Orginal Article

Effects of Preeclampsia on Perinatal Outcome- A Study Done in the Specialized Urban Hospital Set Up in Bangladesh

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Abstract

Preeclampsia is a common pregnancy related disorder in Bangladesh that originates in the placenta and causes variable maternal and fetal problems. A prospective study was designed to see the associated maternal factors and fetal outcome in preeclampsia and to compare with that of normal pregnancy. The study was done in the Department of Obstetrics & Gynaecology, Dhaka Medical College Hospital and Mitford Hospital, Dhaka, from August 2005 to June 2006. Sixty Bangladeshi pregnant women were taken in this study, of which thirty were normal uncomplicated pregnancies (considered as control group or group A) and another thirty having pregnancies complicated by preeclampsia (considered as preeclampsia group or group B), where the patients were normotensive previously. Both the maternal and fetal outcomes were observed and recorded. The mean age of the mother was 26.53±5.26 years and 26.67±5.27 years in group A and group B respectively. The mean gestational age of the mother was 38.27±1.26 weeks and 36.90±1.03 weeks in group A and group B respectively and the difference in between two groups was statistically significant ($p<0.001$). The mean birth weight of the neonate was 2.80±0.27 kg. and 2.26±0.41 kg. in group A and group B respectively and the difference was significant ($p<0.001$). The mean APGAR score of the neonate at one minute was 9.00±1.02 and 8.40±0.93 in group A and group B respectively and the difference was significant ($p<0.05$).

Keywords: Preeclampsia, maternal factors, perinatal outcome, birth weight, APGAR score.

Introduction

Preeclampsia is a relatively common pregnancy disorder that originates in the placenta and causes variable maternal and fetal problems.[1] This condition is characterized by an elevation in blood pressure, proteinuria and by a reduction in plasma volume, an increase in peripheral resistance and a generalized vasoconstriction. It usually develops after 20 weeks of gestation and resolves after delivery of placenta.[2] It affects 3-5% of pregnancies and is a leading cause of maternal and perinatal mortality[3] and an important factor in fetal growth retardation as it is commonly associated with placental insufficiency.[4] Approximately 10-15% of maternal deaths in developing countries are associated with preeclampsia leading to eclampsia.[5]

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There is no concrete data found on incidence of preeclampsia in our country, but calculated from the US Census Bureau, International Data Base, 2004, the extrapolated annual incidence of preeclampsia in Bangladesh is 76,032.[6] Our neighbouring country, India has the incidence of preeclampsia, as recorded from hospital statistics, varying widely from 5 to 15%.[7] Bangladesh, the most densely populated country in South East Asia has high maternal mortality as well as fetal mortality rate. According to Bangladesh Demographic and Health Survey 2004, the maternal mortality rate (MMR) is 3.21 per 1000 live births and neonatal mortality rate (NMR) is 41 per 1000 live births.[8] There are number of causes for this high maternal mortality rate. Preeclampsia, being one of them, is well known to complicate pregnancy and thus contribute to maternal and fetal death. Moreover, a primigravida or women pregnant for the first time is at higher risk of developing preeclampsia than women in subsequent pregnancies.[8,9] Proteinuria and increased blood pressure in preeclampsia are associated with a lower fetal birth weight and a lower APGAR score and an increased risk of adverse perinatal outcome. The present study was aimed to correlate preeclampsia with maternal age, gestational age, parity and to see the fetal outcome e.g. neonatal weight and APGAR score.

Methods

A prospective study was carried out in the Department of Obstetrics and Gynaecology, Dhaka Medical College Hospital and Mitford Hospital,

Dhaka, from August 2005 to June 2006 on 60 Bangladeshi women, of which 30 having normal uncomplicated pregnancies (considered as control group or group A) and another 30 from pregnancies complicated by preeclampsia (considered as preeclampsia group or group B), where the patients were normotensive previously. Data of perinatal outcome were collected from the delivery notes (either normal vaginal delivery or Caesarean section), conducted in the Department of Obstetrics & Gynaecology, Dhaka Medical College Hospital and Mitford Hospital, Dhaka. Pregnant women were selected between 35-40 weeks of gestation. Selection of the control and the study group was done on the basis of diagnosis by a registered physician or from the hospital record.

