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Prevalence of Late Onset IUGR and Association with Maternal Weight Gain

Dr. Praveen Sharma[1], Dr. Shalini Gupta[2*]
[1]Assistant professor, J. K. Loan Hospital, Govt. Medical College, Kota Rajasthan, India
[2]Senior resident, J. K. Loan hospital, Govt. Medical College, Kota Rajasthan, India

Original Research Article

***Corresponding author**
Dr. Shalini Gupta

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Abstract: Intrauterine growth restriction of fetus is defined as the inability of a fetus to achieve its genetically determined growth potential at a given gestational age that means the birth weight is below the 10th percentile or birth weigt less than 2 standard deviation for that gestational age. IUGR is a major source of perinatal morbidity and mortality and this continues to pose a challenging problem for both obstetrician and pediatrician. This prospective observational study was done in j. k hospital, kota medical college from 1st July 2017 to 30th june 2018. All singleton pregnant patients, who were diagnosed of late onset iugr were taken and followed till delivery them. Prevalence of late onset iugr diagnosed in our hospital was. The prevalence of late onset iugr was 2.92%. Patient with lugr associated with poor maternal weight gain during pregnancy.
Keywords: IUGR, late onset, weight gain, uterine artery doppler, perinatal mortality.

INTRODUCTION

IUGR or fetal growth restriction is a condition in which fetus fails to achieve its genetic growth potential. It is a second most common cause of perinatal morbidity and mortality following prematurity [1]. IUGR is used to describe a fetus whose birth weight is below the 10th percentile or <2SD below the mean weight for that gestational age [2] or abdominal circumference is less than 10th percentile. IUGR cases which develop before 32 weeks gestationcanusuallybe managed conservatively because the complications of premature birth outweigh the potential benefit of delivery from a hypoxic and undernourished fetal environment [3].

Difference between SGA and IUGR

Small gestational age

- Babies small for gestational age since conception
- Weight<10%
- Babies with no fetal restriction or pathological conditions
- Babies that have a continuous growth
- SGA includes healthy and constitutionally small fetuses that have o lower risk of abnormal perinatal outcome

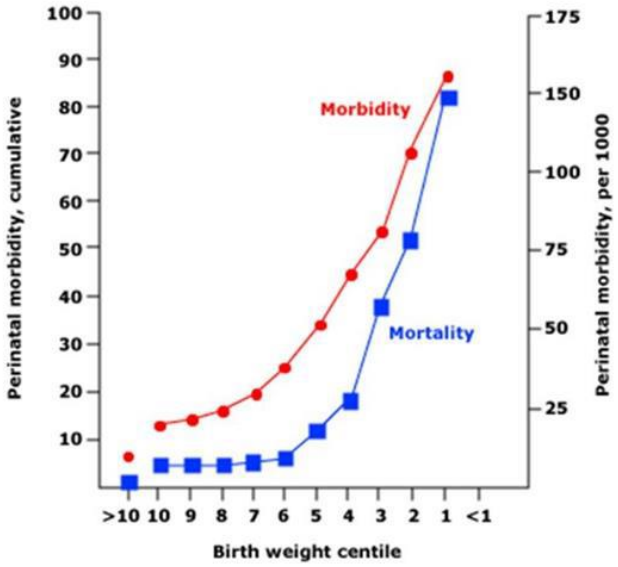
IUGR

- Intrauterine Growth Restriction appears when the fetus does not reach its intrauterine potential for growth and development that was genetically determined as a result of a compromised placental function
- It regards newborns with clinical characteristics of malnutrition and with intrauterine growth retardation independently from birth weight percentile.

Incidence

The overall frequency of IUGR is around 6-8 % About 30-50% of extremely preterm neonates are also IUGR [7] It is responsible of almost 50% of perinatal mortality It is the second fetal risk factor after premature birth Growth-restricted fetuses have an augmented risk of mortality and morbidity:

- Intraventricular hemorrhage
- Bronchopulmonary dysplasia
- Necrotizing enterocolitis
- Infections
- Pulmonary hemorrhage
- Hypothermia and hypoglycemia Data from 2013 in USA reveals an increase with 10% of low birth rate between 1990 and 2006 in singletons.



There are two phenotypes of IUGR: E-IUGR and L-IUGR that are distinct by:

- The moment of onset,
- Evolution
- Doppler parameters modifications and
- Postnatal outcome.

The best cut-off between the two IUGR forms is 32 weeks in terms of perinatal outcome. The antenatal diagnosis, treatment and timely delivery could diminish the risks significantly.

Early onset IUGR in which growth of fetus is affected before 16 week of pregnancy. This is the time when cellular hypoplasia is arrested resulting in symmetrically growth restricted fetus.it is often due to exposure to chemical, chromosalor structural abnormality, aneuploidy, maternal disease, intrauterine infection [4,5].

Late onset IUGR:- Late onset IUGR :IUGR occurring after 32 weeks of gestation, cellular hypertrophy is affected but head spared. The morbidity associated with preterm birth is much less significant in late onset IUGR.

