INFANTS OF DIABETIC MOTHERS AND HYPOGLYCEMIA

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Main Topics

- Introduction & Etiology
- Concerns regarding Diabetes in pregnancy
- Complications
- Diagnosis & Work up
- Management

Presented By :
Duha Mohammed

Introduction

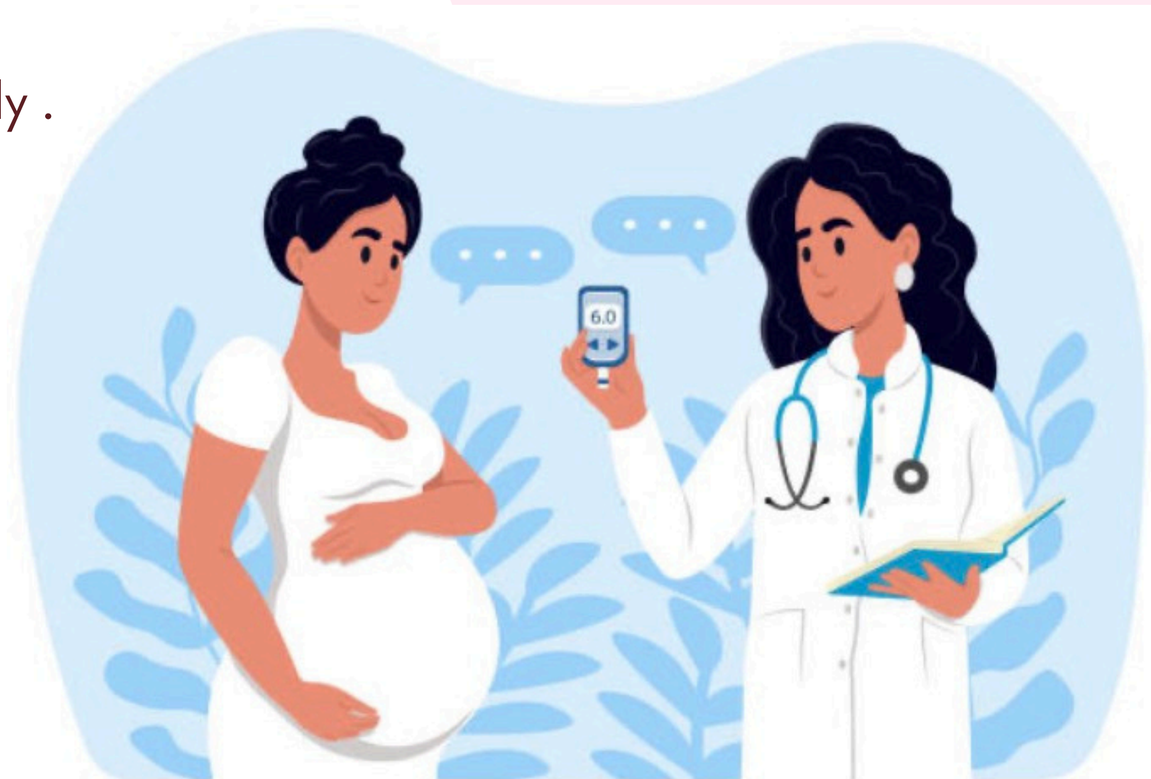
- The fetal and neonatal mortality and morbidity rates due to diabetes whom
affects the mother were as high as 60-65 %.

- but nowadays with the development of specialized maternal, fetal, and neonatal care for women with diabetes and their offspring by (Careful diet
management, blood glucose monitoring, and insulin therapy can help keep a mother's blood glucose levels at normal levels and decrease many of the risks to her baby).
- rates decreased by 30 folds.

Diabetes in pregnancy

According to the most recent (2018) International Diabetes Federation (IDF) estimates, GDM affects approximately 14% of pregnancies worldwide , representing approximately 18 million births annually .

About 5 % of all pregnant women in the U.S. are diagnosed with gestational diabetes . Some pregnant women require insulin to treat their diabetes .



Diabetes in pregnancy

Is a Baby born to a mother who has diabetes (persistently high blood glucose level) during pregnancy 5% of all pregnancies associated with maternal DM, Usually begins 20-24 weeks

There are two types of diabetes that occur in pregnancy :

1) Gestational diabetes : This term refers to a mother who does not have diabetes before conception but develops a resistance to insulin because of the hormones of pregnancy .

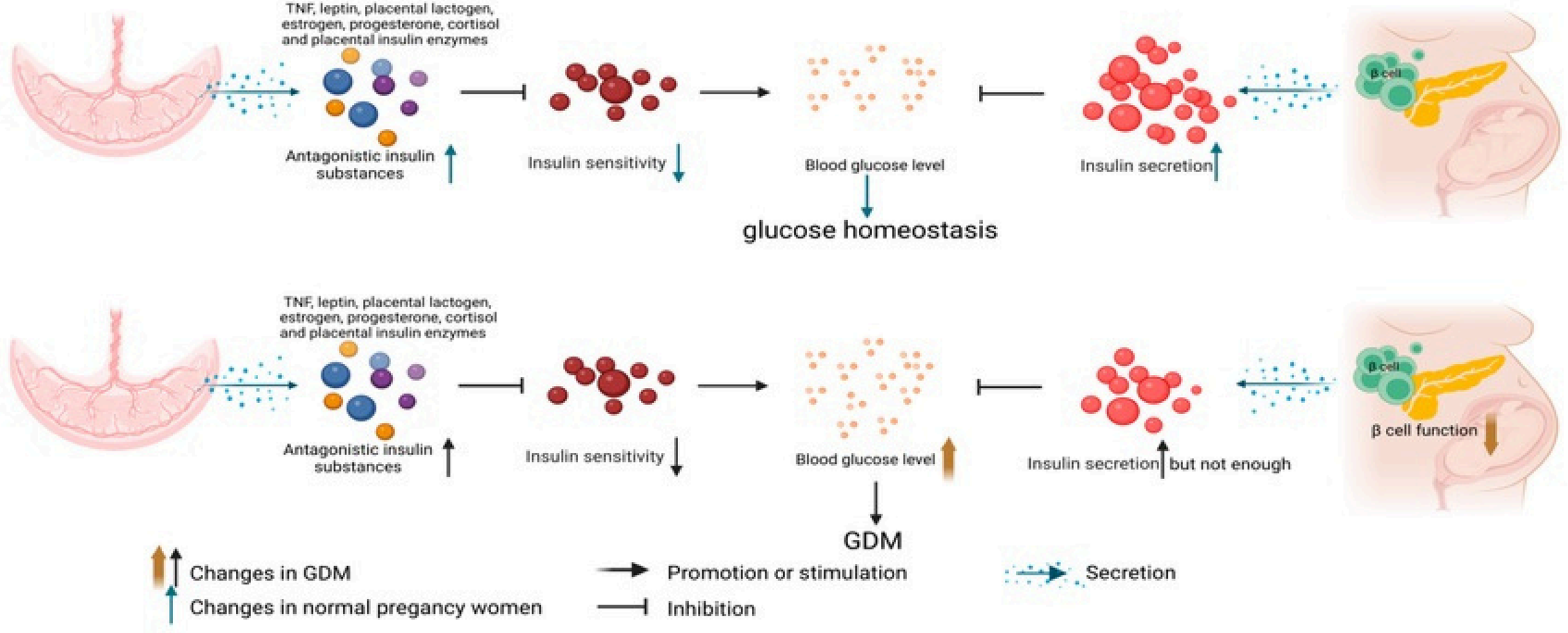
2) Pregestational diabetes : This term describes women who already have insulin-dependent diabetes (type 1 insulin dependent DM) and become pregnant

. Gestational diabetics make up the vast majority of pregnancies with diabetes (80%-88%).

DM in pregnancy

Pregestational DM
TYPE 1(35%)
TYPE 2 (65%)

GDM 80-88%

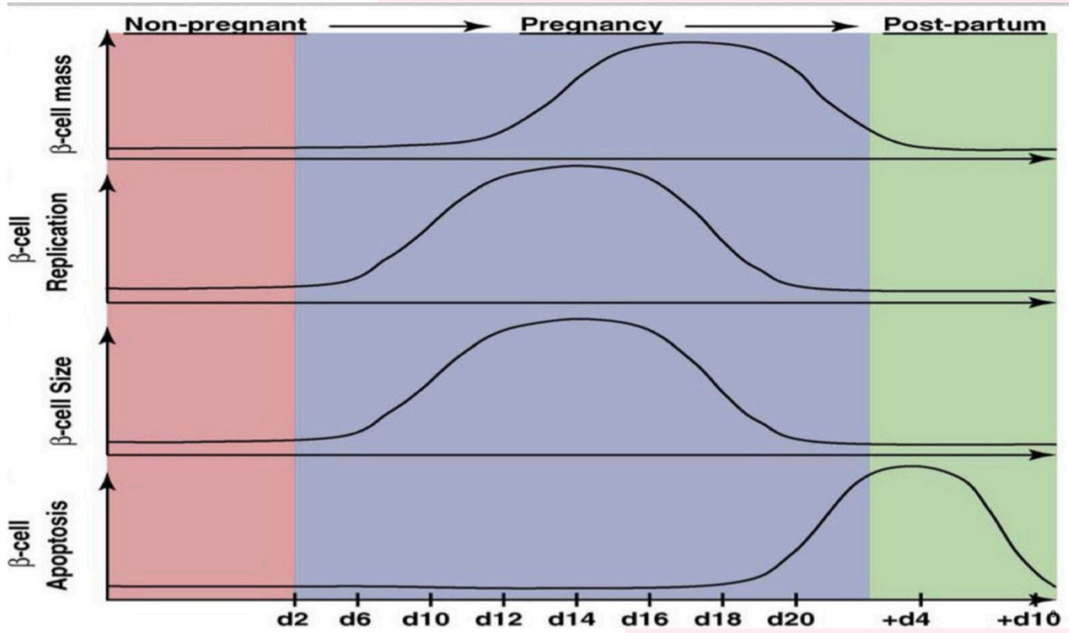

TNF, leptin, placental lactogen, estrogen, progesterone, cortisol and placental insulin enzymes
Antagonistic insulin substances
Insulin sensitivity
Blood glucose level
glucose homeostasis
Insulin secretion
β cell
TNF, leptin, placental lactogen, estrogen, progesterone, cortisol and placental insulin enzymes
Antagonistic insulin substances
Insulin sensitivity
Blood glucose level
GDM
Insulin secretion
but not enough
β cell
β cell function
Changes in GDM
Changes in normal pregancy women
Promotion or stimulation
Inhibition
Secretion

What causes diabetes in pregnancy?

- The placenta supplies a growing fetus with nutrients and water . It also produces a variety of hormones to maintain the pregnancy .
- Some of these hormones (estrogen , cortisol , and human placental lactogen i.e. human chorionic somatomammotropin) can block insulin activity .

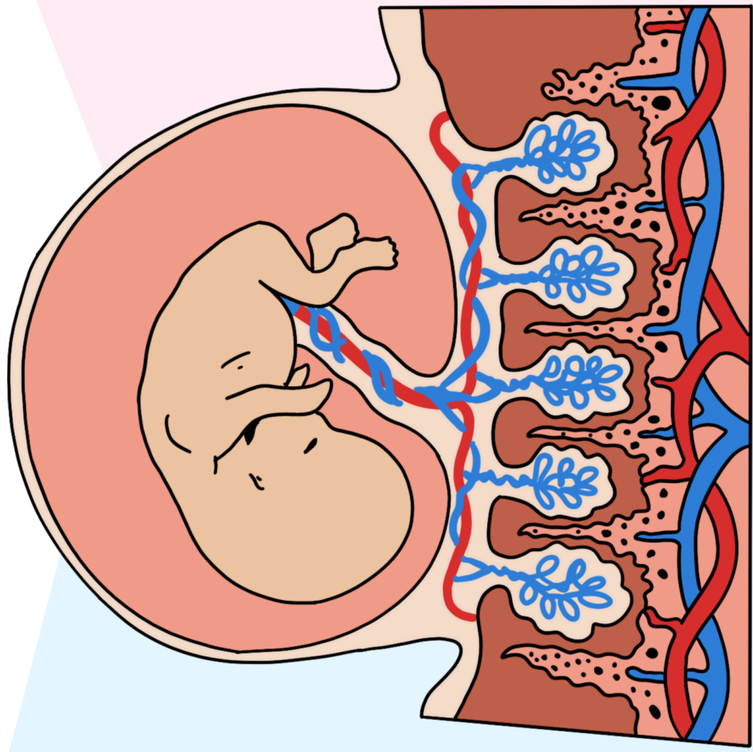
This usually begins about 20 to 24 days into the pregnancy .

The mechanism of β-cell mass expansion i.e. proliferation , neogenesis or hyperplasia as with obese individuals, when compensatory β-cell mass expansion fails during gestation , diabetes results .



As the placenta grows, more of these hormones are produced, and insulin resistance becomes greater .
Normally, the pancreas is able to make additional insulin to overcome insulin resistance, but when the production of insulin is not enough to overcome the
effect of the placental hormones , gestational diabetes results .

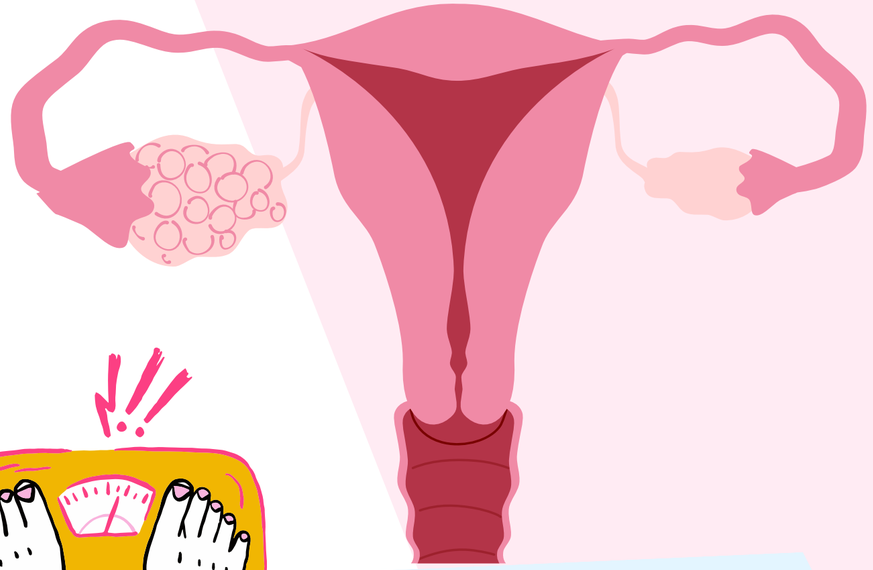
>>>>> leading to insulin resistance, hyperglycemia, and an increased supply of glucose to the growing fetus .
Pregnancy also may change the insulin needs of a woman with preexisting diabetes.
Insulin-dependent mothers may require more insulin as pregnancy progresses



Pre- GDM

Risk Factors :

- **over weight/ obesity**
- **westernized diet and micronutrient deficiencies**
- **advanced maternal age**
- **family history of insulin resistance and/or diabetes**
- **pcos**



Why is diabetes in pregnancy a concern?

The mother's excess amounts of blood glucose are transferred
to the fetus during pregnancy . This causes the baby's body to secrete increased amounts of insulin, which results in increased tissue and fat deposits (insulin-like growth factor) . The infant of a diabetic mother is often larger than expected for the gestational age (LGA i.e. fetal macrosomia) .

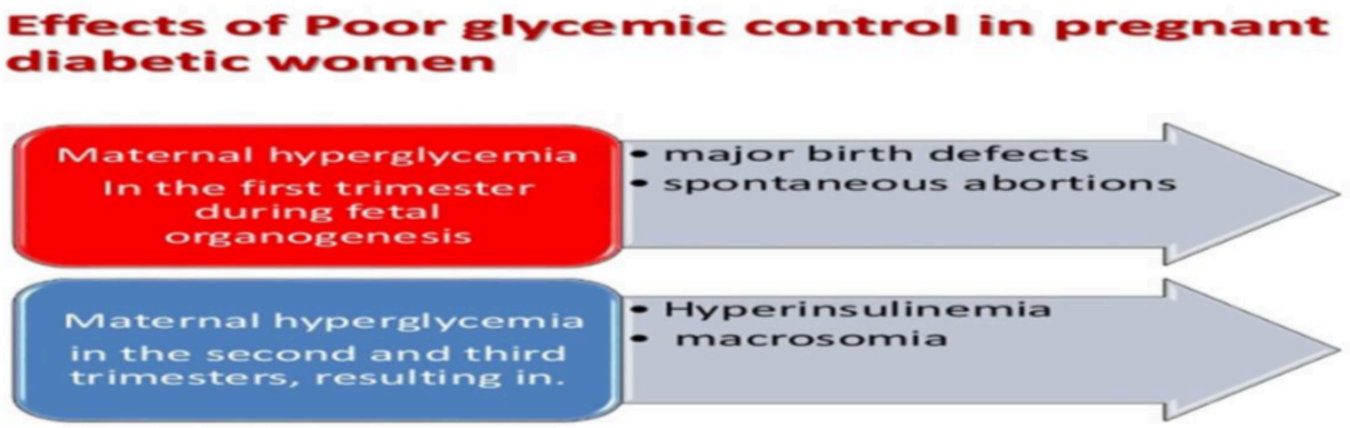
Gestational DM

usually resolves following delivery, but it can have long-lasting health consequences, including increased risk for type 2 diabetes (T2DM), future obesity, and cardiovascular disease (CVD) in the mother .

The infant of a diabetic mother may have higher risks for serious problems during pregnancy and at birth. Problems during pregnancy may include increased risk for birth defects and stillbirth, including problems with the formation of the heart, brain, spinal cord, urinary tract, and gastrointestinal system .

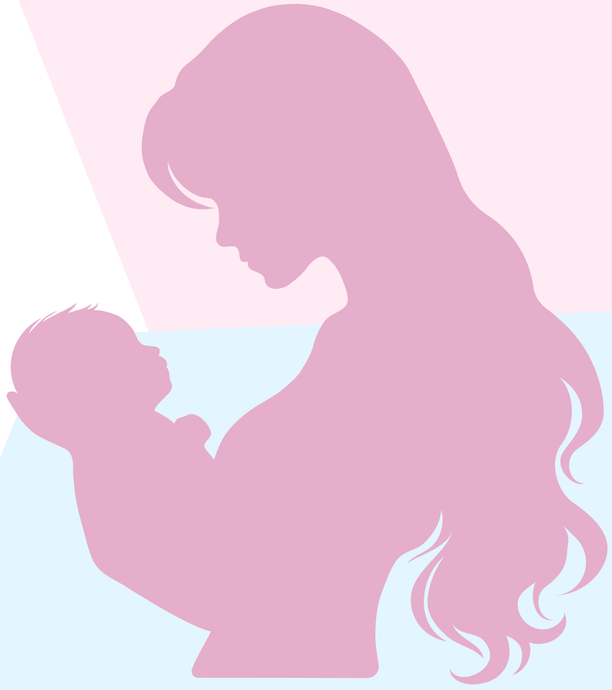
Unlike insulin-dependent diabetes, gestational diabetes generally does not cause birth defects .

Women with gestational diabetes generally have normal blood glucose levels during the critical first trimester when baby's organs form .



Complications :

- **Intrauterine growth restriction**
- **Fetal macrosomia**
- **Hypoglycemia**
- **Hypocalcemia and hypomagnesemia**
- **Pulmonary disease**
- **Hematologic problems**
- **Cardiovascular anomalies**



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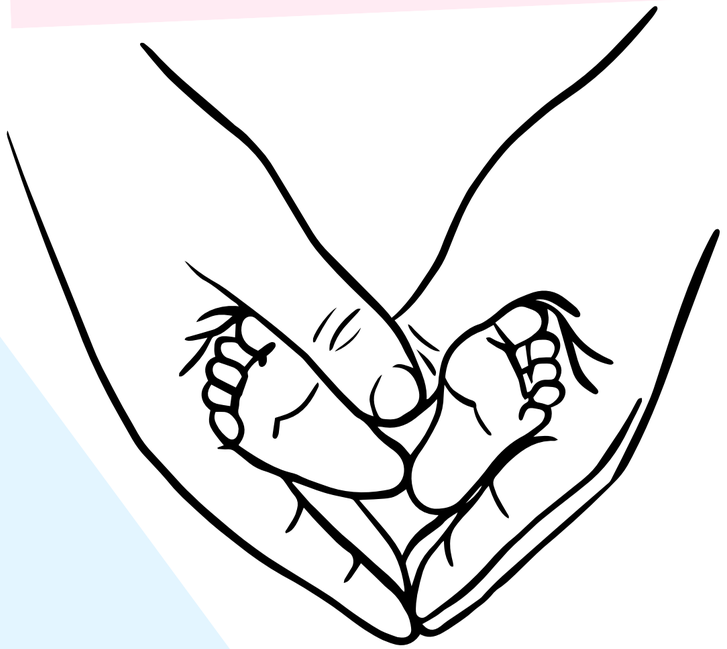
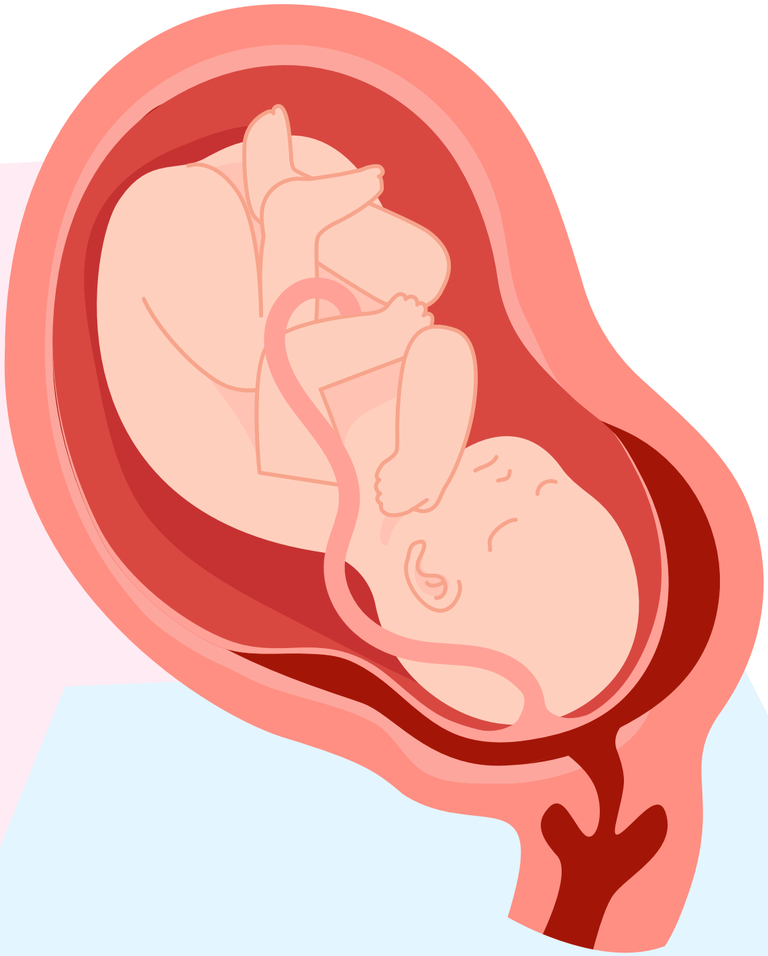
Rama Al.Qaisi

INTRAUTERINE GROWTH RESTRICTION :



The major long-term complications of DM is related to damage to the Blood vessels Results when a problem or abnormality prevents cells and tissues from growing or causes cells to decrease in size Intrauterine growth restriction can result in a baby being Small for Gestational age (SGA) which defined as weight below the 10thpercentile for the gestational age .

Symmetrical IUGR is commonly known as global growth restriction, and indicates that the fetus has developed slowly throughout the duration of the pregnancy and was thus affected from a very early stage .



- **One of the causes of IUGR is PreGDM , and also it can occur with (GDM , HTN , pulmonary disease , cardiovascular disease , renal disease etc...) .**

- **Perinatal death may occur, most commonly as a result of poor placental perfusion due to vascular impairment .**

- **Since most neurons are developed by the 18th week of gestation, the fetus with symmetrical IUGR is more likely to have permanent neurological sequleae .**

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Fetal Macrosomia :

macrosomia:
Macrosomia is the most constant consequence of diabetes and its severity is mainly influenced by maternal blood glucose level. 15-45% of diabetic pregnancies

Macrosomia is defined by a birth weight (BW) of 4000 or 4500 g and more , which is characterized by excess body fat, an increased muscle mass and organomegaly, without increase in brain size.

Fetal Macrosomia :

•Fetal overgrowth is related to increased transplacental transfer of maternal glucose, which stimulates the release of insulin. Insulin is a major factor of fetal growth and it up-regulates the Insulin-like Growth Factor (IGF) system, subsequently leading to fetal macrosomia.

•the infant appears puffy, fat, ruddy, and often hypotonic.

•all types of maternal diabetes are risk factors for macrosomia

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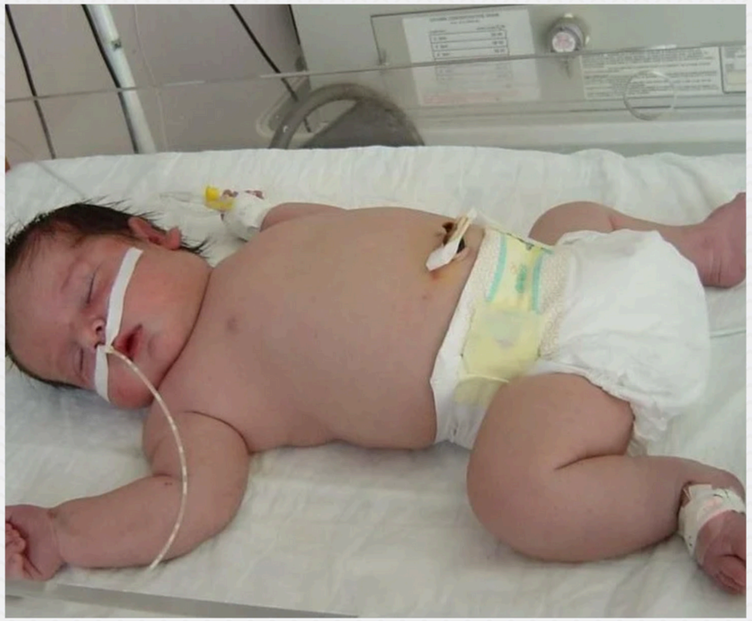
Complications of macrosomia :

Macrosomia may lead to potential birth injuries such as : shoulder dystocia , brachial plexus injury , clavicular or humeral fractures , Cephalohematoma , subdural hemorrhage , facial palsy .



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Hypoglycemia :

Hypoglycemia in infants defined as blood glucose concentration less than 40 mg/dl.

Occurs in 25 to 50% of IDM in the first 24 hours after birth.

Hypoglycemia :

Neonatal hypoglycaemia was strongly associated with elevated cord serum C-peptide levels[that reflects fetal insulin secretion]

Several studies also suggest that the IDM fails to release glucagon or catecholamines in response to hypoglycemia.

Neonatal hypoglycaemia is the main metabolic disorder that should be prevented as soon as possible after birth.

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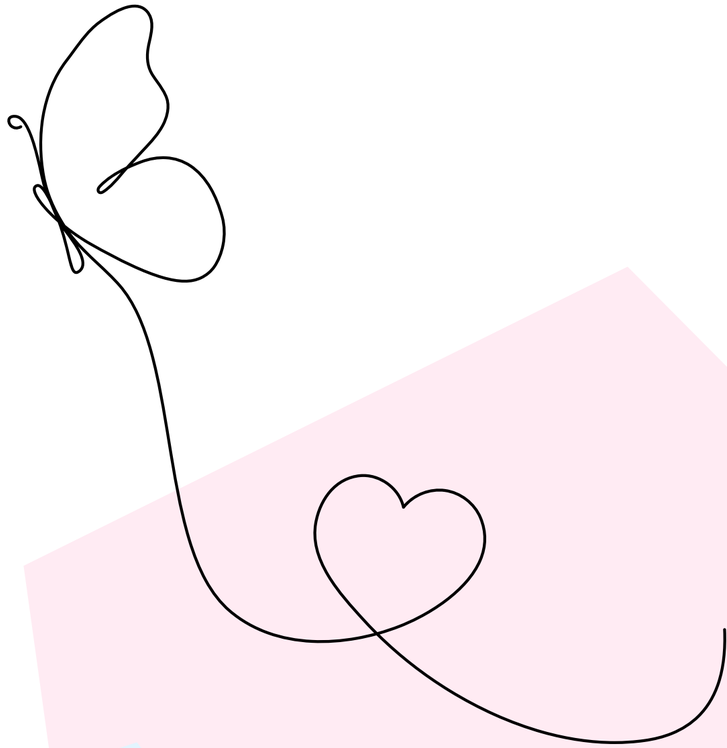
Hypoglycemia :

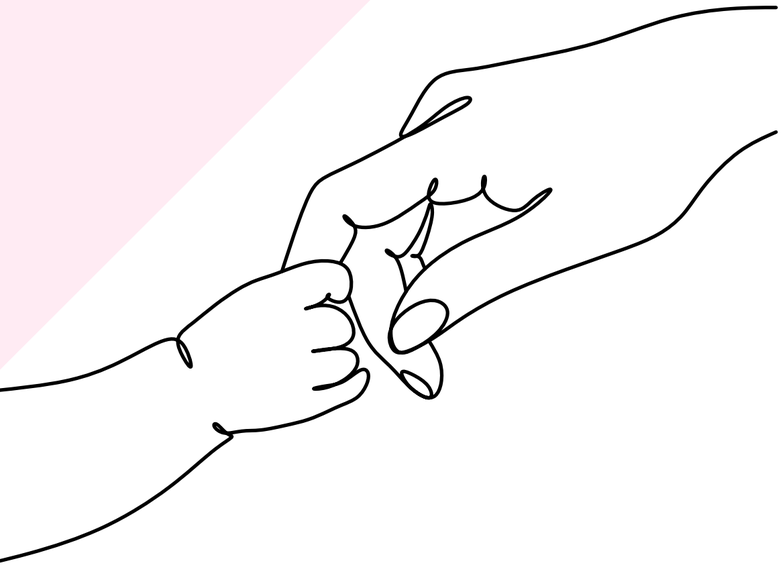


The infant of a diabetic mother is at risk of transient hyperinsulinism, which prevents at birth the normal activation of metabolic pathways producing glucose and ketone bodies, and causes increased glucose consumption by tissue.

Hypoglycemia :

Infants in the first or second day of life may be asymptomatic or may have life-threatening central nervous system (CNS) and cardiopulmonary disturbances.

Symptoms can include the following :



- **Hypotonia**
- **Lethargy, apathy**
- **Poor feeding**
- **Jitteriness, seizures**
- **Congestive heart failure**
- **Cyanosis**
- **Apnea**
- **Hypothermia**

Hypoglycemia :

- **IDM are sometimes asymptomatic despite having blood glucose concentration less than 30mg\dl.**

- **Because hypoglycemia ,even in the absence of symptoms, may cause brain damage and lead to long-term neurologic impairment, it is recommended that blood glucose concentration be maintained above 40mg\dl for all IDM.**

HYPOCALCEMIA AND HYPOMAGNESEMIA :

Hypocalcemia occurs in 10 to 20% of IDM during the neonatal period , usually is associated with hyperphosphatemia and sometimes with hypomagnesemia.

- hypocalcemia can be defined by plasma calcium concentration below 2 mmol/L or ionized calcium concentration below 1.1 mmol/L, regardless of GA or BW
- Transient neonatal hypocalcemia has been mainly reported in neonates of pregestational insulin dependent- diabetic mothers and may be partly related to maternal hypomagnesemia and subsequent fetal hypomagnesemia
- The mechanism is still unclear but seems to involve an abnormal calcium phosphorus metabolism during pregnancy with a decrease in calcium and vitamin D concentrations especially during the third trimester

Presented By :
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HYPOCALCEMIA AND HYPOMAGNESEMIA :

• Plasma parathormone concentration in IDM have been reported to be less than in infants of normal mothers during the first 4 days after birth , this may result from hypomagnesemia , which limits parathormone secretion even in the presence of hypocalcemia.

• Hypocalcemia in neonates can manifest as:
- irritability
- jitteriness,
- clonus or seizures
- Electrocardiography may show prolonged QT interval.

Pulmonary disease :

• It’s a big and Major problem

These infants are at an increased risk of neonatal respiratory distress and may present within the first few hours after birth with tachypnea, nasal flaring, intercostal retractions, and hypoxia and its due to inhibition of surfactant by the insulin, so the function and the concentration of the surfactant will be effected.

Pulmonary disease :

- In cultured fetal lung tissue, insulin blocks the development of enzymes necessary for the synthesis of lecithin, a principal ingredients of surfactant.

Asphyxia and meconium aspiration may end with pneumonia and RDS (the meconium itself can inhibit the synthesis and can effect the function of surfactant)

Cardiac, neural and other complication

Cardiovascular abnormalities:

• Cardiomyopathy with ventricular hypertrophy and outflow tract obstruction may occur in many fetuses of mothers affected by GDM.

These infants are also at an increased risk of congenital heart defects, including (most commonly) ventricular septal defect
(VSD) and transposition of the great arteries (TGA).

• Single umbilical artery is also seen. Neonates born with this condition are considered normal, but seeing such condition may alert us to search for other abnormalities. In short, single umbilical artery has no effect on fetus development but it can be associated with other anomalies.

, The majority of these infants are asymptomatic and the thickening is detected only by an ECG or .echocardiogram

An ejection systolic murmur is heard at the mid-to upper left .sternal border

May lead to left ventricular failure in the first few days after .birth

Cardiac, neural and other complication

Neurological defects :

- Numerous abnormalities may be seen, but one of the most common is neural tube defects.
- Evidence of early poor neurological maturation maybe demonstrated as poor suckling in babies.
- Caudal dysplasia may also be seen.
- Brachial plexus injury is seen in such neonates as they usually have macrosomia, which leads to birth injury during delivery, particulary C5 and C6 injury which is also know as Erb-Duchenne Palsy.

Presented By :
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Hematological abnormalities and hyperbilirubinemia

Platelet abnormalities was major hematological problem in neonates, in particular when complicated by the usual deficiency of Vit K in the past, which further complicated the problem. It was proved that platelet defects is due to thrombocytopenia due to the increase in red blood cells precursors. Platelet function may also be inhibited by the fact that there is polycythemia which interferes with normal platelet plug formation.

•Polycythemia is seen in neonates of GDM because the body of the fetus is not receiving enough oxygen to fulfill the demands, making the body appear to be in hypoxic conditions. This drives erythropoietin up making more RBCs precursors and hence the polycythemia

•Hyperbilirubinemia is multifactorial and is maybe due to the combined effect of immature liver, polycythemia, or the decreased RBC life span.

Presented By :
Rita Al.Qaisi

Hematological abnormalities and hyperbilirubinemia

Several factors appear contributing hyper viscosity :the hematocrit of umbilical cord blood at birth tend to be elevated , because of increase erythropoiesis, IDM often have enhanced placental transfusion at delivery , and elevated plasma fibrinogen concentration increase blood viscosity.. inceasre incidence of renal vein thrombosis

Other associated complications

Renal anomalies include: hydronephrosis, renal agenesis or uretral duplication

•Gastrointestinal defects maybe seen as: duodenal atresia, anorectal atresia, small left colon syndrome, etc..

•Small left colon syndrome will be further discussed upcoming ahead.

Sudden fetal death syndrome

is the death of child less than 1 year old due to an unknown cause, and with autopsy that is unable to identify the exact cause. It is usually silent and the baby would show no signs of struggle or any sounds.

- The exact cause is not known until now, but studies have showed that many risk factors could be a factors playing a role, such as smoking, not sleeping on the back, nonvaccinated child, etc.
- Recent studies shows that there is a correlation between neonates of mothers having GDM are at risk of sudden fetal death syndrome.

Work up :

Glucose concentration (serum or whole-blood)

• **Seizures, coma, and long-term brain damage may occur if neonatal hypoglycemia is Glucose concentration (serum or Whole-blood)unrecognized and untreated. Most centers recognize levels lower than 20-40 mg/dL within the first 24 hours after birth as abnormal, but the precise level remains controversial.**

• **A policy to screen infants of diabetic mothers (IDMs) for hypoglycemia should be in place in every hospital. Operational thresholds were proposed by Cornblath et al.**

• **They suggested that an infant with compromised metabolic adaptation (ie, IDM) undergo blood *glucose* measurements**

• **(1) as soon as possible after birth.**
• **(2) within 2-3 hours after birth and before feeding**
• **(3) at any time abnormal clinical signs are observed.**

Work up :

CBC count

• Polycythemia, commonly defined as a central **hematocrit** level higher than 65%, is a potential concern. Maternalfetal hyperglycemia and fetal hypoxia is a strong stimulus for fetal **erythropoietin** production and subsequent increase in fetal **hemoglobin concentration.**

• Thrombocytopenia may occur because of impaired thrombopoiesis due to"crowding-out" of thrombocytes by the excess of erythroid precursors in the bone marrow.

Work up :

CBC count Cont'...

• Magnesium concentration (serum)

• Hypomagnesemia is related to younger maternal age, severity of maternal diabetes, and prematurity. Neonatal **magnesium** levels are also related to maternal serum magnesium, neonatal calcium and **phosphorus** levels, and neonatal parathyroid function. The clinical significance of low magnesium levels in these infants remains controversial and uncertain.

• Calcium concentration (serum, ionized or total levels).

• Low **serum calcium** levels in IDMs are common. They are speculated to be caused by a functional hypoparathyroidism; however, their clinical relevance remains uncertain and controversial.

Work up :

Bilirubin level (serum, total and unconjugated)

• Hyperbilirubinemia (**see bilirubin**) is notably more common in IDMs than in the general population of neonates.

Causative factors include:
prematurity, hepatic enzyme immaturity, polycythemia, and reduced RBC half-life.

Work up :

ABGs are ordered in many cases depending on the situation, for instance, a neonate having ARDS must undergo ABGs

Work up :

Fetal macrosomia

- **A glucose tolerance test** at 24-28 weeks of gestation screens for gestational diabetes, as a known risk factor for macrosomia

- **Ultrasonographic measurements** to obtain estimated fetal weights are indicated when clinical assessments indicate a uterine size greater than that expected for the gestational age.

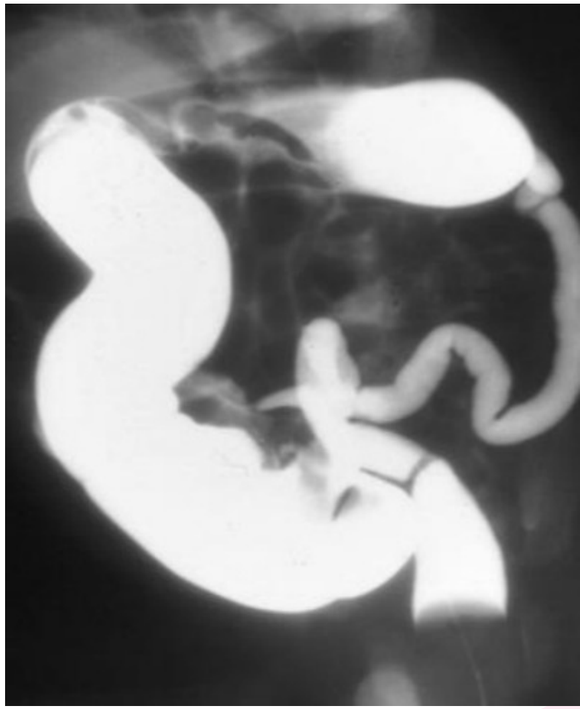
An examination within 1-2 weeks of delivery showing an abdominal circumference of 35 cm or larger should alert the clinician to anticipate a fetus with a birthweight of 4000 g or more.

The definitive diagnosis can only be made after delivery of the neonate.

Work up :

small left bowel syndrome

Apart from the difficulties to feed because of poor suckling pattern, **neonates of diabetic mothers may also exhibit neonatal small left colon**, a cause of functional lower intestinal obstruction that can mimic Hirschsprung disease.
It is associated with maternal DM. The treatment is always conservative as long as intestinal perforation does not happen.



Contrast enema is both diagnostic (abrupt transition zone at the splenic flexure) and curative, promoting the evacuation of meconium relieving the intestinal obstruction.

Work up :

Cardiac anomalies in GDM are plenty, but transposition of great vessels, truncus arteriosus, VSD or even hypertrophic cardiomyopathy
are fairly common and can be easily picked up using many techniques but most easiest and the one with a good cost effectiveness is **echocardiography**.

A powerful advantage that using such tool **can be used in the intrauterine period**

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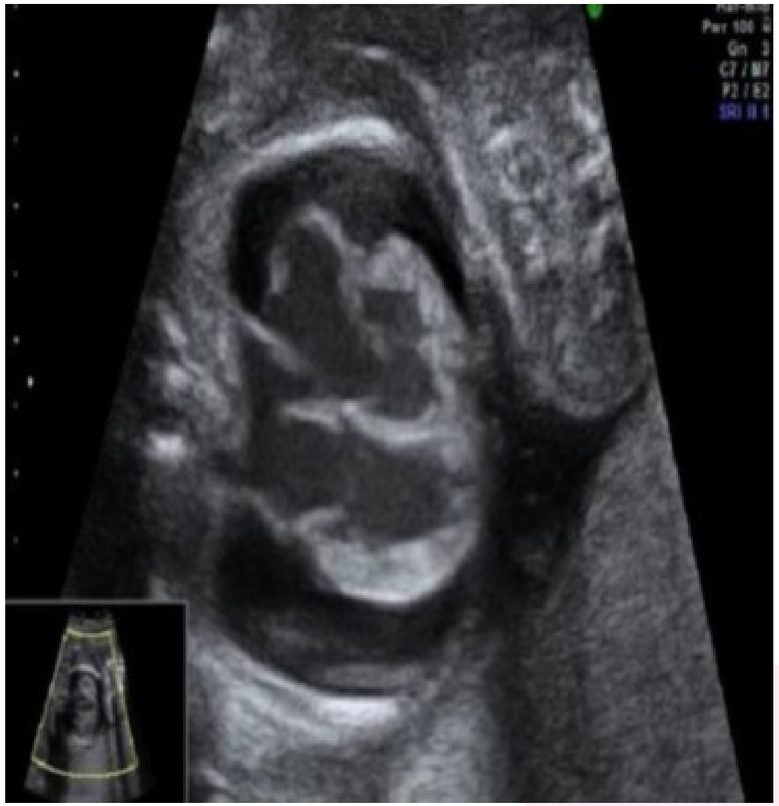


