Diabetes

Diabetes: established disease in pregnancy

1• Established diabetes affects 1–2% of pregnancies.

2• Without good glycaemic control there is increased fetal and neonatal morbidity and mortality.

3• Management should be by a multidisciplinary team including:

- obstetrician
- physician/diabetologist
- diabetic specialist nurse/midwife
- dietitian.

4• Glucose metabolism is altered by pregnancy.

5• Many pregnancy hormones are diabetogenic (human placental lactogen, cortisol, glucagon, oestrogen, and progesterone).

6• Insulin requirements increase throughout and are maximal at term.

Table 13.2 The influence of maternal hyperglycaemia on mother, fetus, neonate and young adult.

First trimester	
Implantation	Inhibits trophectoderm differentiation
Embryogenesis	Increases oxidative stress affecting expression of critical genes essential for embryogenesis
Organogenesis	Activates the diacylglycerol–protein kinase C cascade, increasing congenital defects
Miscarriage	Increases premature programmed cell death of key progenitor cells of the blastocyst
Second trimester	
Endocrine pancreas	Stimulates fetal β cells
Fetal growth	Stimulates fetal hyperinsulinaemia that results in growth acceleration seen on ultrasound by 26 weeks
Third trimester	
Fetal growth	A major fetal substrate and determinant for accelerated fetal growth
Adipose disposition	Stimulates hyperinsulinaemia that promotes fat disposition including intra-abdominal fat
Lung maturation	Stimulates hyperinsulinaemia that delays lung maturation by inhibiting surfactant proteins
Stillbirth	Is associated with defects in placental maturation that increase the risk of fetal hypoxia
Delivery	
Birth trauma	By causing accelerated fetal growth there is an increased risk of shoulder dystocia predisposing to birth trauma and asphyxia

Effect of diabetes on pregnancy

1• Maternal hyperglycaemia: leads to fetal hyperglycaemia.

2• Fetal hyperglycaemia: leads to hyperinsulinaemia (through β-cell hyperplasia in fetal pancreatic cells). Insulin acts as a growth promoter:

- macrosomia
- organomegaly
- increase erythropoiesis
- fetal polyuria (polyhydramnios).
- Neonatal hypoglycaemia: caused by the removal of maternal glucose supply at birth from a hyperinsulinaemic fetus.
- Respiratory distress syndrome: more common in babies born to diabetic mothers due to surfactant deficiency occurring through reduced production of pulmonary phospholipids.

Effect of pregnancy on diabetes

1• Ketoacidosis: rare, but may be associated with hyperemesis, infection,tocolysis (β-sympathomimetics), or steroid therapy.

2• Retinopathy: there is a two-fold increased risk of development or progression of existing disease. Rapid improvement in glycaemic control leads to increased retinal blood flow, which can cause retinopathy. All diabetic women should have assessment for retinopathy in pregnancy, and proliferative retinopathy requires treatment. Early changes usually revert after delivery.

3• Nephropathy: affects 5–10% of women. Renal function and proteinuria may worsen during pregnancy. This is usually temporary. There is increased maternal risk of pre-eclampsia and fetal risk of IUGR in this population and increased surveillance is required.

4• Ischaemic heart disease: pregnancy increases cardiac workload. Women with symptoms should be assessed by a cardiologist before conception.

Complications of diabetes in pregnancy

1.Maternal

- UTI.
- Recurrent vulvovaginal candidiasis.
- Pregnancy-induced hypertension/pre-eclampsia.
- Obstructed labour.
- Operative deliveries: CS and assisted vaginal deliveries.
- increase Retinopathy (15%).
- increase Nephropathy.
- Cardiac disease.

2.Fetal

- Miscarriage*
- Congenital abnormalities:*
- neural tube defects
- microcephaly
- cardiac abnormalities.
- sacral agenesis
- renal abnormalities.
- Preterm labour.
- Polyhydramnios (25%).
- Macrosomia (25–40%).
- IUGR.
- Unexplained IUD.

3.Neonatal

- Polycythaemia.
- Jaundice.
- Hypoglycaemia.
- Hypocalcaemia.
- Hypomagnesaemia.
- Hypothermia.
- Cardiomegaly.
- Birth trauma: shoulder dystocia, fractures, Erb's palsy, asphyxia.
- Respiratory distress syndrome.

* In diabetics with poor control.

Diabetes: antenatal management

Prepregnancy counselling

Offer to all diabetic women of reproductive age; include:

1• Achievement of optimal control: (increase risk of miscarriage and congenital abnormalities with poor control).

Targets for therapy pre-pregnancy should be to maintain HbA1c at 6.5 per cent and pre-meal glucose levels of 4–7 mmol/L

2• Assessment of severity of diabetes: check for hypertension, retinopathy (fundoscopy, ophthalmology assessment), nephropathy (U&E, urinalysis, urinary protein:creatinine ratio, 24h urine for protein, creatinine clearance), neuropathy (clinical assessment), and cardiacdisease.

3• Education: ensure understanding of effects of hyperglycaemia on fetus and need for tight control—instruct to inform doctor as soon as pregnancy confirmed; some drugs may need stopping (ACEIs).

4• General health: stop smoking, optimize weight (aim for a normal BMI).

5• Folic acid: increase risk of neural tube defects, so start on 5mg folic acid.

6• Rubella status: offer vaccination if not rubella immune.

7• Contraception: ensure effective contraception until good control achieved and pregnancy desired.

Antenatal care

Manage by a multidisciplinary team with a diabetologist.

1• Control: as for prepregnancy, aim for normoglycaemia. Monitor glucose at least 4 times/day, usually before meals, but post-meal glucose may give tighter control. Women can alter their own insulin based on their glucose. Insulin can be given as SC injections 2 or 4 times/day or as a continuous infusion. The latter is no better than injections.

2• HbA1c every month: this gives an objective measurement of control over the preceding 2mths.

3• Dietitian review: low sugar, low fat, high fibre diet—low glycaemic index.

4• Dating ultrasound: to confirm viability and gestation.
** Down's syndrome screening: consider nuchal translucency or invasive testing. Serum screening is affected by diabetes (decrease AFP); therefore, less accurate unless appropriate normograms used

5• Anomaly scan: 5–10-fold i risk of congenital anomalies. Risk depends on glycaemic control prior to conception and early pregnancy.

6• Fetal echocardiography: at 20–24wks.

7• Antenatal surveillance: individualize care. Serial USS every 2–4wks to detect polyhydramnios, macrosomia, or IUGR. Increased surveillance if problems detected. The use of umbilical artery Doppler should be restricted to cases of IUGR; it is not of value as a screening test.

8• Hypoglycaemia: awareness of hypoglycaemia may be lost. Educate patient and family and supply with glucagon.

9. If antenatal corticosteroids are indicated, an increase o
40 per cent at the time of the first dose and until 24 hours
after the second dose will usually prevent loss of control.

Diabetes: labour and post-partum care

Timing and mode of delivery should be individualized and based on EFW and obstetric factors (previous mode of delivery, gestation, glycaemic control, and antenatal complications).

Timing of delivery

** Some obstetricians advise elective delivery by induction of labour at 38–39wks if there are no maternal or fetal complications and good glycaemic control. Outcomes may not be better than awaiting spontaneous labour. Delivery should be expedited if complications occur.

Mode of delivery

1.Vaginal delivery is preferred.
2.Continuous electronic fetal monitoring is advised in labour.
3.Consider elective CS if EFW is >4.5kg. If EFW is 4–4.5kg,use obstetric factors to influence decision.
4.CS rates are high: 50–60%.
5.Give antibiotic and thromboprophylaxis if CS is carried out.

**Shoulder dystocia is more common at all birth weights than in the non-diabetic population. Experienced obstetricians should perform instrumental deliveries because this is an independent risk factor.

Glycaemic control in labour

- Diet controlled: check blood glucose hourly. If glucose >6.0mmol/L,start sliding scale.

