Gestational diabetes mellitus: Obstetric issues and management

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INTRODUCTION

The clinician caring for pregnant patients with gestational diabetes mellitus (GDM) should be knowledgeable about the maternal and fetal consequences of the disorder (eg, increased risk of preeclampsia and macrosomia), management of hyperglycemia, pregnancy monitoring, management of pregnancy complications, postpartum care, and long-term follow-up. This topic will discuss most of these issues; glucose management is reviewed in detail separately:

- (See "Gestational diabetes mellitus: Glucose management and maternal prognosis".)

- (See "Pregestational (preexisting) and gestational diabetes: Intrapartum and postpartum glucose management".)

Screening for and diagnosis of GDM are also reviewed separately. (See "Gestational diabetes mellitus: Screening, diagnosis, and prevention".)

CONSEQUENCES OF GDM

In addition to routine pregnancy issues, the prenatal care of patients with GDM focuses on preventing, identifying, and managing conditions that are increased among patients with impairment of glucose metabolism. In contrast to patients with pregestational diabetes, patients with true GDM are not at increased risk of congenital anomalies in offspring because the onset of the disorder is after the major period of organogenesis. Similarly, they should not experience diabetes-related vasculopathy because of the short duration of the disorder. However, it is important to note that some patients diagnosed with GDM actually have preexisting diabetes that was unrecognized because they were not screened prior to or early in pregnancy, thus they may experience these complications.

Short-term consequences — Complications of pregnancy more common in patients with GDM include:

- **Large for gestational age (LGA) newborn and macrosomia** – LGA (commonly defined as fetal or neonatal weight at or above the 90th centile for gestational age) and macrosomia are commonly associated with GDM. Obesity and gestational weight gain are major contributing factors.

  - The overall rates of LGA and macrosomia (defined as birth weight >4000 g) were 18.0 and 10.5 percent, respectively, in a series including over 1.5 million singleton, nonanomalous live births to individuals with GDM in the United States (2014 to 2020) [1].

  - GDM doubles the risk for LGA, and patients with obesity are at even higher risk. In one report [2]:

    - In normal weight individuals, LGA prevalence with versus without GDM was 13.6 versus 7.7 percent, respectively
    - In individuals with obesity, LGA prevalence with versus without GDM was 22.3 versus 12.7 percent, respectively

  - Excessive gestational weight gain (>40 pounds [18 kg]) doubled the risk of LGA in another study [3].

  Randomized trials have consistently demonstrated that maternal hyperglycemia significantly increases the chances of having a LGA or macrosomic newborn [4-7]. The risks of these outcomes increase along a continuum as maternal fasting plasma glucose levels increase. Maternal hyperglycemia leads to increased transplacental

transfer of glucose and other nutrients, which induce fetal hyperinsulinism and, in turn, accelerated fetal growth that is asymmetric (normal head size but broader shoulders and increased thoracic and abdominal diameters compared with newborns of mothers without diabetes). Accelerated fetal growth began as early as 20 to 28 weeks of gestation in a prospective cohort study [8].

Macrosomia and fetal truncal asymmetry are associated with an increased risk of operative birth (cesarean or instrument-assisted vaginal), maternal trauma, and adverse neonatal outcomes, such as shoulder dystocia and its associated complications: brachial plexus injury, fracture, and neonatal depression [9-16]. (See "Fetal macrosomia" and "Shoulder dystocia: Risk factors and planning birth of high-risk pregnancies" and "Large for gestational age (LGA) newborn".)

- **Preeclampsia and gestational hypertension** – Patients with GDM are at higher risk of developing preeclampsia and gestational hypertension (overall frequency 12 percent [1]) compared with patients without GDM. Insulin resistance causes GDM and also appears to be associated with development of preeclampsia and gestational hypertension, which may account for this association [17-26]. (See "Preeclampsia: Clinical features and diagnosis" and "Gestational hypertension".)

- **Polyhydramnios** – Polyhydramnios is more common in patients with GDM (incidence 18 percent [27]). The pathogenesis is unclear; fetal polyuria secondary to fetal hyperglycemia is one potential mechanism. Its impact on pregnancy outcome is also uncertain. Two studies reported GDM-related polyhydramnios, which is typically mild, did not significantly increase perinatal morbidity or mortality [28,29]. However, polyhydramnios in patients with or without GDM has been associated with an increased risk for adverse pregnancy outcome in some studies [27]. (See "Polyhydramnios: Etiology, diagnosis, and management", section on 'Outcome'.)

- **Stillbirth** – Patients with GDM appear to have a small absolute increase in risk of stillbirth compared with the general obstetric population. In a systematic review including 103,000 pregnancies with GDM, the stillbirth rate was approximately 6 stillbirths per 1000 GDM pregnancies compared with 4 stillbirths per 1000 nonGDM pregnancies [30]. However, the increased risk of stillbirth appears to be related to poor glycemic control; it does not appear to be increased in patients with good glycemic control, though ascertainment of good control can be challenging [31-34].

- **Neonatal morbidity** – Neonates of pregnancies complicated by GDM are at increased risk of multiple, often transient, morbidities, including hypoglycemia, hyperbilirubinemia, hypocalcemia, hypomagnesemia, polycythemia, respiratory disorders, and/or cardiomyopathy [35,36]. These risks are related, in large part, to maternal and in turn fetal hyperglycemia. (See "Infants of mothers with diabetes (IMD)".)

In contrast to most studies, a secondary analysis of data from the Antenatal Late Preterm Steroids (ALPS) trial found that GDM was not associated with a clinically significant difference in neonatal respiratory disorders; however, information about each participant's glucose control and diabetes treatment was not available [37]. Good glycemic control may have reduced the risk of respiratory problems in the newborns of patients with GDM. Baseline differences between patients with GDM versus those without GDM in this study may also account for the findings.

Long-term consequences — Risks associated with GDM extend beyond the pregnancy and neonatal period. For example:

- **Maternal risks** – GDM is a strong marker for future maternal development of diabetes mellitus (primarily type 2), metabolic syndrome, and cardiovascular disease. (See "Gestational diabetes mellitus: Glucose management and maternal prognosis", section on 'Long-term risk'.)

- **Offspring risks** – GDM increases the offspring's risk for developing obesity and abnormal glucose tolerance. Poorly controlled maternal diabetes during pregnancy may impact neurodevelopmental outcome; however, evidence is circumstantial and of poor quality. (See "Infants of mothers with diabetes (IMD)", section on 'Long-term outcome'.)

CLASSIFICATION/TERMINOLOGY

Patients with GDM are classified into two groups [38], which have been associated with pregnancy risk and thus guide obstetric management:

- A1: glycemic control achieved **without** medication
- A2: glycemic control achieved **with** medication

PRENATAL CARE

The author's general approach to pregnancy management in GDM is shown in the algorithm (algorithm 1).

Glucose management — Glucose management to achieve glucose levels in the target range is the key intervention for reducing the frequency and/or severity of complications related to GDM.

- **Self-monitoring** – Glucose levels are checked before breakfast (ie, fasting glucose level) and at one or two hours after the beginning of each meal.

- **Glycemic targets** – Commonly used antepartum glycemic targets are:

  - Fasting blood glucose concentration: <95 mg/dL (5.3 mmol/L)
  - One-hour postprandial blood glucose concentration: <140 mg/dL (7.8 mmol/L)
  - Two-hour postprandial glucose concentration: <120 mg/dL (6.7 mmol/L)

  Using tighter postprandial targets did not reduce the overall rate of having a large for gestational age (LGA) newborn, but reduced the risk of perinatal death, birth trauma, or shoulder dystocia in a randomized trial [39].

  There are no standard criteria for describing suboptimal versus poor glucose control. We consider glucose values 20 to 30 percent above the target range suboptimal.

