.5-fold more at maternal age less than 20 years (77.8% vs 31.4; *P* = 0.00). Nulliparas were in majority (201/312; 64.3%), and 70% (71/102) of those who had eclampsia were nulliparas. The median GA at delivery was 35 weeks, while 88.1% **(**275/312) of them presented at GA 34 weeks or more (Table 1). Two-thirds of the eclamptics were at GA between 32 and 36 weeks (information not in Table).

In 20.5% (64/312) of the women, admitting mean arterial blood pressure (MABP) was at least 140 mmHg, and 21.8% (68/312) had proteinuria of at least 3+, while 32.7% (102/312) had no proteinuria. Severe anemia was found in 4.5% (14/312) of the women, while 7.7% (24/312) had packed cell volume (PCV) greater than 40%. (Table 1).

Eclampsia occurred 1.5-fold more with proteinuria of 3+ or more (44.0% vs 29.5%; *P* = 0.00) and 1.6-fold more with a MABP of 120 mmHg or more (39.1% vs 24.6%; *P* = 0.00). Eclampsia was also 1.6-fold more associated with severe anemia than PCV greater than 40% (71.4% vs 45.8; *P* = 0.00). Eclampsia was 1.1-fold less when delivery occurred before 34 weeks than beyond 34 weeks but this difference did not reach statistical significance (33.1% vs 29.7; *P* = 0.13) (Table 2).

Table 1. Frequency of clinical and laboratory parameters in the patients

Variable	Frequency (n=312)	Percentage
Eclampsia	102	32.69
GA at delivery (week)		
<32	14	4.49
32-33	23	7.37
34-36	155	49.67
>36	120	38.46
MABP at admission (mmHg)		
<100	12	3.84
100-119	123	39.42
120-139	113	36.21
>139	64	20.51
Maternal mortality	15	4.80
Perinatal death		
Stillbirth	11	3.53
ENND	20	6.41
Proteinuria at admission		
None	102	32.69
+	24	7.69
2+	118	37.82
3+	68	21.79
PCV at admission (%)		
<19	14	4.48
19-25	15	4.80
26-29	25	8.01
30-40	234	75.00
>40	24	7.69

Abbreviations: GA- Gestational Age, MABP- Mean Arterial Blood Pressure, PCV- Packed Cell Volume, ENND- Early Neonatal Death

Maternal death was 2-fold less in those admitted with MABP in excess of 120 mmHg (4.3% vs 2.1; *P* = 0.00). Severe anemia was 7.7-fold more associated with maternal mortality than higher PCV (28.5% vs 3.7; *P* = 0.00). Similarly,

maternal mortality was 9.5-fold more associated with proteinuria of at least 3+ (7.6% vs 0.8; *P* = 0.00). Only those who had eclampsia suffered maternal mortality (14.7% vs 0; *P* = 0.00). And delivery before 34 weeks was 3.2-fold more associated with maternal death than delivery beyond 34 weeks (16.2% vs 5.1; *P* = 0.00) (Table 2).

Table 2. Determinants of adverse maternal outcome

Outcome	Determinant	P value
Eclampsia	MABP ≥ 120 mmHg	0.003
	Proteinuria ≥ 3+	0.001
	PCV < 19%	0.001
	GA at delivery < 34weeks	0.138
Maternal mortality	MABP ≥ 120 mmHg	0.001
	Proteinuria ≥ 3+	0.001
	PCV < 19%	0.001
	Eclampsia	0.001
	GA at delivery < 34weeks	0.001

Abbreviations: GA- Gestational Age, MABP- Mean Arterial Blood Pressure, PCV- Packed Cell Volume

Stillbirth was 3.3% more in women admitted with MABP 120 mmHg or more though this difference was not statistically significant (7.0% vs 3.7; *P* = 0.60). Similarly, proteinuria of 3+ or more was 3.4% more associated with stillbirth (6.6% vs 3.2; *P* = 0.046). Severe anemia was 32.8% more associated with stillbirth than PCV of 30% and above (35.8% vs 3.0; *P* = 0.00) (Table 3).

