

# Diabetes In Pregnancy

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Before Insuline (1921)

-↑ M.M

-PNM 40-60%

After Insuline -- ↓ MM
-PNM <5%

Inidence:

-IDD 1/1000 -G.D 2-3%
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# **Diabetes in pregnancy**

#### □ Increase in prevalence

- -increase number of women of childbearing age with pregestational diabetes type 2
- -increase in the diagnosis of gestational D

# Categories of diabetes ncountered in obstetric practice

- Type 1 Diabetes
- Type 2 Diabetes
- Monogenetic Diabetes
- Mitochondrial Diabetes
- Secondary Diabetes
- Gestational Diabetes



# **Monogenetic Diabetes**

- Maturity onset of the young
- Single gene mutation----defect in pancreatic
   B-cell insulin secretion
- Autosomal dominant
- Not associated with obesity



#### **Mitochondrial Diabetes**

- Mutation in the mitochondrial DNA----defect in insulin secretion
- Associated with other medical problems sensrineural deafness, Tendency to stroke and lactic acidosis
- Develops in midtherties
- No obesity



# **Secondary diabetes**

Associated with other medical conditions Like pancreatitis, cystic fibrosis, Glucocorticoids and other drugs.

# **Screening & Diagnosis**

Random Blood Sugar

-Booking & 28wks

7.2 or >mmol/L  $\Rightarrow$  GTT

**Osullivan Test** 

50g(non fasting) at booking

1hr blood glucose >7.8mmol/L GTT

**GTT** 



# <u>GTT</u>

10% → Have indication for GTT



45% of women with G.D have one or more of the predisposing factors.

## GTT-----Indications

- -History of D in first degree relative
- -Glucosuria 2or >occasions(2nd fast.)
- -Maternal BMI >30kg/m square.
- -A previous baby wt 4.5kg or more.
- -Congenital abnormalities, IUD, Ndeath
- -Large for date -polyh. -prev.G.D
- -Recurrent candidal vulvovaginitis



# Three hours GTT (100gm)

Fasting < 95mg/dl 5.3mmol/l

1 hour 180mg/dl 10mmol/l

2 hours 155mg/dl 8.6mmol/l

3 hours 140mg/dl 7.8mmol/l



# 75 gms OGTT at 24-32 wks

- □ Fasting 5.1mmol/L (91.8 mg/dl)
- □ 1 hour 10 mmol/L (180 mg/dl)
- □ 2 hours 8.5 mmol/L (153 mg/dl)

# Pregnancy & CHO Metabolism

- sensitivity to insuline, with gest.
  - HPL
  - Estrogen & Progesterone
  - Cortisol
  - Degradation of insulin by plac.



# Effect Of Pregn.On Diab.

#### **Control is more difficult:**

- Lowered renal threshold.
- Nausea &Vomiting early in preg.
- Infection(e.g.UTI )--- Res.to insul.
- Labour Need glucose.
- Post partum → Req.of insuline.



#### □ First trimester

Implantation----inhibits trophectoderm differentiation

Embryogenesis---Activates the diacylglycerol

protein-kinase C cascade increasing

congenital defects

Miscarriage -----Increase premature programmed cell death of key progenitor cells of the blastocyst



#### Second Trimester

Endocrine pancreas---Stimulate fetal B-cells

Fetal growth----Stimulate fetal hyperinsulinemia that

results in growth acceleration seen

on U/S by 26 wks



#### □ Third Trimester

Fetal growth —A major fetal substrate an determinant for accelerated fetal growth

Adipose disposition----Stimulates hyperinsulinemia that promotes fat disposition including intra- abdominal fat.

Lung maturation---hyperinsulinemia delay lung maturation by inhibiting surfactant protein

Stillbirth---Is associated with defects in placental maturation that increase the risk of fetal hypoxia



- Delivery
  - Birth trauma----causing accelerated fetal growth shoulder dystocia—Trauma & asphyxia
- Neonate
  - hypoglycemia, Hypocalcemia, Polycythemia Hypomagnesemia, Cardiomyopathy, RDS
- Adolescence/adulthood
  - Obesity---Intrauterine exposure predisposis to metabolic syndrome independent of genetic susceptability Type 2 Diabetes



# Other effect Of Diab. On Preg.

Infection -UTI -Asym.baceruria

Monilial vulvo vaginitis:

- Clucose content of vag. epith.
- -Glucosuria

PET 8% - Renin&aldost.

-Angiotensir ~ Blood glucose.



## Effect of Diab.----cont.

Polyhydramnios:-25%

-foetal polyuria

**Preterm Labour** 

**Perinatal Death:** 

- -Unexplained IUFD
- Idiopathic RDS
- -Congenital Abnormalities



# Management

- Preconception counseling
  - -5 mg folic acid before conception and for 12wks
  - -Achieve the best possible HbA1c
  - -Ensure that all medications are safe for preg.
  - -Screened for possible eye and kidney diseases

# 

#### □ First trimester

- -Combined Clinic.
- -Dating scan
- -Screening for diabetic complications
- -Screening for non-diabetic morbidities
- -Assessment and optimization of glycemia (fasting 6mmol/l, 1 hour postprandial 7.8mmol/l)
- -Advice on hypoglycemia prevention
- -Experienced Dietition.



- Second trimester
  - -Optimization of gycemic control
  - -Screening for congenital abnormalities
  - -Surveillance for medical/obstetric complications
  - -Assessment of fetal growth.

#### □ Third trimester

- -Optimization of glycemic control
- -Assessment of fetal growth
- -Timing and mode of delivery

# Obestetric Manag.----cont.

- -Delivery:
  - Uncomplicated, well controlled,

and normal growth 40 wks.

- -Bad obestetric history 38 wks.
- -Mode of delivery:
  - -c/s is not indicated.
  - insulin infusion -5% dextrose.

# Obestetric Manag.----cont.

- -Induction of Labour:
  - -If unfavorable -> PG.
  - -If favorable ARM & Oxytocin.
  - -Aim Delivery within 12hours.
  - -Insulin → 1/2 dose
  - -Hourly blood glucose.
- -Postpartum -> prepreg. dose.



# Breast feeding Family planning

- -Breast feeding:
  - CHO by 50 gm/day.
  - -Oral hypoglycemic → contraindic.
- -Family planning:
  - -Barrier methods -IUCD -OCP
  - -Sterilization &Vasectomy.