Common exclusion criteria

1. Chronic hypertension (hypertension before current pregnancy)
2. Eclampsia
3. Pregnancy induced hypertension
4. Placenta Praevia
5. Antepartum haemorrhage (APH)
6. Presence of Diabetes Mellitus
7. Rh-negative mother
8. VDRL-positive mother
9. Gestational age below 35 weeks and above 40 weeks.

Exclusion criteria were followed meticulously as per hospital records and investigations.

Statistical processing of data

The collected data were processed and statistical analyses were done by unpaired Student's 't' test. All the statistical analyses were done by using the SPSS 11.0 version.

Results

Maternal variables

The mean age of the mother was 26.53±5.26 years and 26.67±5.27 years in group A and group B respectively. No difference was found in age of mothers between two groups. The mean gestational age of the mother was 38.27±1.26 weeks and 36.90±1.03 weeks in group A and group B respectively. The mean difference in gestational age of mothers between two groups was statistically significant ($p<0.001$) (Fig.1). It was found that in group A, 14 (46.7%) were primiparous and 16 (53.3%) were multiparous women, and in group B, 19 (63.3%) were primiparous and 11 (36.7%) were multiparous women (Fig.2).

Neonatal variables

The mean birth weight of the neonate was 2.80±0.27 kg. and 2.26±0.41 kg. in group A and group B respectively. The mean difference between the two groups was significant at $p<0.001$. The mean APGAR score of the neonate at one minute was 9.00±1.02 and 8.40±0.93 in group A and group B respectively. Statistical analysis between the two groups was significant as $p<0.05$. (Fig.3).

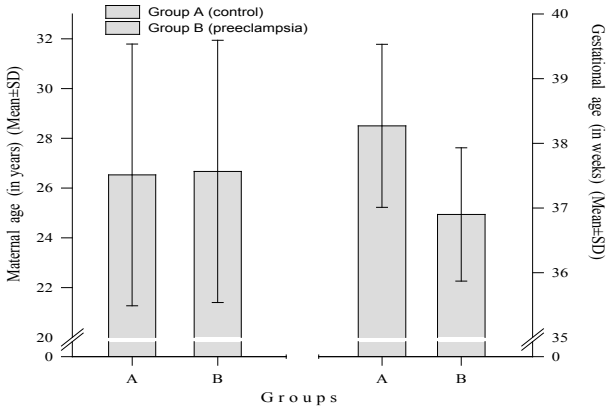

Fig. 1: Maternal age and gestational age in normal (control) and preeclamptic women.

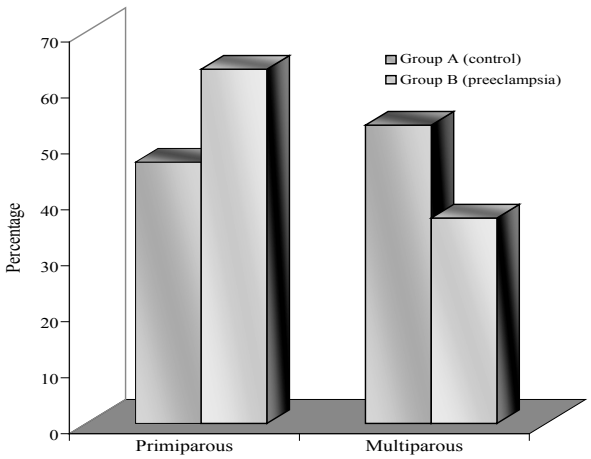

Fig. 2: Parity distribution in normal (control) and preeclamptic women.

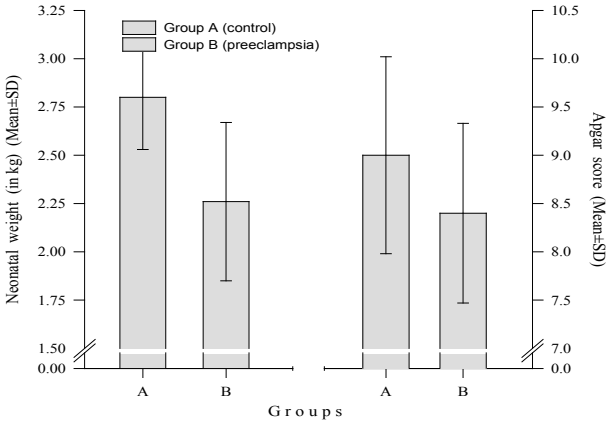

Fig. 3: Neonatal weight and APGAR score found after delivery of normal (control) and preeclamptic women.

