Fetal and Neonatal Effects of Maternal Diabetes

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Fetal and Neonatal Effects of Maternal Diabetes

- Introduction
- Diabetic Pregnancy
- Pathophysiology
- Fetal Effects (Diabetic Embryopathy, Diabetic Fetopathy)
- Neonatal Effects
  1. Congenital anomalies
  2. Prematurity
  3. Perinatal asphyxia
  4. Macrosomia and birth injury
  5. Intrauterine growth restriction
  6. Respiratory distress syndrome
  7. Metabolic complications
  8. Cardiomyopathy
Introduction

- Diabetes is the most common medical complication of pregnancy.

- Incidence of diabetes complicating pregnancy;
  - 5% - 14%, in USA & Europe
  - 15–20%, in developing world

- Epidemiological and experimental data strongly suggest that diabetes in pregnancy in turn induces long-term consequences in offspring that include ↑incidence of diabetes & obesity in adulthood.

- Diabetes in pregnancy is divided into 2 types.
  1. Gestational DM
  2. Pre-gestational DM (PGDM); more severe because it is present before pregnancy [T1DM or T2DM or MODY (maturity onset diabetes of the young)]
Diabetic Pregnancy

- **Gestational diabetes mellitus**
  - Usually diagnosed in the 2nd half of pregnancy
  - **Affects mainly fetal growth.**
  - Maternal complications include preterm labor, pre-eclampsia, birth trauma, cesarean section, ---.
  - Fetal complications include macrosomia, shoulder dystocia, stillbirth, and metabolic complications.

- **Pre-Gestational Diabetes Mellitus (PGDM)**
  - Effects begin as soon as fertilization and implantation
  - An ↑risk of CMs, Perinatal mortality, obstetric complications, and severe neonatal morbidity.

- **Perinatal outcome is related to the onset and duration of glucose intolerance and to the severity of the disease.**
# White’s Classification of Diabetes in Pregnancy (Modified)

<table>
<thead>
<tr>
<th>G. Diabetes</th>
<th>Abnormal GTT, but euglycemia maintained by diet alone; if diet alone insufficient, insulin required</th>
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<tbody>
<tr>
<td>Class A</td>
<td>Diet alone, any duration or age of onset</td>
</tr>
<tr>
<td>Class B</td>
<td>Age of onset: &gt;20 y; duration: &lt;10 y</td>
</tr>
<tr>
<td>Class C</td>
<td>Age of onset: 10 to 19 y; duration: 10 to 19 y</td>
</tr>
<tr>
<td>Class D</td>
<td>Age of onset: &lt;10 y; duration: &gt;20 y; background retinopathy or hypertension (not preeclampsia)</td>
</tr>
<tr>
<td>Class R</td>
<td>Proliferative retinopathy or vitreous hemorrhage</td>
</tr>
<tr>
<td>Class F</td>
<td>Nephropathy, with &gt;500 mg/d proteinuria</td>
</tr>
<tr>
<td>Class RF</td>
<td>Criteria for both classes R and F coexist</td>
</tr>
<tr>
<td>Class H</td>
<td>Arteriosclerotic heart disease clinically evident</td>
</tr>
<tr>
<td>Class T</td>
<td>Prior renal transplantation</td>
</tr>
</tbody>
</table>

- **Class B through T require Insulin**
Pathophysiology

- During pregnancy, diabetic women develop a pronounced Peripheral insulin resistance; decreased numbers of insulin receptors; and decreased binding of insulin to target cells because of insulin resistance.

- The diabetic mother experiences frequent episodes of hyperglycemia and high levels of amino acids, and transfer of these nutrients to the fetus is increased.
Effects on the Developing Fetus

- During Organogenesis, at 3–8 wks gestation, the abnormal metabolic environment is **teratogenic** ↑ incidence of **Cong. Malformations**
- ↑ carbohydrates that enter the fetal circulation **Hyperinsulinemia**
- Altered Arachidonic acid & Myoinositol levels or fetal hyperglycemia promote;
  - ↑ formation of O2 radicals in cells **Mitochondrial damages**
  - Inhibits prostacyclin ↑ oxidative stress
  - ↑ thromboxanes and other prostaglandins disrupts vascularization of developing tissues.
- Hyperglycemia alters the expression of regulating genes altered cellular mitosis and normal apoptosis (**Programmed Cell Death**).
- Exaggerated apoptosis **Fetal Anomalies**.
Effects of Maternal Hyperglycemia on the Fetus.

Maternal Hyperglycemia

Fetal Macrosomia

↑ Visceral Enlargement

↑ Deposition of Fat

High Rate of Fetal Growth

↑ Protein, Lipid, & Glycogen Synthesis

Excess Fuels To Fetus

Pancreatic β-cell Hyperplasia

Fetal Insulin Growth Factors

↑ High Rate of Fetal Growth

↑ Deposition of Fat

↑ Visceral Enlargement

↑ Protein, Lipid, & Glycogen Synthesis
Fetal Hyperinsulinemic State.

- ↑ Hematocrit Levels
- Fetal Hyperinsulinemia
- Red Blood Cell Hyperplasia
- ↑ Erythropoiesis
- Cardiac Hypertrophy
- ↑ Hypertension
- ↑ Adrenal Catecholamines
- ↑ Fetal Catabolism
- ↑ Energy Usage
- ↓ Fetal Oxygen Stores
- Fetal Hypoxia
Maternal Diabetes
  ↓
Fetal hyperglycemia
  ↓
Fetal islet and β-cell hyperplasia
  ↓
Fetal hyperinsulinism
  ↓
O₂ demand, Uteroplacental insufficiency
  ↓
Chronic hypoxemia
  ↓
Erythropoietin
  ↓
Polycythemia
  ↓
FFA & AA transferred to fetus
  ↓
Fetal substrate uptake
  ↓
Macrosomia, LGA
  ↓
Fetal Asphyxia
  ↓
Neonatal Asphyxia
  ↓
Stillbirth
  ↓
Dystocia
  ↓
RDS
  ↓
Congential anomalies
  ↓
Delayed lung maturation
  ↓
Peripheral glucose uptake
  ↓
Lipolysis
  ↓
counter-regulatory hormone production
  ↓
Hypoglycemia (no defect in GNG; adequate glycogen)
Fetal Effects

- Diabetic Embryopathy
  A spectrum of malformations and spontaneous abortions

- Diabetic Fetopathy
  - Occur during fetal development (after the 10th wk of gestation)
  - Not associated with malformations
  - Fetal Hyperinsulinism
Diabetic Embryopathy

- Spontaneous Abortions and Congenital Malformations.
- Incidence of CM in infants of diabetic mothers is ↑2 to 5 times compared to general population.
- Malformations are frequently multiple.

**Typical Congenital Malformations reported:**
- **Heart defects:** DORV, transposition of the great vessels, TOF
- **Skeletal defects:** caudal regression syndrome; has the strongest association with diabetes (occurring more than 200 times more frequently in IDM than in other infants):
- **NTD** (anencephaly, spina bifida)
Diabetic Embryopathy

Deleterious effect of poor glycemic control on fetal outcome
Combined incidence of major malformation and spontaneous abortion according to the hemoglobin A1 (HbA1) value during the first trimester of pregnancy in 315 women with type 1 diabetes. The risk rose markedly at HbA1 values above 11 percent (approximately equivalent to a HbA1c value of 8.5 percent). Other studies have found an increase in risk at HbA1c values above 9.5 percent. (Data from Greene, MF, Hare, JW, Cloherty, JP, et al, Teratology 1989; 39:225.)
Diabetic Fetopathy

Fetal Growth

- Fetal growth in diabetic and non-diabetic women is similar during the 1st & early 2nd trimesters.
- After 24/52 Gestn, hyperglycemia disproportionally ↑ abdominal circumference, while head growth remains normal.
- ↑ fetal growth, particularly of insulin-sensitive tissues (liver, muscle, cardiac muscle, and subcutaneous fat), resulting in Macrosomia.

Fetal Hypoxemia

- Stimulates the synthesis of erythropoietin Polycythemia
- Promotes catecholamine production (HT, Cardiac Hypertrophy)
- May contribute to the 20 - 30 % rate of Stillbirth
Major Morbidities in the IDM

- Macrosomia
- Shoulder dystocia
- Asphyxia
- Birth injury
- RD / RDS
- Congenital anomalies
- Caudal regression
- Small left colon syndrome
- Hypertrophic Obstructive Cardiomyopathy
- Septal hypertrophy
- DORV / Heart failure
- Truncus Arteriosus

- Hyperbilirubinemia
- Hypocalcemia
- Hypoglycemia
- Hypomagnesemia
- Organomegaly
- Increased blood volume
- Polycythemia and hyperviscosity
- Transient hematuria
- Renal vein thrombosis
- Neurologic instability (short- and long-term)
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Neonatal Effects

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1- Congenital Anomalies In Diabetic Pregnancy

- 3–6% of all infants at birth, representing a 2-5 fold ↑, when compared with non-diabetics.

