Obstetrics & Gynecology
TEAM

Intra Uterine Growth Restriction (IUGR) + Intra Uterine Fetal Death (IUFD)

Leader: Sarah Al-Haddab

Done By: Sadeem Al-Dawas

◆very important ◆mentioned by doctor ◆team notes ◆not important

Intra Uterine Growth Restriction (IUGR) + Intra Uterine Fetal Death (IUFD)

- **Low Birth weight:** Is defined by the WHO simply as birth weight < 2.5kg, so does not correct for gestation.

- **Small for gestation age:** is used variably prenatally and postnatally to describe a fetus or neonate with growth parameters (e.g EFW, AC, birthweight) below a given centile for gestational age.

- **Implications of Growth Restriction**

Growth restricted fetuses

- Have a higher risk of still birth and mortality
- Are most at risk of
  - hypothermia
  - hypoglycaemia
  - pulmonary haemorrhage
  - infection
  - encephalopathy
  - necrotising enterocolitis
- Incidence of fetal heart rate abnormalities
- Higher incidence of operative delivery

- **Aetiology**

There is a wide range of associations:

- Fetal
- Maternal
- Placental

These in turn may have a genetic or environmental basis

➢ Fetal Causes ..

1. Chromosomal

Abnormal fetal karyotype can be responsible for up to 20% of growth restricted fetus

- Early pregnancy, triploidy 58%
- Trisomy 46%
- Trisomy 21 and Turners – second trimester

The reason is probably because of lack of cell division or cell growth in either the fetus or placenta

2. Structural anomalies / Structural defects

- Central nervous system
- Cardiovascular system
- Gastro intestinal system
- Genito urinary system
- Muskilo skeletal

Are associated with an increased risk of IUGR

3. Infection:

- Malaria – major cause of IUGR – its treatment reduces the incidence of IUGR
- Rubella
- Cytomegalovirus
- Toxoplasmosis
- Syphilis

Can affect cell division and have all been implicated

4. Nutrient Supply

Inadequate maternal nutrition can restrict growth in the 3rd trimester

- examples are the Dutch Famine and the Leningrad sledge
- Leningrad sledge 700gm Glucose, amino acids and lactate are the major substrates for the fetus
- Oxygen : Babies born at higher altitude are smaller than those born at sea level

➢ Placental Causes

1. Lack of conversion of spiral arteries into utero placental arteries
2. The low-resistance circulation thus created allows high blood flow to the placenta.

In normal pregnancies, end diastolic flow is usually present (umbilical arteries by the early second trimester And increases until term

3. Growth restricted fetuses often have absent or reversed end-diastolic flow in the umbilical artery – this suggests increased resistance in the feto-placental circulation

➢ Maternal Causes

1. Smoking

- Active and passive smoking is a major cause of IUGR
- Such babies weigh between 100-300gm less than other babies
- > 10 cigarettes/ day is significant
- Male fetus more affected than females
- Mechanism is probably via the higher levels of carboxy haemoglobin in such fetuses.

2. Alcohol:

Moderate to heavy alcohol consumption can reduce fetal weight by up to 500 gm.

3. Drugs

Heroin and methadone use are associated with growth restriction <490 gm and 280 gm respectively.

4. Chronic Diseases

- Congenital heart disease – especially if cyanotic
- Chest disease e.g. cystic fibrosis, bronchitasis, kyphoscoliosm and asthma in severe cases where there is marked respiratory compromis.
- Chronic renal diseases – especially if there is hypertension, proteinuria
- Diabetes mellitus – if there is renal disease and vascular disease.

▪ Screening:

- **Clinical Examination**
  - Palpation
  - Symphyseal – fundal height – higher sensitivities than palpation

- **Ultrasound**
  - Has a better detection rate for IUGR than clinical examination
  - Only disadvantage is that the work load will be great if all pregnant women were to be subjected to it too often.

▪ Management

The terms symmetric and asymmetric growth restrictions are descriptive

- Growth restriction detected at any gestation without associated anomaly is most likely to represent true growth restriction as a result of utero placental dysfunction
- The earlier the gestation the more likely the fetus is to be aneuploidy or infected.

The gestational age should be checked using the last menstrual period and any early scans.

- Diagnosis of IUGR should be made on serial scans – every 2 weeks
- Thorough survey of the fetus for associated anomalies is undertaken
- Liquor volume should be quantified (amniotic fluid index)
- Doppler waveforms of the uterine and umbilical artery should be obtained.

➢ Early –Onset Growth Restriction (<32 Weeks)

The principle differential diagnosis are:

(a) Chromosomal abnormality or some other genetic problem
(b) Congenital infection
(c) Utero placental dysfunction

Findings that would make a chromosomal problem more likely include:

- Normal uterine artery doppler findings
- Normal liquor volume
- Presence of a structural abnormality

The commonest infection associated with IUGR is **cytomegalovirus** (CMV)

- Mother may have complained of flu-like illness
- Fetus has sonographic findings compatible with CMV (e.g. microcephaly and cerebral calcification).

Utero placental dysfunction is a diagnosis of exclusion, Factors supporting this are:

- A history of growth restriction in a previous pregnancy
- Reduced liquor volume
- Abnormal uterine umbilical artery waveforms

➢ Late-Onset Growth Restriction (>32 Weeks)

Most likely cause is utero placental insufficiency, often associated with the development of pre-eclampsia

▪ Fetal Monitoring

Monitoring the growth-restricted fetus involves serial fetal measurement

- Abdominal circumference
- Amniotic fluid index
- Cardiotocography
- Doppler ultrasound

- Fetuses with absent end-diastolic flow are hypoxaemic, these changes may appear up to 5 weeks before demise
- Reversed end-diastolic flow is suggestive of preterminal compromise ; the fetus may die within 1-2 days if not delivered.

