

# Diabetes in pregnancy

metabolic disease result from underproduction of insulin which effect CHO , fat &protein metabolism

During pregnancy

- 1.preexisting
- 2.gestational

## Pathophysiology

1-Insulin resistance due placental secretion of

Anti insulin hormones

- 2-Maternal hepatic glucose production by 15% to meet fetal demand
- 3-Pancreatic B cells dysfunction due to
  - genetic , autoimmune

## Homeostasis during pregnancy

NP - FBS maintained 4-5 mmol/l

- insulin level double to maintained in the 2-3<sup>rd</sup> trimesters
- preg is insulin resistant state
- causes of resistant
  - a. placental hormones
  - b. changes in periphrel insulin receptors

Glucose cross the placenta by facilitated diffusion

# Gestational Diabetes

State of glucose intolerance which occurs at the end of second trimester or early third trimester met WHO criteria for diabetes and revert to normal after peurperium

## prevalence :

22 million women between 20-39 years have diabetes 2010 data

Expected to rise by 20% in next 10 year

## WHO criteria

	<u>fasting</u>	<u>2hr postprandial</u>
Diabetic	$\geq 8\text{mmol}$	$\geq 11\text{mmol} \setminus \text{L}$
Normal	$< 8\text{mmol}$	$< 11\text{mmol} \setminus \text{L}$

## screening

- No single test has been shown to be perfect
- Aim of screening is to identify asymptomatic patients with high propability
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**GTT** high risk or potential diabetic

- h\o 1 st degree relative or 2 nd
- poor obstetrical history
- glycoseuria on two occasion first in the morning
- polyhydramnias
- macrosomic infant
- obese mother
- advance maternal age

GTT on low risk group cannot be justified

- Glucose challenge test
- HbA1C
- Glycosylated protein
- Glucoseuria
- Standard meal test

### **Effect of diabetes on pregnancy**

**Feta & neonatal complication**

- miscarriage
- 2<sup>nd</sup> trim. Fetal death
- congenital fetal abnormalities – 3 times
- PIH
- Fetal macrocosmic
- Unexplained still birth
- Polyhydramnia & preterm labour

Neonatal : hypoglycemia , polycythemia ,  
hyperbilirubinemia  
RDS  
birth asphyxia & trauma  
hypocalcaemia&magnesiumemia

**maternal**

Maternal mortality & morbidity

\_MMR improved after insulin

- nephropathy
- retinopathy
- PE
- Infection : UTI , moniliasis ,chest infection
- Sever hypo & hyperglycemia
- operative delivery : 50%

### **Effect of pregnancy on diabetes**

- Difficulty in control
- lower renal threshold ,
- nausea and vomiting of early pregnancy
- diminish sensitivity to insulin as pregnancy advance

- Nephropathy may worsen
- Retinopathy previous proliferative retinopathy was contraindication for pregnancy

#### Management issue :

- education
- Medical nutrition
- Pharmacological therapy
- Glycemic control
- Fetal monitoring
- Plan delivery
- Postnatal care
  1. Required diabetic team
  2. Strict metabolic control before and during pregnancy
  3. Primary management :dietry changes and exercise

#### To achieve euglycemia

-Diet 1800 calories should be prescribe i.e30-35kcl \kg if ideal body weight + 300kcl to anticipate wt gain during pregnancy

-50-60 % CHO complex

-18-20% as protein

-25% as fat ( important to have bed time snaks )

#### Insulin

- Insulin should be first line treatment
- If uncontrolled uses of metformin
- 50% of women initially treated with diet only required insulin ( must be individualized)

-better to use combination 2\3th in the morning And 1\3th in the evening,  
with ratio 1:2 sort to intermediate or long acting

-Short acting +intermediate in two divided dose

-long acting rarely used

Special formula to calculate insulin requirment:

$$\begin{aligned} \text{unit insulin} &= \text{Bwt} \times 0.6 \text{ 1}^{\text{st}} \text{ trimester} \\ &= 0.7 \text{ 2}^{\text{nd}} \\ &= 0.8 \text{ 3rd} \end{aligned}$$

#### Dosage and schedule

2/3 in the morning , 1/3 in the evening

A.M 2/3 intermediate + 1/3 short acting

P.M 1/2 intermediate + 1/2 short acting

Aim : is to keep blood sugar pre-prandial < 6mmol/l i.e less than 100mg/dl , FBS 60-90 mg  
exercise should be encourage 1/2hr after meal

#### Antenatal obstetrical management

Surveillance should be maintained to avoid risk of maternal and fetal complication

Detailed USS at 1st trimester dating scan , nuchal translucency , biochemical screening ,  
uterine artery Doppler to detect early PE, middle cerebral artery to detect early IUGR 16-20 weeks

then 28-32 weeks (biophysical profile )

Fetal ECHO

- 2<sup>nd</sup> scan to detect anomaly at 19 weeks , triple marker , amniotic fluid index and fetal echo at 22 wks
- 3<sup>rd</sup> at 28,32 assess growth, AFI, non- stress
- Fetal monitoring
- Baseline USS , cong. Mal, fet. Echocardiogram
- At 26 growth and liquor

-Serum alfa fetoprotein

-maternal : renal ,cardiac , ophthalmic ,function are monitor

- glycosylated ( hba1c) monthly

### Timing of delivery

-maternal state is stable

- Blood glucose level is euglycemic
- Fetal growth is satisfactory

Wait until term (38-40wks) not beyond, if condition not met so intervention

### Intrapartum

Need to keep the mother glycemic in labor

CTG , ARM

- Continues infusion 5%- 10% dextrose + 0.5-2 unit of insulin
- Measure blood glucose every 2hrs AIM : 80-120 mg, adjust insulin accordingly aim is to keep level 80-100mg/dl
- Fetal scalp electrode, continuous monitoring
- 2<sup>nd</sup> stage anticipate —shoulder dystocia , assisted vaginal delivery
- 3<sup>rd</sup> stage —active management---PPH atonic
- Aim is vaginal delivery unless there is obstetrical complications
- c/s rate 50%
- Postpartum
- After delivery of placenta insulin requirement drops sharply
- 1<sup>st</sup> 48hrs most pt do not require insulin
- After depend on sliding scale
- Encourage breast feeding with addition of 70 calories
- Counsel about contraception

Barrier better

Combine pills risky

Progesterone only pills failure rate

IUCD infection

## Sterilization with advance vascular involvement

- 1.