

Diabetes Mellitus (DM)

Classification during pregnancy:-

Type 1 DM :-

Formerly known as juvenile-onset or insulin-dependent DM.
absolute insulin deficiency. Pancreatic B-cell destruction
can begin any age more <30 years but in 5-10% >30 years.

Type 2 DM (NIDDM) :-

Formerly known as adult onset or NIDDM.

Peripheral resistance to insulin and unable to compensate
for the resistance by increasing insulin production.

Gestational DM :-

Similar to Type 2 DM. Carbohydrate intolerance first
occurs during pregnancy or first detected during pregnancy.
It's either due peripheral insulin resistance or
insulin deficiency.

4-5% of pregnancies are complicated by DM.

90% → cause is GDM

Incidence of GDM is increasing 3-3% Canada,
5% USA, 14% South Africa.

Before introduction of insulin DM can lead to
increased perinatal mortality and morbidity.

GDM :-

increased insulin resistance in 2nd trimester due
to placental Lactogen, Prolactin, Cortisol, estrogen
and progesterone. Also increased insulin
clearance.

Diabetic Screening in Non Pregnant Patients:-

Normal Fasting $< 110 \text{ mg/dl}$ 2h. after 75g glucose < 140

Glucose intolerant 110-125 " " = 140-190

DM ≥ 126 ≥ 200

Symptoms of DM: polydipsia, polyphagia,
polyuria, weight loss. Recurrent vaginal infections, blurred vision

Gestational D.M Risk group:

Obese, H/o G.D.M, Family H.D.M, >25y
Previous Macrosomic baby, PCO, Twin pregnancy.
Racial (Asian, Hispanic, African-Caribbean)

I) UK: (NICE)

Whom: Selective if +ve Risk Factors without regard to age. 10% Missed.

How: At 24-28 weeks 2 hour 75 gm OGTT

Screening & diagnostic. Fasting 7.2h 7.8

IF 1+ G.D.M.

one reading required > abnormal

Screening 16-18 weeks if Normal Repeat 28 weeks

II) ACOG:

2 step approach.

Universal, more practical. Sensitive. No screening if <25y

1st Step:

IF NO Risk Factor

50gm oral glucose challenge

↓

Check serum glucose at 1 Hour (No Fasting required)

>130 mg/dl 7.2 → Discover 90% of G.D.M

↓

Do

3 hour G.T.T glucose Tolerance Test. or 75 gm.

If abnormal Fasting or any two abnormal →

Diagnostic D.M.

Follow-up G.T.T can be done 32-34w (To identify Late onset D.M)

USA

ADA
75 gm

ADA (American Diabetes association)
100 gm

F 5.3

5.3 95

1 10

10 180

2 8.6

8.6 155

3 —

7.8 140

Pregpregnancy Counselling .. For Type 1 & 2

1. High dose Folic acid 5mg (4000ug) up to 12/52.
2. Evaluate renal function (24H urine collection for Protein & Creatinine Clearance)
3. Full history and Examination.
4. Ophthalmology referral.
5. Echo (>30y, smoker, hypertensive)
6. Cardiologist referral if suspected cardiac illness.
7. Monitor Medications: ACE I (cause oligohyd.)
Renal failure. Skull defects.
8. Aspirin if Risk of Preclampsia.
9. HbA1c < 6.1 if Decreased Less Congenital anomalies (HbA1c in preg not sensitive)
HbA1c > 9.5% carries > 20% Fetal maj or anomalies. (advise women HbA1c > 10% to Avoid Pregnancy)
10. Stop OHA and start insulin if required (apart from Metformin).
FBS is Low in pregnancy due increased Renal Clearance, in non diabetic T1w insulin to 5%
To overcome The Resistance.

Type 1 D.M 1 Insulin Requirement 3 Times the normal dose.
pt at risk of:

Hypoglycemia < 3.9

Common 45% of DM1

Vomiting in Early pregnancy.

nausea, headache, Tremor, blurred or double

vision, weakness, hunger, confusion,

Paraesthesia, Stupor.

Rx: give glucose tablet or 4 ounces of juice.

20 min check Blood sugar.

Reapt feeding until RBS > 7 mmol/L

if cant take orally

10% Dextrose amp. IV push then.

5% Dextrose. ... calcium injection.

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- Rapid progression of Microvascular and Atherosclerotic Disease (IHD, HF, cerebral ischaemia)
 - Nephropathy: complicates 5-10% of D.M.
 - Chronic hypertension
 - Preeclampsia
 - Preterm more in uncontrolled D.M.

DKA: Diabetic ketoacidosis

Life Threatening, can occur at lower blood glucose < 200

Fetal mortality 10-30%. Maternal mortality is rare due to poor R.

Causes:-

50% infection 20% Non compliance on R. 30% No cause.

Steroids & Furosemides can precipitate DKA.

• \uparrow blood sugar \rightarrow glucosuria \rightarrow Osmotic diuresis \rightarrow loss of fluids, Na, K.