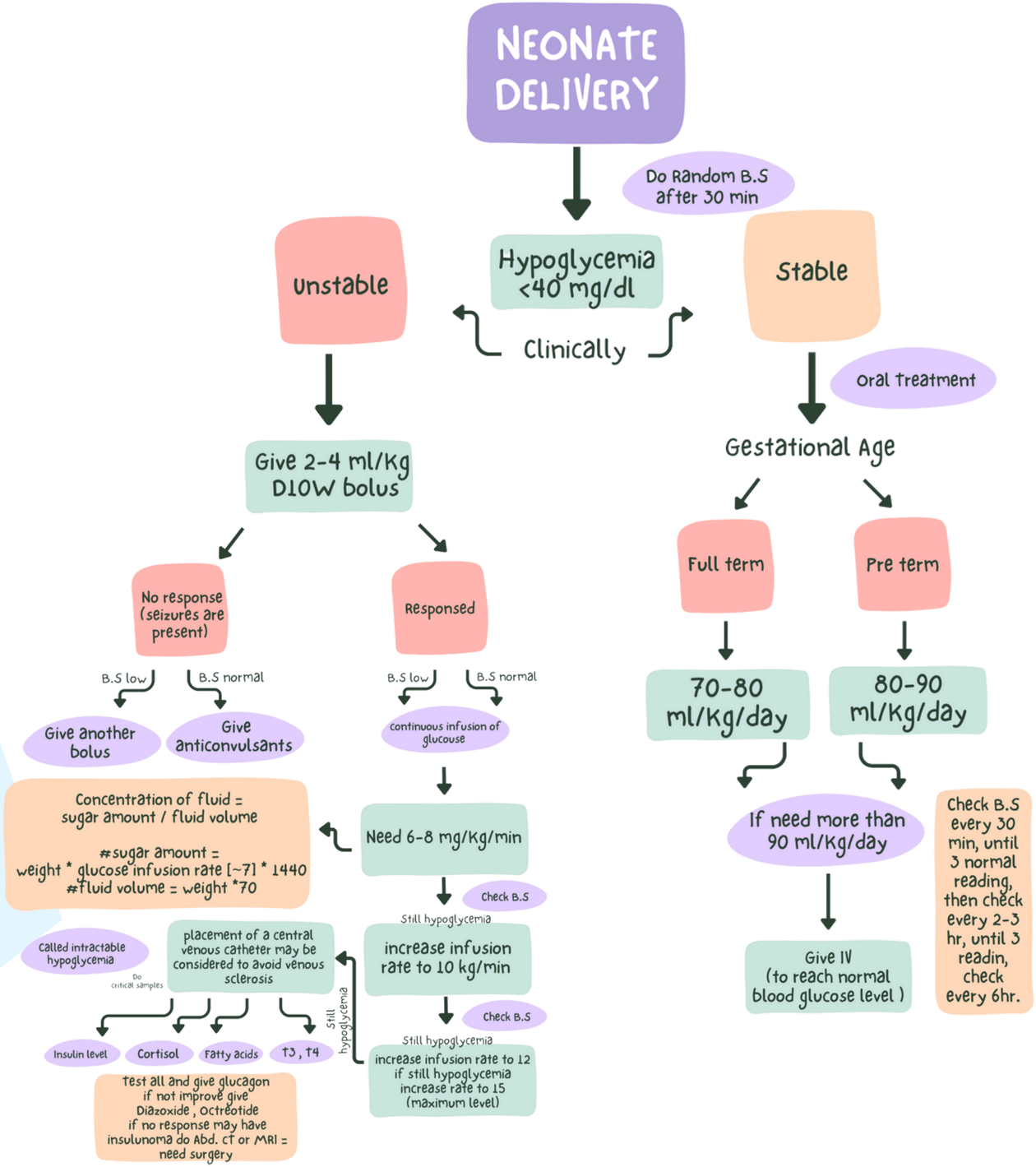
Presented By :
Daniah Sarayrah

Work up :

Immediately after labor, some anomalies will present their selves such as **caudal dysplasia,** which may include flexion contracture of lower limb, club foot, tibia and fibula defects, hypoplastic femur or even complete fusion of legs. Such complications are the most common orthopedic anomaly in IDM. **Plain radiographs for the abdomen, pelvis and lower extremity** are needed to evaluate the extent of such anomalies.

MANAGEMENT

Presented By :
Sadeen Mohammad


NEONATE DELIVERY
Do Random B.S after 30 min
Hypoglycemia <40 mg/dl
Clinically
Unstable
Stable
Oral treatment
Give 2-4 ml/Kg D10W bolus
Gestational Age
Full term
Pre term
No response (seizures are present)
Responsed
70-80 ml/Kg/day
80-90 ml/Kg/day
B.S low
B.S normal
B.S low
B.S normal
Give another bolus
Give anticonvulsants
continuous infusion of glucouse
Concentration of fluid = sugar amount / fluid volume
#sugar amount = weight * glucose infusion rate [~7] * 1440
#fluid volume = weight *70
Need 6-8 mg/Kg/min
Check B.S
Still hypoglycemia
increase infusion rate to 10 kg/min
Check B.S
Still hypoglycemia
increase infusion rate to 12 if still hypoglycemia increase rate to 15 (maximum level)
Still hypoglycemia
placement of a central venous catheter may be considered to avoid venous sclerosis
Called intractable hypoglycemia
Do critical samples
Insulin level
Cortisol
Fatty acids
T3 , T4
test all and give glucagon if not improve give Diazoxide , Octreotide if no response may have insulunoma do Abd. CT or MRI = need surgery
If need more than 90 ml/Kg/day
Give IV (to reach normal blood glucose level)
Check B.S every 30 min, until 3 normal reading, then check every 2-3 hr, until 3 readin, check every 6hr.

ACT FAST IN TREATMENT!

10 mins Hypoglycemia may lead to brain insult

- **Normal blood glucose in neonate 45 mg/dl**
- **Hypoglycemia<40mg/dl**

WHEN TO DO RANDOM BLOOD SUGAR?

- If the baby is hypoglycemic repeat the test again after 20-30 minutes.

- Hypoglycemia:glucose<40mg/dl,in infant of diabetic mother it starts after 1 hour of delivery

GOALS!

THE GOAL OF TREATMENT

Is to maintain a blood glucose level of at least 45mg/dL(2.5 mmol/L).

•Note:Hypoglycemia with marked acidosis(pH<7.1)suggests shock or serious underlying disease (mostly inborn of metabolism diseases) and should be treated appropriately.

PRESENTATION AND TREATMENT

- We have two modes of presentation:

1. Asymptomatic
2. Symptomatic:

- We have two modes of treatment:

1) Enteric
2)Intravenous

IS THE BABY SYMPTOMATIC OR NOT?

• Most important symptom is:
neurological manifestations as convulsion, jitteriness

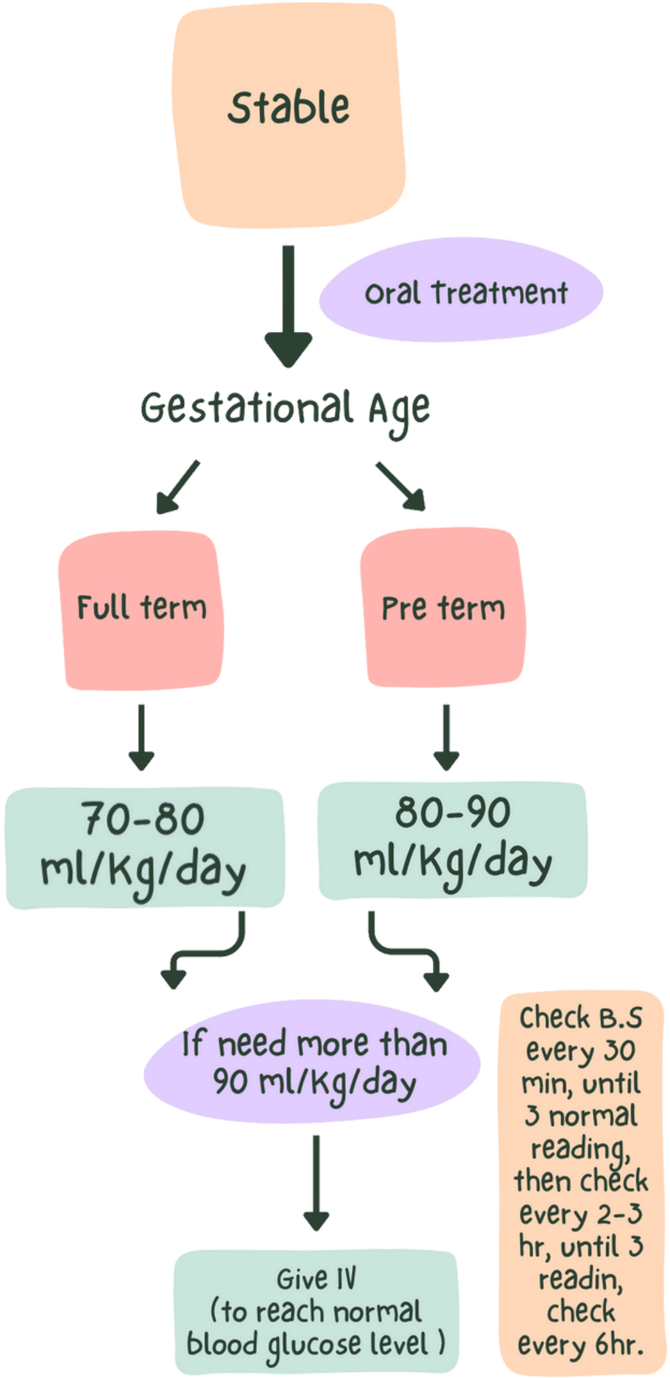
• Other symptoms:
1- poorfeeding
2- tremor like movements in the limbs
3- respiratory distress
4- tachypnea>70 (normal RR in neonateis:40-60/min)

IF THE PATIENT IS ASYMPTOMATIC

Check respiratory rate :

if its >70breath\min don't give oral feeding because of risk of aspiration
(this is an indication of I.V feeding) .

if RR <70breath\min then start oral feeding.


Stable
Oral treatment
Gestational Age
Full term
Pre term
70-80 ml/Kg/day
80-90 ml/Kg/day
If need more than 90 ml/Kg/day
Give IV (to reach normal blood glucose level)
Check B.S every 30 min, until 3 normal reading, then check every 2-3 hr, until 3 readin, check every 6hr.

