Gestational diabetes mellitus

Gestational diabetes is glucose intolerance first recognized in pregnancy. Its prevalence is rising. There are well-recognized associations between gestational diabetes and increased risks to the fetus and the mother. This review looks at the presentation, risks and management of gestational diabetes including recent guidelines.

Gestational diabetes mellitus is defined by the World Health Organization as 'carbohydrate intolerance resulting in hyperglycaemia of variable severity with onset or first recognition during pregnancy' (World Health Organization, 1999). This does not exclude the possibility that unrecognized carbohydrate intolerance may have antedated or begun concomitantly with the pregnancy. Gestational diabetes affects 1–14% of all pregnancies depending on the population studied and the diagnostic tests used. The prevalence is higher in populations with a high frequency of diabetes and when the diagnostic tests have lower threshold glucose values for diagnosis (Buchanan and Xiang, 2005).

For more than a century, it has been known that diabetes antedating pregnancy can have severe adverse effects on fetal and neonatal outcomes resulting in high fetal and neonatal mortality. By the 1950s the term 'gestational diabetes' was applied to what was thought to be a transient condition that abated after delivery. A decade later O'Sullivan and Mahan (1964) found that the degree of glucose intolerance during pregnancy related to the risk of developing diabetes after pregnancy. They proposed criteria for the interpretation of oral glucose tolerance tests during pregnancy that were fundamentally statistical, establishing cut-off values – approximately two standard deviations for diagnosing glucose intolerance during pregnancy and for predicting diabetes after pregnancy. The current diagnostic criteria used aim to identify pregnancies at risk of high perinatal morbidity and mortality.

In most cases gestational diabetes mellitus is characterized by mild glucose intolerance, with mild fasting and typical postprandial hyperglycaemic events. Intrauterine fetal death during the last 4–8 weeks of gestation has been associated with fasting hyperglycaemia (American Diabetes Association, 2004). The most common complication of gestational diabetes mellitus is fetal macrosomia. Neonatal hypoglycaemia, jaundice, polycythemia and hypocalcaemia are the metabolic abnormalities that can be seen in an infant of a mother with gestational diabetes mellitus, while shoulder dystocia, birth trauma, respiratory distress syndrome as a result of decreased lung surfactant synthesis and cardiac septal hypertrophy are also associated with gestational diabetes mellitus. During pregnancy gestational diabetes mellitus is associated with maternal hypertension, polyhydramnios and pre-eclampsia. Caesarean delivery is more common in women with gestational diabetes mellitus (Stella et al, 2008), mainly because of macrosomia and alterations in obstetric management as a result of the knowledge that the mother has gestational diabetes mellitus. In terms of future morbidity, women are at increased risk of impaired glucose tolerance and frank diabetes after delivery, and gestational diabetes mellitus in a future pregnancy. Offspring of women with gestational diabetes mellitus are at increased risk of obesity, glucose intolerance and diabetes in late adolescence and young adulthood.

Dr A Theodoraki *is Specialist Registrar in Diabetes and Endocrinology, North Thames Central London Rotation and* **Dr SE Baldeweg** *is Consultant Physician in Diabetes and Endocrinology, Department of Endocrinology, University College London Hospitals, London NW1 2BU*

Correspondence to: Dr SE Baldeweg

Pathogenesis

Even normal pregnancy can be viewed as a state of progressive insulin resistance, with dynamic changes during gestational stages. Early pregnancy is largely an anabolic state with small increases in insulin sensitivity, whereas late pregnancy is better characterized as a catabolic state with increased insulin resistance (Lain and Catalano, 2007).

Insulin resistance appears to result from a combination of increased maternal adiposity and the insulin-desensitizing effects of hormonal products of the placenta. The fact that insulin resistance rapidly abates following delivery suggests that the major contributors to this state of resistance are placental hormones. Potential hormones include human placental lactogen, progesterone, prolactin and cortisol (Yamashita et al, 2000). Tumour necrosis factor-α (TNF-α) and adiponectin in insulin resistance of pregnancy has been investigated, especially in altering insulin post-receptor signalling mechanisms and resulting in insulin resistance. TNF-α is associated with decreased insulin sensitivity in a number of conditions, such as obesity and sepsis. TNF-α down-regulates insulin receptor signalling in pregnancy via different mechanisms and is mainly placentally derived. Adiponectin, a collagen-like protein expressed by adipocytes, is negatively associated with obesity, hyperinsulinaemia, and insulin resistance. Adiponectin increases tyrosine phosphorylation of the insulin receptor, and levels are decreased during pregnancy. The ratio of adiponectin:TNF-α may be an important factor for insulin sensitivity (Lain and Catalano, 2007).

Insulin resistance is the main scenario used to explain the pathophysiology of gestational diabetes mellitus. However, other settings with predominant β-cell dysfunction rather than insulin resistance involve autoimmune β-cell destruction (in <10% of cases) and monogenic diabetes (in <10% of cases) (Buchanan and Xiang, 2005).

Nevertheless, a large defect in pancreatic cell function is a consistent finding in women with prior gestational diabetes mellitus, which suggests that gestational diabetes mellitus represents the detection during pregnancy of metabolic abnormalities that antedate pregnancy.

Increased insulin resistance in pregnancy results in increases in maternal glucose and free fatty acid concentrations. High maternal glucose load is transferred to the fetus by the placental circulation, as well as per os when the fetus swallows glucose-enriched amniotic fluid after the 20th week of gestation. It has been suggested that the gut stimulus for insulin production in the fetus may be more potent than the transient intravenous hyperglycemia allowing for greater substrate availability for fetal growth (Jovanovic et al, 2007). Excessive transfer of glucose to the fetus results in fetal hyperglycaemia, with a consequent fetal pancreatic cell hypertrophy, beta-cell hyperplasia and fetal hyperinsulinaemia. Hyperinsulinaemia leads to fetal adiposity and visceromegaly, resulting in macrosomia. Macrosomia has been correlated with postprandial hyperglycaemia (De Veciana et al, 1995).

Diagnosis

Gestational diabetes mellitus is usually a clinically quiescent condition diagnosed via screening of asymptomatic pregnant women. Different criteria have been proposed for selective population screening. Risk assessment should be ascertained at the first prenatal visit.

According to the American Diabetes Association (2004) women with clinical characteristics consistent with a high risk of gestational diabetes mellitus should undergo glucose testing as soon as feasible. Such clinical characteristics include: marked obesity, personal history of gestational diabetes mellitus, glucosuria or a family history of diabetes in a first degree relative. If high-risk women are found not to have gestational diabetes mellitus in the initial screening they should be retested between 24 and 28 weeks of gestation. Women who meet all of the following characteristics are stratified as low risk and do not need glucose testing:

- Age <25 years
- Weight normal before pregnancy
- Member of an ethnic group with a low prevalence of gestational diabetes mellitus
- No known diabetes in first degree relatives
- No history of abnormal glucose tolerance
- No history of poor obstetric outcome.

Ethnic groups with relatively high rates of carbohydrate intolerance in pregnancy and later life include women of Hispanic, African, Native American, South or East Asian, Middle Eastern, Pacific Islands or indigenous Australian ancestry (Metzger and Coustan, 1998; National Institute for Health and Clinical Excellence, 2008). Women who fall between the two above categories of high and low risk are thought to have average risk for the development of gestational diabetes mellitus and a test should be undertaken at 24–28 weeks of gestation.

Recently released guidance from the National Institute for Health and Clinical Excellence (NICE) and the National Collaborating Centre for Women's and Children's Health suggests selective screening of the pregnant population using the following risk factors (National Institute for Health and Clinical Excellence, 2008):

- Body mass index >30 kg/m^2
- Previous macrosomic baby (≥ 4500 g)
- Previous gestational diabetes
- Family history of diabetes (first degree relative with type 1 or type 2 diabetes)
- High-risk ethnic group.

According to a 1999 survey (Mires et al, 1999), 67% of UK maternity service providers currently screen using a combination of these factors. The NICE guidance suggests that the evidence for screening using risk factors is unclear. However, while screening using risk factors is less sensitive than performing a glucose tolerance test in an unselected pregnant population, it is more practical and less disruptive for women. Women who have had gestational diabetes in a previous pregnancy should be offered early self-monitoring of blood glucose or an oral glucose tolerance test at 16–18 weeks, and a further oral glucose tolerance test at 28 weeks if the results are normal. Women with any other risk factors for gestational diabetes should be offered an oral glucose tolerance test at 24–28 weeks (National Institute for Health and Clinical Excellence, 2008).

In terms of screening tests, either of two different approaches is advised (Metzger and Coustan, 1998). In the one-step approach a diagnostic oral glucose tolerance test without prior serum glucose screening is performed. In the two-step approach initial screening is done with a glucose challenge test during which serum glucose concentration is measured 1 hour after a 50 g oral glucose load. A diagnostic oral glucose tolerance test is then performed on women exceeding the glucose threshold value on the glucose challenge test. A glucose threshold value of 7.8 mmol/litre 1 hour after the glucose load identifies approximately 80% of women with gestational diabetes mellitus, while using a cutoff of >7.2 mmol/litre increases the yield to 90% (American Diabetes Association, 2004).

With either of these approaches the diagnosis of gestational diabetes mellitus is based on an oral glucose tolerance test. Definitive consensus about the glucose threshold on an oral glucose tolerance test to diagnose gestational diabetes mellitus does not exist. According to the American Diabetes Association (2004) a 100 g or 75 g oral glucose tolerance test can be administered. In a 75 g oral glucose load two or more of the below venous plasma glucose concentrations must be met or exceeded for a positive diagnosis: fasting ≥ 5.3 mmol/litre, 1 hour ≥ 10.0 mmol/litre, 2 hour ≥8.6 mmol/litre. All tests should be done in the morning after an overnight fast of between 8 and 14 hours and after at least 3 days of unrestricted diet and activity (*Table 1*).

In contrast, different glucose threshold values have been proposed by the World Health Organization (1999).

They advise the use of a 75 g oral glucose tolerance test during which if either of the plasma glucose threshold values is met or exceeded a positive diagnosis can be made: fasting ≥7.0 mmol/litre or 2 hours ≥7.8 mmol/litre (*Table 2*). However the diagnosis is reached, using either the World Health Organization or the American Diabetes Association criteria, both identify populations with adverse pregnancy outcomes (Schmidt et al, 2001).

According to recent NICE guidance screening via fasting plasma glucose, random blood glucose, glucose challenge test and urinalysis for glucose is not advisable (National Institute for Health and Clinical Excellence, 2008). NICE advises using the World Health Organization criteria to interpret the oral glucose tolerance test.

Management

It is now known that adverse pregnancy outcome correlates with the degree of hyperglycaemia and especially postprandial serum glucose. In the diabetes in early pregnancy study in a type 1 diabetic population birth weight correlated with the maternal fasting serum glucose and haemoglobin A_{1c} (HBA_{1c}) during the first trimester (Jovanovic et al, 1998). However, during the third trimester 1 hour postprandial serum glucose levels were a stronger predictor of fetal macrosomia and infant body weight. In women with gestational diabetes, improved fetal outcome with decreased neonatal hypoglycaemia, macrosomia and caesarean delivery rates are achieved when controlling 1 hour postprandial serum glucose, as opposed to only fasting values (De Veciana et al, 1995). Controlling even mild hyperglycaemia in women with gestational diabetes mellitus reduces perinatal complications without increasing the rate of caesarean delivery (Crowther et al, 2005). Finally, in a recent study no threshold glucose value for gestational diabetes mellitus could be identified, but showed instead that the risk for a number of adverse pregnancy outcomes gradually increases with increasing glucose levels (Jensen et al, 2008).

Table 1. Diagnosis of gestational diabetes mellitus with a 75 g oral glucose load, American Diabetes Association criteria*

Plasma glucose	mmol/litre	mg/dl
Fasting	5.3	95
1 hour	10.0	180
2 hour	8.6	155

*Two or more venous plasma concentrations must be met or exceeded for a positive diagnosis

Table 2. Diagnosis of gestational diabetes mellitus with a 75 g oral glucose load, World Health Organization criteria

Plasma glucose	mmol/litre	mg/dl
Fasting	7	126
2 hour	7.8	140

*One venous plasma concentration must be met for a positive diagnosis

There is consensus that diagnosis and intervention for gestational diabetes improves maternal and fetal outcomes. In terms of cost-effectiveness, in high-income countries reductions in perinatal mortality and in serious perinatal complications would justify additional health service and personal monetary charges. Cost-consequence analysis of the Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS) study showed that over the whole lifespan, the incremental cost per extra life-year gained is favourable (Moss et al, 2007). NICE developed a health economic model to evaluate the cost-effectiveness of screening pregnant women and treating gestational diabetes, which concluded that screening based on risk factors and treatment as outlined in the ACHOIS trial is cost effective. In the ACHOIS trial, apart from diet, insulin was the only means of therapy. Since oral hypoglycaemic agents are cheaper than insulin NICE concludes that if oral agents achieve the glycaemic targets set, they are cost effective alternatives to insulin therapy (National Institute for Health and Clinical Excellence, 2008).

According to the American Diabetes Association (2004) guidelines the aim when treating women with gestational diabetes is to maintain the following plasma glucose levels: fasting glucose ≤5.8 mmol/litre, 1 hour postprandial glucose ≤8.6 mmol/litre and 2 hour postprandial glucose ≤7.2 mmol/litre. In view of the correlation of macrosomia with even mild hyperglycaemia, NICE sensibly advises women with diabetes to aim to lower targets: to keep fasting blood glucose between 3.5 and 5.9 mmol/litre and 1-hour postprandial blood glucose below 7.8 mmol/litre (National Institute for Health and Clinical Excellence, 2008). Achieving these targets relies on diet, exercise, regular capillary glucose monitoring, and insulin or oral hypoglycaemic agents. Insulin or oral hypoglycaemics should be considered if diet and exercise fail to maintain blood glucose targets after 1–2 weeks.

Diet

Individualization of diet is recommended depending on maternal weight and height. Provision of adequate calories and nutrients to meet the needs of pregnancy should be ensured and should be consistent with the maternal blood glucose goals. For obese women (body mass index >30 kg/m^2) a 30–33% caloric restriction, i.e. about 25 Kcal/kg actual body weight per day, reduces hyperglycaemia and plasma triglycerides with no increase in ketonuria (American Diabetes Association, 2004). Restriction of carbohydrates to 35–40% of calories decreases maternal glucose levels and improves maternal and fetal outcome. NICE advises a diet that is high in unrefined carbohydrates, that has been shown to improve overall glucose control and that reduces postprandial glucose excursions.

Exercise

Moderate exercise is well tolerated in pregnancy. Thirty minutes daily exercise is advised (National Institute for Health and Clinical Excellence, 2008). If the maternal

heart rate is maintained below 140 beats/min and if the mother is well hydrated and does not overheat there is no risk for the fetus. Exercise three times a week achieves glycaemic control and fetal weight similar to those seen in women treated with insulin (Artal, 2003).

Insulin

Human insulin and rapid-acting insulin analogues are safe and effective in maintaining glycaemia within targets and in reducing maternal and fetal adverse outcomes. Regular human insulin has a slow onset of action of 30–60 minutes with a peak concentration at 90 minutes and a 5–7-hour duration. The rapid-acting insulin analogues lispro and aspart have largely substituted regular insulin as their pharmacokinetics (onset of action at 0–20 minute, peak at 45 minutes, duration 3–4 hours) match the peak postprandial serum glucose better and result in fewer hypoglycaemic events (Gamson et al, 2004). They achieve satisfactory glucose control with better compliance, while their safety has also been established. Insulin aspart was found to be superior to human insulin in lowering postprandial glucaemic peaks (Pettitt et al, 2003, 2007), with similar perinatal and fetal outcomes (Hod et al, 2008). Insulin lispro has also been studied during gestation with benefits similar to aspart (Homco and Reece, 2006). Rapid insulin analogues are used before every meal, i.e. three injections/day in most patients.

Neutral protamine hagedorn is an intermediate acting human insulin that needs to be administered between one and three times/day to ensure basal euglycaemia. Insulin glargine has not been studied systematically in pregnancy. However, there have been several case reports and series that have not demonstrated any adverse pregnancy outcomes (Homco and Reece, 2006). No published reports exist for use of the long-acting insulin analogue detemir in pregnancy. From the above analogues, only insulin aspart has UK marketing authorization specifically for pregnant and breastfeeding women (National Institute for Health and Clinical Excellence, 2008), while the *British National Formulary* (Joint Formulary Committee, 2008) advises that limited evidence of safety of newer insulin analogues exists in pregnancy. In practice careful glycaemic control to ensure the set targets and individualized needs in certain cases are met takes priority over the unlicensed use of insulin. The choice of insulin should be discussed with the pregnant women with gestational diabetes mellitus.

In women with gestational diabetes mellitus, insulin requirements increase progressively in response to rising placental hormone levels during the second and third trimesters. It has been estimated that the total 24-hour insulin requirement, symbolized as 'Big I', depends directly on the patient's weight and number of weeks of gestation (Gamson et al, 2004) (*Table 3*). A simplified formula for the insulin dosage regimen is outlined below:

Big I = weight (kg)x k,

where k= 0.7, 0.8 and 0.9 units for the first, the second and third trimester respectively.

From this, 50% of Big I is usually required as daily basal insulin requirement (provided by a long-acting insulin) and 50% as daily bolus insulin requirement (provided by a rapid-acting insulin). Two more detailed schemes are presented in *Tables 4* and *5* (Jovanovic, 2004).

Oral hypoglycaemics

Oral hypoglycaemics are theoretically very attractive as they are cheaper than insulin, do not need special storage and no specific education is needed (Coetzee et al, 2007). The second generation sulphonyluria glibenclamide (glyburide) is minimally transferred across the placenta and is an effective alternative to insulin therapy (Langer et al, 2000). Glibenclamide enhances insulin secretion and is administered 1 hour before each meal three times a day. In two cohorts of women with gestational diabetes glibenclamide failure was reported to be 20% (Chmait et al, 2004; Kremer and Duff, 2004). In the Chmait et al study glibenclamide success was predicted if dietary failure occurred after 30 weeks, or fasting plasma values were <6.1 mmol/litre and 1-hour postprandial values were <7.8 mmol/litre. In a retrospective study of women treated with either

Table 3. Commonly used insulin therapies

Type	Peak action	Effective duration
Regular insulin	2–3 hours	5–8 hours
Neutral protamine hagedorn	6–10 hours	10–16 hours
Lispro	30–90 minutes	3–5 hours
Aspart	30–90 minutes	3–5 hours
Glargine	None	20–24 hours

Table 4. Three injections of insulin scheme

Type	Prebreakfast	Prelunch	Presupper	Prebed
Neutral	8/18 I*			3/18 I*
Regular, lispro or aspart	4/18 I*		3/18I*	
Totals	2/3 I*		1/6I*	1/6I*

Big I is defined as the total 24-hour insulin requirement (neutral protamine hagedorn twice daily calculated as 4/9 I* before breakfast and 1/6 I* before bed, along with two injections a day of regular, lispro or aspart at 2/9 I* before breakfast and 1.6 I* before dinner)

Table 5. Four injections of insulin scheme

Type	Prebreakfast	Prelunch	Presupper	Prebed
Glargine				1/2 I*
Regular, lispro or aspart	4/20 I*	3/20 I*	3/20 I*	
Totals	1/5 I*	3/20 I*	3/20 I*	1/2 I*

Big I is defined as the total 24-hour insulin requirement (Glargine once daily = 1/2 I* along with three injections of regular, lispro, or aspart calculated as 4/20 I* at breakfast, + 3/20 I* at lunch, + 3/20 I* at dinner). *BIG I for pregnancy depends on weight and weeks of gestation, *I = 0.7 U/kg = total daily insulin for weeks 6–18, *I = 0.8 U/kg = total daily insulin for weeks 18–26, *I = 0.9 U/kg = total daily insulin for weeks 26–36, *I = 1.0 U/kg = total daily insulin for weeks 36–40. Recent literature suggests that glargine may be taken every 24 hours, and the glycaemic response is similar if it is taken before breakfast, before lunch, in the evening, or before bedtime.

insulin or glibenclamide, glibenclamide was associated with a higher incidence of pre-eclampsia and neonates were more likely to receive phototherapy (Jacobson et al, 2005). In this study more women in the glibenclamide group achieved mean fasting and postprandial goals, and glibenclamide was at least as effective as insulin in achieving glycaemic control and similar birth weights in women with gestational diabetes mellitus who failed diet therapy.

Metformin is an insulin-sensitizing agent, thus reduces hepatic glucose production and increases glucose uptake by the tissues. It is known to cross the placenta. Almost 30 years ago Coetzee and Jackson (1979) reported comparable perinatal mortality between metformin- and insulin-treated pregnant women with diabetes, metformin failure in women with gestational diabetes mellitus was 28.6% and infant morbidity was low. In a study among women with diabetes treated with metformin, sulphonylureas or insulin, treatment with metformin during pregnancy was associated with increased prevalence of pre-eclampsia and a high perinatal mortality, with no difference in the perinatal morbidity (Hellmuth et al, 2000). In a retrospective review no difference in perinatal outcomes (pre-eclampsia, perinatal loss or neonatal morbidity) was found between pregnant women with type 2 diabetes treated with metformin or insulin (Hughes and Rowan, 2006). In a recent randomized controlled trial metformin was not associated with increased perinatal complications as compared with insulin (Rowan et al, 2008). In the same study 46.3% of the women treated with metformin required supplemental insulin to meet the glycaemic targets set.

It is widely accepted that adverse pregnancy outcomes correlate to the degree of hyperglycaemia and especially to postprandial glucose peaks. In view of their pharmacokinetic profile, oral hypoglycaemics have been criticized for not adequately controlling postprandial hyperglycemia and thus possibly resulting in higher macrosomia rates (Jovanovic et al, 2007). Nevertheless, glibenclamide and metformin were included in the NICE guidance about the pharmacological armamentarium available for diabetes in pregnancy (National Institute for Health and Clinical Excellence, 2008).