Glucose monitoring, medical nutritional therapy, exercise, and the use of insulin and/or oral antihyperglycemic medications to achieve and maintain these targets are discussed in detail separately. (See "Gestational diabetes mellitus: Glucose management and maternal prognosis".)

Fetal surveillance

  A1 GDM with good glucose control — Patients who are euglycemic with nutritional therapy alone (ie, class A1 GDM) and have no other pregnancy complications (eg, no macrosomia, preeclampsia, growth restriction, polyhydramnios, or oligohydramnios) do not appear to be at increased risk of stillbirth [40]; therefore, omitting antenatal fetal surveillance (nonstress test [NST] and amniotic fluid index, biophysical profile [BPP]) is a reasonable approach for these patients and this author's approach, but practice patterns

vary given the range of existing data on this issue. If the practitioner chooses to order NSTs or BPPs in these pregnancies, the tests should probably be begun no earlier than 36 weeks rather than at 32 weeks since no increased risk of stillbirth has been demonstrated in this population. (See "Overview of antepartum fetal assessment".)

The American College of Obstetricians and Gynecologists (ACOG) made no specific recommendations for fetal assessment in patients with well-controlled glucose levels on nutritional therapy, except for assessment of amniotic fluid volume; this decision was left to local practice patterns [38].

A2 GDM or A1 GDM with suboptimal glucose control — We obtain twice weekly NSTs plus an amniotic fluid index beginning at 32 weeks of gestation for (algorithm 1):

- All patients who use insulin or an oral antihyperglycemic medication to achieve good glycemic control.

- All patients with suboptimal glycemic control. Ideally, patients with suboptimal glucose control will be brought under better control with appropriate nutritional therapy and/or medication.

The evidence supporting antenatal fetal testing in pregnancies complicated by GDM consists primarily of data from older observational series that reported no or rare fetal losses in pregnancies complicated by diabetes monitored by various antenatal testing regimens [41,42]. No randomized trials have evaluated fetal surveillance in patients with GDM, and findings from the small number of cohort and case-control studies are inconclusive.

The practice pattern that has evolved over decades is to base use of fetal testing on (1) the severity of GDM (ie, whether euglycemia is achieved and whether it is achieved by nutritional therapy or by pharmacotherapy) and (2) the presence of other risk factors for adverse pregnancy outcome (eg, past history of stillbirth, presence of comorbidities such as chronic hypertension, LGA/macrosomia). As some studies have reported that patients with GDM are at increased risk of stillbirth [43-45], we agree with expert opinion, which generally recommends that patients who require insulin or an oral antihyperglycemic medication (ie, class A2 GDM) to maintain euglycemia or who have suboptimally controlled blood glucose levels should be managed the same way as patients with pregestational diabetes or other conditions placing the pregnancy at increased risk of

adverse outcome. These patients typically undergo periodic fetal testing, usually initiated at approximately 32 weeks of gestation.

The timing for initiating testing in the third trimester, the frequency of testing, and the test utilized (NST, BPP, or both) vary by institution and practice setting. Although we perform NSTs with an amniotic fluid index twice per week, no strong evidence favors twice weekly over weekly testing or initiating testing at 32 weeks versus later in gestation. For example, some medical centers begin NSTs weekly at 32 weeks and increase to twice weekly at 36 weeks.

ACOG suggests antenatal fetal assessment beginning at 32 weeks of gestation for (1) patients treated with insulin or oral medications, even when good glycemic control is achieved with drug therapy, and (2) patients with suboptimal glucose control on medical nutritional therapy [38].

Monitoring fetal growth — We perform a single third-trimester ultrasound examination at 36 to 39 weeks to estimate fetal weight in all patients with GDM, regardless of degree of metabolic control or requirement for insulin or oral antihyperglycemic medications. Macrosomia is a factor in decision-making about the route of birth. (See 'Route of birth' below.)

A broad spectrum of practice patterns has evolved, given the limitations of available data, which are observational and do not strongly support a specific approach [39,46,47]. For example:

- Some clinicians use the 36- to 39-week scan to identify maternal-fetal pairs that may benefit from induction of labor before the fetus grows too large. (See "Shoulder dystocia: Risk factors and planning birth of high-risk pregnancies", section on 'Patients with diabetes'.)

- Some clinicians also obtain one or more earlier ultrasound examinations (between diagnosis of GDM and 36 weeks) to identify fetal growth acceleration, as it appears to be a sign of suboptimal glycemic control and may prompt tightening glycemic control to reduce the risk of macrosomia. However, early growth acceleration does not accurately predict LGA at birth [47] and a randomized trial comparing tight versus less tight control in GDM failed to show a difference between groups in LGA, although it did reduce serious neonatal morbidity [39].

- Some clinicians do not monitor fetal growth sonographically in euglycemic patients with A1 GDM (medical nutritional therapy alone) because of concerns that false-positive findings will lead to iatrogenic complications. As an example, one study reported an increase in cesarean birth among patients who had a third-trimester ultrasound examination, even after controlling for birth weight [48].

Estimation of fetal weight is challenging because no method of fetal growth assessment performs well; all current methods are neither sensitive nor specific, especially for identifying the LGA fetus [49-51]. One review of pregnant patients with diabetes treated with insulin found that the sonographically estimated fetal weight had to be ≥4800 grams for there to be at least a 50 percent chance that the newborn's birth weight would be ≥4500 grams [52]. Studies in nondiabetic pregnancies report similar results [53]. Investigators have tried to find a more sensitive modality to estimate fetal weight, but there is little evidence that these experimental modalities can improve on existing two-dimensional ultrasound technology [54-57]. The diagnosis of LGA/macrosomia is discussed in detail separately. (See "Fetal macrosomia", section on 'Diagnosis' and "Fetal macrosomia", section on 'Patients with diabetes'.)

Management of selected antenatal complications — As discussed above, the following antenatal complications are more prevalent in patients with GDM. (See 'Consequences of GDM' above.)

Preeclampsia and gestational hypertension — Management of preeclampsia and gestational hypertension is similar to that in patients without GDM. Labetalol can be used to treat severe hypertension; hypoglycemic symptoms are unlikely to be masked in patients with GDM. (See "Preeclampsia: Antepartum management and timing of delivery" and "Gestational hypertension" and "Hypoglycemia in adults with diabetes mellitus".)

Preterm labor — Preterm labor is managed the same as in patients without GDM. Indomethacin and nifedipine are first-line medications for tocolysis, with the choice dependent on gestational age. If these medications are contraindicated, terbutaline can be used, but maternal glucose levels need to be monitored closely since beta agonists increase glucose levels. For fasting levels >100 mg/dL (5.5 mmol/L) or postprandial levels >140 mg/mL (7.8 mmol/L), we treat with subcutaneous insulin. (See "Inhibition of acute preterm labor".)

Coadministration of antenatal corticosteroids accentuates the increase in glucose levels.

(See 'Hyperglycemia related to antenatal corticosteroid administration' below.)

Hyperglycemia related to antenatal corticosteroid administration — Administration of antenatal corticosteroids (ACS), if indicated, has hyperglycemic effects, beginning approximately 12 hours after the first steroid dose and lasting for approximately five days [58,59]. We monitor capillary blood glucose concentrations regularly (eg, at least every four times daily, but more frequently depending on glucose levels and difficulty in obtaining control) beginning 12 hours after the first dose of betamethasone and continuing for 24 hours after the second dose. We then reduce the frequency to four times per day if glucose levels are reasonably well controlled. If a fasting level exceeds 100 mg/dL (5.5 mmol/L) or a postprandial level exceeds 140 mg/mL (7.8 mmol/L), we would treat with subcutaneous insulin. We initiate a continuous intravenous insulin infusion on the labor unit if values are persistently elevated despite escalation of the subcutaneous regimen or if initial values are above 180 mg/dL (10.0 mmol/L). (See "Pregestational (preexisting) diabetes mellitus: Obstetric issues and management", section on 'Antenatal glucocorticoids in patients at risk for preterm birth'.)