Represents the failure of the fetus to reach its growth potential at term, fetal hypoxemia/hypoxia secondary of placental insufficiency represents the main cause of L-IUGR. In most cases the placental lesions have a late onset and/or do not have a significant extent in order to increase the resistivity of the placenta and translated into augmented UA IP. In terms of frequency L-IUGR is far more prevalent than E-IUGR. It has placental anomalies such as villous immaturity with less impact upon placental resistance; therefore the umbilical Doppler indices can be unaffected [6].

The diagnostic is more difficult, due to the large variability of fetal parameters on growth charts in the third trimester. In fetuses with L-IUGR, EFW appears to remain within the limits for AGA fetuses at >10% L-IUGR can be suspected when:

- The individual growth curve slows down or even become flat.
- An increase of the HC/AC is detected in a previous "normal" growing fetus. The fetal brain at term has increased requirements of oxygen; therefore the first hemodynamic alteration in the presence of hypoxia is cerebral vasodilatation. This method of neuro-protection cannot completely compensate the effects of hypoxia. The cardiac insufficiency does not have enough time to arise, the severity of the cerebral lesions taking place faster and determining severe CTG alterations. Therefore, the classical sequences of Doppler modifications are not present.

During the last 20 weeks of pregnancy the brain increases in size 17-fold L-IUGR fetuses are very fragile, due to increased oxygen requirements of their brain. They present multiple risks due to their inability to tolerate hypoxia. The reactive cerebral redistribution in L-IUGR fetuses is associated with an alteration of the brain metabolism. Undetected IUGR in the third trimester of pregnancy represents the main cause of unexplained stillbirths in low-risk pregnancies. Also probably a number of idiopathic cases of cerebral neonatal palsy where an acute intrapartum hypoxic event could not be identified are in fact caused by an undetected L-IUGR [7].

L-IUGR is a challenging diagnosis associated with:

- Increased fetal morbidity and mortality,
- Impaired postnatal outcome
- Suboptimal neuro-development.

In fetuses with L- IUGR the placental insufficiency seems not to be not reflected in UA Doppler. Umbilical artery Doppler and the sequence of Doppler modifications in multiple fetal vessels are not reliable for the fetal surveillance

MATERIALS AND METHODS

A prospective observational study was conducted at J.k. loan hospital, a tertiary care centre, kota medical college, for a period of 1 year from July 2017 to June 2018. All pregnant women diagnosed of late onset IUGR, who fitted in the study criteria were selected for the study.

All singleton pregnant patients with vertex presentation (after 32 weeks of gestation) undergoig regular antenatal checkup (with accurate dates which were substantiated by first trimester dating scan were enrolled) which were diagnosed of late onset iugr were taken and followed till delivery.

Exclusion criteria

- Autoimmune disease
- Eclampsia
- Multiple pregnancy
- Malpresentation
- Constitutional small baby
- Congenital malformation
- Exposure to teratogenic drugs

Diagnosis is based on 1. Effective fetal weight <10th percentile, 2. after 32 weeks of gestation

Once diagnosis of late onset iugr was made weekly follow ups were done and following parameters were studied;- Effective fetal weight, amniotic fluid index, cardiotocography, doppler study and maternal weight gain.

RESULTS AND OBSERVATIONS

Prevalence:- the total number of deliveries at J.K Loan hospital, kota between July 2017 to June 2018 were 2868.

Prevalence = total no. of diagnosed late onset IUGR cases\ total no. of deliveries at J.K. loan 84\2868= 2.92%

Table-1: Distribution of study subjects according to risk factors: (n = 84)

Risk factor	N	Percentage
Anaemia	48	57.14%
Gestational hypertension	46	54.84%
Gestational Diabetes	16	19.35%
Previous IUGR	12	14.28%
Sickling	30	36.25%
Jaundice	03	3.57%

Table-2: Distribution of study subjests in different weight gain group

Weight gain group	n
≤ 5.0 kg	13(15.47%)
5.1-8.0 kg	54(64.28%)
8.1-10 kg	14(16.66%)
10.1-15kg	3(3.57%)

DISCUSSION

The prevalence of late onset IUGR in our study was 2.92% majority of the women (64.28%) in our study had weight gain of less than 8 kg during their pregnancy. This indicates the high incidence of IUGR being in those a poor maternal weight gain in pregnancy. Strauss and associate did a study on low maternal weight gain and its association with IUGR. The study was done on 10696 women enrolled in national collaborative perinatal project (NCPP) and the child health and development study (CHDS) and found out that low weight gain in 3rd trimester was associated with a relative risk of IUGR of 1.7(1.3- 2.3) in the NCPP cohort and 2.59 (1.7 - 3.8) in CHDS cohort. Low birthweight is defined as less than 0.1 kg per week for first trimester and less than 0.3 kg per week for second and third trimester. The importance of weight gain had been studied by Abrams and Salving and they observed that lack of weight gain in second trimester is strongly correlated with decreased weight gain. The maternal weight gan in pregnancy highly significant for prevention of IUGR.

CONCLUSION

In our study we found that weight gain seems to bestrong prognostic factor in terms of association with IUGR, so diagnosis of decreasein weight gain should bemadeat an earlist. A deviation from normal growthcurve should make us think in the directon of fetus getting hampered. Early diagnosisof iugr isvery important, because it enables the identification of etiology of the condition andadequate moitoring of fetal status, thereby minimizing risks of premature birth and intrauterine hypoxia.

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