# Management of infants with intra-uterine growth restriction

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**Abstract**

Intra-uterine growth restriction (IUGR) contributes to almost two-thirds of LBW infants born in India. Poor nutritional status and frequent pregnancies are common pre-disposing conditions in addition to obstetric and medical problems during pregnancy. Growth restriction may be symmetrical or asymmetrical depending on the time of insult during pregnancy. The pathological insult in an asymmetrical IUGR occurs during the later part of the pregnancy and has a brain-sparing effect. Common morbidities are more frequent in <3$^{rd}$ percentile group as compared to 3$^{rd}$–10$^{th}$ percentile group. Guidelines for management of IUGR neonates in these two groups have been provided in the protocol.



 

**Introduction**

Nearly one third of neonates born in India are low birth weight (LBW), weighing less than 2500 grams at birth. A baby's low birth weight is either the result of preterm birth (before 37 completed weeks of gestation) or due to intrauterine growth restriction (IUGR). Later condition is akin to malnutrition and may be present in both term and preterm infants. Neonates affected by IUGR are usually undernourished, undersized and therefore, low birth weight. Two-third LBW neonates born in India fall in this category[1]. Since IUGR neonates are more likely to suffer complications including cold stress and hypoglycemia, it is important that these infants are identified and managed appropriately at birth[2]. Even after recovering from neonatal complications, they remain more prone to poor physical growth, poor neurodevelopmental outcome, recurrent infection and chronic diseases (hypertension, hyperlipidemia, diabetes mellitus, coronary heart disease) later in life[3].

**IUGR and SGA (Small-for-gestational age)**

Although both the terms are used inter-changeably and both denote malnutrition, there is a minor difference in the terminology. SGA a statistical definition, is used for neonates whose birth weight is lower than (less than 10$^{th}$ percentile for that particular gestational age) population norms. IUGR is a clinical definition and includes neonates with clinical evidence of malnutrition. This may be in the form of loose skin folds on the face and in the gluteal region, absence of subcutaneous fat and peeling of skin. Although most IUGR infants would also be SGA, it is possible that a small minority of IUGR infants may have birth weights above the 10$^{th}$ percentile. These morphological IUGR infants would behave like SGA infants and should be managed along the same lines as SGA infants. For purposes of discussion in this paper, the term IUGR would include both the groups of infants .



 

**Etiology**

Poor nutritional status of the mother and frequent pregnancies are the major cause of IUGR. Mothers with a weight of less than 40 kg and a height of less than 145 cm often give birth to SGA infants. Insufficient nutritional intake during pregnancy also has an adverse effect on fetal weight. Maternal hypertension, pre-eclampsia, post-maturity, frequent pregnancies, multiple pregnancy, anemia, malaria and tobacco use are other causes of IUGR[4-6]. Chronic maternal diseases of heart, kidneys, lungs or liver may also lead to IUGR.

**Types of IUGR**

Infants with IUGR are often classified as having symmetrical (head circumference, length and weight equally affected) or asymmetrical (with relative head growth sparing) growth restriction. Infants with symmetric IUGR often have an earlier onset and are associated with causes that affect total fetal cell number including chromosomal, genetic, teratogenic, intra-uterine infections and severe hypertensive etiologies. Asymmetric IUGR is often of a later onset, demonstrates preservation of blood flow to brain and is associated with poor maternal nutrition or late onset exacerbation of maternal vascular disease (pre-eclampsia, chronic hypertension)7.

**Clinical features**

IUGR or SGA infants are often term or near-term in gestation. Their birth weight usually falls below the 10$^{th}$ percentile[8] The neonate has an emaciated look and loose skin because of lack of subcutaneous tissue. These are particularly prominent over the buttocks and the thighs. They look alert and are often plethoric. Comparison of the head circumference with chest circumference is helpful in the identification of a SGA infant. In infants with appropriate growth, the head size is usually bigger than the chest by about 2-cm. In SGA infants, the head circumference usually



 

exceeds the chest circumference by more than 3 cm. A preterm SGA infant would have a combination of clinical features suggestive of both, prematurity and IUGR[9].

**Problems of SGA infants**

Common neonatal morbidities encountered in SGA infants born in our hospital are given in Table 1. The common morbidities encountered in IUGR neonates include: (a) perinatal asphyxia, (b) hypothermia, (c) hypoglycemia and (d) polycythemia. These morbidities are commoner in the more severely gr

owth restricted babies (<3rd percentile) as compared to babies in the 3rd to 10th percentile category. From August 2004 to July 2005, 144 SGA babies were born in our hospital. 24 (17%) developed hypoglycemia and 14 (10%) had polycythemia requiring partial exchange transfusion. Amongst 24 babies with hypoglycemia 50% of the total episodes occurred at 2 h, 22% at 48 h, 11% each at 6 and 12 h and only 4% each at 24 and 72 h of age. 12 (50%) had multiple episodes of hypoglycemia. 3 babies were symptomatic and required intravenous fluid therapy. Rest were managed with supplementary oral feeds. Of 14 babies with polycythemia, only 3 were symptomatic. Polycythemia was detected at 2 h in 50%, at 6 h in 29%, at 12 h in 14% and only 7% at 24 h of life. No cases of polycythemia were detected at 48 and 72 h of life.

**Management**

Early delivery is indicated if there is arrest of fetal growth and pulmonary maturity is satisfactory. Fetal hypoxia may necessitate emergency cesarean section and one should be prepared to receive an asphyxiated infant. If liquor is meconium stained and the neonate is depressed, endotracheal suctioning is essential[10]. Infant should be screened for any congenital malformations. Based on initial assessment, decision is taken to either keep the infant in nursery or with mother.

***Birth weight 3rd – 10th percentile***



 

In the absence of complications including perinatal asphyxia and respiratory distress, these neonates may be managed with the mother (Table 2). Skin-to-skin care helps in maintaining temperature and facilitates breast-feeding. Early initiation of breastfeeding and/ or assisted feeding helps in averting hypoglycemia. Term SGA infants usually do not pose any serious difficulties because they have no problems in direct breast-feeding.To avoid hypoglycemia, they should be put to breast within one hour of birth. However these infants are at risk of morbidities and should be monitored regularly for hypoglycemia and polycythemia in the first 48-72 hours.

Neonates with asymptomatic hypoglycemia should be supplemented with sugar fortified formula feeds. This may be given with the help of a cup and spoon/ *paladai*. Neonates with normoglycemia on regular feeds should be gradually weaned to exclusive breast-feeding within the next 3-4 days. Failure to maintain normoglycemia despite regular oral feeds should be treated with IV fluids. Neonates with symptomatic hypoglycemia should be shifted to a special care nursery and managed appropriately with a glucose bolus followed by a continuous glucose infusion at 6-8 mg/kg/min.

Neonates with asymptomatic polycythemia and a hematocrit <75 maybe managed conservatively by increasing fluid intake. The infant should receive regular (2-3 hourly) breast feeds with extra supplementation. Infants with symptomatic polycythemia or hematocrit >75 should be managed by partial exchange transfusion in the neonatal intensive care unit[11].

***Birth weight <3rd percentile, gestation <35 weeks***

Neonates with severe growth restriction (<3rd percentile) or with presence of complications should be managed in the intensive care unit (Table 2). This group would include infants with perinatal asphyxia, symptomatic hypoglycemia, symptomatic polycythemia, prematurity (<35 weeks), respiratory distress and hypothermia. They should be monitored for hypoglycemia, polycythemia and feed intolerance in the initial few days.



 

Infants with gestation <30 weeks (birth weight <1200 grams) should be started on IV fluids initially and gradually weaned to oral feeds over the next few days. In the absence of other complications, oro-gastric feeds should be started for neonates ≥30 weeks (≥1200 grams) and gradually shifted to katori-spoon/ *paladai* feeding. An infant on full oral feeds with spoon-feeding may be tried on direct breast-feeding. These high-risk infants need to be observed for a minimum of 72 hours for hypoglycemia. Infants on full katori-spoon feeding and/ or breast-feeding may be shifted to the mother after 72 hours if she is confident of on going-care.

