Incidence of Intrauterine Growth Restriction and Perinatal Outcome in Pregnancy Induced Hypertension

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Abstract

Introduction: Pregnancy induced hypertension is associated with increased incidence of uteroplacental insufficiency and intrauterine growth restriction. Incidence and severity of IUGR depends upon the severity of hypertension or preeclampsia, onset of hypertension or preeclampsia in relation to gestational age, duration of hypertension and associated complications.

Methods: The study was conducted in the Department of Obstetrics and Gynecology at a tertiary care teaching hospital in Rangareddy District, Telangana State. It was a prospective observational study. All antenatal patients attending outpatient department at Hospital were screened for hypertension and those having blood pressure \geq 140/90 mm of Hg on two occasions at an interval of 4-6 hours presenting after 20 weeks of gestation are taken into study group.

Results: Among the 50 cases of pregnancy induced hypertension 29 (58%)were gestational hypertension, 17 (34%) were pre-eclampsia, 3 (6%) were eclampsia, 1 (2%) pre-eclampsia superimposed on chronic hypertension. Respiratory distress was found in majority of cases i.e 62.5% of babiesassociated with IUGR compared to 17% of babies not associated with IUGR. 75% of babies affected by IUGR required NICU admission compared to 38% of babies not affected by IUGR. There were no perinatal deaths in PIH cases not associated with IUGR in the present study compared to 25% in babies affected by IUGR.

Conclusions: IUGR is the most important complication of PIH, which is responsible for increasedperinatal morbidity and mortality. Majority of these conditions are preventable with good antenatal care.

Keywords: Pregnancy induced hypertension (PIH), Perinatal outcome, Intrauterine growth restriction (IUGR)

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I. Introduction

Intra uterine fetal growth restriction (IUGR) is a condition where the fetus fails toachieve its genetic growth potential and consequently is at risk of increased perinatalmorbidity and mortality.¹

IUGR is defined as any baby with birth weight below 10th percentile for the gestationalage or two standard deviations below the population mean who fails to achieve itsgrowth potential.²

IUGR is clinically important because the fetus with IUGR is at increased risk of perinatalmorbidity and mortality. This can be mitigated by appropriate fetal surveillance and timely delivery. IUGR fetuses are frequently described as symmetric or asymmetric in terms of theirbody proportion.

Symmetrically small fetuses are usually associated with factors that directly impair theintrauterine growth potential of fetus (i.e., chromosomal abnormalities, viral infectionsetc.), while asymmetric growth restriction is classically associated with uteroplacentalinsufficiency.

Hypertension is a common medical problem that affects 20% -30% of the adultpopulation and more than 5% - 8% of all pregnancies in the world. Hypertensivedisorders of pregnancy are most common causes of

maternal mortality and morbidity.³The fetuses of hypertensive mothers are at increased risk for IUGR, premature delivery, placental abruption, still birth and neonatal deaths.

The American college of Obstetricians and Gynecologists (ACOG) has classifiedPregnancy induced hypertension (PIH) into four groups of disorders ³:

• Gestational hypertension: where resting blood pressure is 140/90 mm Hg orhigher after the 20th week of gestation;

• Chronic hypertension: if hypertension exists before pregnancy or begins in thefirst 20 weeks of gestation;

• Pre-eclampsia- where hypertension is associated with edema or proteinuria;

• Eclampsia – where preeclampsia is associated with convulsions;

• Preeclampsia superimposed on chronic hypertension.

If preeclampsia develops before 34 weeks of gestation it is called early onsetpreeclampsia.

If it develops after 34 weeks of gestation it is called late onset preeclampsia.

If BP is between 140/90 mmHg and < 159/109 mmHg, It is classified as mildpreeclampsia.

If BP is \geq 160/110 mmHg. It is classified as severe preeclampsia.

Pregnancy induced hypertension, both protienuric and non protienuric, is associated with increased incidence of uteroplacental insufficiency and intrauterine growthrestriction.