Table 3. Determinants of adverse perinatal outcome

Outcome	Determinant	P value
Stillbirth	MABP ≥ 120 mmHg	0.597
	Proteinuria ≥ 3+	0.046
	PCV < 19%	0.001
	Eclampsia	0.001
	GA at delivery < 34 weeks	0.030
Early neonatal death	MABP ≥ 120 mmHg	0.181
	Proteinuria ≥ 3+	0.004
	PCV < 19%	0.001
	Eclampsia	0.019
	GA at delivery < 34 weeks	0.001

Abbreviations: GA- Gestational Age, MABP- Mean Arterial Blood Pressure, PCV- Packed Cell Volume

Stillbirth occurred 1.3% more in those with eclampsia (6.1% vs 4.8; *P* = 0.00). Stillbirth was also 5.7% more in women delivered beyond 34 weeks gestation (8.6% vs 2.9; *P* = 0.03) (Table 3).

Early neonatal death (ENND) occurred 3.6% more in those admitted with MABP in excess of 120 mmHg but the difference was not statistically significant (10.6% vs 7.0; *P* = 0.18). ENND also occurred 9.8% more with proteinuria of 3+ or more (11.4% vs 1.6; *P* = 0.00), 13.2% more with severe anemia (21.4% vs 8.2; *P* = 0.00), and 8.1% more in those with eclampsia (14.3% vs 6.2; *P* = 0.01). Furthermore, delivery before 34 weeks was 16.6% more associated with ENND (23.5% vs 6.9; *P* = 0.00) (Table 3).

A logistic regression analysis was done to predict the roles of PCV at admission, proteinuria at admission, MABP at admission and eclampsia as confounding variables in the determination of maternal mortality in the mothers, and ENND in the babies by GA at delivery. Prediction success overall for maternal mortality was 97.4% and for ENND was 95.8%.

The Wald criterion demonstrated that only MABP at admission made a contribution, though not significant (P=0.080), to the prediction of maternal mortality; however, all the variables were significant in the 'variables not in the equation' table (MABP: P=0.00; proteinuria: P=0.00; PCV: P=0.00; eclampsia: P=0.00 and GA: P=0.00); while PCV at admission, eclampsia and GA at delivery made significant contributions to the prediction of ENND (Table 4).

Table 4. Logistic regression table to predict maternal mortality and early neonatal death

Variable	P value	
	Maternal mortality	Early neonatal death
MABP	0.080	0.432
Proteinuria	0.710	0.375
PCV	0.999	0.001
Eclampsia	0.990	0.034
GA at delivery	0.756	0.001
Constant	0.993	0.003

Abbreviations: GA- Gestational Age, MABP- Mean Arterial Blood Pressure, PCV- Packed Cell Volume

4. DISCUSSION

The rate of eclampsia in this study was 2.5%. The majority of women studied were unbooked, nulliparous and young. Eclampsia was more likely with maternal age less than 20 years, nulliparity, unbooked status, and at GA 34 weeks

or higher. Higher rates of eclampsia, maternal mortality, stillbirth and ENND were significantly associated with severe proteinuria and severe anemia. Maternal mortality and ENND occurred more with deliveries before 34 weeks while eclampsia and stillbirth rates were higher at 34 weeks or more. Eclampsia, severe anemia and delivery below 34 weeks significantly contributed to the occurrence of ENND. Case fatality was 4.8% while perinatal mortality was 9.9%.

The contribution of inadequate antenatal attendance, young age and nulliparity to the development and severity of preeclampsia was shown in this study and same has often been documented in previous studies [17,18]. Similar to other reports, our findings also suggested a significant association of nulliparity and unbooked status with eclampsia [17-19]. The median GA at delivery for these women was 35 weeks. Our definition of early-onset preeclampsia is 32 weeks gestation, and this will suggest that most of our cases of severe preeclampsia occur as late-onset disease, as has often been alluded to by previous authors [19-21]. Even then, a redefinition in line with the current evidence and practice from elsewhere in Europe and America [10], where 34 weeks has been more commonly accepted as beginning of late-onset disease, will still accommodate most of our cases of severe preeclampsia.

The 2.5% incidence of eclampsia in this study is higher than 1.32% previously reported from this hospital by Onuh and Aisien [4], much higher than 0.9% found by Bhalerao et al. [17], but lower than 4.4% reported by Adamu et al. [18]. The risk of developing eclampsia was found to be significantly higher with severe hypertension and severe proteinuria, which agrees with the findings of Al-Mulhim and colleagues [19]. Severe anemia was also significantly associated with eclampsia, a similar finding to what was reported by Liu et al. [20]. GA at delivery less than 34 weeks did not significantly reduce the risk of eclampsia in our study population. This observation may suggest that occurrence of preeclampsia earlier than 34 weeks in our women does not confer a fulminant course, as has been often depicted by previous researchers [21-23]. This description of rapid progression has often been projected as the indication for immediate delivery without much consideration for conservative management in cases of severe disease. Expectedly, the fetal survival at lower GA may be a serious cause for concern when embarking on early delivery in our environment.

We found significantly higher rates of maternal mortality were associated with severe anemia and severe proteinuria. Previous studies have also associated these factors with adverse outcome [20,24]. On the other hand, severe hypertension at admission was found to be associated with lower risk of maternal death in this study. This observation is in sharp contrast to the findings of previous authors who have consistently highlighted the role of blood pressure (BP) control in mitigating the level of morbidity and mortality encountered in severe preeclampsia and eclampsia [19]. It is likely that aggressive treatment of hypertension improved outcome in those women who initially presented with severe hypertension, so that their eventual outcome reflected the level of blood pressure control achieved.

The contribution of eclampsia to maternal death in preeclampsia has been shown consistently in the literature [2-5], and this was also found in this study. Delivery at GA less than 34 weeks was significantly associated with a higher rate of maternal mortality in our series. In the report by Onah et al. [22], early-onset preeclampsia was noted to be associated with excess maternal death compared to late-onset disease. Similarly, Madazli et al. [21] showed that preeclampsia occurring late was unlikely to lead to maternal death as uterine Doppler abnormalities as well as serious maternal organ dysfunction is not commonly encountered.

Stillbirth rate was significantly higher in patients with severe hypertension, severe proteinuria and severe anemia. The role of these factors in adverse perinatal outcome has previously been documented by many researchers [19,20,24]. Similarly, the presence of eclampsia was shown to be associated with higher rates of stillbirth, which agrees with the report of previous authors [20]. The rate of stillbirth in women delivered at GA below 34 weeks was significantly lower than those delivered at higher GA. However, Madazli et al. [21] and Lisonkova [23] had earlier shown that stillbirth occurred significantly more in early-onset preeclampsia. Our observation is probably explained by the fact that most of our women had late-onset disease, and with a universal policy of immediate delivery, intrauterine demise is expected to be generally low. Furthermore, the toll on the fetus from the disease is likely to be more marked as gestation advanced.

Severe hypertension was associated with lower rates of ENND in our women in contrast to

previous reports which implicated severe disease with worse neonatal outcome [21,23]. Similar to the findings of Liu et al. [20], the proportion of babies who stayed alive up to 7 days after delivery was significantly reduced by the presence of severe anemia and occurrence of eclampsia in the mothers. Again, the survival of babies up to 7 days was more observed in women delivered at GA 34 weeks or higher. This better chance of neonatal survival beyond 34 weeks has been highlighted by authors working in our environment [22] and others working elsewhere [21,23]. In fact, a recent study noted that infants born at 34 weeks gestation were 18 times more likely to require oxygen supplementation, and over 19 times more likely to require assisted ventilation than those born at 38 to 40 weeks gestation [25]. Perinatal morbidity and mortality associated with lower GA have been linked to mainly prematurity and development of fetal growth restriction.

The contribution of GA at delivery to the rates of adverse maternal and perinatal outcome was tested with logistic regression analysis which revealed that none of the independent variables of severe hypertension, severe proteinuria, severe anemia, eclampsia and delivery below 34 weeks gestation significantly predicted the occurrence of maternal mortality. In contrast, eclampsia, severe anemia and delivery at GA less than 34 weeks significantly contributed to the presence of ENND. More babies stayed alive up to 7 days when delivery occurred at 34 weeks or higher. This again, probably reflects the overwhelming influence of fetal lung maturation in determining neonatal survival.

