New-Onset IDDM Presenting With Diabetic Ketoacidosis in a Pregnant Adolescent

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OBJECTIVE — To describe the presentation of insulin-dependent diabetes mellitus (IDDM) as ketoacidosis during pregnancy in a teenager.

CASE — A 14-year-old pregnant girl presented with ketoacidosis (bicarbonate 14 nM, 14 meq/l, pH 7.27, glucose 67 mM, 1,208 mg/dl) during the last month of pregnancy with a fetal demise. Two years of follow-up has confirmed that she has IDDM.

CONCLUSIONS — Diabetes presenting in pregnant adolescents is likely due to IDDM. Immediate insulin therapy and proper education about managing diabetes should be initiated to hopefully prevent the outcome described in this patient.

Diabetes first appearing during pregnancy is classified as gestational diabetes mellitus (GDM), a self-limited disorder occurring during 2–3% of pregnancies that terminates when the pregnancy ends (1). Many women with GDM will, years later, go on to develop permanent diabetes, usually non-insulin-dependent diabetes mellitus (NIDDM) (1).

Insulin-dependent diabetes mellitus (IDDM) may also be diagnosed during pregnancy (2–5), although less commonly than is GDM. Diabetic ketoacidosis as the initial presentation of IDDM has been reported in pregnant adults (6). We report a previously well teenager who was found to be in diabetic ketoacidosis during pregnancy and who was subsequently proven to have IDDM.

CASE — We were asked to consult on a 14.5-year-old African-American girl with a 35-week pregnancy who presented to another hospital complaining of leg muscle aches, lower abdominal pain, and blurry vision. She had felt no fetal movements for 24 h. She had not sought prenatal care until 2 months before hospitalization, at which time a 50-g glucose challenge test was 6.7 mM, 120 mg/dl. Ten days before her presentation, glycosuria was detected during a prenatal visit. She had not returned for a scheduled follow-up visit 1 week later.

Further history revealed that she had had increased thirst and increased urination during the previous month. Family history was positive for insulin-requiring diabetes in her grandmother and diabetes treated with oral hypoglycemic agents in an aunt who died at 40 years of age.

On physical examination, her temperature was 98.8°F, pulse was 100/min, respirations were 20/min, and blood pressure was 140/100. Her height was 156 cm. She was slim and had poor skin turgor, sunken eyes, and dry oral mucosa. Her uterus had a fundal height of 34 cm. No fetal heart sounds were heard.

Initial laboratory evaluation revealed a glucose of 67 mM (1,208 mg/dl), sodium 127 mM (127 meq/l), K 5.8 mM (5.8 meq/l), chloride 96 mM (96 meq/l), blood urea nitrogen 7.5 mM (21 mg/dl), creatinine 132 μM (1.5 mg/dl), bicarbonate 14 nM (14 meq/l), and pH 7.27. Serum acetone was positive. Concomitant serum insulin was 143 pM (23.8 mIU/ml) (normal: 36–180 pM), and C-peptide was <0.05 nM (<0.15 ng/ml) (normal: 0.1–1.2 nM). Islet cell antibodies (ICAs) (measured by indirect immunofluorescence assay) and insulin antibodies (measured by serum insulin binding capacity) were undetectable. Thyroid antimicrosomal antibodies were positive at 400. Glycosylated hemoglobin was 15.5% (normal 4.3–7.7%).

She was diagnosed as having mild ketoacidosis with hyperosmolar hyperglycemia, preeclampsia, and a pregnancy with fetal demise. She was treated with magnesium sulfate, insulin, and rehydration. When her clinical condition was stabilized, she delivered a female stillborn

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GDM, gestational diabetes mellitus; IDDM, insulin-dependent diabetes mellitus; NIDDM, non-insulin-dependent diabetes mellitus; ICA, islet cell antibody.

infant. Pathology of the placenta showed it was in the third trimester with eccentric insertion of a trivascular cord.

She was discharged on insulin, which was discontinued in 2 weeks. Eleven months later, she began insulin treatment again because she developed hyperglycemia, weight loss, and ketosis. She has continued to require insulin therapy for 2 years.

CONCLUSIONS — Diabetes diagnosed during pregnancy is classified as GDM and usually implies a disorder of carbohydrate tolerance related to the diabetogenic effect of the pregnancy. Many women with GDM do go on, years later, to develop NIDDM (1,5). Although NIDDM may present in teenagers, GDM in pregnant teenagers is reported to be so low as to not warrant routine screening (7).

IDDM presenting during pregnancy in adults is less common than is GDM, but it does occur (2–5). The incidence of new-onset IDDM presenting during pregnancy may, however, be underestimated. In women with presumed GDM, ICAs have been detected in 10–38% of the subjects (8–11). ICAs are believed to be predictors of IDDM, suggesting that in adult women with presumed GDM, it is possible that the hyperglycemia may be secondary to early IDDM. The stress of a third-trimester gestation may cause the insulin deficiency to be expressed sooner than if the person had not become pregnant.

IDDM commonly presents as a new diagnosis during the teenage years (12). However, IDDM presenting during pregnancy in adolescents has only rarely been reported. Of the 60 women reported by Buschard et al. (2), three were <20 years of age. New-onset IDDM presenting as diabetic ketoacidosis during pregnancy has been reported in two adults. The first patient was 29 years of age and presented at 29 weeks gestation with signs of ketoacidosis, glucose 19.5 mM (351 mg/dl), and bicarbonate 6.0 nM (6.0 meq/l). Her ketoacidosis was treated, and insulin therapy was begun. At 38 weeks gestation, labor was induced, and she delivered a healthy baby girl. She did not require insulin in the puerperium but required insulin 3 months after the delivery. The second patient was 31 years of age and was admitted at 29 weeks gestation with ketoacidosis, glucose 29.5 mM (531 mg/dl), and bicarbonate 11.1 nM (11.1 meq/l). The ketoacidosis was treated, and she was started on insulin therapy. At 37 weeks gestation, labor was induced, and she delivered a baby boy with transposition of the great vessels (6). Ketoacidosis during pregnancy is associated with a rate of fetal mortality up to 70–90% (13,14).

Our teenage patient presented with symptoms of diabetes and glycosuria. Two months before her presentation of diabetic ketoacidosis, she had a negative screening test for diabetes. Once she developed symptoms of hyperglycemia, the time course to ketoacidosis was characteristically brief. Even though she had negative ICAs and insulin antibodies, she was ultimately proven to have IDDM, and she has remained insulin-dependent and ketosis-prone years later.

We think our experience should alert health-care workers to the concept that diabetes presenting in pregnant adolescents is likely due to IDDM. Immediate insulin therapy and proper education about managing diabetes should be initiated to hopefully prevent the adverse outcomes of fetal demise and maternal diabetic ketoacidosis.

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