Incidence and severity of IUGR depends upon the severity of hypertension orpreeclampsia, onset of hypertension or preeclampsia in relation to gestational age, duration of hypertension and associated complications. The most important consequences of IUGR are fetal compromise with perinatal morbidity and mortality, principally still birth.^{4,5}

Ultrasound remains the best method for diagnosis, characterization and follow up of IUGR. Single estimate of fetal size, amniotic fluid volume, umbilical artery resistance are poor predictors of IUGR. Serial estimation of the fetal weight and abdominal circumference are more useful to detect the growth lag in the fetus.

Hence it is necessary to identify the growth restricted fetuses prior to delivery in order toreduce the perinatal morbidity and mortality by intensive surveillance and appropriate and timely intervention.

Objectives of the study

• To study the incidence of IUGR in pregnancy induced hypertension.

• To study the effects of various factors in development of IUGR like age, parity, socioeconomic status, onset of PIH in relation to gestational age and duration of PIH.

II. Patients And Methods:

The study was conducted in the Department of Obstetrics and Gynecology at a tertiary care teaching hospital inRangareddyDistrict, Telangana State.

It was a prospective observational study over a period of 18 months from January 2016 toJuly 2017.

Study population: All antenatal patients attending outpatient department at Hospital were screened for hypertension and those having blood pressure $\geq 140/90$ mm of Hg on two occasions at an interval of 4-6 hours presenting after 20 weeks of gestation are taken into study group.

Inclusion criteria:

• All diagnosed cases of pregnancy induced hypertension (raised blood pressure of \geq 140/90 mm of Hg on two occasions at an interval of 4-6 hours including both proteinuric and non proteinuric) between 20-40 weeks of gestation.

• Singleton pregnancies.

Exclusion criteria:

• Congenital anomalies, Multiple pregnancy

• Cases of chronic hypertension, Renal diseases, Adrenal diseases, diabetes mellitus, connective tissuedisorders, APLA, Other causes of secondary hypertension, Essential hypertension, sickle cell anemia and known case of epilepsy.

Sample size was 50 cases of pregnancy induced hypertension; which included gestational hypertension, pre-eclampsia, eclampsia and pre-eclampsia superimposed on chronic hypertension.

Women fulfilling the selection criteria were included in the study after taking writteninformed consent. Patients name, age , husband's name, socioeconomic status, residential address were recorded. Detailed medical history of patient was taken regardinghypertension, history of pregnancy induced hypertension in previous pregnancies, diabetes, chronic kidney disease, connective tissue disorders or sickle cell anemia.

Detailed obstetric history regarding gravidity, parity, LMP,EDD were noted. Weight of thepatient was recorded at the booking visit and at every subsequent visits.Patient's vitals wererecorded and systemic examination carried out in detail. When patient was diagnosed tohave high blood pressure, she was monitored at frequent intervals i.e, every week forblood pressure status and fetal status. At each visit along with blood pressure urine waschecked for presence of protienuria. Mild PIH cases were monitored on outpatient basis.

When the patient is found to have high blood pressure she was admitted in hospital forevaluation and close monitoring. Fetal surveillance was done to prevent complications of effectively controlling blood pressure.

All the patients with blood pressure \geq 150/100 mm of Hg, the following investigationswere done to rule out hematological, hepatic, renal and coagulation abnormalities and repeated periodically depending upon the results -

• Complete blood picture with platelet count, Complete urine examination, Blood grouping and Rh typing, Bleeding time, clotting time, HIV 1 & 2, HBsAg, VDRL, Thyroid profile, Screening blood sugar,Liver function tests,Blood urea,Sr. creatinine,Sr. uric acid,PT INR, APTT.

USG of gravid uterus for-

Fetal lie, Fetal position, Gestational age, Biometric parameters- BPD, HC, AC, FL, EFW. AFI, Placental location and grading, Growth chart at an interval of 3-4 weeks, Fetal Doppler, Fetal biophysical profile.

Patients were monitored for the development of symptoms like headache, visualdisturbances, epigastric pain, oliguria or convulsions.

