Diabetes and Pregnancy

Preface

Lois Jovanovic, MD, Guest Editor

What is so bad about a big baby? Although the concept of a large, fat neonate conjures up joy, wealth, and a bouncing baby of health, when large-for-gestational-age (LGA) infants are the result of intrauterine overnutrition, the infants become pathologically overweight. Then, the consequences are not so happy. When obese neonates have metabolic aberrances, and if the majority of the fat concentration is in their viscera, these infants are destined to develop the metabolic syndrome and most likely will eventually develop diabetes.

Because the most immediate concern surrounding the birth of a large infant is trauma, such as shoulder dystocia, brachial palsy, and hypoxia with subsequent cerebral palsy, obstetricians have concentrated their treatment, management strategies, and concerns on determining the optimal timing and mode of delivery to ensure that such babies are alive and born without injury. However, once these babies are given to their pediatrician and the obstetricians are relieved of responsibility for them, the obstetricians redirect decisions about subsequent pregnancies and focus again on labor and delivery and not the fate of the infants after birth. This disconnect is resolving as more perinatologists understand the importance of intrauterine nutrition, glucose metabolism, and fetal overgrowth. Evidence is accumulating in the literature that neonatal glucose-mediated macrosomia is one of the major reasons that childhood obesity is on the rise and the rate of diabetes has grown exponentially.

Still, because the risk of an overgrown fetus is only 40% for those fetuses who have been exposed to hyperglycemia rather than 100%, many feel that intensive maternal glucose control may not be cost-effective in the general population. This phenomenon has been named the “fidgety fetus hypothesis” and suggests that the infants who are at highest risk of becoming macrosomic and thus predisposed to subsequent diabetes are sluggish in utero and thus cannot metabolize the extra glucose if there is exposure of hyperglycemia. Perhaps, therefore, because 60% of fetuses can compensate for the overnutrition by auto-regulating their own energy expenditure by increasing their activity to “burn off” extra calories, compulsive attention to minor elevations of maternal glucose in a general population is not considered a priority.

This lack of concern about LGA infants may come back to haunt us. Ignoring the abnormal growth and metabolic outcomes of macrosomic neonates who are born large secondarily to lack of attention to maternal glucose control dooms these infants to becoming the next generation of people with diabetes. To avoid this, we must reinforce our efforts to diagnose, monitor, and treat all hyperglycemia that occurs in pregnancy.

This Diabetes Spectrum From Research to Practice section focuses on new issues related to the challenge of providing intensive care to all pregnant women with hyperglycemia. Our first article (p. 85) reviews the literature on polycystic ovary syndrome (PCOS). Women with this syndrome have insulin resistance and develop gestational diabetes as soon as they become pregnant. By waiting for the usual time for an oral glucose tolerance test to be performed, we may be allowing the first half of these pregnancies to be affected by undiagnosed hyperglycemia. In this article, Howard Craig Zisser, MD, suggests that the
treatment plan for these women should include continuation of metformin throughout the first half of pregnancy to decrease the risk of spontaneous abortion and to treat any minor hyperglycemia. In our second article (p. 89), Julie D.L. Dupak, MD, PhD, and Angelina L. Trujillo, MD, focus on the utility of ultrasonography to monitor fetal well-being and guide therapy if there is any indication that a fetus is experiencing accelerated growth. The next article in this series (p. 94), also by Dr. Trujillo, reviews the safety and efficacy of insulin analogs in the treatment of diabetes in pregnancy. Of particular note, this article heralds the new U.S. Food and Drug Administration approval of insulin aspart as a Category B drug, placing it at the same status as insulin lispro. Elsewhere in this issue of Diabetes Spectrum (p. 117), readers can find a comprehensive review of preconception counseling for pregnant women with type 2 diabetes. The last article (p. 101) is written by the champion of the old “new” use of oral hypoglycemia agents. Oded Langer, MD, PhD, reviews his philosophy on the use of oral agents for the treatment of gestational diabetes and quotes his landmark randomized trial to underscore his reasoning.

Given the current evidence regarding hyperglycemia in pregnancy, we can no longer wait and hope that maternal excessive glucose will not affect fetuses or take our chances that these infants will be unscathed by the maternal glucose environment in which they develop. We need to take an active role in protecting today’s fetuses, tomorrow’s infants, from possible harm. Following the advice offered in this research section will positively change the destiny of future generations.

Suggested Readings

McCance DR, Pettitt DJ, Hanson RL, Jacobsson LT, Knowler WC, Bennett PH: Birth weight and non-insulin dependent diabetes: thrifty genotype, thrifty phenotype, or surviving small baby genotype? BMJ 308:942–945, 1994