We have shown in this study that late-onset severe preeclampsia is still predominantly encountered in our practice, and that maternal outcome does not appear to differ between patients who are delivered earlier than 34 weeks or later. However, neonatal survival beyond 7 days is influenced by the timing of delivery, so that delivery after 34 weeks confers a survival advantage to the babies. In the light of recent evidence suggesting poorer neonatal outcome at GA below term [25], and the increasing likelihood that maternal morbidity and mortality are not excessive when cases are carefully selected for conservative management for the sake of the fetus [26], it has become important to review our policy of universal immediate delivery for severe preeclampsia occurring before 34 weeks. This will afford the opportunity to improve our neonatal survival figures, while heightening the surveillance on the mothers. There is also need to review our definition of early-onset preeclampsia in accordance with changing international practice. Uniform definition will be useful in documentation, future audit and international data comparison. However, the fear exists that preeclampsia in our environment is rapidly progressive. We, therefore, recommend a prospective study designed to evaluate the progression of mild and severe preeclampsia diagnosed preterm and classified into early and late onset disease, to confirm the existence of a cohort of women with rapidly progressive clinical course, and to determine the associations with such phenotype.

This study was retrospective, a design that may affect the validity of the results mainly because of inadequate documentation in case notes. However, the information retrieval in this study was made relatively easy and reliable because the computer-coded departmental obstetric data sheets captured details of management of most of our patients. The sample size of this study is adequate considering that a homogeneous group of severe preeclamptics was studied. Though this is a hospital-based study, its findings may be representative of the general obstetric population because of the referral status of our hospital.

5. CONCLUSION

In conclusion, severe preeclampsia is associated with significant maternal and perinatal adverse outcome. Delivery at or beyond 34 weeks confers improved neonatal outcome. We recommend an approach of selective conservative management till 34 weeks to improve neonatal survival rate. Clear endpoints for delivery should include eclampsia, placental abruption, inability to control hypertension, severe fetal growth restriction, deteriorating platelet count, non-reassuring fetal status, intravascular hemolysis, deteriorating liver function, deteriorating renal function, persistent neurological symptoms, persistent epigastric pain, nausea or vomiting, and pulmonary edema.

CONSENT

It is not applicable.