For assessing the growth of the fetus, accurate gestational age was calculated by noting the last menstrual period provided the dates were good or excellent and by doingUSG examination during 1st trimester or early 2nd trimester depending upon thepatient's booking visit.

Growth of the fetus is assessed by measuring the symphysiofundal height and bymeasuring the biometric parameters and EFW on USG examination, periodically at aninterval of 3-4 weeks. The estimated fetal weight was plotted on the growth charts.

When the estimated fetal weight is < 10th centile for the gestational age, the babies werenoted as growth restricted and intensive monitoring was carried out. Growth scan wasdone every two weeks to know the rate of growth and to identify if there is any growthlag. Well being of the fetus, identified as growth restricted is monitored by daily fetal kickcount and weekly biophysical profile and fetal Doppler studies. Non stress test wasperformed twice in a week to identify any early features of intrauterine hypoxia. Timeand mode of delivery was decided by the gestational age and the results of the Dopplervelocimetry of fetal vessels and NST.

For the patients who were allowed vaginal delivery, continuous cardiotocographymonitoring was done.Decision to take emergency caesarian section was made in theevent of non-reassuring fetal heart rate or development of any other complication likeaccidental hemorrhage, PROM with non-progression of labour, etc. Elective caesareansection was done for the following indications: Severe uncontrolled hypertension, Severeoligohydramnios, Abnormal fetal umbilical artery Doppler with absent diastolic flow.

At the time of delivery either vaginal or by caesarian section, the neonate'sAPGAR score was recorded. Weight, sex, placental weight, any infarcts in placentawere noted. Presence or absence of meconium in the liquor, staining of the cord and placenta noted. Third stage of labour was managed by standard protocol.

Baby's outcome was noted by recording whether it required admission to NICU, development of HIE and perinatal death.

Statistical analysis:Data entry was done by Microsoft Excel 2010 version and analysis using EPI INFO version 7. Data was presented in percentages and proportions. Association between categorical variables was done using Chi-square test with p value less than 0.05 was considered statistically significant.

III. Results

In the present study, 42 cases (84%) of the total cases of pregnancy induced hypertension were in the age group of 19-25 years and two thirds (n=33) belonged tolow socioeconomic group. Majority of the patients, 43(86%) cases had BMI between 21 and 29.9 kg/m².

With regards to Gravida, 40 (80%) PIH cases were primigravidae. Multigravida constituted only 20%. In the present study, among 13 cases of PIH with the onset before 34 weeks ofgestation, 46% developed IUGR in the fetus. Among the patients who developed PIHbetween 34 and 36+6 weeks, 28% developed IUGR and among the cases whodeveloped PIH after 37 weeks of gestation only 22% developed IUGR. This implies that the development of IUGR is more common in the early onsetpregnancy induced hypertension.

Among the 50 cases of pregnancy induced hypertension 29 (58%)were gestational hypertension, 17 (34%) were pre-eclampsia, 3 (6%) were eclampsia.One case (2%) had pre-eclampsia superimposed on chronic hypertension. Among these 20 of gestational hypertension cases, 11 of pre-eclampsia cases, 2 of eclampsiacases were not affected by IUGR. Whereas 9 of gestational hypertension cases, 6 of preeclamsia cases and one of eclampsia case developed IUGR. The only one case with pre-eclampsia superimposed on chronic hypertension did not have IUGR.

Amniotic fluid index was more than 8 cm in 34 cases of PIH not associated with IUGR whereas among the cases associated with IUGR 16(100%), 5 cases had an AFI > 8 cm, 9 (56.25%) cases had AFI between 6 and 7 cm and 2 cases (12.5 %) had AFI lessthan 5 cm.

With regards to admission to delivery interval, in 50% of the PIH cases not affected with IUGR, the pregnancywas prolonged for 2 to 4 weeks. Where as in PIH cases associated with IUGR thepregnancy was

prolonged for 2 to 4 weeks only in 25 % of the cases. In one case withIUGR, the pregnancy was prolonged for 5 weeks. In 2 cases (5.8%) of PIH without IUGRpregnancy was prolonged for 5 weeks and in another 2 (5.8%) cases the pregnancywas prolonged for 6 weeks. Delivery was conducted immediately in 32% cases of PIH without IUGR and 43% cases of PIH with IUGR.