How much will I feed the baby to maintain nsormal B.S ?

Volume:

Any full term baby should be fed 70-80 ml/kg in the first 24 hours .

but in premature babies it's more (80-90 ml/kg

How much will I feed the baby to maintain nsormal B.S ?

frequent times:

normal babies are usually fed every 3-4hours

but hypoglycemic babies should have frequent times of feeding , every 2 hours

Example

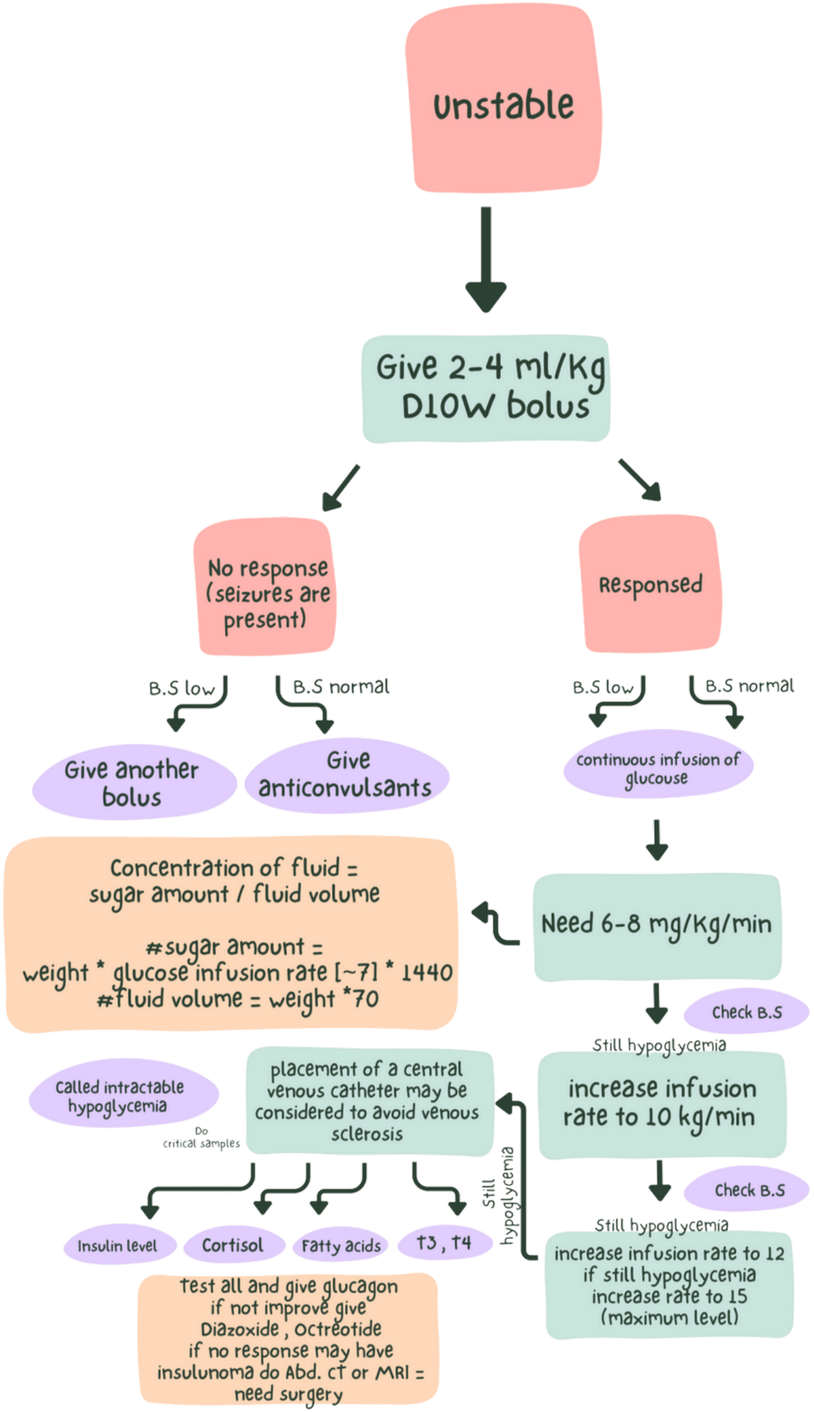
A full term baby whose weight is 4kg
In this baby ,
total milk is 4 x70 = 280 ml in 24 hours
which means 280/12= 25 ml every 2 hours

start enteric feeding with follow up of blood sugar every 30 min, until we have 3 normal readings (improve with time as 30,40,50 mg\dl)
-then check B.S every 2-3 hours, until we have 3 normal readings. -then every 6 hours .

Check random blood sugar every 30 minutes , if the baby is still hypoglycemic what is the next step ?
Increase the amount of feeding to 80 ml/kg , so
4x80= 320 ml,
320/12 = 30 ml every 2 hours
If we increased the amount of feeding but the baby is still hypoglycemic what is next step ?
-Don't increase the amount again , start I.V feeding

INDICATIONS FOR I.V GLUCOSE

1- Neurological manifestations (symptomatic)
2- Respiratory unstable (RR>70)
3- Not responding to oral feeding (Feeding as tolerated)
4- Glucose <25mg/dl


Unstable
Give 2-4 ml/Kg D10W bolus
No response (seizures are present)
Responsed
B.S low
B.S normal
Give another bolus
Give anticonvulsants
B.S low
B.S normal
continuous infusion of glucouse
Need 6-8 mg/Kg/min
Concentration of fluid = sugar amount / fluid volume
#sugar amount = weight * glucose infusion rate [~7] * 1440
#fluid volume = weight *70
Check B.S
Still hypoglycemia
increase infusion rate to 10 kg/min
Check B.S
Still hypoglycemia
increase infusion rate to 12
if still hypoglycemia increase rate to 15 (maximum level)
Still hypoglycemia
placement of a central venous catheter may be considered to avoid venous sclerosis
Called intractable hypoglycemia
Do critical samples
Insulin level
Cortisol
Fatty acids
T3 , T4
Test all and give glucagon
if not improve give Diazoxide , Octreotide
if no response may have insulunoma do Abd. CT or MRI = need surgery

WHAT IF SEIZURES ARE PRESENT?

Give (2- 4 mL/kg) bolus of D10W (=200-400mg \kg) Over 5 to 10 min If the baby (still seizure):

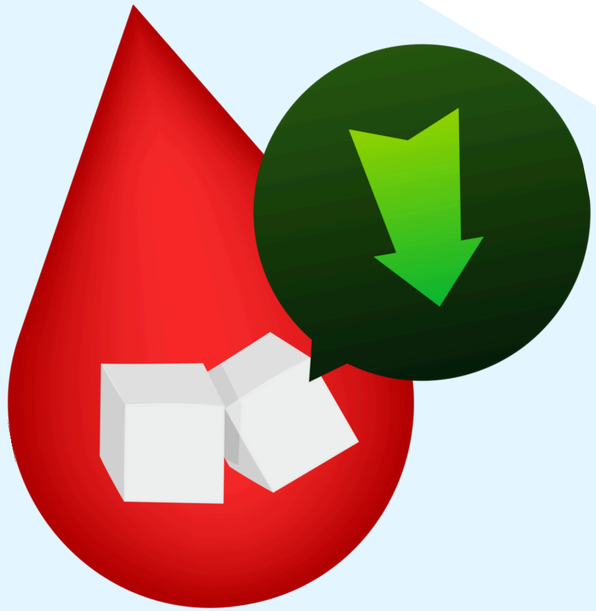
Check B.S :

If it is low give another bolus

If it is normal BS,,, give anticonvulsants Phenobarbital

if the baby still seizure give antiepileptic and still give glu tx

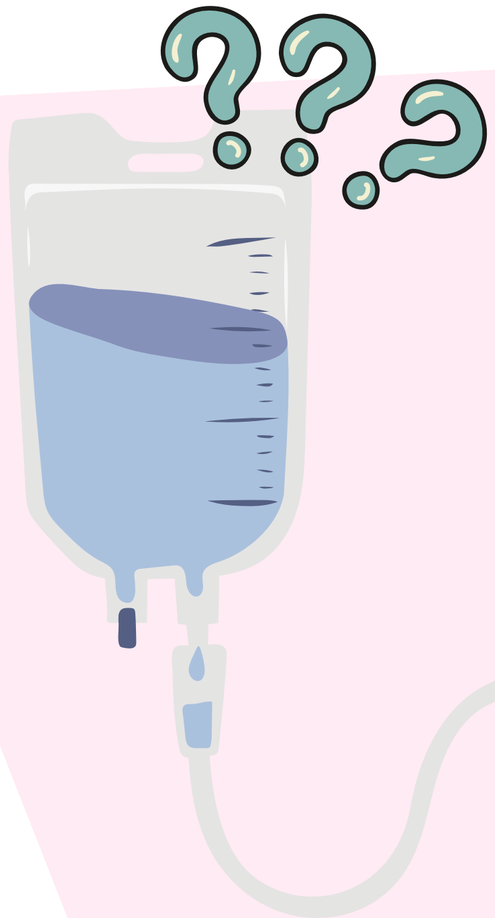
No seizure but still hypoglycemia



Normal glucose infusion rate is :4-6 mg/kg/minute
If we give bolus and the baby responded (no seizure) but he is still in .hypoglycemia , start I.V fluid (follow by a continuous infusion of glucose at 6-8 mg/kg/min
they are at high risk of hypoglycemia
glucose infusion rate (mg/kg/min) = % dextrose * I.V rate (mL/h) /6 x body weight (kg)Determination Of Fluid Type

