Diabetes In Pregnancy

Before Insuline (1921)

- ↑ M.M

-PNM 40-60%

After Insuline → - ↓ MM

-PNM <5%

Inidence :

-IDD 1/1000 -G.D 2-3%

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

GTT

10% ⇨ Have indication for GTT

⇩

20-25% Abnormal

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.

Effects of maternal hyperglycaemia

- **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

Effects of maternal hyperglycaemia

- **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

Effects of maternal hyperglycaemia

- **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

Effects of maternal hyperglycaemia

- 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.

-Angiotensin ↑ ~ 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

Management

- **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.