Among 50 cases of PIH, 11 (22%) cases had spontaneous vaginaldelivery, 32 (64%) cases had lower segment caesarian section, 7 (14%) cases hadinstrumental vaginal delivery. Among the PIH cases which were not affected by IUGR 19 (55.88%) cases underwent lower segment caesarian section, whereas (44%) caseshad vaginal and instrumental vaginal delivery put together. Among PIH cases who wereaffected by IUGR 13 (81.25%) underwent lower segment caesarian section and only 3(18%) had vaginal delivery.

Among the 32 cases who underwent lower segment caesarian section, indications are PROM and failure to progress in 6 (12%) cases, meconium stainedliquor in 7 (14%) cases, non-reassuring fetal heart rate in 17 (34%) cases and breechpresentation in 2 (4%) cases. The overall caesarian section rate in PIH cases notassociated with IUGR was 55.8% (9 cases) whereas the overall caesarian section rate in PIH cases associated with IUGR was 81.2% (13 cases). Commonest indication inboth the groups was non reassuring fetal heart rate followed by meconium stainedliquor and failure to progress.

Distribution of birth weight of the babies in relation to PIH and IUGR: Majority of babies, 35 (70%)cases, had birth weight between 2500 to 3499 gm. The birth weight of the babies of PIHcases not affected by IUGR was between 2500 and 3499 gm in 97% of the cases. Among the babies affected by IUGR, 10 (62.5%) babies weighed between 2000 and2499 gm, 2 (12.5%) cases weighed between 1000 and 1999 gm and 2 (12.5%) caseshad birth weight between 2500 to 2999gm and the association was significant statistically. The mean birth weight in PIH cases not associated with IUGR was 2830gm and meanbirth weight of PIH cases affected by IUGR was 2050gm.

Distribution of PIH and IUGR cases based on APGAR at 5 min: APGAR score at 5 minutes was between 8 to 10 in 28 (56%)cases of PIH cases and between 4 to 7 in 20 (40%) cases. Among 34 cases of PIH not affected by IUGR APGAR score at 5 minutes was between 8 and 10 in 28 (82.35%) cases and in 6 (17.46%) of cases the APGAR score at 5 minutesbetween 4 and 7 . Among the 16 PIH cases affected by IUGR, 14 cases (87%) with APGAR score at 5 minutes were between 4 and 7 and there were 2 cases (12.5%) of still birth. There was statistically significant difference between the two groups.

<u>Distribution of respiratory distress in relation to PIH and IUGR</u>: Over all there was incidence of 16 (32%) of respiratory distress atbirth among the babies of 50 PIH cases and only 6 (17%) of babies who were notaffected by IUGR had respiratory distress at birth, whereas 10 (62.5%)cases of babieswho were affected by IUGR had respiratory distress at birth. More number of neonates affected by IUGR developed respiratory distress which was statistically significant.

Distribution of PIH and IUGR cases based on NICU admission: 25 (50%) were admitted in NICU for various reasons such as 14 were for respiratory distress, 8 were admitted in view of sepsis and 3 in view of low birth weight. Among those who were not affected by IUGR only 38% were admitted inNICU, whereas 12 (75%) cases of the babies who were affected by IUGR wereadmitted in NICU for various indications. The association between distribution of PIH & IUGR and requirement for NICU admission was significant statistically.

<u>Distribution of PIH and IUGR cases based on Perinatal death</u>: Overall there were 4 cases (8%) of perinatal death among 50 cases of PIH. There wereno perinatal deaths among babies who were not affected by IUGR and all the 4 cases ofperinatal deaths were among the babies who were affected by IUGR which constituted25% of IUGR cases.

Out of 4 perinatal deaths one elderly primigravida with gestational hypertension withsevere IUGR and oligohydramnios was admitted at 31 weeks of gestation which wasclinically corresponding to only 24 weeks of gestation. Abnormal umbilical arteryresistance was noted on doppler and it ended up with IUD in two days. Labour wasinduced by ethacredyl lactate and a dead baby was delivered.

Second case was primigravida diagnosed with severe pre eclampsia at 30 weeks ofgestation. Emergency caesarian section was done for severe uncontrolled hypertension and a female baby weighing 1250 gm was delivered with good APGAR score. Thegestational age was assessed to be between 28 to 30 weeks. Baby was referred toNiloufer children's speciality hospital but died after 3 days.

Third case was a second gravida with previous caesarian section and Rh negativestatus, got admitted at 34 weeks of gestation with severe pre-eclampsia andpolyhydramnios. Fetal anomaly, meningomyelocele at lumbar region, was diagnosed on ultrasound. Elective caesarean section was done in view of severe uncontrolled pre-eclampsia and polyhydramnios. Delivered a male baby, 1.5 kg with rupturedmeningomyelocele and paraplegia. Baby was referred to Niloufer children's specialtyhospital and baby died after 2 days.

Fourth was a case of primigravida with 37 weeks of gestation with pre-eclampsia, IUGR and mild oligohydramnios. Labour was induced in view of pre-eclampsia and IUGR.Labour progressed well. ARM was done at 3 cm dilatation of the cervix, when CTGshowed non reassuring fetal heart rate. Thick meconium stained

liquor was detected and emergency LSCS was done but ended with fresh still birth. Weight of the baby was2.4 kg.

TABLE 1. Distribution of permatar outcomes in relation to Tim and rook				
Perinatal outcomes	No of cases of PIH	Neonates not affected by	Neonates affected by	p value
	(n=50)	IUGR (n=34)	IUGR (n=16)	
Respiratory distress				
Yes	16 (32%)	06 (17.6%)	10 (62.5%)	0.001*
No	34 (68%)	28 (82.4%)	06 (37.5%)	
NICU Admission				
Yes	25 (50%)	13 (38.2%)	12 (75%)	0.01*
No	25 (50%)	21 (61.8%)	04 (25%)	
Perinatal death				
Yes	04 (8%)		04 (25%)	0.002*
No	46 (92%)	34 (100%)	12 (75%)	

TABLE 1: Distribution of perinatal outcomes in relation to PIH and IUGR

IV. Discussion

Pregnancy induced hypertension (PIH) continues to be responsible for largestproportion of perinatal deaths resulting from prematurity and IUGR.50 cases of pregnancy induced hypertension which included gestational hypertension, pre-eclampsia, eclampsia and pre-eclampsia superimposed on chronic hypertensionwere followed up through pregnancy and labour and analysed for the incidence of IUGRand perinatal morbidity and mortality.

In the present study, majority 42 (84%) of the cases of PIH were in the agegroup of 19-25 years which was similar to Zafar Het al study 6 (2005- 2006) where 78.6% of cases were in the age group of 21-30 years.

In the present study, among 13 cases of PIH with the onset before 34 weeks of gestation, 46% developed IUGR in the fetus. In Srinivas SK et alstudy 7 (2009) showed increased oddsof IUGR (AOR=1.7) in cases delivered before 34 weeks compared to the cases delivered after 34 weeks.

In the present study, Birth weight of the babies of PIH cases not affected byIUGR was between 2500 and 3499 gm in 97% of the cases. Among the babiesaffected by IUGR, 2 (12.5%) cases weighed between 1000 and 1999 gm, 10(62.5%) babies weighed between 2000 and 2499gm and 2 (12.5%) cases hadbirth weight between 2500 to 2999gm. The P value is 0.0000001 which isstatistically highly significant.

But in contrast, in KazimGezging et al study⁸, mean birth weight was 1180 \pm 250 gm withsevere doppler changes and 1490 +/- 310 gm in those without doppler changes. The difference between the above study and present study is due to inclusion of only severe pre-eclampsia cases in the above study.

Among 50 PIH cases, 25 (50%) babies were admitted inNICU for various reasons. Among those who were not affected by IUGR only38% were admitted in NICU, whereas 12 (75%) cases of the babies who wereaffected by IUGR were admitted in NICU for various indications. The P value is0.01 which is statistically significant.

