Objectives

1. To understand the natural progression and treatment of type 1 diabetes during pregnancy and postpartum
2. To understand the hormonal changes throughout pregnancy and how they may affect blood sugar levels
3. To understand the adverse sequelae of poor or suboptimal glycemic control during pregnancy

Case Presentation

A 28-year-old woman diagnosed with type 1 diabetes mellitus 18 years prior, who is on an insulin pump, presents to her endocrinologist stating that she is planning a pregnancy. Her glycemic control has been suboptimal over recent years and she has background retinopathy, which has been stable and has not required laser treatment. She has no obvious neuropathy or nephropathy. Currently her insulin pump is set at basal rates such that her total daily basal dose is 14.2 units/24 hours, and she takes boluses of Lispro with each meal (4 to 10 units). Her blood pressure is stable and physical exam unremarkable. Her fasting blood sugars range from 140 to 180, and throughout the day her premeal values are 130 to 215. Her current hemoglobin A1c(HbA1c) is 7.8%.

She is instructed to refrain from getting pregnant until glycemic control improves, as documented by an A1c <7%, and to start prenatal vitamins. She is also requested to maintain food and glucose records documenting fasting, premeal, 1 hour postmeal, bedtime, and 3 a.m. fingersticks for 1 to 2 weeks. She is referred for nutritional and carbohydrate counting counseling. Over the course of the next 3 to 4 weeks, insulin basals and boluses were adjusted according to pre- and postmeal sugars. The basal rate of insulin was increased to 16.6 units/24 hours, and premeal

C. Levy
Assistant Professor, Weill Cornell Medical College, New York, NY
boluses were adjusted to an insulin to carbohydrate ratio of 1:10, with correction of 1:40 for hyperglycemia. Two months later, her A1c was 6.4%.

After 3 months, the patient notifies her endocrinologist that she is 3 weeks pregnant. Laboratory evaluation revealed an A1c of 6.1%, her retinopathy was stable, and there were no signs of nephropathy. The patient was instructed regarding the importance of maintaining optimal glycemic goals in pregnancy, that is, fasting blood levels <90 and 1-hour postprandial blood sugars of <120. The risks of hypoglycemia during the first trimester were discussed, as well as appropriate nutritional guidelines for both the prevention of ketosis and appropriate nutrition for the mother and fetus.

At 9 weeks’ gestation the patient develops low fasting glucose levels and high postprandial sugars. She is instructed to decrease her overnight basal rate (total dose 15.5 units/24 hours basal) and increase her premeal insulin-carbohydrate ratio to 1:8. At 15 weeks’ gestation (second trimester), she is normotensive and is exhibiting appropriate weight gain. Fasting glucose levels and postprandial values exceed the outlined goals. The basal rate, therefore, is increased to a total daily dose of 16.5 units and the insulin/carbohydrate ratio for different meals is adjusted as follows: breakfast 1:8, lunch 1:5, and dinner 1:6.

At 24 weeks, her hemoglobin A1c was 6.1% and she is normotensive with adequate weight gain. She exhibits no retinopathy or proteinuria. Fasting and postmeal blood sugars are elevated, and her insulin is adjusted as follows: basal rates were increased overnight (total daily basal now 17.1 units/24 hours) and premeal ratios were adjusted to breakfast 1:7, lunch 1:4, and dinner 1:5. At 26 weeks, she calls with complaints of abdominal pain, nausea, and fever with minimal solid food intake. She took no insulin boluses the prior day and glucose levels had been steadily increasing. Her morning sugar was 221 mg/dL with moderate urine ketones. She is able to drink fluids. She is instructed to take a correction dose using an algorithm of 1 unit of insulin for each 20 mg/dL of glucose decrement desired. Other instructions included to increase fluid intake and recheck her blood glucose in 1 hour to ensure the pump site is not occluded. Three hours later her glucose was 147 mg/dL, and urine ketones were “trace,” but she still felt nauseous. She is instructed to take her insulin boluses after meals so that that she does not vomit before taking the bolus, thus preventing hypoglycemia. Ketones resolved and her glucose levels stabilized.

At 32 weeks she starts to undergo nonstress testing to assess placental function. An ultrasound reveals a fetal size in the 60th percentile without polyhydramnios. At 37 weeks, she started to note increased episodes of hypoglycemia in the 50- to 60-mg/dL range. Her nonstress tests were normal. She is reassured and instructed to decrease her bolus and basal insulin rate administrations.

At 39 weeks, she notes contractions. In the hospital, the obstetrician is instructed to discontinue the pump and an insulin drip is initiated with frequent blood glucose monitoring to maintain euglycemia (goal 70–110 mg/dL). She has a normal vaginal delivery and is instructed to resume her pump at 50% of her latest pregnancy doses for both basal and boluses.

At 2 weeks postpartum, the patient is breast-feeding. She notes variability in blood glucose levels and increased frequency of hypoglycemia after the baby is fed.