Teratogenicity of maternal DM in human

- Occurring during embryogenesis at the end of blastogenesis & organogenesis between 3rd & 7th wks of gestation.

- Leading to a spectrum of malformations known as Diabetic Embryopathy
<table>
<thead>
<tr>
<th>Location</th>
<th>Malformations in Infants of Diabetic Mothers</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Open Neural Tube Defects, holoprosencephaly, absent Corpus Callosum, microcephaly, macrocephaly, agenesis of olfactory tracts, <strong>bizarre</strong> undergrowth or overgrowth of brain</td>
</tr>
<tr>
<td>CVS</td>
<td>Transposition of great vessels, VSO, ASO, TOF, CoA, hypoplastic left heart, <strong>Cardiomyopathy (thickened IVS)</strong></td>
</tr>
<tr>
<td>GIT</td>
<td>Pyloric stenosis, duodenal atresia, microcolon, anorectal atresia, omphalo-enteric cyst/fistula, hernias</td>
</tr>
<tr>
<td>Uro/Gen</td>
<td>Renal agenesis, renal cysts, hydronephrosis, uterine agenesis, micropenis, hypospadias, cryptorchidism, hypoplastic testes, ambiguous genitals</td>
</tr>
<tr>
<td>MS/SK</td>
<td>Caudal Regression Syndrome/Sacral Agenesis, costovertebral anomalies, limb reduction, cleft palate, club foot, <strong>polysyndactyly</strong></td>
</tr>
<tr>
<td>Others</td>
<td>Sinus inversus, microphthalmia, colobomas of iris or chorioretina, diaphragmatic hernia, branchial arch anomalies, choanal atresia, aplasia cutis, Cutaneous vascular dysplasia</td>
</tr>
</tbody>
</table>
Caudal Regression Syndrome / Sacral Agenesis

- Complete or partial agenesis of sacrum and lumbar vertebrae
- Femoral hypoplasia, clubbed feet, & flexion contractures of LE
- Anomalies of GIT, GUT, heart and NTD

- > 1% of all diabetic pregnancies, 22% of CRS are IDMs
- Correlates with poor glycemic control (elevated HbA1c) in early pregnancy
- H/O maternal diabetes is obtained in 16% of infants with sacral agenesis
- Absent lumbo-sacral spine
- ‘Double Bubble’
- Small pelvis
- Hypoplastic femur
- Healing fracture
Central Nervous Malformations;

- 16 times more likely in these infants.
- Risk of Unencephaly is 13 times higher
* Risk Of Spina Bifida Is 20 Times Higher in IDM.
Neonatal Small Left Colon Syndrome, or Microcolon

- A transient anomaly, unique to the IDM.
- Presents as gastrointestinal obstruction and may mimic congenital aganlionic megacolon or Hirshprung disease.
- Normal innervation of the bowel and ultimately have normal intestinal function.
Small Left Colon Syndrome

Barium enema
Holoprosencephaly

- Semilobar

Holoprosencephaly; typical facial characteristics: Hypotelorism, a Flat nose, & median Cleft lip.
- 2-year-9-month old girl with Diabetic Embryopathy
- left club hand with hypoplastic thumb and sacral agenesis with contractures of knees
Prenatal diagnosis

- ↑1\textsuperscript{st} trimester HbA1c (>8%)

- Maternal serum \( \alpha \)-fetoprotein for the presence of associated NTD

- \textbf{Prenatal USS}; Estimation of GA, Evaluation of CM & Evaluation of fetal growth; Macrosomia / IUGR.

- Fetal echocardiography.

- Accordingly; Plan for the delivery.
Prevention of the Malformations

- **Inositol supplementation**. No studies have been performed in human diabetic pregnancy.

- **Arachidonic acid supplementation**. No studies

- **Antioxidant supplementation**; Supplementation of antioxidants diminishes embryonic maldevelopment in experimental diabetic pregnancy, both in vivo and in vitro. No studies have been performed in human diabetic pregnancy

- **Folic acid supplementation**; Supplementation of folic acid diminishes embryonic maldevelopment in high-glucose-exposed embryos in vitro, as well as in fetuses of diabetic rats. No studies have been performed in human diabetic pregnancy.
Premature labor occurred in 31% of IDMs compared to 20% in a control population.

Poor glycemic control may have contributed to the development of premature labor.