Discussion

Moldenhauer et al (2003)[10] stated that preeclampsia affects 6% to 7% of nulliparous women. The incidence of preeclampsia increases as pregnancy approaches term, with the majority of cases developing at or near term. The development of mild hypertension or preeclampsia at or near term is associated with minimal maternal and neonatal morbidities. In contrast, the onset of severe preeclampsia before 35 weeks' gestation is associated with significant maternal and perinatal complications. Barua (2002)[11] found mean gestational age of eclampsia group was 37.55 weeks and that of control group was 38.60 weeks. Boyd and Scott (1985)[12] found that preclampsia group was significantly more likely to be primiparous than control group. They also found that the mean gestational age of preeclampsia group was 33.4 weeks and that of normal group was 34.5 weeks. The findings of the present study on gestational age in preeclampsia correlate the previous findings. The present study has also shown that neonatal birth weight has been reduced in pregnancies complicated by preeclampsia. Mayhew et al. (2003)[13] who studied placental morphology in pregnancies complicated by preeclampsia with or without intrauterine growth restriction and observed that fetal weights were reduced in all complicated pregnancies but only intrauterine growth restriction was accompanied by a significantly smaller placenta. Barua (2002)[11] had an observation on gestational diabetes and eclampsia. She found the mean neonatal weight in the control and eclampsia group was 2940 gm and 1940 gm respectively. Barton et al. (2001)[14] observed that the mean birth weight of the neonate was significantly lower in the group with preeclampsia than that of gestational hypertension. Odegard et al. (2000)[15] found that preeclampsia was associated with a 5% reduction in birth weight. In severe preeclampsia, the reduction was 12% and in early onset disease, birth weight was 23% lower than expected. The risk of small for gestational age (SGA) was four times higher in infants born after preeclampsia than in control pregnancies. Among nulliparous, preeclampsia was associated with a nearly threefold higher risk of SGA than that of normal pregnancy. However, in multiparous women, the risk of SGA was particularly high after recurrent preeclampsia. In the year 1985, Teasdale studied with 10 placentae of which 5 from primigravid women with severe preeclampsia and 5 from normal healthy women. He found that the infants of the preeclampsia group were significantly smaller than the control infants in terms of the mean birth weights i.e. 2466 gm and 3315 gm respectively ($p<0.001$).[16] Later in 1987, he again examined 10 placentae of which 5 from women of second gravida with severe preeclampsia and 5 from healthy women. He found that severe preeclampsia group gave birth to infants who were severely growth retarded with birth weights that ranged between 1330 and 1960 gm. On the other hand, infants from healthy women were appropriate for gestational age (ranging from 3000 to 3335 gm).[17] Boyd and Scott (1985)12 observed that the small for dates' (SFD) infants and those from pregnancies complicated by preeclampsia had significantly reduced birth weight compared to those from normal pregnancies. The mean infant birth weight was 2714 gm and 1998 gm in control and preeclampsia group respectively, as studied within 35-38 weeks of gestational age. The underlying cause was described by Soma et al. (1982)[4], who suggested that low birth weight of the neonate in hypertensive pregnancy was due to placental insufficiency resulting in fetal growth retardation. Cibils (1974)[18] found that the infants born to normal patients weighed an average of 3321 grams. They lost an average of only 4.14 per cent of their weight in the first few days of life, and this was regained within the fifth to seventh days. Those born to mothers with transient or moderate hypertension weighed an average of 2770 grams. The infants born to mothers with severe or chronic hypertension weighed an average of 2353 grams. The last two figures are much lower than the normal average and are indirect evidence of the deficient somatic growth of these infants who presented the so-called "intrauterine growth retardation syndrome". They found invariably a low APGAR score for those newborn. Masodkar et al. (1985)[19] also observed a low APGAR score in toxaemia of pregnancy. The present study has got similar findings as well.

