Foetal/Neonatal Survival with Hypertensive Disorders during Pregnancy

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Introduction

Hypertensive disorders during pregnancy, dangerous, multifactorial disorders with multigorgan effects on the mother and baby, continue to occur globally. Baby gets affected in many ways. Etiology, course of disorders, effects on the baby, interventions and medication which affect the baby, are not well understood. Also effects on future life of baby are not very well known. Though perinatal survival is poorly affected because of many reasons, effects on baby depend on duration of hypertension, gestation at occurrence, at diagnosis, at therapy, effects of HDsP on mother, interventions, therapy and gestation at delivery. In addition foetal and new born survival also depend on mode of delivery and neonatal care, available and used. They continue to be persisting dilemmas. So research about various issues continues globally Various studies are being done about prediction, prevention and best therapy so that at least fatality is prevented, till the time HDsP and their severity can be prevented.

Some local research has been done in relation to foetal/ neonatal survival. A study (published), was done to know the differences in the fetal/ neonatal survival in cases of HDsP in relation to gestation at diagnosis of HDsP and gestation at delivery. In the study 1046 cases of HDPs, all the women admitted beyond 20 weeks of pregnancy with HDsP were followed. These cases were divided into Early Onset (EO), all cases beyond 20 weeks of pregnant to less than 34 weeks of pregnancy and Late Onset (LO) women with HDsP, 34 weeks of pregnancy onwards EO cases were further divided in two, more than 20 weeks to less than 28 weeks, Group A, 28 weeks to less than 34 weeks, Group B. Similarly LO cases were further divided into completed 34 weeks to less than 37 weeks. Group C, and more than 37 weeks , Group D. Mean gestation at birth in Group A. EO HDsP cases (> 20 - <28 weeks pregnancy ) was 30+1 weeks, in Group B EO(≥28 - <34 weeks), 32+6 weeks. In LO, Group C (≥34-<37 weeks), 35+4 weeks and in LO, Group D (≥37 weeks), it was 38+4 weeks. In Group A there were 97.5% preterm births, with caesarean section rate (CSR) of 42.5%. In Group B. there were 84.29%, preterm births with CSR of 53.93% in Group C 31.37%, preterm births and CSR of 40.63%, and in Group D, CSR was 46%.

Mean birth weight in Group A was 1741.54 gms, B 1936.31 gms, C 2633.38 gms and in D 2677.30 gms. In Group A there were 45%, perinatal deaths. However perinatal deaths were 100% in births before 28 weeks, with 100% survival if pregnancy continued till term, but it was only in 2 women in whom the disease was diagnosed before 28 weeks, (Group A EO). In Group B 25.13% perinatal deaths occurred, in Group C 14.32%, and in and D also 14.00% in Group D. Critical gestation at birth for neonatal survival was little beyond 32 weeks. In babies of mothers of Group C and D perinatal-mortality was similar. Peguero [1] did a study to know about added prognostic value of longitudinal changes of angiogenic factors in early- onset severe preeclampsia, for the prediction of adverse outcome and reported that levels of placental growth factor [PIGF], soluble fms-like tyrosine kinase sFlt-1 and s Flt-1/PIGF ratio. added to baseline characteristics in the prediction of adverse outcome, specially in EO cases. In another local study of HDsP in women with complete HELLP or partial HELLP, perinatal mortality rate was 275 in women with HDPs and HELLP or partial HELLP. In women with HDsP without HELLP it was 110. Overall PMR in women with HDPs was 150. During the same period PMR was 50. HELLP in the mother affected the baby very badly.

In yet another analysis of cases of Eclampsia where conservative treatment was tried in women in whom convulsions occurred at less than 34weeks of pregnancy and mother’s condition improved after being given therapy This was to try increase the chances of neonatal survival in neonatal intensive care areas with limited resources. In the study 33 women were kept on conservative management. They got admitted with eclampsia at less than 34 weeks of pregnancy with live baby and without any anomalies, which could be diagnosed with sonography and convulsions responded to treatment with no onset of labour pains and mothers doing well. As there was no problem with the mother, labour was not induced. Induction was done either when intrauterine death occurred, or pregnancy reached beyond 34 weeks to maximum completed 37 weeks. Overall 14 intra uterine deaths occurred. After spontaneous labor one fresh still birth and three neonatal deaths occurred. However 15 mothers went home with healthy babies. All 33 mothers remained healthy. Baby weight/survival got affected by prematurity and dysmaturity. Interval to delivery by pregnancy prolongation in early-onset preeclampsia was associated with improved perinatal outcome and survival in other studies too. These effects did not appear to be
deleterious to short-term maternal cardiovascular and metabolic function, but were associated with a modest increase in risk of residual albuminuria. In another local study of HDsP in which both lipid and glucose metabolism were studied for relationship to severity of disease and baby weight, it was found that severity of HDsP and glucose and lipid metabolism derangement mostly went parallel. However baby weight got badly affected with glucose and lipid metabolism derangement, even in otherwise less severe cases of HDsP.

A study by Behrman2 showed that the risk of delayed neuro developmental and neonatal death decreased with increased gestational age, even in late preterm births. By various tests. Prediction is being tried to know risk factors in view of the dangers of HDsP. Glycosylated fibronectin (GlyFn) is being investigated for late onset cases. There is no single test which can surely predict the disorder. So prevention has limitations presently. Not only attempts are being made to prevent the HDsP but also their severity and fatality of mother and baby too by various means. Vitamin C, vitamin E have been tried.

Many other things including Vit. D, Low-molecular-weight heparin have also been studied with no effect on onset and severity. Calcium large doses were advocated but has become controversial. Aspirin is being tried for prevention of HDsP and seems to be beneficial, so continues to be recommended. Controversy now is when to start Aspirin and what is the optimum dose. Also there are nonresponders. Since HDsP are dangerous both for the mother as well as the baby and are common globally, attempts continue to be made for prediction and try prevention accordingly. More research is needed about various aspects of HDsP.

References:
