Original Article

FREQUENCY OF IUGR IN PREGNANCY INDUCED HYPERTENSION

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ABSTRACT

Objective:

To find out the frequency of Intrauterine fetal growth restriction (IUGR) in patients with pregnancy induced hypertension.

Design:

Observational study.

Place and duration of study:

This study was conducted in Gynaecology unit 1 at Allied Hospital Faisalabad from March 2005 to February 2006. Fifty patients with pregnancy induced hypertension presented to gynae unit 1 were enrolled using convenience sampling technique.

Patients and methods:

After informed consent and history these patients were examined. Other causes of IUGR were excluded. Per abdominal examination was done to assess Symphysis fundal height, lie, presenting part, amount of liquor and USG was done to assess BPD, FL, abdominal circumference, head circumference and amount of liquor. Fetal growth monitoring was done by measuring serial Symphysis fundal height (SFH) and serial ultrasound (USG) after every two weeks.

Results:

The frequency of IUGR in patients with PIH was found to be 28%.

Conclusion:

IUGR is a major neonatal health issue. A prevalence of IUGR in excess of 20% has been recommended as cutoff point for triggering public health action. The prevalence in Pakistan is 25%. Maternal factors have been found to have great impact on IUGR. Studying these factors can help in reducing the mortality and morbidity associated with IUGR by timely intervension.

Keywords: Intra uterine growth restriction, pregnancy induced hypertension.

INTRODUCTION

IUGR is a concept defining the fetus that has failed to fulfill its programmed growth potential. IUGR is clinically important because the fetus with IUGR is at increased risk of perinatal morbidity and mortality.[1, 2] This can be mitigated by appropriate fetal surveillance and timely delivery.[3]

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The incidence of intrauterine growth restriction is estimated to be approximately 5% in general obstetrics population.[4] However the incidence varies depending on the population under examination (including its geographic location) and standard growth curve used as reference.[5]

IUGR fetuses are frequently described as symmetric or asymmetric in term of their body proportion. Symmetrically small fetuses are usually associated with factors that directly impair the intrauterine growth potential of fetus (i.e. Chromosomal abnormalities, viral infections etc.) while asymmetric growth restriction is classically associated with uteroplacental insufficiency.[6]

Pregnancy induced hypertension and pre eclampsia are closely associated with placental dysfunction[7] that result in fetal growth restriction.

By SFH curve we are able to predict IUGR in about 25-70% of fetuses. Ultrasound remains the best method of diagnosis, characterization and follow up of IUGR.[8]

Single estimate of fetal size, amniotic fluid volume, umbilical artery resistance are poor predictor of IUGR.[9,10,11] However growth velocity of the fetal abdominal area is useful[12] which is measured during serial monitoring.

There continues to be need to accurately identify the growth restricted infants prior to delivery in order to reduce the incidence of antepartum fetal loss by instituting close monitoring and expediting deliveries.

PATIENTS AND METHODS

This study was conducted in the department of Obstetrics and Gynaecology Allied Hospital, affiliated with Punjab Medical College Faisalabad. Study duration was one year. Fifty patients which were diagnosed cases of pregnancy induced hypertension (raised blood pressure of $\geq$140/90 mmhg 4 to 6 hours apart on two occasions including both proteinuric and non proteinuric) presented between 22-32 weeks of gestation were included in this study.

Fifty patients diagnosed as a case of pregnancy induce hypertension were enrolled in study after strict exclusion criteria i.e. mistaken dates (ruled out by early dating scan), patients with chronic hypertension, rupture membranes, systemic diseases (renal respiratory, congenital heart disease) and with congenital anomalies. First a detailed history of the patient was taken regarding the period of gestation, duration of raised blood pressure, headache, vertigo and body swelling. Examination was done to check blood pressure and pallor. The assessment of these patients for IUGR was done clinically and by USG. During clinical assessment abdominal examination was done to assess SFH, lie, presenting part, amount of liquor, estimated fetal weight.

USG was done to assess biparital diameter (BPD), femur length (FL), abdominal circumference (AC), head circumference (HC), amount of liquor and any congenital abnormality.

One group of patients with PIH had IUGR at first visit as their growth velocity was less than 10th centiles according to customized growth chart at that particular gestation while remaining develop fetal growth restriction subsequently that was detected during follow up visits.