Monitoring

All women with gestational diabetes should be taught how to monitor capillary glucose values and how to interpret the results. In terms of frequency of testing, women should be advised to test fasting blood glucose levels and blood glucose levels 1 hour after every meal during pregnancy and at bedtime for the individuals treated with insulin (National Institute for Health and Clinical Excellence, 2008). Women on insulin should also be taught hypoglycaemia management and should be provided with glucagon in case of an emergency. Regular ultrasound scans should take place to measure the fetal growth and the amniotic fluid volume (Metzger et al, 1998). Ultrasound scans are advised every 4 weeks from the 28th until the 36th week, during which the estimated fetal weight and the abdominal circumference are measured. Obstetric management should include blood pressure measurements, urine testing for protein and planning of the delivery. Routine ketone monitoring is not recommended. However, women with gestational diabetes should be provided with urine and blood ketone strips to test ketone levels if they become unwell or hyperglycaemic. During delivery maternal glucose monitoring should take place and be maintained at between 4 and 7 mmol/litre. An intravenous dextrose and insulin infusion might be required. Neonates should be monitored for hypoglycaemia (National Institute for Health and Clinical Excellence, 2008).

After delivery

Postpartum approximately 10% of women with gestational diabetes remain diabetic. The remaining, however, are at increased risk of impaired glucose tolerance and type 2 diabetes with an estimated risk of up to 70%, depending on the cohort studied and the length of follow up. The risk for type 2 diabetes increases in the first 5 years after delivery and appears to plateau after 10 years. Fasting hyperglycaemia during pregnancy and higher plasma glucose on oral glucose tolerance test appears to increase the future risk of type 2 diabetes (Kim et al, 2002). The recurrence rate for gestational diabetes mellitus in a future pregnancy lies between 30 and 84% and in insulin treated women is as high as 75% (National Institute for Health and Clinical Excellence, 2008).

After delivery, the American Diabetes Association advises the conduction of an oral glucose tolerance test 6–8 weeks post delivery to differentiate between diabetes, impaired glucose tolerance and normoglycaemia. The oral glucose tolerance test needs to be repeated at 3-yearly intervals.

In stark contrast to this, according to NICE guidance women with gestational diabetes should be offered glucose testing postpartum and before their discharge. Women should be informed about the symptoms of diabetes, offered lifestyle advice and follow up in 6 weeks with fasting plasma glucose that should be repeated annually. In case of a subsequent pregnancy early testing of plasma glucose or oral glucose tolerance test at 16–18 weeks is advised and if the result is normal, the oral glucose tolerance test should be repeated at 28 weeks of gestation.

It will be interesting to observe if and how the new NICE guidance changes local practice.

Conclusions

Gestational diabetes is a common clinical scenario in the antenatal period. It is ideally managed in multidisciplinary specialist clinics. It is important for women and health-care professionals to acknowledge the need for accurate management during pregnancy for a healthy pregnancy outcome and to focus on education and post-partum follow-up regarding the high-risk of future diabetes. BJHM

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KEY POINTS

- Gestational diabetes mellitus involves hyperglycaemia that starts or is first recognized during pregnancy. Fetal macrosomia is the most common complication of gestational diabetes mellitus. Diagnosis and treatment aims to lower the perinatal and maternal complications.
- Gestational diabetes is usually diagnosed with an oral glucose tolerance test at 24–28 weeks of gestation, using the World Health Organization or the American Diabetes Association criteria.
- The aim is to keep fasting blood glucose between 3.5 and 5.9 mmol/litre and 1-hour postprandial blood glucose below 7.8 mmol/litre. The means to meet the above targets rely initially on diet and exercise. If these fail pharmacological treatment insulin or oral hypoglycaemics, is the next step.
- Rapid-acting insulin analogues (aspart and lispro) or less often regular human insulin are used to cover meal-time glucose peaks. The intermediate acting insulin neutral protamine hagedorn is most commonly used for fasting hyperglycaemia. Metformin or glibenclamide are recommended by the National Institute for Health and Clinical Excellence as alternatives to insulin therapy.
- After delivery women with gestational diabetes are at high risk of impaired glucose tolerance, diabetes and gestational diabetes mellitus in a future pregnancy. Therefore glucose testing should be undertaken 6 weeks postpartum and annually thereafter.