

Diabetes In Pregnancy

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Before Insuline (1921)

-↑ M.M

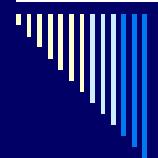
-PNM 40-60%

After Insuline - MM

-PNM <5%

Inidence:

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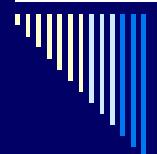
Diabetes in pregnancy

■ 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 encountered in obstetric practice

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



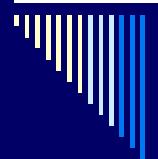
Monogenetic Diabetes

- 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

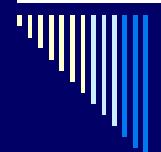
7.2 or >mmol/L | GTT

Osullivan Test

50g(non fasting) at booking

1hr blood glucose >7.8mmol/L GTT

GTT

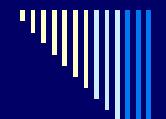


<u>GTT</u>

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

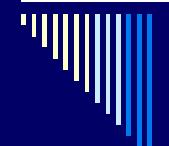


75 gms OGTT at 24-32 wks

- □ 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

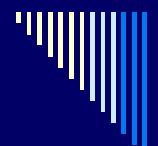
- sensitivity to insuline, with gest.
 - HPL
 - Estrogen & Progesterone
 - Cortisol
 - Degradation of insulin by plac.



Effect Of Pregn.On Diab.

Control is more difficult:

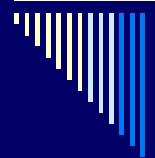
- Lowered renal threshold.
- Nausea &Vomiting early in preg.
- Infection(e.g.UTI)--- Res.to insul.
- Labour Need glucose.
- Post partum → Req.of insuline.



First trimester

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Implantation----inhibits trophectoderm differentiation
Embryogenesis---Activates the diacylglycerol
protein-kinase C cascade increasing
congenital defects
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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



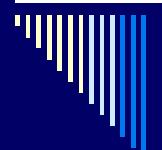
□ Third Trimester

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

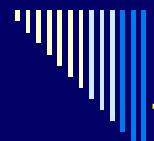
Adipose disposition-----Stimulates hyperinsulinemia that promotes fat disposition including intra- abdominal fat.

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:

- Glucose content of vag. epith.
- -Glucosuria

PET 8% - Renin&aldost.

-Angiotensi ~ Blood glucose.



Effect of Diab.----cont.

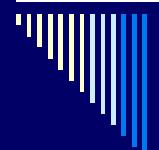
Polyhydramnios:-25%

-foetal polyuria

Preterm Labour

Perinatal Death:

- -Unexplained IUFD
- Idiopathic RDS
- -Congenital Abnormalities

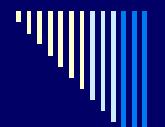


Management

- 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.



Management

- 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,

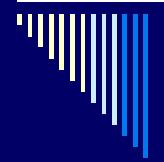
and normal growth 40 wks.

- -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.