- Insulin dependent: continue SC insulin until in established labour, then convert to insulin sliding scale .If induction of labour or CS,continue normal insulin until day of procedure, then start sliding scale in early morning.

Avoid maternal hyperglycaemia l causes fetal hypoglycaemia. If steroids are given for threatened preterm labour, monitor glucose closely—hyperglycaemia should be anticipated.

maternal blood glucose levels maintained at 4–8 mmol/L to reduce risks of neonatal hypoglycaemia

Post-partum care

- Encourage breast-feeding.

**Avoid oral hypoglycaemic drugs if breastfeeding metformin and insulin are safe.

- Baby needs early feeding and glucose monitoring.

Contraception

- Avoid the COCP if breast-feeding or vascular complications.

Progesterone-based contraception is safe and there are no contraindications to an IUCD. This should be fitted from 6wks post-partum onwards. Sterilization or vasectomy should be considered if the family is complete.

- Review sliding scale regularly.
- Renew insulin syringe every 24h.
- IV fluids should always be given with the sliding scale
- stable situations—5% glucose
- high blood glucose—normal saline.

Post-partum insulin requirements

*Insulin requirements fall dramatically after delivery of the placenta.

*Halve the sliding scale initially. Change back to SC insulin when eating and drinking. Start with the prepregnancy dose of SC insulin. If this is not known, it is roughly half the last dose. The dose may need to be further reduced if breast-feeding. Stop the sliding scale 1h after giving the SC dose.

** Aim for blood sugar monitoring (BM) 4–9mmol/L in the post-partum period.

Gestational diabetes

The World Health Organization (WHO) now includes gestational impaired glucose tolerance (IGT) with gestational diabetes. A proportion of women diagnosed in pregnancy will actually have previously unrecognized type 1 or 2 diabetes (20–30%). WHO does not advocate universal screening. Selective screening should be based on risk factors.

Risk factors for gestational diabetes

- BMI above 30kg/m2.
- Previous macrosomic baby weighing 4.5kg or above.
- Previous gestational diabetes.
- First-degree relative with diabetes.
- Family origin with a high prevalence of diabetes (South Asian, black Caribbean, and Middle Eastern).

The diagnosis is based on an oral glucose tolerance test (OGTT) , usually undertaken at 26–28wks gestation. A normal result in early pregnancy does not mean that gestational diabetes will not develop, and an OGTT should be repeated at 34wks if there are concerns.

Oral glucose tolerance test

- Overnight fasting (8h minimum):
- water only may be consumed during this time
- no smoking.
- 75g Glucose load in 250–300mL water.
- Plasma glucose measured fasting and at 2h.

Results

- Diabetes:
- fasting glucose ≥7.0mmol/L
- 2h glucose ≥11.1mmol/L.
- IGT:
- fasting glucose <7.0mmol/L
- 2h ≥7.8 <11.0mmol/L.

Only one value needs to be abnormal to make the diagnosis.

Management

1• Management by a multidisciplinary team.

2• Measure glucose 4–6 times/day (1h post-prandial measurements may be more effective in preventing macrosomia than pre-meal glucose).

3• Diet should be first-line treatment:

* aim for normoglycaemia and avoid ketosis.
* weight should remain steady if diet followed
* compliance is often poor—dietitian input may help.

4• Start insulin if:

- pre-meal glucose >6.0mmol/L
- 1h post-prandial glucose >7.5mmol/L.
- AC >95th centile despite apparent good control.

5• There is no increased risk of miscarriage or congenital anomalies; other fetal and neonatal risks are similar to established diabetes (IUGRis less likely).

6• Antenatal and intrapartum care as for established diabetes.

7• Post-partum:
* stop insulin and glucose infusions
* check glucose prior to discharge to ensure normal (risk of previously undiagnosed type 2 diabetes)
* arrange OGTT at 6wks post-partum
* education—50% risk of developing type 2 diabetes mellitus over next 25yrs (this risk can be reduced by maintaining physical activity and avoiding obesity).