ETHICAL APPROVAL

This study was approved by the Research and Ethics Committee of the hospital.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. Obstet Gynecol. 2003;102: 181-92.
2. Kullima AA, Kawuwa MB, Audu BM, Usman H, Geidam AD. A 5-year review of maternal mortality associated with eclampsia in a tertiary institution in northern Nigeria. Ann Afr Med. 2009;8:81-4.
3. Onyiriuka AN, Okolo AA. Perinatal outcome in patients with preeclampsia in Benin City, Nigeria. Trop J Obstet Gynaecol. 2004;21:148-52.
4. Onuh SO, Aisien AO. Maternal and fetal outcome in eclamptic patients in Benin City, Nigeria. J Obstet Gynaecol. 2004; 24(7):765-8.
5. Omo-Aghoja LO, Aisien OA, Akuse JT, Bergstrom S, Okonofua FE. Maternal mortality and emergency obstetric care in Benin City, South-south Nigeria. J Clin Med Res. 2010;2(4):55-60.
6. 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 Gynaecol. 1994;171(3):818-22.
7. Publications Committee, Society for Maternal-Fetal Medicine, Sibai BM. Evaluation and management of severe preeclampsia before 34 weeks' gestation. Am J Obstet Gynaecol. 2011;205(3):191.
8. Lowe SA, Bowyer L, Lust K, McMahon LP, Morton M, North RA, et al. Guideline for the management of hypertensive disorders of pregnancy. Society of Obstetric medicine of Australia and New Zealand; 2014.
   Available:https://somanz.org/documents/HTPregnancyGuidelineJuly2014.pdf.
   (Last accessed 19th March, 2015)
9. Wikstrom AK, Larsson A, Eriksson UJ, Nash P, Norden-Lindeberg S, Olovsson M. Placental growth factor and soluble FMS-like tyrosine kinase-1 in early-onset and late-onset preeclampsia. Obstet Gynecol. 2007;109(6):1368-74.
10. Tranquilli AL, Brown MA, Zeeman GG, Dekker G, Sibai BM. The definition of severe and early-onset preeclampsia. Statements from the International Society for the Study of Hypertension in Pregnancy (ISSHP). Hypertens Pregnancy. 2013; 3(1):44-7.
11. Duddel GG, Jain L. Hypoxic respiratory failure in the late preterm infant. Clin Perinatol. 2006;33(4):803-30.
12. Engle WA. Late preterm infants: A population at risk. Pediatrics. 2008;121(2): 451.
13. Igberase GO. Perinatal mortality in a rural referral hospital in the Niger Delta, Nigeria. Afr J Med Health Sci. 2014;13:47-50.
14. Tukur J, Ahonsi B, Ishaka SM, Araoyinbo I, Okereke E, Babatunde AO. Maternal and fetal outcomes after introduction of magnesium sulphate for treatment of preeclampsia and eclampsia in selected secondary facilities: A low-cost intervention. Matern Child Health J. 2013; 17(7):1191-8.
15. Orhue AA. Active management of labour: A five-year experience from a university hospital in a developing country. J Obstet Gynaecol. 1997;17(Suppl11):S40
16. Zuspan FP. Problems encountered in the treatment of pregnancy-induced hypertension. Am J Obstet Gynecol. 1978; 131:591-7.
17. Bhalerao A, Kulkarni S, Ghike S, KawthalkarJoshi S, Somalwar S. Eclampsia: Maternal and fetal outcome. JSAFOG. 2013;5(1):19-21.
18. Adamu AN, Ekele BA, Ahmed Y, Mohammed BA, Isezuo SA, Abdullahpi AA. Pregnancy outcome in women with eclampsia at a tertiary centre in northern Nigeria. Afr J Med Med Sci. 2012;41(2): 211-9.
19. Al-Mulhim A-A, Abu-Heija A, Al-Jamma F, El-Harith el-HA. Preeclampsia: Maternal risk factors and perinatal outcome. Fetal Diagn Ther. 2003;18:275-80.
20. Liu S, Joseph KS, Liston RM, Bartholomew, Walker M, Leon JA, et al. Incidence, risk factors and associated complications of eclampsia. Obstet Gynaecol. 2011;118(5):987-94.
21. Madazli R, Yuksel MA, Imamoglu M, Tuten A, Oncul M, Aydin B, et al. Comparison of clinical and perinatal outcomes in early- and late-onset preeclampsia. Arch Gynaecol Obstet. 2014;290(1):53-7.

22. Onah HE, Iloabachie GC. Conservative management of early-onset preeclampsia and fetomaternal outcome in Nigerians. J Obstet Gynaecol. 2002;22(4):357-62.
23. Lisonkova S. Severe maternal morbidity associated with early- and late-onset preeclampsia. Abstract at the International Health Data Linkage Conference, held in Vancouver, Canada, 26 to 30 April, 2014. Available:http://www.ihdlnconference2014.org/abstract/Lisonkova
(Last accessed 14th March, 2015)
24. Franceschini N, Savitz DA, Kaufman JS. Maternal urine albumin excretion and pregnancy outcome. Am J Kidney Dis. 2005;45(6):1010-18.
25. Escobar GJ, Clark RH, Greene JD. Short-term outcomes of infants born at 35 and 36 weeks gestation: We need to ask more questions. Semin Perinatol. 2006;30(1): 28-33.
26. Oettle C, Hall D, Roux A, Grove D. Early onset severe preeclampsia: Expectant management at a secondary hospital in close association with a tertiary institution. BJOG. 2005;112(1):84-8.

Peer-review history:
The peer review history for this paper can be accessed here:
http://sciencedomain.org/review-history/10413