• Lipolysis, hepatic oxidation of Fatty acids \rightarrow Ketones \rightarrow Metabolic acidosis (\downarrow bicarbonate), \rightarrow organ impairment

Cardiac Arrhythmias

Ketones \rightarrow Placenta \rightarrow Fetal distress

R: Fluids 1lit N/S \approx 1h. Then 1lit in next 2-4 hours. change then D5NS (RBS < 250)

• insulin infusing, K supplement, bicarbonate if pH < 7.1

• Risk of infection: UTI, wound, candida due to impaired neutrophil function

Fetal complications:

Miscarriage \uparrow HbA1c due to congenital A. For DKA

congenital malformation

30-50% of PN Mortality

hypoglycaemia is prime factor (hypoglycaemia and hypokalaemia is suspected)

6-10% of Diabetic mothers have Major Congenital A.

Cardiac (Transposition of Great vessels)

VSD, ASD, hypoplastic left ventricle

- CNS anomalies ↑ 10-fold

NTD

- GIT malformation.

- Genitourinary Anomalies (polycystic kidneys)

- Sacral agenesis / caudal regression.

Rare 400 Times more
Frequent in D.M.

- Macrosomia:

WT 4-4.5 90th percentile.

25-42% of diabetic

Shoulder dystocia ↑ 3-fold.

IUGR

- IUGR 32-36w in uncontrolled D.M.

Complications of G.D.M.:

Preterm Labor, ↑ B.P., ↑ C/S Rate. ↑ Fetus

Recurrent G.D.M., Type 2 D.M.

Macrosomia, Shoulder dystocia (Fracture + palsy)

neonatal hypoglycemia, ↑ bilirubin. (Late Onset,
impaired GTT, intellectual).

Cause of Macrosomia:

glucose it will pass to fetus by facilitated
Diffusion → ↑ insulin production by fetus (act
as growth F) → ↑ growth of cells.

Neonatal complications in infants of Diabetic Mother:-

↓ Ca

↓ Blood sugar

Neonatal Death.

↓ Mg

• "33% polycythemia; ~~in 10-20%~~ HCT > 65%

chronic intrauterine hypoxia →

increases Erythropoietin Production.

• hypobilirubinemia, neonatal jaundice. (Delay in

Fetal liver maturation in poor glycaemic control;

• RDS → ↑ Fetal hypoinsulinemia → suppress production
of surfactant

Fetal Cardiac Septal hypoplasia and hypotrophic
Pulmonary myopathy.

Calculation and dose of initial insulin management

Don't more 60 U/day

0.7 U/kg (6-18 weeks)

0.8 U/kg (18-26)

0.9-1.1 (>26)

$\frac{1}{2}$ dose Am.

$\frac{2}{3}$ NPH

$\frac{1}{3}$ Novolog or Humalog

$\frac{1}{2}$ Pm

$\frac{1}{2}$ NPH

$\frac{1}{2}$ Novolog

eg 60

30 \leftarrow 20 NPH
10 N

30 \leftarrow 15

if steroids used (↑ insulin)

Antenatal follow-up.

1. 1st Trimester \leftarrow Control blood sugar.
Retinal + Renal checkup

2. 7-8w U/s For viability.

3. 16 weeks \rightarrow Retinal Ex if abnormal 1st U/S & L

4. 20w U/s For heart and other structure

5. 28 U/s For Growth + A.F.

Retinal Ex if normal in 1st T.

6. 32 U/s For Growth.

7. 36 U/s For growth.

D/W made of Delivery Timing

8. 38 IOL or C/S if wt > 4.5 kg. Blood sugar
4-7 during labor.

Delivery \rightarrow $\frac{1}{2}$ dose insulin.

Modify life style. Breast feeding. wt Reduction. Diet

GDM \rightarrow Risk of D.M 20-50% within 10y
GTT 6h, post P.

Management

Multidisciplinary (Physician, Midwife, Obst. nurse
nutrition consultation)

Referral urgently.

Diet:

CHO 40% of Total calories.

Vegetables + Fruit of high fibres

1800 kcal/day \rightarrow 2400 kcal/d.

Exercises:

Walking, yoga, Swimming, upper arm Ex } 30 min/day

glucose monitoring "glucometer" at home and
To be reviewed every 1-2 weeks.

Fasting, 1h or 2h after each meal (4 Times)

Target:

UK Fasting 3.5 - 5.9

1h.p.p < 7.8

ACOG: F \rightarrow < 5.3

1h. < 7.2

2h < 6.7

Insulin:

(1) x 4 injections: 3 Fast acting insulin before meals.
1 long acting at bed time.

fast acting:

• Standard soluble insulin [Humulin S, Actrapid]

• OR Fast acting insulin analogue [Novorapid, humalog] better

NPH is insulin of choice (intermediate acting) (onset 15 min, peak 2-4h) less hypoglycaemia

is Neutral Protamin Hagedorn Peak 6h Last 12.

Humulin I.

(2) 2 injections [mixed long + short] \rightarrow Neonatal Complications