Both groups were monitored serially after every two weeks. At each visit fetal growth assessment was done by measuring SFH and USG. The parameters observed at USG were BPD, FL, AC growth velocity (10 mm per week).

Patients were followed till delivery or 38 weeks.

RESULTS

Fifty patients of PIH were selected for the study. We found that 28% out of them proved to have IUGR. The effects of various factors like age, parity, socioeconomic status, duration of PIH were evaluated.

Out of 14 cases having IUGR, we found that maximum frequency of problem was found to be in age group 21-30 years i.e. 78.6% (Table 1). 14.3% cases were found to be in the age group of 30 years and above, only one case (7.1%) was in a group of age 20 years and below (Table 2).

Table 1. Frequency of IUGR in PIH

Patients of PIH	50	Percentage
Normal outcome	36	72%
Diseased IUGR	14	28%

n=total number of patients=50
IUGR frequency=28%

57.1% of patients with IUGR were primigravida. 35.8% patients were in parity of G2-G5. 7.1% were in the parity of G6 and above (Table 3).

35.7% of patients with IUGR presented at gestation of less than 28 weeks. 21.4% patients presented at gestation of 28-30 weeks. 42.9% patients presented at gestation of 30 weeks or above (Table 4).

Amongst 14 patients with IUGR 78.6% were with low socioeconomic status and 21.4% patients with high socioeconomic status, (Table 5).

Table 2. Distribution of patients with PIH and IUGR according to the age

Age group	IUGR				Total	
	Non–effected		Effected			
	N	%age	n	%age	N	%age
Upto 20 years	4	11.1%	1	7.1%	5	10%
20–30 years	21	58.3%	11	78.6%	32	64%
>30 years	11	30.6%	2	14.3%	13	26%
Total	36	72%	14	28%	50	100%

Chi square value = 1.830 Degree of freedom = 2 P value = 0.401

Table 3. Distribution of patients with PIH and IUGR according to the parity

Gravida group	IUGR				Total	
	Non–effected		Effected			
	N	%age	n	%age	N	%age
G1	8	22.2%	8	57.1%	16	32%
G2–G5	25	69.5%	5	35.8%	30	60%
G6 & above	3	8.3%	1	7.1%	4	8%
Total	36	72%	14	28%	50	100%

Chi square value = 5.771 Degree of freedom = 2 P value = <0.50

Table 4. Distribution of patients of PIH and IUGR according to the gestational age at admission

Gestational group	IUGR				Total	
	Non–effected		Effected			
	N	%age	n	%age	N	%age
<28 weeks	7	19.4%	5	35.7%	12	24%
28-30 weeks	14	38.9%	3	21.4%	17	34%
>30 weeks	15	41.7%	6	42.9%	21	42%
Total	36	72%	14	28%	50	100%

Chi square value = 2.019 Degree of freedom = 2 P value = 0.364

Table 5. Distribution of patients with PIH and IUGR according to the socio economic status

Socioeconomic status	IUGR				Total	
	Non–effected		Effected			
	N	%age	n	%age	N	%age
Low socioeconomic status	28	77.8%	11	78.6%	39	78%
High socioeconomic status	8	22.2%	3	21.4%	11	22%
Total	36	72%	14	28%	50	100%

Chi square value = 0.00371 Degree of freedom = 1 P value = <0.957

Table 6. Distribution of patients with PIH and IUGR according to the previous history of the same problem

Past history of PIH/IUGR	IUGR				Total	
	Non–effected		Effected			
	N	%age	n	%age	N	%age
H/O PIH present	11	30.6%	3	21.5%	14	28%
No H/O PIH/IUGR	25	69.45	10	71.4%	35	70%
H/O IUGR present	0	0	1	7.1%	1	7.1%
Total	36	72%	14	28%	50	100%

Chi square value = 2.877 Degree of freedom = 2 P value = <0.10

Table 7. Distribution of patients with PIH and IUGR according to the USG findings

USG	IUGR				Total	
	Non-effected		Effected			
	N	%age	N	%age	N	%age
Oligohydramnios	2	5.5%	4	28.6%	6	12%
Placental calcification	0	0	3	21.4%	3	6%
Normal	34	94.5%	7	50%	41	82%
Total	36	72%	14	28%	50	100%