ACS are generally not recommended at ≥34 weeks of gestation in patients with diabetes. While the Antenatal Late Preterm Steroids (ALPS) trial found a modest benefit in late preterm gestations, this trial specifically excluded patients with diabetes because of concerns for impact on glycemic control. (See "Antenatal corticosteroid therapy for reduction of neonatal respiratory morbidity and mortality from preterm delivery".)

Macrosomia and prediction/prevention of shoulder dystocia — Macrosomia (which is commonly defined as ≥4500 grams in GDM) is diagnosed by ultrasound and often accompanied by polyhydramnios. A major concern is the risk of shoulder dystocia during labor and birth. Scheduled cesarean birth is typically offered to patients with GDM and estimated fetal weight ≥4500 grams to prevent shoulder dystocia, particularly its potential maternal and newborn morbidities. (See 'Route of birth' below.)

Decision-making regarding induction of fetuses <4500 grams versus expectant management are reviewed separately. (See "Shoulder dystocia: Risk factors and planning birth of high-risk pregnancies", section on 'Patients with diabetes'.)

Timing of birth — One of the key issues of managing patients with GDM is whether to induce labor and, if so, when. The major potential benefits of induction are avoiding late stillbirth and avoiding birth-related complications of continued fetal growth, such as

shoulder dystocia or a cesarean birth for failure to progress. The potential disadvantages are the risks of induction (eg, longer labor, increased tendency for intervention) and greater neonatal morbidity if the induction is before 39 weeks [43]. Increasing evidence suggests that induction itself does not result in higher cesarean birth rates than expectant management, even in patients with GDM [60-62].

The optimal timing of birth in GDM has not been evaluated in well-designed trials. The available data [60,63-68] are inadequate to allow a strong evidence-based recommendation; thus, practice varies worldwide [69,70]. Our approach is similar to the practice pattern that has evolved at many institutions and is based primarily on whether the patient is A1 or A2 GDM and the adequacy of glucose control.

A1 GDM with good glucose control — For patients who remain euglycemic with nutritional therapy and exercise alone (A1 GDM), we discuss the possibility of induction and the tradeoffs of induction at ≥39+0 weeks versus expectant management until 41+0 weeks. This relatively noninterventional approach is based on the favorable outcomes reported in a classic uncontrolled case series of 196 patients with class A diabetes managed this way [40]. Although clinical practice varies from institution to institution, the general consensus is that these patients should not be electively delivered before 39+0 weeks of gestation [71] or after 41+0 weeks. Timing of induction between 39+0 and 41+0 weeks is more controversial.

While a study using decision analysis found that fetal and neonatal mortality may be minimized by birth at 38 weeks of gestation, this mathematical model alone is insufficient for changing our clinical practice [72]. ACOG has opined that birth should not be planned before 39+0 weeks of gestation unless otherwise indicated and that expectant management up to 40+6 weeks is generally appropriate with antepartum testing [38].

A2 GDM and A1 GDM with suboptimal glucose control — For patients with GDM whose glucose levels are medically managed with insulin or oral antihyperglycemic medications (A2 GDM) and patients with A1 GDM with suboptimal glucose control, we suggest induction at 39+0 weeks of gestation based on data from a retrospective cohort study of patients with GDM suggesting that the infant mortality rate at 39+0 weeks (8.7 out of 10,000) was statistically lower than the risk of stillbirth plus infant mortality with expectant management over an additional week (15.2 out of 10,000) [43]. In addition, induction may reduce the risk of shoulder dystocia compared with later birth since birth

weight should be less in the absence of ongoing growth in utero [63,64].

Early term birth (37+0 to 38+6 weeks) is not indicated in uncomplicated A2 GDM with well-controlled glucose levels as the risk of stillbirth is low while neonatal morbidity rates are increased at this gestational age [73]; however, if a concomitant medical condition (eg, hypertension) is present or glycemic control is suboptimal on pharmacotherapy, birth should be undertaken as clinically indicated prior to 39+0 weeks of gestation [63,64]. Fetal weight also needs to be considered. The risk of stillbirth appears to be increased among those who are LGA near and at term compared with those who are appropriate for gestational age (AGA). For example, a study of pregnancies complicated by GDM reported the absolute risks of stillbirth at 38 weeks in LGA and AGA fetuses were 21.5 and 4 per 10,000 pregnancies, respectively; and at 39 weeks 20.7 and 5.7 per 10,000 pregnancies, respectively [45]. Limitations of these data included the absence of information about glycemic control and about patients requiring medication versus those managed with nutritional therapy and exercise alone.

ACOG suggests birth at 39+0 to 39+6 weeks of gestation for patients with A2 GDM that is well controlled with medication [73]. However, guidance for patients with suboptimal glycemic control on pharmacologic therapy is less precise. They suggest birth at 37+0 to 38+6 weeks of gestation may be reasonable, but that birth prior to 37+0 weeks should only be initiated when more aggressive efforts to control blood glucose levels, such as hospitalization, have failed [38].

Route of birth — Estimated fetal weight is an important consideration in decision-making.

- **Estimated fetal weight ≥4500 grams**– Scheduled cesarean birth to avoid birth trauma is typically offered to patients at 39+0 weeks with GDM and estimated fetal weight ≥4500 grams. The fetal weight threshold at which scheduled cesarean birth should be performed to reduce the risk of birth trauma from shoulder dystocia is controversial. It has been estimated that in diabetic pregnancies with an estimated fetal weight of ≥4500 grams, 443 cesareans would need to be performed to prevent one permanent brachial plexus injury [74]. Whether this tradeoff justifies the increased risks of cesarean birth is unclear. The ACOG practice bulletin on GDM recommends discussing the risks and benefits of scheduled cesarean birth with patients with GDM and estimated fetal weight ≥4500 grams [38].

If a patient with GDM and estimated fetal weight ≥4500 grams decides to undergo a trial of labor, we follow labor progress closely and perform an instrument-assisted vaginal birth only if second-stage descent has progressed normally because forceps- or vacuum-assisted birth is associated with a higher risk of shoulder dystocia and brachial plexus injury, with the risk even higher with the use of vacuum as compared with forceps [75,76]. (See "Shoulder dystocia: Risk factors and planning birth of high-risk pregnancies", section on 'Planning birth in high-risk pregnancies'.)

- **Estimated fetal weight <4500 grams** — Cesarean birth is performed for standard obstetric indications.

Glycemic monitoring during labor and birth — Insulin requirements usually decrease during labor as oral caloric intake is typically reduced and the work of labor, particularly uterine contractions, requires extra energy. A reasonable target range for intrapartum glucose levels is 70 to 125 mg/dL (3.9 to 6.9 mmol/L). Hyperglycemia is treated to reduce the risk of neonatal hypoglycemia. Although prolonged neonatal hypoglycemia is primarily due to fetal exposure to chronic hyperglycemia during pregnancy and resultant fetal pancreatic hyperplasia, transient hypoglycemia can be caused by intrapartum maternal hyperglycemia, which induces an acute rise in fetal insulin [77-81].

- **A1 GDM** – Patients with GDM who were euglycemic without use of insulin or oral antihyperglycemic medications during pregnancy generally do not develop hyperglycemia during labor and birth and thus do not need their blood glucose levels checked. However, some practitioners and centers choose to check a few blood glucose levels during labor because of concerns about the validity of outpatient self-monitored glycemic assessments.