- 24hrs=1440min
- Concentration =g/l

DETERMINATION OF FLUID TYPE



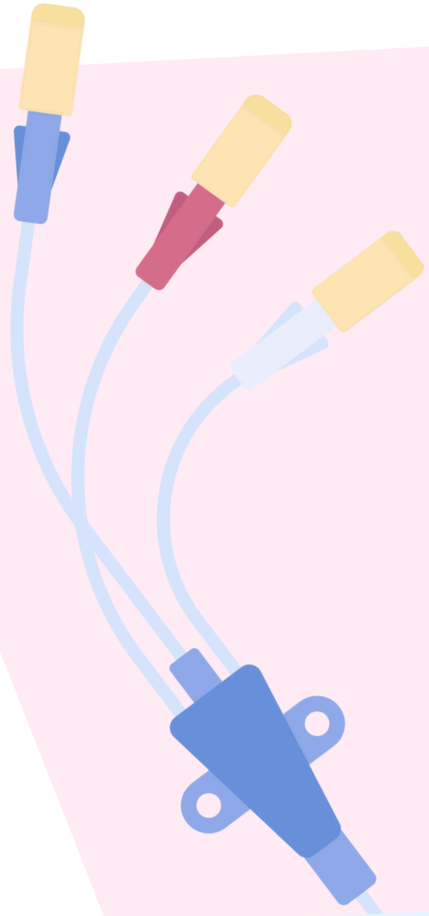
Concentration of fluid = (amount of sugar per day / volume of fluid per day) = (wt * glu infusion rate 6-8 mg / kg / min * minutes in 24 hrs) / (70-80 *wt)

So if baby is 4 kg ,
4*7*1440 =
40320 /1000 = 40 g /day (amount per day
Volume per day = 70*4 =280 ml
concentration of fluid = 40/280 =14 % glu (15%glu)

Continue

how many ml per hrs should I give ?
280/24=11ml per hrs of glu water 15%

If the infant requires a dextrose concentration of more than 12.5 , placement of a central venous catheter may be considered to avoid venous sclerosis



If the Baby is still hypoglycemic , what is next step ?

increase glucose infusion rate by 2 each time 6-8-10-12-15 -
Glucose concentration = amount/volume
so (10x4x1440)/1000 = 58 g
= 58 /280 = 20% glucose

NO RESPONSE

If after 30 minutes still hypoglycemic ?

incrase glucose infusion rate by 2mg/kg=12 mg/kg/min ,

- so (12x4x1440)/1000 = 69 g
- concentration=69/280 = 25% glucose

If still not responding

NO RESPONSE

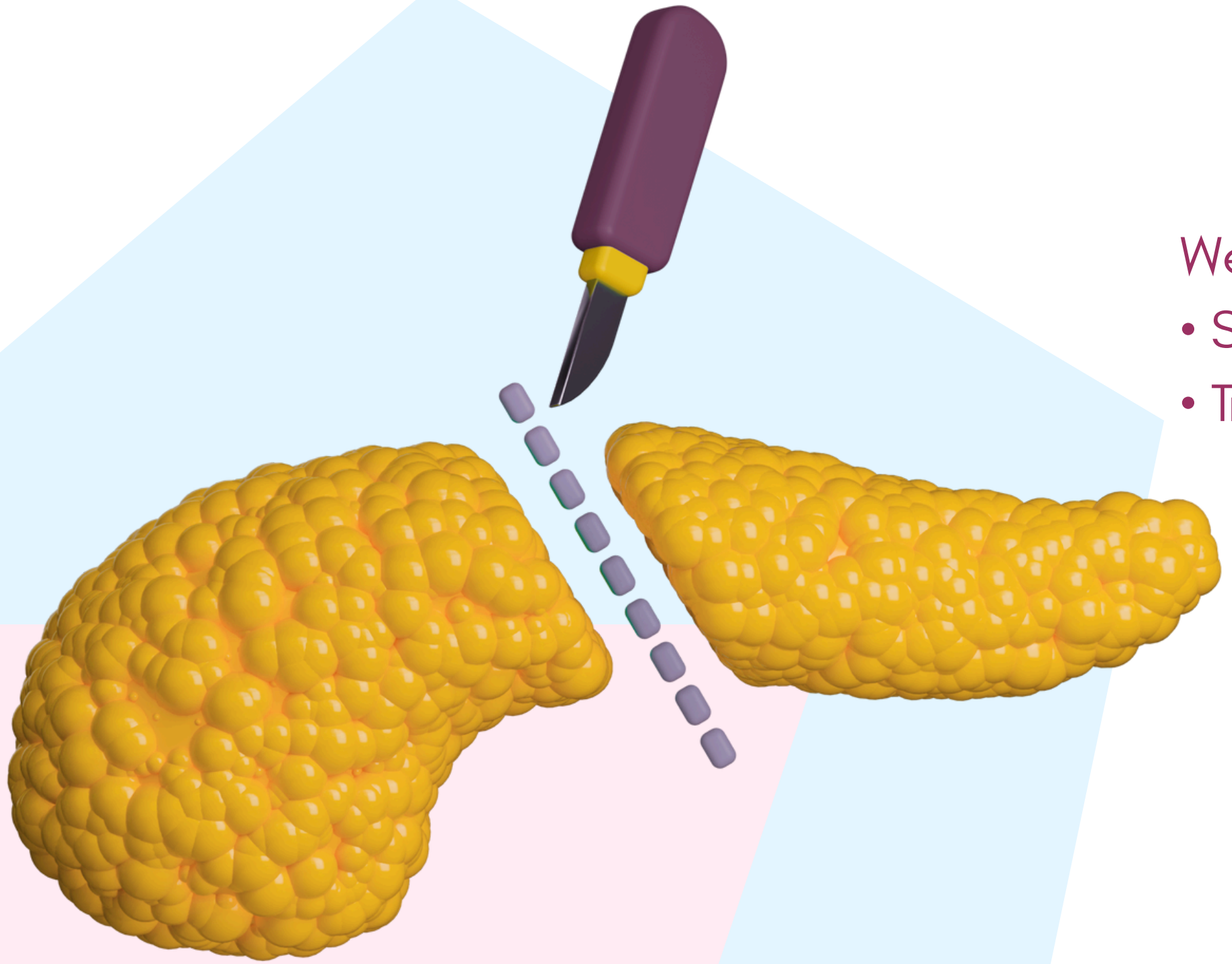
Intractable hypoglycemia:
long standing hypoglycemia
ddx?

• Maximum glucose infusion rate = 15 mg/kg/min
If baby still not responding now its consider (intractable hypoglycemia)
and send critical sample for patient :
(insulin ,cortisol, FA, T3,T4)
• Then:
give Glucagon 1 mg I.V infusion (best) or IM
Contraindication in preterm baby
main clinical reason is lack of liver glycogen for Glucagon to act on).

NO RESPONSE

If still not responding

Hydrocortisone (5 mg/kg/day divided in two doses intravenously) or
Prednisone (2 mg/kg /day orally).
It affects in the baby- adrenal insufficiency
Octreotide (somatostatin analogue): (2 - 10 mg/kg/day SC/IV divided q 12hr ; increase on basis of response)
diazoxide : which is antihypertensive drug (arterial vasodilator) , but one of its side affect is hyperglycemia
2-5 mg/kg orally every 8 hrs



If still not responding

We suggest that the diagnosis is : insulinoma

- So we do pancreatic CT
- Treatment : subtotal pancreatectomy

Other Measures of treatment

In infants of diabetic mothers (IDMs), calcium and magnesium levels are commonly measured within the first hours after birth. Ideally, ionized levels of these electrolytes should be obtained and used to properly manage these electrolyte disturbances..

Other Measures of treatment

Low levels may be treated by adding calcium gluconate to the IV solution to deliver 600–800mg/kg/day of calcium gluconate

Bolus therapy should be avoided unless cardiac arrhythmia is present. Bolus therapy may result in bradycardia.

Thank You

«عـزمٌ على الخيـراتِ هذا دأبُنا ... نسعى لتحصيلِ العلومِ بلا كللْ

نــدري بأن العلمَ صعـبٌ نيـله ... ولنَحْن نحيـا باليقيـنِ وبالأملْ»

اللهمَّ ألِّف بيننا وبين الحقِّ، وقرِّبنا من معادن الصدق، واعصمنا من بوائق الخلق