***Paladai*/ spoon feeding**

Feeding with a spoon (or a similar device such as '*paladai*') and katori (or any other receptacle such as cup) has been found to be safe in SGA infants[8]. This mode of feeding is a bridge between gavage feeding and direct breast-feeding. It is based on the premise that neonates with a gestation of 30-32 weeks or more are in a position to swallow the feeds satisfactorily even though they may not be good at sucking or coordinated sucking and swallowing. A medium sized katori and a small (1-2 ml size) spoon should be used. The spoon should be filled just short of the brim with expressed milk, should be placed at the corner of mouth and milk should be allowed to flow into the infant's mouth slowly, avoiding any spillage. The infant would actively swallow the milk. This process should be repeated till the required amount has been fed. If the infant does not actively accept and swallow the feed, an attempt should be made to wake the infant with gentle stimulation. If he is still sluggish, do not insist on this method. It is better to switch back to gavage feeds till the infant is ready.

**SGA/IUGR babies with absent or reversed end-diastolic flow (AREDF) in umbilical artery**

In the IUGR fetus, hypoxaemia produces circulatory redistribution towards the brain and away from the viscera and placenta, culminating in umbilical artery or aortic AREDF in the most



 

severely affected fetuses. The combination of antenatal and persisting postnatal disturbances of gut perfusion, interacting with the metabolic demands of feeding, may adversely affect intestinal tissue oxygenation, combining with stasis and immunological factors to contribute to the development of NEC. A review by Dorling et al including 14 studies and 1178 neonates found higher risk of NEC in IUGR babies with AREDF (odds ratio: 2.13; 95% CI: 1.49 to 3.03).[12] Although evidence for feeding strategy to be adopted in these babies is limited, it may be prudent to start and persist with minimal enteral nutrition for first 48-72 h of life.

**Long-term outcome and follow-up**

IUGR babies are at risk for poor growth and neuro-developmental outcome.[3] We routinely follow IUGR babies with birthweight below <3$^{rd}$ percentile and those with birthweight 3-10$^{th}$ percentile if they develop significant morbidities (e.g. hypoglycemia, polycythemia, birth asphyxia) during hospital stay.



 

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| Table 1: Common morbidities in SGA neonates | | | |
|---|---|---|---|
| | **Period: Jan 1999 to Dec 00 (n=156)** | | **Period: Aug 04 to Jul 05 (n=144)** |
| | Weight <3rd percentile (n=47) | Weight 3rd-10th percentile (n=109) | Weight <10th percentile (n=144) |
| Birth asphyxia | | | |
| Total | 4 (8.5%) | 10 (9.2%) | 25 (17%) |
| Moderate | 2 | 8 | - |
| Severe | 2 | 2 | - |
| Hypoglycemia | | | |
| Total | 12 (25.5%) | 14 (12.8%) | 24 (17%) |
| Symptomatic | 6 | 3 | 3 |
| Asymptomatic | 6 | 11 | 21 |
| Polycythemia | | | |
| Total | 14 (29.8%) | 17 (15.6%) | 14 (10%) |
| Symptomatic | 3 | 8 | 3 |
| Asymptomatic | 11 | 9 | 11 |
| Hypothermia | 0 | 4 (3.7%) | 19* (13.2%) |

*Includes 1 baby with hypothermia and 18 babies with cold stress

**Table 2: Management of SGA infants**

**Criteria for admission to Nursery**

- All SGA infants < 2 SD (3rd percentile)
- Infants with gestational age < 35 wks
- Infants with birth asphyxia, respiratory distress etc.