- **A2 GDM** – Patients with GDM who used insulin or oral antihyperglycemic medications antepartum may need insulin during labor and birth to maintain intrapartum glucose levels in the target range. Periodic assessment of maternal glucose levels during labor and treatment of hyperglycemia are prudent, although intrapartum maternal hyperglycemia infrequently leads to an adverse neonatal outcome [82].

Intrapartum glucose monitoring and management of fluids, insulin, and oral antihyperglycemic medications during spontaneous labor, before induction, and before scheduled cesarean birth are discussed in detail separately. (See

"Pregestational (preexisting) and gestational diabetes: Intrapartum and postpartum glucose management".)

POSTPARTUM CARE

Patients with GDM should be able to resume a normal diet postpartum.

Breastfeeding — Breastfeeding should be encouraged since it benefits both the mother and child. (See "Maternal and economic benefits of breastfeeding" and "Infant benefits of breastfeeding".)

Breastfeeding improves maternal glucose metabolism. It may reduce the glucose levels obtained during a postpartum glucose tolerance test (GTT) [83-85], especially if the patient breastfeeds during the test [86]. Theoretically, this could lead to a spurious result.

Several prospective studies have reported that breastfeeding decreased the long-term incidence of type 2 diabetes after a diagnosis of GDM compared with not breastfeeding [87-90]. Higher lactation intensity and longer duration were inversely associated with the risk, independent of weight loss and after adjusting for risk factors for type 2 diabetes (sociodemographic characteristics, prenatal metabolic status and course, perinatal outcome, lifestyle behaviors).

Contraception — While any type of contraception is acceptable as long as the usual medical contraindications to use are absent, the advantages of long-acting reversible contraception (eg, intrauterine device [IUD], contraceptive implant) are the minimal risk of unplanned pregnancy and convenience [91]. Choosing contraceptives with lower systemic hormone levels should, in theory, minimize any changes in metabolic parameters. If a patient is still concerned about hormonal issues, a copper-releasing IUD is a good alternative. However, there is no convincing evidence that hormonal contraceptives (estrogen-progestin or progestin-only) increase the user's risk of developing type 1 or type 2 diabetes [92]. (See "Contraception: Counseling and selection".)

Screening for overt diabetes — After birth, the hyperglycemic effects of placental hormones dissipate rapidly. Thus, most patients revert back to their prepregnancy glycemic status shortly after birth, ranging from almost immediately to a week postpartum. Most patients who were taking antihyperglycemic medications antepartum

do not need them postpartum.

- **Check postpartum glucose levels** – Since some patients with GDM may have previously unrecognized type 2 diabetes mellitus, we check glucose concentrations postpartum to exclude ongoing hyperglycemia. In a patient with A1 GDM, it is common to check only a single fasting glucose and, if normal, stop checking blood glucose values. In a patient with A2 GDM, we usually check glucose levels for 24 hours, obtaining both fasting and postprandial blood glucose values; if all are normal, then we stop checking. However, if any values are abnormal, we continue to check levels during hospitalization and possibly at home to determine whether ongoing medical therapy is needed.

  If fasting glucose concentrations suggest overt diabetes (fasting glucose ≥126 mg/dL [7 mmol/L] or a postprandial glucose is ≥200 mg/dL [11.1 mmol/L]), hyperglycemia must be treated; the type of treatment (diet, exercise, weight reduction, medication) should be decided on a case-by-case basis, often with consultation from a diabetologist or the patient's primary care provider. Postpartum glucose monitoring and therapy, if indicated, are reviewed separately. (See "Pregestational (preexisting) and gestational diabetes: Intrapartum and postpartum glucose management", section on 'Gestational diabetes'.)

- **Obtain a GTT at 4 to 12 weeks** – Patients with normal/near normal postpartum should undergo a two-hour 75 gram oral GTT 4 to 12 weeks after delivery to check for diabetes or prediabetes (table 1). (See "Gestational diabetes mellitus: Glucose management and maternal prognosis", section on 'Follow-up'.)

  - Those diagnosed with diabetes (table 1) are managed as medically appropriate. (See "Initial management of hyperglycemia in adults with type 2 diabetes mellitus".)

  - Those with a normal GTT or prediabetes (table 2) are counseled about their future risk of developing type 2 diabetes and cardiovascular disease, encouraged to adopt lifestyle changes for risk reduction (eg, healthy diet, weight loss, exercise), and informed about the importance of close follow-up with their primary care provider and rescreening at appropriate intervals. These issues are discussed in detail separately. (See "Gestational diabetes mellitus: Glucose management and maternal prognosis", section on 'Follow-up' and "Screening for

type 2 diabetes mellitus" and "Clinical presentation, diagnosis, and initial evaluation of diabetes mellitus in adults".)

Screening for depression — Although all postpartum patients should be screened for depression, clinicians should be aware that postpartum depression is more common among patients with diabetes (pregestational or gestational) than in postpartum patients without diabetes [93]. The validated questionnaire most commonly used for screening pregnant and postpartum individuals is the Edinburgh Postnatal Depression Scale (　figure 1A-B), but other validated tools can be used (　table 3). (See "Postpartum unipolar major depression: Epidemiology, clinical features, assessment, and diagnosis", section on 'Screening'.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Diabetes mellitus in pregnancy" and "Society guideline links: Shoulder dystocia and macrosomia".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Gestational diabetes (The Basics)")

- Beyond the Basics topics (see "Patient education: Gestational diabetes (Beyond the

Basics)")

SUMMARY AND RECOMMENDATIONS

- **Screening for and birth of the macrosomic fetus**

  - **Ultrasound** – We perform a single third-trimester ultrasound at 36 to 39 weeks to screen for macrosomia in all patients with gestational diabetes mellitus (GDM). (See 'Monitoring fetal growth' above.)

  - **Scheduled cesarean birth** – Scheduled cesarean birth to avoid birth trauma is typically offered at 39+0 weeks to patients with GDM (any class) and an **estimated fetal weight ≥4500 grams**. These patients should be counseled about the poor predictive ability of ultrasound estimates of fetal weight and the risks and benefits of cesarean birth in the current and future pregnancies. (See 'Route of birth' above.)

- **Fetal surveillance and timing of birth in patients with A1 GDM well controlled with nutritional medical therapy alone** – These patients are not at increased risk for stillbirth.

  - **Antenatal fetal surveillance** – We do not order antenatal fetal testing (nonstress test, biophysical profile) in these patients unless they have a standard obstetric indication for fetal surveillance (eg, growth restriction). (See 'Fetal surveillance' above.)

  - **Timing of induction** – For candidates for vaginal birth, we offer induction of labor at 39+0 weeks of gestation and suggest performing induction by 41+0 weeks of gestation (**Grade 2C**), as with other late term pregnancies. (See 'Timing of birth' above and "Postterm pregnancy".)

- **Fetal surveillance and timing of birth in patients with A2 GDM (ie, on pharmacologic therapy) or A1 GDM with suboptimal glucose control** – These patients may be at increased risk for stillbirth.

  - **Antenatal fetal surveillance** – We suggest a standard form of antenatal fetal testing. The optimal testing regimen has not been established from rigorous

studies. We order twice weekly antenatal testing, using a nonstress test with an amniotic fluid index, starting at 32 weeks of gestation. Ideally, patients with suboptimal glucose control will be brought under better control with diet and/or medication. (See 'Fetal surveillance' above.)

- **Timing of induction** – For candidates for vaginal birth, we suggest induction of labor at 39+0 weeks of gestation (**Grade 2C**). Potential benefits include lower rates of: macrosomia and large for gestational age (LGA) infants, shoulder dystocia, cesarean birth, and stillbirth. If a concomitant medical condition (eg, hypertension) is present or glycemic control is suboptimal on pharmacologic therapy, birth should be undertaken as clinically indicated prior to 39+0 weeks of gestation. (See 'Timing of birth' above.)