Chi square value = 14.33 Degree of freedom = 2 P value = <0.001

Table 8. Distribution of patients with PIH and IUGR according to the onset of PIH

Raised BP group	IUGR				Total	
	Non–effected		Effected			
	N	%age	N	%age	N	%age
Early onset	21	58.3%	9	64.3%	30	60%
Late onset	15	41.7%	5	35.7%	20	40%
Total	36	72%	14	28%	50	100%

Chi square value = 0.1489 Degree of freedom = 1 P value = <0.75

78.6% patients had no previous history of PIH and 21.4% patients were with history of PIH in previous pregnancies (Table 6).
On USG 28% patients with IUGR were found to have oligohydramnios (Table 7).
64.3% patients with IUGR presented with early onset of PIH whereas 35.7% patients presented with late onset of PIH (Table 8).

DISCUSSION

Hypertensive disorders in pregnancy account for increased perinatal morbidity and mortality when compared to uneventful gestations.[13]
IUGR is the main complication of the fetus in hypertensive pregnancies.[14] The degree of intrauterine growth restriction also has a negative effect on early morbidity.[15] During embryogenesis and development fetus obtains oxygen and nutrients from the mother through placental microcirculation. Pregnancy induced hypertension and pre eclampsia are closely associated with placental dysfunction.[16]
Pathogenesis of pregnancy induced hypertension and intrauterine growth restriction is strictly connected with poor supply of the fetomaternal unit with well oxygenated blood rich in all nutritional substances.[17] There was an association between pregnancy induced hypertension and parity of mother.[18] PIH is common among primigravida and probably the main factor in the genesis of IUGR and reduced placental weight.[19]

In our study 57% patients with IUGR were primigravida and remaining 42% were multigravida, this is comparable with study conducted at Agha Khan medical university hospital where primigravida were 57.5% and multigravida were 42. 5%.
The frequency of intrauterine growth restriction was 22.1% at Agha Khan University Hospital Karachi.[20]
In a study conducted at Jinnah hospital Lahore out of 200 patients 46 cases (23%) were detected to have IUGR.[21]
Our results are comparable with study conducted at postgraduate medical institute and Hayat shaheed teaching hospital Peshawar. In this study frequency of intrauterine growth restriction was 25%.[22]
Pre eclampsia, in particular, is associated with substantial risk to both the mother and fetus. [23]
The presence of fetal growth restriction among women with severe early growth restriction is not associated with increased severity of maternal disease. However the incidence of stillbirth and perinatal death is significantly increased in this sub-population.[24] In our study 60% of patients with IUGR have pre eclampsia. This is comparable to the study conducted in Catholic University of the Sacred Heart, Rome, Italy reported that rate of IUGR

was 22.8% in PIH and 50.7% in pre eclampsia.[13]

In a study conducted at the two centers at Aurora Health Care, USA IUGR was detected in 20% (4 of 20) and 28% (5 of 18) in patients with PIH.[25]

Another study is compared that is conducted at Nagoya City University, Nagoya, Japan where 57% cases with IUGR were having early onset pre-eclampsia and 43% patients with IUGR were having late onset pre-eclampsia.[26] According to our analysis 64.3% had early onset PIH and 35.7% had late onset PIH.

In our study 28% patients had oligohydramnios, this is comparable with the study conducted at Aga Khan University Hospital Karachi, where 20% patients had oligohydramnios with amniotic fluid index below 5th percentile for their respective gestational age. Significant morbidity has been found to exist in pregnancies with an amniotic fluid index value of less than 5 cm.[27]

Hypertension is a common complication of pregnancy. Perinatal outcome in hypertensive disorders of pregnancy is dependent on gestational age and/or the presence of fetal growth restriction. Increased surveillance should be undertaken in patients with PIH so that perinatal outcome can be improved by appropriate intervention.

CONCLUSION

Intrauterine growth restriction is frequently a sequel of hypertensive disorders of pregnancy. Assessment of this growth disorder is an easy task in modern obstetrics. It can be conveniently diagnosed and monitored using clinical and ultra sonographic assessment. Timely intervention in the form of delivery can prevent the hostile consequences of PIH on fetus and decreasing the perinatal morbidity and mortality.

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