*Care of SGA infants with mothers (birth weight between 3rd and 10th percentile, gestation >35 wks)*

- Early initiation of breast feeding (within 1 hour)
- Skin-to-skin care to maintain temperature, monitoring of cold stress by mother and health professionals.
- Monitor blood sugar, hematocrit
- Prevent infections

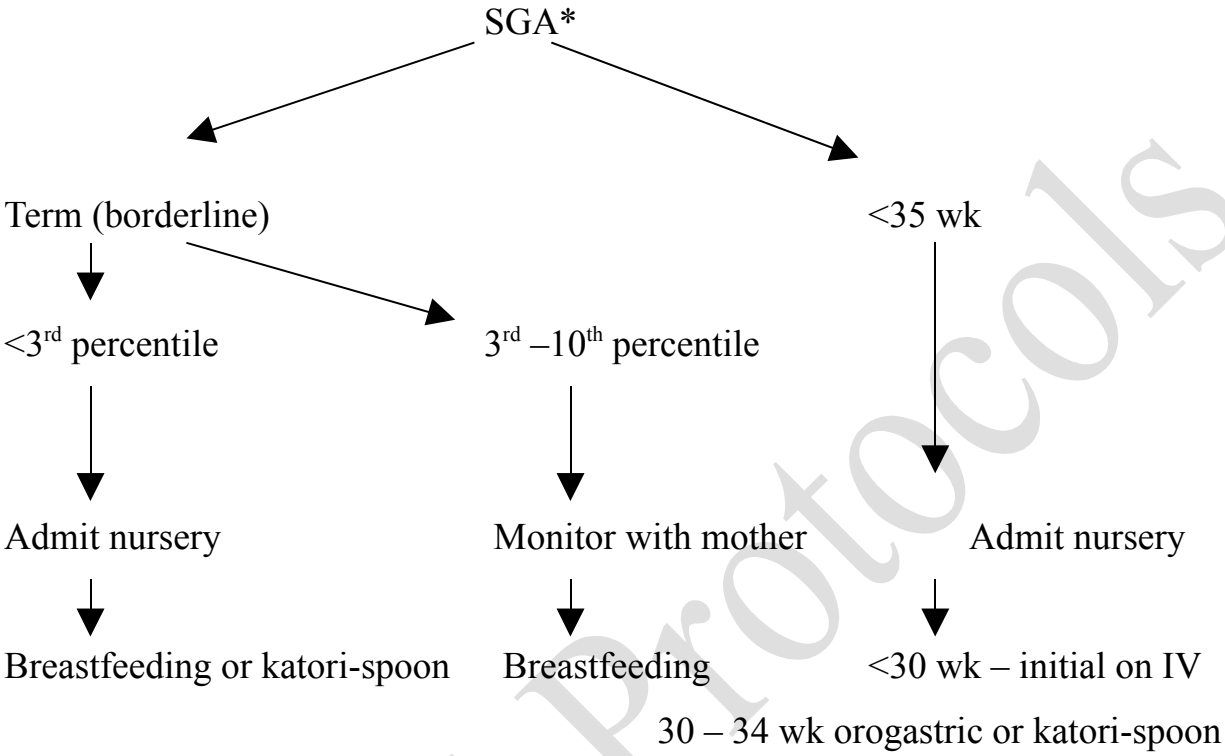
*Care of SGA infants in Nursery (birth weight <3rd percentile or gestation <35 wks)*

- Nurse in thermo neutral environment
- It stable, early initiation of feeds ( EBM).
- Feed by orogastric tube or katori-spoon /paladai if gestation ≥32 wks
- Initial intravenous fluids followed by orogastric or katori-spoon /paladai if gestation <32 wks
- Monitor blood sugar, hematocrit

*Care of SGA infants with absent or reversed en-diastolic blood flow*

- At higher risk of development of NEC
- If preterm (gestation <32 weeks): Nil per oral or on minimal enteral nutrition for first 48-72 h of life followed by gradual advancement of feed volume

**Figure 1: Algorithm for management of SGA infants**




**Blood sugar, hematocrit, temperature monitoring*