  The American College of Obstetricians and Gynecologists suggests birth at 39+0 to 39+6 weeks of gestation for patients with A2 GDM that is well controlled with medication. For patients with suboptimal glycemic control on pharmacologic therapy, birth at 37+0 to 38+6 weeks may be reasonable, but that birth prior to 37+0 weeks should only be done when more aggressive efforts to control blood sugars, such as hospitalization, have failed.

- **Postpartum care**

  - **Breastfeeding** – All patients should be encouraged to breastfeed. A potential benefit of breastfeeding is that it improves glucose metabolism in the short term. (See 'Breastfeeding' above.)

  - **Contraception** – While any type of contraception is acceptable as long as the usual medical contraindications to use are absent, long-acting reversible contraception is convenient and has minimal risk of unplanned pregnancy. (See 'Contraception' above.)

  - **Postpartum testing for diabetes** – All patients with GDM should have a two-hour 75 gram oral glucose tolerance test between 4 and 12 weeks postpartum (see 'Postpartum care' above):

    - Those with a normal test or prediabetes (table 2) should be informed of their future increased risk for diabetes and cardiovascular disease, counseled about preventive measures that they can adopt, and informed of the need for

rescreening at periodic intervals.

- Those with diabetes (table 1) are managed as medically appropriate. (See "Initial management of hyperglycemia in adults with type 2 diabetes mellitus".)

ACKNOWLEDGMENT

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REFERENCES

1. Venkatesh KK, Lynch CD, Powe CE, et al. Risk of Adverse Pregnancy Outcomes Among Pregnant Individuals With Gestational Diabetes by Race and Ethnicity in the United States, 2014-2020. JAMA 2022; 327:1356.

2. Black MH, Sacks DA, Xiang AH, Lawrence JM. The relative contribution of prepregnancy overweight and obesity, gestational weight gain, and IADPSG-defined gestational diabetes mellitus to fetal overgrowth. Diabetes Care 2013; 36:56.

3. Hillier TA, Pedula KL, Vesco KK, et al. Excess gestational weight gain: modifying fetal macrosomia risk associated with maternal glucose. Obstet Gynecol 2008; 112:1007.

4. HAPO Study Cooperative Research Group, Metzger BE, Lowe LP, et al. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med 2008; 358:1991.

5. Crowther CA, Hiller JE, Moss JR, et al. Effect of treatment of gestational diabetes mellitus on pregnancy outcomes. N Engl J Med 2005; 352:2477.

6. Kwik M, Seeho SK, Smith C, et al. Outcomes of pregnancies affected by impaired glucose tolerance. Diabetes Res Clin Pract 2007; 77:263.

7. Garner P, Okun N, Keely E, et al. A randomized controlled trial of strict glycemic control and tertiary level obstetric care versus routine obstetric care in the management of gestational diabetes: a pilot study. Am J Obstet Gynecol 1997; 177:190.

8. Sovio U, Murphy HR, Smith GC. Accelerated Fetal Growth Prior to Diagnosis of

Gestational Diabetes Mellitus: A Prospective Cohort Study of Nulliparous Women. Diabetes Care 2016; 39:982.

9. Dooley SL, Metzger BE, Cho NH. Gestational diabetes mellitus. Influence of race on disease prevalence and perinatal outcome in a U.S. population. Diabetes 1991; 40 Suppl 2:25.

10. Lipscomb KR, Gregory K, Shaw K. The outcome of macrosomic infants weighing at least 4500 grams: Los Angeles County + University of Southern California experience. Obstet Gynecol 1995; 85:558.

11. Lazer S, Biale Y, Mazor M, et al. Complications associated with the macrosomic fetus. J Reprod Med 1986; 31:501.

12. Rouse DJ, Owen J. Prophylactic cesarean delivery for fetal macrosomia diagnosed by means of ultrasonography--A Faustian bargain? Am J Obstet Gynecol 1999; 181:332.

13. Bérard J, Dufour P, Vinatier D, et al. Fetal macrosomia: risk factors and outcome. A study of the outcome concerning 100 cases >4500 g. Eur J Obstet Gynecol Reprod Biol 1998; 77:51.

14. Stotland NE, Caughey AB, Breed EM, Escobar GJ. Risk factors and obstetric complications associated with macrosomia. Int J Gynaecol Obstet 2004; 87:220.

15. Cohen BF, Penning S, Ansley D, et al. The incidence and severity of shoulder dystocia correlates with a sonographic measurement of asymmetry in patients with diabetes. Am J Perinatol 1999; 16:197.

16. Nesbitt TS, Gilbert WM, Herrchen B. Shoulder dystocia and associated risk factors with macrosomic infants born in California. Am J Obstet Gynecol 1998; 179:476.

17. Casey BM, Lucas MJ, Mcintire DD, Leveno KJ. Pregnancy outcomes in women with gestational diabetes compared with the general obstetric population. Obstet Gynecol 1997; 90:869.

18. Yogev Y, Xenakis EM, Langer O. The association between preeclampsia and the severity of gestational diabetes: the impact of glycemic control. Am J Obstet Gynecol 2004; 191:1655.

19. Innes KE, Wimsatt JH, McDuffie R. Relative glucose tolerance and subsequent development of hypertension in pregnancy. Obstet Gynecol 2001; 97:905.

20. Joffe GM, Esterlitz JR, Levine RJ, et al. The relationship between abnormal glucose tolerance and hypertensive disorders of pregnancy in healthy nulliparous women.

Calcium for Preeclampsia Prevention (CPEP) Study Group. Am J Obstet Gynecol 1998; 179:1032.

21. Carpenter MW. Gestational diabetes, pregnancy hypertension, and late vascular disease. Diabetes Care 2007; 30 Suppl 2:S246.

22. Yogev, Chen, Hod, et al. Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study: preeclampsia. Am J Obstet Gynecol 2010; 202:255.e1.

23. Parretti E, Lapolla A, Dalfrà M, et al. Preeclampsia in lean normotensive normotolerant pregnant women can be predicted by simple insulin sensitivity indexes. Hypertension 2006; 47:449.

24. Sierra-Laguado J, García RG, Celedón J, et al. Determination of insulin resistance using the homeostatic model assessment (HOMA) and its relation with the risk of developing pregnancy-induced hypertension. Am J Hypertens 2007; 20:437.

25. Hauth JC, Clifton RG, Roberts JM, et al. Maternal insulin resistance and preeclampsia. Am J Obstet Gynecol 2011; 204:327.e1.

26. Bryson CL, Ioannou GN, Rulyak SJ, Critchlow C. Association between gestational diabetes and pregnancy-induced hypertension. Am J Epidemiol 2003; 158:1148.

27. Pilliod RA, Page JM, Burwick RM, et al. The risk of fetal death in nonanomalous pregnancies affected by polyhydramnios. Am J Obstet Gynecol 2015; 213:410.e1.

28. Shoham I, Wiznitzer A, Silberstein T, et al. Gestational diabetes complicated by hydramnios was not associated with increased risk of perinatal morbidity and mortality. Eur J Obstet Gynecol Reprod Biol 2001; 100:46.

29. Biggio JR Jr, Wenstrom KD, Dubard MB, Cliver SP. Hydramnios prediction of adverse perinatal outcome. Obstet Gynecol 1999; 94:773.

30. Ye W, Luo C, Huang J, et al. Gestational diabetes mellitus and adverse pregnancy outcomes: systematic review and meta-analysis. BMJ 2022; 377:e067946.

31. Girz BA, Divon MY, Merkatz IR. Sudden fetal death in women with well-controlled, intensively monitored gestational diabetes. J Perinatol 1992; 12:229.

