

# Intrauterine Growth Restriction (IUGR)

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## Definition

Fetal growth which is less than the 10<sup>th</sup> percentile for that gestational age.

Incidence : 10%

## What is the importance to diagnose and manage IUGR ?

**1. There are increased chances of perinatal morbidity and mortality with IUGR.**

**2. Intrapartum fetal acidosis :**

Fetal monitoring signs of acidosis such as late deceleration, severe variable decelerations, decreased beat-to-beat variability and episodes of bradycardia are more frequent in IUGR than in normal growth fetuses. Acidosis occurs during labour in as many as 40% of IUGR fetuses. As a result the incidence of caesarean section rate is high in IUGR fetuses.

**3. Neonatal complications;**

There is increased incidence of asphyxia, acidosis, persistent fetal circulation, meconium aspiration syndrome, metabolic alterations like hypoglycemia, hypocalcemia, hyperviscosity syndrome, hyperglycemia and hypothermia. Other problems are infection, congenital malformations and chromosomal abnormalities.

## e. Maternal infections

Intrauterine infection is not a common cause of IUGR. Viral infections may be chronic and may affect fetal growth, however, the only viral infection clearly associated with IUGR is congenital rubella.

*Classification:* There are two types of IUGR

1. Symmetric
2. Asymmetric

## Comparison of Symmetric and Assymmetric IUGR fetuses:

### Symmetric

Symmetrically small normal

head / abd and Femur / abd. Ratio

Cause may be - Genetic, Infection

Complicated neonatal course, poor prognosis

### Assymmetric

Head larger than abdomen elevated head / abd. & femur / abd. Ratio

Cause may be - Placental vascular insufficiency: Usually do well if complications are prevented or treated.

### 1. Diagnosis:

Have a high index of suspicion when there is a maternal history of PIH, diabetes, chronic hypertension, previous stillbirth, chronic renal disease, Twin pregnancy.

### 2. Weight gain:

The association between poor maternal weight gain and small babies is not very clear.

### 3. Uterine fundal height:

Measurement of the uterine fundal height is the most common method used to clinically estimate fetal growth. Fundal height measurements should be measured in centimeters from the upper border of pubic symphysis to the top of the fundus of uterus after correction of the dextrorotation and with empty bladder. This measurement should be plotted against a standard curve from a normal obstetric population.

### 4. Ultra sound

a. Estimated fetal weight- This is one of the most

common and logical methods of identifying IUGR baby. Obviously, accurate knowledge of gestational age is essential for proper interpretation of the estimated fetal weight. Studies of Hadlock et al have shown that fetal weight estimation has the best sensitivity and specificity for the identification of IUGR fetus.

(b) Fetal measurements "slow growth type" and "late flattening type" could be identified if fetal measurements are plotted against a normal growth rate. Abdominal circumference is the best single measurements which has a negative predictive value of 99%. This means that finding a normal AC practically rules out the possibility that the baby is small.

(c) Amniotic fluid volume

Amniotic Fluid Index (AFI) for assessing the four quadrants is a good method of assessing amniotic fluid volume. Less than 8 cms is low normal and less than 5 cm is low. Low AFI can be associated with IUGR.

(d) Doppler

There is evidence suggesting that Doppler waveforms be used to assess resistance to blood flow.

Vessels with low resistance will produce waveforms with significant flow during diastole, whereas vessels with high resistance will show decreased diastolic flow. Doppler assessment of umbilical vessels, cerebral vessels and Ductus venosus is usually done for deciding on the time of delivery of an IUGR baby.

### Customized Growth Curves

The need for early recognition of growth restriction is highlighted by its strong association with antepartum still birth. Each fetus has its own potential and has its own ideal growth curve. Models like Rossavik model where data from two early scans 6 to 8 weeks apart is used to project an individual growth curve. The use of projected ideal fetal weight does appear to have some validity and

may be a useful tool for the investigation, but additional research in this area still needs to be done.

### Management

Once an IUGR is diagnosed the important step is to establish adequate methods of fetal surveillance for women with IUGR fetus and deliver them under optional conditions. Antepartum surveillance of the IUGR fetus plays an important role in deciding the time of delivery.

#### 1. Fetal movements

A one hour period post meal three times a day showing > 10 FM is reassuring.