In Nehamunniyar et al (2017) study 9 60% of babies with IUGR wereadmitted to NICU and another 22.2% were admitted in NICU which requiredventilatory assistance. The results of the present study correlate with the above study.

Among PIH cases the overall perinatal deaths were 4 cases and all these 4belonged to the group affected by IUGR. In 34 cases of PIH not affected byIUGR there were no perinatal deaths, whereas among 16 cases of PIH affected by IUGR the perinatal deaths were 4 (25%). The P value is 0.002 which is statistically significant.

Among these 4 deaths, 2 were related to IUGR associated with prematurity, 1 case was severe IUGR with intrapartum hypoxia and acidosis and 1 wasbecause of congenital anomalies along with IUGR.

In KazimGezging et al study⁸, which included only severe pre-eclampsiacases with IUGR, the perinatal deaths were 21% in those affected with abnormalfetal Doppler velocimetry changes and only 7% in the group where they were notaffected with abnormal fetal Doppler velocimetry.

In Nehamunniyar et al study ⁹, which included various risk factors for IUGRincluding PIH, the perinatal deaths were 10% which is not correlating with the present study because 50% of cases in this study were not associated with PIH.

V. Conclusion

IUGR is the most important complication of PIH, which is responsible for increasedperinatal morbidity and mortality.Majority of these conditions are preventable with good antenatal care.Early booking for the antenatal care, regular antenatal checkups help in identification of the development of pre-eclampsia and IUGR which helps in the appropriate management by intensive maternal and fetal surveillance and deciding the mode and time of delivery. All the high risk cases for the development of hypertensive disorders of pregnancyshould be counselled for regular and frequent checkups. This goes a long way in reducing the perinatal morbidity and mortality. ANM's and midwife's should be trained to check BP in primary health centre and subcentresand to identify the high risk cases and refer to the higher facilities, where these cases can be managed appropriately to prevent the complications.

References

- [1]. Lubchenco, L., Hansman, C. and Boyd, E. Intrauterine growth as estimated for liveborn birth weight data at 24-42 weeks of gestation. Pediatrics 1963; 32:79.
- [2]. Donald, I. (2014). Practical obstetric problems. 7th ed. WoltersKluwer India pvt ltd,p.339.
- [3]. Cunningham, F.G., Leveno, K., Bloom, S., et al. (2010) Williams obstetrics. 23 rdEditio, McGraw-Hill, Medical Publishing Division, New York.
- [4]. Villar J, Carroli G, Wojdyla D, et al. Preeclampsia, gestational hypertension and intrauterine growth restriction, related or independent conditions?. Am J Obstet Gynecol. 2006;194(4):921-931. doi:10.1016/j.ajog.2005.10.813.
- [5]. Shenoy HT, James SX, ShenoyST. Maternal risk factors and perinatal outcomes infetal growth restriction using obstetric Doppler inSouth Kerala, India.Int J ReprodContraceptObstetGynecol 2019;8:6-13.
- [6]. Zafar H, Naz M, Fatima U, Irshad I. Frequency of IUGR in pregnancy induced hypertension. J Univ Med Dent Coll 2012;3(2):8– 13.
- [7]. Srinivas SK, Edlow AG, Neff PM, Sammel MD, Andrela CM, Elovitz MA. Rethinking IUGR in preeclampsia: dependent or independent of maternal hypertension? J Perinatol. 2009;29(10):680-684.
- [8]. KazimGenging, Ali Acar, Harun Peru, RenginKaratayli, Cetin Celik, MetinCapar.How to manage Intrauterine growth restriction associated with severePreeclampsia at 28-34 weeks of gestation. Eur J Gen Med 2008; 5(4):212-215
- [9]. Muniyar N, Kamble V, Kumar S (2017) IUGR Pregnancies Feto-Maternal Outcome. GynecolObstet (Sunnyvale) 7: 440. doi: 10.4172/2161-0932.1000440.

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