32. Aberg A, Rydhström H, Källén B, Källén K. Impaired glucose tolerance during pregnancy is associated with increased fetal mortality in preceding sibs. Acta Obstet Gynecol Scand 1997; 76:212.

33. Dudley DJ. Diabetic-associated stillbirth: incidence, pathophysiology, and prevention. Obstet Gynecol Clin North Am 2007; 34:293.

34. Langer O, Rodriguez DA, Xenakis EM, et al. Intensified versus conventional management of gestational diabetes. Am J Obstet Gynecol 1994; 170:1036.

35. Blank A, Grave GD, Metzger BE. Effects of gestational diabetes on perinatal morbidity reassessed. Report of the International Workshop on Adverse Perinatal Outcomes of Gestational Diabetes Mellitus, December 3-4, 1992. Diabetes Care 1995; 18:127.

36. Hod M, Merlob P, Friedman S, et al. Gestational diabetes mellitus. A survey of perinatal complications in the 1980s. Diabetes 1991; 40 Suppl 2:74.

37. Werner EF, Romano ME, Rouse DJ, et al. Association of Gestational Diabetes Mellitus With Neonatal Respiratory Morbidity. Obstet Gynecol 2019; 133:349.

38. ACOG Practice Bulletin No. 190: Gestational Diabetes Mellitus. Obstet Gynecol 2018; 131:e49.

39. Crowther CA, Samuel D, Hughes R, et al. Tighter or less tight glycaemic targets for women with gestational diabetes mellitus for reducing maternal and perinatal morbidity: A stepped-wedge, cluster-randomised trial. PLoS Med 2022; 19:e1004087.

40. Gabbe SG, Mestman JG, Freeman RK, et al. Management and outcome of class A diabetes mellitus. Am J Obstet Gynecol 1977; 127:465.

41. Kjos SL, Leung A, Henry OA, et al. Antepartum surveillance in diabetic pregnancies: predictors of fetal distress in labor. Am J Obstet Gynecol 1995; 173:1532.

42. Landon MB, Gabbe SG. Antepartum fetal surveillance in gestational diabetes mellitus. Diabetes 1985; 34 Suppl 2:50.

43. Rosenstein MG, Cheng YW, Snowden JM, et al. The risk of stillbirth and infant death stratified by gestational age in women with gestational diabetes. Am J Obstet Gynecol 2012; 206:309.e1.

44. Cheng YW, Chung JH, Block-Kurbisch I, et al. Treatment of gestational diabetes mellitus: glyburide compared to subcutaneous insulin therapy and associated perinatal outcomes. J Matern Fetal Neonatal Med 2012; 25:379.

45. McElwee ER, Oliver EA, McFarling K, et al. Risk of Stillbirth in Pregnancies Complicated by Diabetes, Stratified by Fetal Growth. Obstet Gynecol 2023; 141:801.

46. Kjos SL, Schaefer-Graf UM. Modified therapy for gestational diabetes using high-risk and low-risk fetal abdominal circumference growth to select strict versus relaxed maternal glycemic targets. Diabetes Care 2007; 30 Suppl 2:S200.

47. Ben-Haroush A, Chen R, Hadar E, et al. Accuracy of a single fetal weight estimation at

29-34 weeks in diabetic pregnancies: can it predict large-for-gestational-age infants at term? Am J Obstet Gynecol 2007; 197:497.e1.

48. Little SE, Edlow AG, Thomas AM, Smith NA. Estimated fetal weight by ultrasound: a modifiable risk factor for cesarean delivery? Am J Obstet Gynecol 2012; 207:309.e1.

49. Engstrom JL, Work BA Jr. Prenatal prediction of small- and large-for-gestational age neonates. J Obstet Gynecol Neonatal Nurs 1992; 21:486.

50. Humphries J, Reynolds D, Bell-Scarbrough L, et al. Sonographic estimate of birth weight: relative accuracy of sonographers versus maternal-fetal medicine specialists. J Matern Fetal Neonatal Med 2002; 11:108.

51. Johnstone FD, Prescott RJ, Steel JM, et al. Clinical and ultrasound prediction of macrosomia in diabetic pregnancy. Br J Obstet Gynaecol 1996; 103:747.

52. McLaren RA, Puckett JL, Chauhan SP. Estimators of birth weight in pregnant women requiring insulin: a comparison of seven sonographic models. Obstet Gynecol 1995; 85:565.

53. Smith GC, Smith MF, McNay MB, Fleming JE. The relation between fetal abdominal circumference and birthweight: findings in 3512 pregnancies. Br J Obstet Gynaecol 1997; 104:186.

54. Hackmon R, Bornstein E, Ferber A, et al. Combined analysis with amniotic fluid index and estimated fetal weight for prediction of severe macrosomia at birth. Am J Obstet Gynecol 2007; 196:333.e1.

55. Scioscia M, Scioscia F, Vimercati A, et al. Estimation of fetal weight by measurement of fetal thigh soft-tissue thickness in the late third trimester. Ultrasound Obstet Gynecol 2008; 31:314.

56. Cromi A, Ghezzi F, Di Naro E, et al. Large cross-sectional area of the umbilical cord as a predictor of fetal macrosomia. Ultrasound Obstet Gynecol 2007; 30:861.

57. Higgins MF, Russell NM, Mulcahy CH, et al. Fetal anterior abdominal wall thickness in diabetic pregnancy. Eur J Obstet Gynecol Reprod Biol 2008; 140:43.

58. Mathiesen ER, Christensen AB, Hellmuth E, et al. Insulin dose during glucocorticoid treatment for fetal lung maturation in diabetic pregnancy: test of an algorithm [correction of analgoritm]. Acta Obstet Gynecol Scand 2002; 81:835.

59. Refuerzo JS, Garg A, Rech B, et al. Continuous glucose monitoring in diabetic women following antenatal corticosteroid therapy: a pilot study. Am J Perinatol 2012; 29:335.

60. Feghali MN, Caritis SN, Catov JM, Scifres CM. Timing of delivery and pregnancy outcomes in women with gestational diabetes. Am J Obstet Gynecol 2016; 215:243.e1.

61. Alberico S, Businelli C, Wiesenfeld U, et al. Gestational diabetes and fetal growth acceleration: induction of labour versus expectant management. Minerva Ginecol 2010; 62:533.

62. Sutton AL, Mele L, Landon MB, et al. Delivery timing and cesarean delivery risk in women with mild gestational diabetes mellitus. Am J Obstet Gynecol 2014; 211:244.e1.

63. Kjos SL, Henry OA, Montoro M, et al. Insulin-requiring diabetes in pregnancy: a randomized trial of active induction of labor and expectant management. Am J Obstet Gynecol 1993; 169:611.

64. Lurie S, Insler V, Hagay ZJ. Induction of labor at 38 to 39 weeks of gestation reduces the incidence of shoulder dystocia in gestational diabetic patients class A2. Am J Perinatol 1996; 13:293.

65. Conway DL, Langer O. Elective delivery of infants with macrosomia in diabetic women: reduced shoulder dystocia versus increased cesarean deliveries. Am J Obstet Gynecol 1998; 178:922.

66. Lurie S, Matzkel A, Weissman A, et al. Outcome of pregnancy in class A1 and A2 gestational diabetic patients delivered beyond 40 weeks' gestation. Am J Perinatol 1992; 9:484.

67. Peled Y, Perri T, Chen R, et al. Gestational diabetes mellitus--implications of different treatment protocols. J Pediatr Endocrinol Metab 2004; 17:847.

68. Alberico S, Erenbourg A, Hod M, et al. Immediate delivery or expectant management in gestational diabetes at term: the GINEXMAL randomised controlled trial. BJOG 2017; 124:669.