#### 2. Nonstress test (NST)

This is one of the important tests in the follow up of an IUGR fetus. As long as the NST shows adequate variability and accelerations and no deceleration, the fetal situation is not deteriorating -expectant management is possible. Decrease in beat to beat variability, loss of reactivity, lack of accelerations, and occurrence of variable deceleration are signs of less fetal reserve. NST is advisable to be done biweekly from 32-34 weeks, and sometimes daily depending on the circumstances. Daily NST is required in severe IUGR babies.

#### 3. Amniotic fluid index:

Severe oligoamnios (<5 cms AFI) is a sign of fetal jeopardy and AFI between 5 - 8 cms is low normal which needs assessment biweekly.

#### 4. Doppler :

With oligoamnios Doppler abnormality in umbilical vessel, ductus venosus is suggestive of severe fetal compromise. Absence of end diastolic flow or reversal in umbilical vessel or abnormal ductus venosus flow is indicative for immediate delivery. Decision of delivery could be taken for a fetus beyond 32 - 34 weeks. However, earlier to this, the management is conservative. However in symmetrical variety (early onset type) since, 20%



of such fetuses have chromosomal abnormalities one should have a proper counselling. In an IUGR fetus, the presence of a coexistent structural malformation raises the risk of chromosomal abnormalities further.

### **Therapy of IUGR during antenatal monitoring**

The growth of the fetus in utero reflects a delicate equilibrium between the mother, the placenta and the fetus. Fetal growth depends on adequate maternal fuel supply and a maternal vascular tree that can deliver these fuels to the fetoplacental unit.

#### **a. Bed rest:**

This is probably the single therapy most commonly recommended for treatment of fetal growth restriction. Theoretically, bed rest results in decreased blood flow to the periphery and an increase in blood flow to the uteroplacental circulation which presumably contributes to improved fetal growth. However, reports recently have not proved this.

#### **b. Maternal nutritional supplementation**

The provision of adequate fuel to the fetoplacental unit has long been a focus of research into fetal growth. Both observational and interventional studies suggest that maternal nutritional deprivation has a modest effect on fetal birth weight.

#### **c. Oxygen therapy**

Studies did not observe any significant birth weight difference of the two groups i.e. those who received antenatal O<sub>2</sub> and those who did not receive. However, they did observe a significant difference in perinatal mortality rates. The control group had a perinatal mortality rate of 68% whereas the fetuses whose mothers received hyperoxygenation had a perinatal mortality rate of only 29%.

These data suggest that maternal hyperoxygenation may prove to be a useful therapy in IUGR.

#### **d. Pharmacologic therapy**

Aspirin and Dipyridamole - The blood supply to the uteroplacental bed is thought to reflect the balance between the vasoconstrictive actions of TxA<sub>2</sub> (Thromboxane A<sub>2</sub>) and the vasodilatory effect of PGA<sub>2</sub> (Prostacyclin).

The ratio between these two vasoactive compounds is subject to modulation by exogenous substances like fish oil, Aspirin, Dipyridamole. Aspirin or acetyl salicylic acid irreversibly inhibits the cyclooxygenase enzyme. Dipyridamole is a phosphodiesterase inhibitor which delays cAMP degradation and cAMP increase may render platelets more sensitive to the effects of prostacyclin and may also stimulate prostacyclin synthesis leading to vaso dilatation. Though efficacy of Aspirin and Dipyridamole is not proved, it brings down the severity of PIH if developed in the course of pregnancy.

#### **Delivery of the IUGR fetus**

The management of labour and delivery is an important part of the care of the IUGR fetus. The reason for excluding congenital defects is that, intrapartum asphyxia is the major cause of perinatal mortality and morbidity for the IUGR fetus.

The full term fetus has a large capacity to tolerate the stress of labor. This is markedly less in IUGR because of the marked depletion of energy stores in the liver and subcutaneous tissues. Hence a caesarean section is advocated for all severe forms of IUGR on reaching 37 weeks or earlier if required according to the antepartum testing. Those mild IUGR who are allowed vaginal delivery should be monitored by continuous cardiotocography and early amniotomy for observation of meconium. A good neonatal set up is required to have a better outcome.