Conclusion

Preeclampsia is a largely preventable condition and the maternal mortality is decreasing, but the perinatal mortality still remains very high (7-10%) even in the developed countries7. As preeclampsia contributes to the high mortality and morbidity of both mothers and neonates in our country, proper antenatal care must be given to all pregnant women to prevent and screen for preeclampsia. Measures should be taken to control this deadly condition through behavioural change communication (BCC) regarding antenatal care, danger signs, delivery plan etc, involving both public and private sectors.

Acknowledgement

We would like to express our gratitude to the participants of the study and special thanks to the authorities of Dhaka Medical College Hospital and Mitford Hospital, Dhaka for their kind permission to carry out the study.

References

1. Redman CW, Sargent IL. Latest advances in understanding preeclampsia. Science. 2005; 308(5728): 1592-4.

2. VanWijk MJ, Boer K, van der Meulen ET, Bleker OP, Spaan JA, VanBavel E. Resistance artery smooth muscle function in pregnancy and preeclampsia. Am J Obstet Gynecol. 2002; 186(1): 148-54.

3. Roberts JM, Cooper DW. Pathogenesis and genetics of pre-eclampsia. Lancet. 2001; 357(9249): 53-6.

4. Soma H, Yoshida K, Mukaida T, Tabuchi Y. Morphological changes in the hypertensive placenta. Contrib Gynecol Obstet. 1982; 9: 58-75.

5. Duley L. Maternal mortality associated with hypertensive disorders of pregnancy in Africa, Asia, Latin America and the Caribbean. Br J Obstet Gynaecol. 1992; 99(7): 547-53.

6. Statistics by country for preeclampsia: extrapolation of incidence rate for preeclampsia to countries and regions. Available from: http://www.wrongdiagnosis.com/p/preeclampsia/sta

ts-country.htm [accessed on 15th April, 2010].

7. Dutta DC. Hypertensive disorders in pregnancy. In: Text book of obstetrics including perinatology and contraception. 6th ed. Calcutta: New Central Book Agency; 2004. p. 221-42.

8. Bangladesh demographic and health survey: summary indicators. National Institute of Population Research and Training (NIPORT), Dhaka, Bangladesh. 2005.

9. Dhananjay BS, Dayananda G, Sendilkumaran D, Murthy N. A study of factors affecting perinatal mortality in eclampsia. J Physiol Biomed Sci. 2009; 22(2): 2-5.

10. Moldenhauer JS, Stanek J, Warshak C, Khoury J, Sibai B. The frequency and severity of placental findings in women with preeclampsia are gestational age dependent. Am J Obstet Gynecol. 2003; 189(4): 1173-7.

11. Barua R. Macroscopic and microscopic changes in human placenta in gestational diabetes and eclampsia [thesis]. Dhaka: BSMMU; 2002.

12. Boyd PA, Scott A. Quantitative structural studies on human placentas associated with preeclampsia, essential hypertension and intrauterine growth retardation. Br J Obstet Gynaecol. 1985; 92: 714-21.

13. Mayhew TM, Ohadike C, Baker PN, Crocker IP, Mitchell C, Ong SS. Stereological investigation of placental morphology in pregnancies complicated by preeclampsia with and without intrauterine growth restriction. Placenta. 2003; 24: 219-26.

14. Barton JR. O'Brien JM, Bergauer NK, Jacques DL, Sibai BM. Mild gestational hypertension remote from term: progression and outcome. Am J Obstet Gynecol. 2001; 184(5): 979-83.

15. Odegård RA, Vatten LJ, Nilsen ST, Salvesen KA, Austgulen R. Preeclampsia and fetal growth. Obstet Gynecol. 2000; 96(6): 950-5.

16. Teasdale F. Histomorphometry of the human placenta in maternal preeclampsia. Am J Obstet Gynecol. 1985; 152: 25-31.

17. Teasdale F. Histomorphometry of the human placenta in preeclampsia, associated with severe intrauterine growth retardation. Placenta. 1987; 8: 119-28.

18. Ciblis LA. The placenta and newborn infant in hypertensive conditions. Am J Obstet Gynecol. 1974; 118(2): 256-70.

19. Masodkar AR, Kalamkar LR, Patki PS. Histopathology of placenta and its correlation with foetal outcome. J Obstet Gynecol India. 1985; 35: 294-300.