69. Biesty LM, Egan AM, Dunne F, et al. Planned birth at or near term for improving health outcomes for pregnant women with gestational diabetes and their infants. Cochrane Database Syst Rev 2018; 1:CD012910.

70. Hod M, Kapur A, Sacks DA, et al. The International Federation of Gynecology and Obstetrics (FIGO) Initiative on gestational diabetes mellitus: A pragmatic guide for diagnosis, management, and care. Int J Gynaecol Obstet 2015; 131 Suppl 3:S173.

71. Spong CY, Mercer BM, D'alton M, et al. Timing of indicated late-preterm and early-

term birth. Obstet Gynecol 2011; 118:323.

72. Niu B, Lee VR, Cheng YW, et al. What is the optimal gestational age for women with gestational diabetes type A1 to deliver? Am J Obstet Gynecol 2014; 211:418.e1.

73. American College of Obstetricians and Gynecologists' Committee on Obstetric Practice, Society for Maternal-Fetal Medicine. Medically Indicated Late-Preterm and Early-Term Deliveries: ACOG Committee Opinion, Number 831. Obstet Gynecol 2021; 138:e35.

74. Rouse DJ, Owen J, Goldenberg RL, Cliver SP. The effectiveness and costs of elective cesarean delivery for fetal macrosomia diagnosed by ultrasound. JAMA 1996; 276:1480.

75. Caughey AB, Sandberg PL, Zlatnik MG, et al. Forceps compared with vacuum: rates of neonatal and maternal morbidity. Obstet Gynecol 2005; 106:908.

76. Demissie K, Rhoads GG, Smulian JC, et al. Operative vaginal delivery and neonatal and infant adverse outcomes: population based retrospective analysis. BMJ 2004; 329:24.

77. Flores-le Roux JA, Sagarra E, Benaiges D, et al. A prospective evaluation of neonatal hypoglycaemia in infants of women with gestational diabetes mellitus. Diabetes Res Clin Pract 2012; 97:217.

78. Barrett HL, Morris J, McElduff A. Watchful waiting: a management protocol for maternal glycaemia in the peripartum period. Aust N Z J Obstet Gynaecol 2009; 49:162.

79. Andersen O, Hertel J, Schmølker L, Kühl C. Influence of the maternal plasma glucose concentration at delivery on the risk of hypoglycaemia in infants of insulin-dependent diabetic mothers. Acta Paediatr Scand 1985; 74:268.

80. Kenepp NB, Kumar S, Shelley WC, et al. Fetal and neonatal hazards of maternal hydration with 5% dextrose before caesarean section. Lancet 1982; 1:1150.

81. Jovanovic L. Glucose and insulin requirements during labor and delivery: the case for normoglycemia in pregnancies complicated by diabetes. Endocr Pract 2004; 10 Suppl 2:40.

82. Ryan EA, Al-Agha R. Glucose control during labor and delivery. Curr Diab Rep 2014; 14:450.

83. Tigas S, Sunehag A, Haymond MW. Metabolic adaptation to feeding and fasting during lactation in humans. J Clin Endocrinol Metab 2002; 87:302.

84. Lenz S, Kühl C, Hornnes PJ, Hagen C. Influence of lactation on oral glucose tolerance in the puerperium. Acta Endocrinol (Copenh) 1981; 98:428.

85. Kjos SL, Henry O, Lee RM, et al. The effect of lactation on glucose and lipid metabolism in women with recent gestational diabetes. Obstet Gynecol 1993; 82:451.

86. Gunderson EP, Crites Y, Chiang V, et al. Influence of breastfeeding during the postpartum oral glucose tolerance test on plasma glucose and insulin. Obstet Gynecol 2012; 120:136.

87. Gunderson EP, Hurston SR, Ning X, et al. Lactation and Progression to Type 2 Diabetes Mellitus After Gestational Diabetes Mellitus: A Prospective Cohort Study. Ann Intern Med 2015; 163:889.

88. Ziegler AG, Wallner M, Kaiser I, et al. Long-term protective effect of lactation on the development of type 2 diabetes in women with recent gestational diabetes mellitus. Diabetes 2012; 61:3167.

89. Ley SH, Chavarro JE, Li M, et al. Lactation Duration and Long-term Risk for Incident Type 2 Diabetes in Women With a History of Gestational Diabetes Mellitus. Diabetes Care 2020; 43:793.

90. Gunderson EP, Lewis CE, Lin Y, et al. Lactation Duration and Progression to Diabetes in Women Across the Childbearing Years: The 30-Year CARDIA Study. JAMA Intern Med 2018; 178:328.

91. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. Medical Eligibility Criteria for Contraceptive Use, 2016. MMWR Recomm Rep 2016; 65:1.

92. Lopez LM, Grimes DA, Schulz KF. Steroidal contraceptives: effect on carbohydrate metabolism in women without diabetes mellitus. Cochrane Database Syst Rev 2014; :CD006133.

93. Kozhimannil KB, Pereira MA, Harlow BL. Association between diabetes and perinatal depression among low-income mothers. JAMA 2009; 301:842.

Topic 4800 Version 68.0

GRAPHICS

General approach to obstetric management of uncomplicated GDM
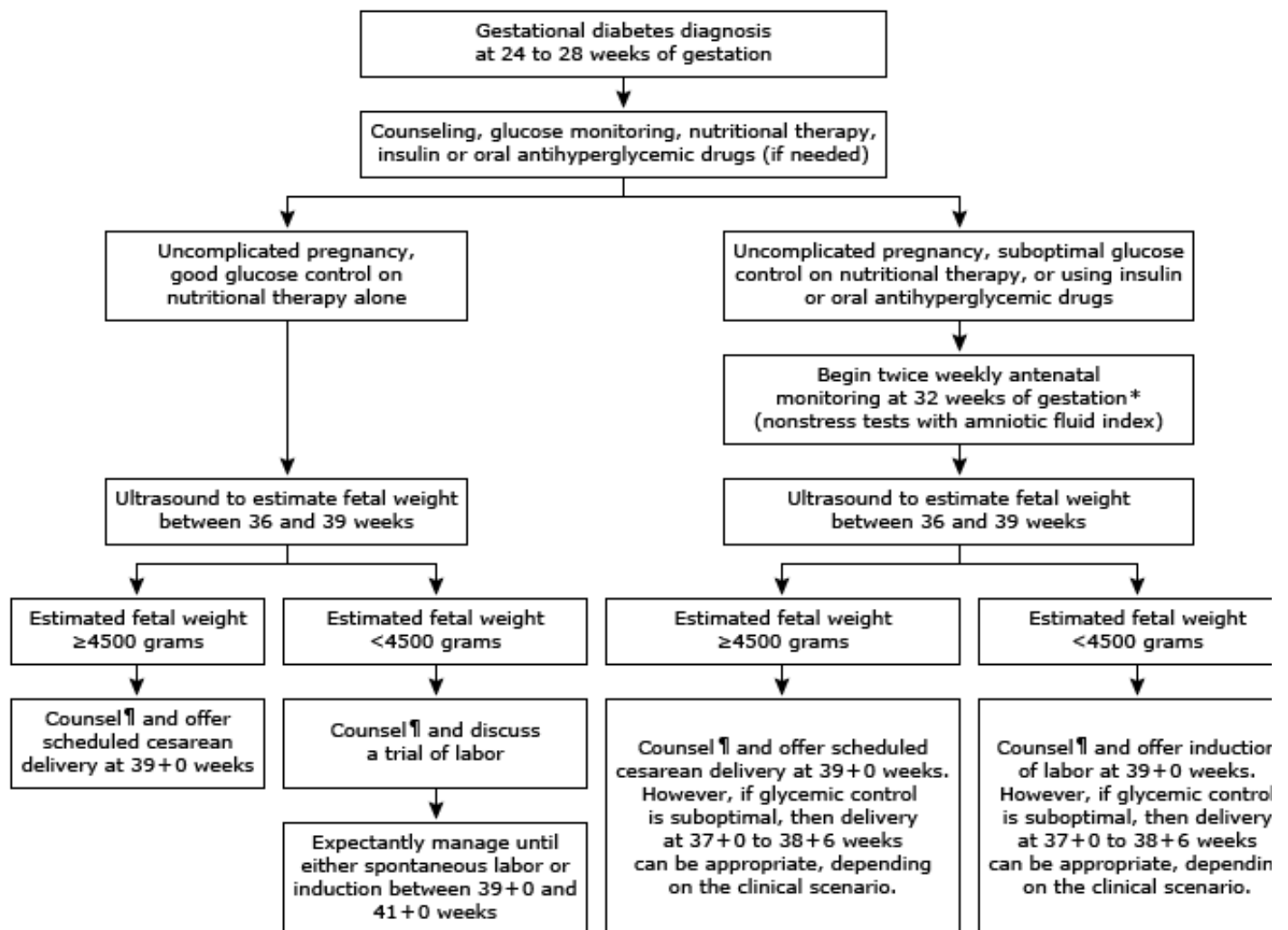
This algorithm reflects the author's general approach to obstetric management of patients with uncomplicated gestational diabetes.