Maternal preeclampsia contributes to premature delivery.
3 - Perinatal Asphyxia:

- Maternal DM increase the likelihood of impaired placental blood flow.

- IDM are at increased risk for intrauterine or perinatal asphyxia.

- Mimouni et al. reported a prospective study of 162 infants born to 149 diabetic mothers: 44 (27%) infants had perinatal asphyxia, defined as fetal distress during labour (late decelerations, persistent fetal bradycardia, or both), 1 min Apgar score 6, or intrauterine fetal death.

- Perinatal asphyxia was correlated with hyperglycemia in labor, prematurity, Maternal vascular disease, and nephropathy.
• Asphyxia may have diverse consequences for the neonate.

• Acutely, it may affect respiratory, renal, and CNS function.

• Decreased fluid intake usually is recommended until the degree of injury to the Renal and CNS can be ascertained.
4 - Macrosomia and Birth Injury:

- B.WT > 90th percentile or >4000 g

- Macrosomia in IDMs is associated with disproportionate growth baby’s body being relatively > head

- More likely to have hyperbilirubinemia, hypoglycemia, and acidosis.

- Macrosomia occurs among all classes of diabetic pregnancies, except those with vasculopathy that results in (IUGR).
4 - Macrosomia and Birth Injury: continued

- Predisposes to birth injury, especially Shoulder Dystocia.
- Shoulder Dystocia occurs in nearly 1/3 of IDMs who weigh ≥ 4000 g
- Shoulder Dystocia can result in:
  - Brachial Plexus Injury
  - Clavicular Or Humeral Fractures
  - Perinatal Asphyxia
  - Cephalohematoma
  - Subdural Hemorrhage
  - Facial Palsy

- Spinal cord --- vulnerable to birth trauma for IDMs;
- Brachial plexus injuries, including Erb’s palsy (roots C5–7), Klumpke palsy (roots C7–8), diaphragmatic nerve paralysis (roots C3–5), & recurrent laryngeal nerve damage (rts T1–2).
A macrosomic infant of a diabetic mother (IDM) has head circumference and length that are at the 90th percentile; body weight greatly exceeds the 90th percentile & fat deposition in the shoulder and intrascapular area.
Macrosomic IDM; Disproportionate growth with a plethoric appearance & excessive fat accumulation.
Intrauterine Growth Restriction Retardation (IUGR)

- Decreased utero-placental blood flow in diabetic vasculopathy

- Underlying chronic placental insufficiency affecting the SGA IDMs that predisposes them to birth depression and/or Perinatal asphyxia.

- Infants from classes D, F, and R (with malignant retinopathy) are often growth restricted at birth, i.e., SGA.
Sequelae of IUGR

Initially at risk for;

- Perinatal Asphyxia, IVH, MAS, RDS
- Impaired thermoregulation, fasting and alimented hypoglycemia,
- Hyperviscosity-polycythemia syndrome, immunodeficiency, thrombocytopenia, leukopenia and NEC.

Potential long-term complications are;

- CP, behavioral & learning problems, & altered postnatal growth.
IUGR / SGA infant;

Typical shrunken or "wizened" appearance of an SGA infant.
Which baby is the infant of diabetic mother?

A

B
Neonatal Effects:

1. Congenital anomalies
2. Premature delivery
3. Perinatal asphyxia
4. Macrosomia and birth injury
5. Intrauterine growth restriction
6. Respiratory distress syndrome
7. Metabolic complications
8. Cardiomyopathy
6 - Respiratory Distress Syndrome (RDS):

- More frequently in IDMs (class A, B, and C) than in normal infants at each gestational age, especially before 38.5 weeks.

- Fetal lung maturation may occur early in diabetic pregnancies complicated by vasculopathy (class F or higher).

- Chronically stressed infants of Class D, F, and R diabetes may suffer from meconium aspiration if depressed or overly stressed before or during the birth process.

- Other causes of respiratory distress:
  - pneumonia
  - hypertrophic Cardiomyopathy
  - Transient Tachypnea of the Newborn (TTN)
6 - Respiratory Distress Syndrome (RDS):

- RDS occurs almost 6 times > IDMs

- Delayed maturation of surfactant synthesis has been observed in IDMs, (! cause is hyperglycemia, hyperinsulinemia, or both,)

- Insulin inhibits glycogen breakdown, decreasing the substrate available for synthesis of phosphatidylglycerol (PG), an important component of surfactant.
Neonatal Effects:

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2. Premature delivery
3. Perinatal asphyxia
4. Macrosomia and birth injury
5. Intrauterine growth restriction
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7. Metabolic complications
8. Cardiomyopathy
7 - Metabolic Complications:

1. Hypoglycemia
2. Hypocalcemia
3. Hypomagnesemia
4. Polycythemia
5. Hyperbilirubinemia
1) **Hypoglycemia**

- **Definition of hypoglycemia:**
  Blood glucose value of <40 mg/dL (2.2 mmol/L) in 1st 24 hrs of life and <45 mg/dL (2.5 mmol/L) after 24 hrs of age.

- Occurs frequently in IDMs (5 -25 %)

- Onset typically occurs in 1st few hrs after birth

- **Most common in macrosomic infants.**

- **Premature or SGA also are at increased risk of hypoglycemia**

- Healthy infants are fed 1st within half an hr of birth.

- Reagent strip monitoring; If normal blood sugar recheck blood sugar before feed x 3 hours.

- In infants who are premature or too ill to begin enteral feedings; begin parenteral glucose infusions at a rate of at least 5-7 mg/kg per minute.
1) Hypoglycemia (Management)

- **Healthy asymptomatic infants:**
  - laboratory measurement of plasma glucose concentration and immediately offer breast feeding.
  - check the glucose value 20 - 30 minutes after the feeding and continue to offer feedings at two- to three-hour intervals / demand.

- **Symptomatic infants or very low glucose concentrations:**
  - Start parenteral glucose therapy:
    - Symptomatic infants (lethargic, apnea, RD, hypotonic shock, cyanosis, seizures)
    - Plasma glucose concentration is less than 20 to 25 mg/dL
    - Remains below 40 mg/dL after feeding
    - Bolus infusion of 300 mg/kg glucose (3 ml/kg 10% dextrose)
    - Followed by a glucose infusion at 6 - 8 mg/kg /minute
    - Check the blood glucose concentration 20 minutes after the bolus
1) Hypoglycemia (Management)

- **Persistent hypoglycemia:**

Pharmacologic treatment with corticosteroids is considered in infants who remain hypoglycemic after 2-3 days of a glucose infusion >12 mg/kg/m:

- **Hydrocortisone** (5 mg/kg/day divided in two doses po or iv)
- **Prednisone** (2 mg/kg/day po)
- **Glucagon** (0.2 to 0.3 mg/kg/dose IV, IM, or SC; maximum dose 1.0 mg)
- **Diazoxide** (8-15 mg/kg/day po) should only be used if there is hyperinsulinemia
- **Octreotide** which suppresses insulin release in hyperinsulinism but expert advice is required before this agent is used.

- **Without adequate glucose, neonate metabolizes other energy sources; lactic acid, FFA, AA, & ketones, leads to cerebral anaerobic glycolysis & hypoxic ischemia**
2) Hypocalcemia:

- Defined as a total serum calcium concentration of <7 mg/dL
- Occurs in at least 10 to 20% of IDMs (50% in some series)
- The lowest serum calcium concentration typically occurs between 24 to 72 hours (hyperphosphatemia)
- Hypocalcemia is thought to be caused by the lower PTH concentrations after birth in IDMs compared to normal infants
2) Hypocalcemia:

- Hypocalcemia in term IDMs usually is **asymptomatic** and resolves without treatment.

- **Serum calcium concentration should be measured in infants with:**
  - Jitteriness
  - Lethargy
  - Apnea
  - Tachypnea
  - Seizures
  - Prematurity asphyxia
  - RDS & Suspected infection
2) Hypocalcemia ( Management )

- Most early hypocalcemia is asymptomatic and resolves without treatment.

- Infants with signs of hypocalcemia (irritability or seizures) are treated with calcium gluconate 10% (100mg/kg or 1 mL/kg IV). Or calcium chloride (20 mg/kg or 0.2 ml/kg).

- Dose can be repeated in 10 minutes if no response occurs.

- Maintenance Ca gluconate should be added to the IV solution (30 to 50 mg/kg/day PO in four divided doses).
3) **Hypomagnesemia**:

- Defined as serum magnesium concentration < 1.5 mg/dl.

- Occurs in up to 40% of IDMs within the first 3 days after birth.

- Mechanism is thought to be maternal hypomagnesemia caused by increased urinary loss secondary to diabetes.

- Usually is transient and asymptomatic and, thus, usually is not treated.

- In some neonates with Hypocalcemia & Hypomagnesemia, Hypocalcemia may not respond to treatment until the Hypomagnesemia is corrected.
4) Polycythemia:

- Defined as a central venous hematocrit of > 65%.
- Hematocrit should be measured within 12 hours of birth.
- Described in 13 - 33% of IDMs.
- Erythropoiesis within bone marrow and thrombocytopenia.
- Polycythemia may lead to Hyperviscosity syndrome:
  - Vascular Sludging
  - Ischemia, NEC...
  - Infarction of Vital Organs
  - Renal Vein Thrombosis
4) Polycythemia:

- Hyperglycemia decreases fetal oxygen tension, which in turn stimulates fetal erythropoietin production, resulting in an increase in red blood cells.

- Sludging of hyperviscous blood in the cerebral microcirculation can be responsible for symptoms of irritability, jitteriness, and a high-pitched cry, symptoms usually ascribed to hypoglycemia or Hypocalcemia.

- Pulmonary vascular bed sludging may manifest as PPHT
Renal Vein Thrombosis

- Rare, but life-threatening illness.

- Occurs more frequently in association with maternal DM.

- Subgroups of IDMs who are at special high risk are those from mothers whose pregnancies were further complicated by toxemia or hydramnios, or in IDMs already suffering from birth trauma, polycythemia, or sepsis.

- Presents with hematuria, flank masses, thrombocytopenia, and hypertension.
5) Hyperbilirubinemia:

- Occurs in 11 to 29% of IDMs
- Increased hemolysis (glycosylation of erythrocyte membranes)
- Increased risk for hyperbilirubinemia because of the expanded red cell mass, a decreased red blood cell life, and immature hepatic bilirubin conjugation and excretion.
- Jaundice is associated with:
  - Poor maternal glycemic control
  - Macrosomic
  - Polycythemia
  - Prematurity
8) Cardiomyopathy:

- IDMIs are at ↑ risk for hypertrophic cardiomyopathy (thickening of the IVS)

- Infants often are asymptomatic (30 to 50 %)

- 5 to 10 % have respiratory distress or signs of poor cardiac output or heart failure.

- CXR may show cardiomegaly,
- Echocardiography --- Hypertrophy
- Symptomatic infants typically recover after 2 - 3 wks of supportive care
- Echocardiographic findings resolve within 6 - 12 months
Cardiac Anomalies in IDM

1. **Cardiomyopathy**
   - Septal hypertrophy
   - Ventricular hypertrophy (bi- or uni-)
   - May cause outlet tract obstruction or hypertrophy may be so severe and essentially result in HLHS physiology
   - May present as CCF
   - Usually transient and resolves spontaneously in 6 - 12 months

2. **Truncus Arteriosus** (with or without aortic arch anomalies)

3. **DORV**

4. **VSD, TGA, Dextrocardia.**
• Chest radiograph of a vaginally delivered, full-term IDM (4.7 kg).
• Cardiomegaly, Hepatomegaly, Congested lung fields, and Fractures of the right humerus & left clavicle.
Diagnostic Investigations

Diagnostic investigations of IDM

1. CBC ---- polycythemia
2. Serum / whole blood **glucose** conctn --- N.N. hypoglycemia
3. Serum **magnesium** conctn --- hypomagnesemia
4. Serum **calcium** conctn --- hypocalcemia
5. Serum **bilirubin** level --- hyperbilirubinemia
6. **ABG** to assess oxygenation and ventilation with evidence of RD
7. **Radiography** for cardiopulmonary distress and skeletal and vertebral anomalies
8. **Echocardiography** for detecting cardiomyopathy and CHD
9. **Renal ultrasonography** for renal anomalies
Offspring of Diabetic Pregnancy: Long-Term Outcomes

- Higher frequency of IGT in offspring of mothers who had pre-gestational T1DM or T2DM or GDM

- 6 fold higher prevalence of IGT at ages 10-16yrs.

- Higher BMI and higher arterial blood pressure

- ↑ risk for obesity during childhood (Even in normal birth weight offspring ) in offspring exposed in utero to diabetes.

- ↑ risk for delayed motor and cognitive development
A Final Message

- Those providing care to IDMs must understand the gravity of the risks;
  - Chance of serious birth injury is doubled in this population
  - Cesarean section rate is tripled
  - Incidence of NICU admission is quadrupled
  - Rate of stillbirths is five times that in the general population
  - Congenital malformations are observed 2-5 times greater in IDMs, with anomalies occurring in any organ system.
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Please take good care of me!

I am the Future!

THANK YOU