▪ Amniotic Fluid Index

- Reduction in amniotic fluid index (the sum of the four deepest vertical pools in each quandrant) is associated with an increase in perinatal mortality.
- Fetal urine production is significantly lower in the SGA fetus than in the AGA fetus.
- Decreased renal perfusion results in oligohydramnios

- **Biophysical Profile**
    - Breathing
    - Tone
    - Movement
    - Amniotic fluid volume
    - Cardiotocography
        - Requires about 40 mins observation of fetal breathing movements.
        - A persistently abnormal biophysical score is associated with absence of end-diastolic flow

- **Prevention:**
    - All women should be encouraged to stop smoking since it is the commonest risk factor
    - Even passive smoking is harmful – husbands should be persuaded to stop.
    - Early aspirin treatment before 17 weeks (100-150mg) for patients with previous IUGR babies (possible role of placental thrombosis)

- **Labour And Delivery**
    - In the preterm failure to delilver poses the risk of chronic hypoxia while delivery exposes the neonate to the risks of prematurity
    - Most fetuses follow a decompesation cascade:
        - Absent end-diastolic flow →decelerative CTG → reversed end diastolic flow → fetal death
    - IUGR fetus is more likely to become more hypoxic in labour
    - With AEDF or reversed EDF, delivery should be by caesarean section

From Kaplan lecture notes

Symmetric IUGR

- All ultrasound parameters (HC, BPD, AC, FL) are smaller than expected.
- Etiology is **decreased growth potential**, i.e., aneuploidy, early intrauterine infection, gross anatomic anomaly.
- Workup should include detailed sonogram, karyotype, and screen for fetal infections.
- **Antepartum tests are usually normal.**

Asymmetric IUGR

- Ultrasound parameters show **head sparing**, but **abdomen is small.**
- Etiology is **decreased placental perfusion** due to chronic maternal diseases (hypertension, diabetes, SLE, cardiovascular disease) or abnormal placentation (abruption and infarction).
- Amniotic fluid index is often decreased, especially if uteroplacental insufficiency is severe.
- **Monitoring** is with serial sonograms, non-stress test, amniotic fluid index, biophysical profile, and umbilical artery Dopplers.

➢ Intra uterine fetal death IUFD

- The term IUFD (Intra uterine fetal death) embraces before the 28th week of pregnancy (delayed) miscarriage) and those occuring later which result in macerated stillbirth.
- **Maceration** is a destructive process which first reveals itself by blistering and peeling of the fetal skin. This appears between 12 and 24 hours after fetal death. The ligaments are softened and the vertebral column is liable to sag. The skull bones overlap each other at the sutures because of the shrinkage of the brain (Spalding's sign). It takes several days for Spalding's sign to appear after intrauterine death, usually a week or more.

▪ Causes:

1. One of the commonest is pre-eclampsia
   - ❖ Hypertensive spasm of the utero placental vessels which results into reduced oxygen supply to the fetus.
2. Chronic hypertension
3. Chronic nephritis
   - ❖ Fetus dies from placental infarction and hypoxia even before the age of viability
4. Hyperpyrexia – a body temperature over 39.40C can kill the fetus directly
5. Diabetes in pregnancy
6. Fetal malformation
7. Placental insufficiency
8. Idiopathic

▪ Management:

- Conservative – await spontaneous labour
- Induction of labour
  - Prostin E2 (Vaginal pessary)
  - IV Nalador
  - Oxytocin
- Exclude coagulation disorder
  - generally hypofibrinogenaemia does not set in until after about 4 weeks after the IUFD.

Causes of Fetal Death Based on Trimester " from First Aid"

T1 (1–13 WEEKS)

1. Chromosomal abnormalities.
2. Environmental factors (eg, medications, smoking, toxins).
3. Maternal anatomic defects (eg, müllerian defects).
4. Endocrine factors (eg, progesterone insuffi ciency, thyroid dysfunction,diabetes).
5. Unknown.

T2 (14–27 WEEKS)

1. Anticardiolipin antibodies.
2. Antiphospholipid antibodies.
3. Chromosomal abnormalities.
4. Anatomic defects of uterus and cervix.
5. Erythroblastosis.
6. Placental pathological conditions (eg, circumvallate placentation, placenta previa).

T3 (28 WEEKS–TERM)

_ Anticardiolipin antibodies.

1. Placental pathological conditions (eg, circumvallate placentation, placenta previa, abruptio placentae).
2. Infections (eg, toxoplasmosis, CMV, parvovirus).

➢ Management from first aid

- D&E may be used if fetal death occurs in T2. D&E has ↓ maternal mortality compared to PGE2 labor induction, but also has the risk of uterine perforation.
- Labor induction if fetal death occurs in T3. Induction of labor with vaginal misoprostol is safe and effective even in patients with a prior cesarean delivery with a low transverse uterine scar.
- Every attempt should be made to avoid a hysterotomy.
- The patient should be encouraged to seek counseling due to emotional stress caused by diagnosis of fetal death and length of time between diagnosis and delivery.

- **Monitoring for infections and coagulation profile if no evidence of problems we can wait for 2-3 weeks but if there is any problem deliver.**
- **Examine the fetus for malformations take blood from the cord + fetal tissue + placental tissue**