GDM: gestational diabetes mellitus.

* Patients who have suboptimal glucose control on nutritional therapy should also be started on insulin.

¶ When counseling patients, key issues to address include:

- The difficulty in accurately predicting birth weight by any method.
- The estimated fetal growth between ultrasound examination and delivery.
- The risks of shoulder dystocia and associated complications.
- The risks of a cesarean delivery in the current pregnancy.
- The risks of cesarean delivery in the current pregnancy on management and outcome of future pregnancies.

Graphic 94336 Version 3.0

Graphic 94336 Version 3.0

American Diabetes Association criteria for the diagnosis of diabetes

1. A1C ≥6.5%. The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay.*

   OR

2. FPG ≥126 mg/dL (7 mmol/L). Fasting is defined as no caloric intake for at least 8 hours.*

   OR

3. 2-hour plasma glucose ≥200 mg/dL (11.1 mmol/L) during an OGTT. The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.*

   OR

4. In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dL (11.1 mmol/L).

A1C: glycated hemoglobin; NGSP: National Glycohemoglobin Standardization Program; DCCT: Diabetes Control and Complications Trial; FPG: fasting plasma glucose; OGTT: oral glucose tolerance test.

* In the absence of unequivocal hyperglycemia, diagnosis requires two abnormal test results from the same sample or in two separate test samples.

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Graphic 61853 Version 18.0

Categories of increased risk for diabetes (prediabetes)*

FPG 100 to 125 mg/dL (5.6 to 6.9 mmol/L) – IFG
2-hour post-load glucose on the 75 g OGTT 140 to 199 mg/dL (7.8 to 11.0 mmol/L) – IGT
A1C 5.7 to 6.4% (39 to 46 mmol/mol)

FPG: fasting plasma glucose; IFG: impaired fasting glucose; OGTT: oral glucose tolerance test; IGT: impaired glucose tolerance; A1C: glycated hemoglobin.
* For all 3 tests, risk is continuous, extending below the lower limit of the range and becoming disproportionately greater at higher ends of the range.

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Graphic 82479 Version 16.0

Appendix A: Edinburgh Postnatal Depression Scale

Name _____

Date: _____

Number of Months Postpartum: _____

As you have recently had a baby, we would like to know how you are feeling. Please mark the answer which comes closest to how you have felt in the past **7 days**, not just how you feel today.

Here is an example, already completed:

I have felt happy:
— Yes, all the time
X Yes, most of the time
— No, not very often
— No, not at all

This would mean "I have felt happy most of the time during the past week". Please complete the following questions in the same way.

In the past 7 days:
1. I have been able to laugh and see the funny side of things
   — As much as I always could   0
   — Not quite so much now   1
   — Definitely not so much now   2
   — Not at all   3

2. I have looked forward with enjoyment to things
   — As much as I ever did   0
   — Rather less than I used to   1
   — Definitely less than I used to   2
   — Hardly at all   3

3. I have blamed myself unnecessarily when things went wrong
   — Yes, most of the time   3
   — Yes, some of the time   2
   — Not very often   1
   — No, never   0

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Graphic 67741 Version 3.0

Appendix A: Edinburgh Postnatal Depression Scale (EPDS) (continued)

In the past 7 days:

4. I have been anxious or worried for no good reason
   __ No, not at all — 0
   __ Hardly ever — 1
   __ Yes, sometimes — 2
   __ Yes, very often — 3

5. I have felt scared or panicky for no very good reason
   __ Yes, quite a lot — 3
   __ Yes, sometimes — 2
   __ No, not much — 1
   __ No, not at all — 0

6. Things have been getting on top of me
   __ Yes, most of the time I haven't been able to cope — 3
   __ Yes, sometimes I haven't been coping as well as usual — 2
   __ No, most of the time I have coped quite well — 1
   __ No, I have been coping as well as ever — 0

7. I have been so unhappy that I have had difficulty sleeping
   __ Yes, most of the time — 3
   __ Yes, sometimes — 2
   __ Not very often — 1
   __ No, not at all — 0

8. I have felt sad or miserable
   __ Yes, most of the time — 3
   __ Yes, quite often — 2
   __ Not very often — 1
   __ No, not at all — 0

9. I have been so unhappy that I have been crying
   __ Yes, most of the time — 3
   __ Yes, quite often — 2
   __ Only occasionally — 1
   __ No, never — 0

10. The thought of harming myself has occurred to me
    __ Yes, quite often — 3
    __ Sometimes — 2
    __ Hardly ever — 1
    __ Never — 0

We suggest a cutoff score of 11, which appears to maximize sensitivity plus specificity in screening for postpartum depression. Women who report depressive symptoms without suicidal ideation or major functional impairment (or score between 5 and 9 on the EPDS) should be re-evaluated within one month.

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Graphic 81407 Version 6.0

PHQ-9 depression questionnaire

Name:		Date:			
Over the last 2 weeks, how often have you been bothered by any of the following problems?		**Not at all**	**Several days**	**More than half the days**	**Nearly every day**
Little interest or pleasure in doing things		0	1	2	3
Feeling down, depressed, or hopeless		0	1	2	3
Trouble falling or staying asleep, or sleeping too much		0	1	2	3
Feeling tired or having little energy		0	1	2	3
Poor appetite or overeating		0	1	2	3
Feeling bad about yourself, or that you are a failure, or that you have let yourself or your family down		0	1	2	3
Trouble concentrating on things, such as reading the newspaper or watching television		0	1	2	3
Moving or speaking so slowly that other people could have noticed? Or the opposite, being so fidgety or restless that you have been moving around a lot more than usual.		0	1	2	3
Thoughts that you would be better off dead, or of hurting yourself in some way		0	1	2	3
Total ___ =		__	+ __	+ __	+ __
PHQ-9 score ≥10: Likely major depression					
Depression score ranges:					
5 to 9: mild					
10 to 14: moderate					
15 to 19: moderately severe					
≥20: severe					
If you checked off any problems, how difficult		Not	Somewhat	Very	Extremel

have these problems made it for you to do your work, take care of things at home, or get along with other people?	difficult at all ___	difficult ___	difficult ___	difficult ___

PHQ: Patient Health Questionnaire.

Developed by Drs. Robert L Spitzer, Janet BW Williams, Kurt Kroenke, and colleagues, with an educational grant from Pfizer, Inc. No permission required to reproduce, translate, display or distribute.

Graphic 59307 Version 12.0

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