Maternal Diabetes Raises Atrial Septal Defect Risk

BY MITCHEL L. ZOLER
Philadelphia Bureau

ORLANDO — Women with either gestational or established diabetes were much more likely to deliver an infant with an atrial septal defect than were those with normal glucose control, based on the results of a retrospective, case-control study that included almost 5,000 women.

Women with established diabetes before they became pregnant were nearly 11-fold more likely to give birth to a child with an atrial septal defect (ASD), compared with women without diabetes, Dr. Creighton W. Don, a cardiologist at the University of Washington, Seattle, and associates reported in a poster at the annual scientific sessions of the American Heart Association.

They used linked birth certificate and hospital discharge data from all nonfederal hospitals in the Comprehensive Hospital Abstract Reporting System in Washington state during January 1987–December 2005. Cases were live-born singleton infants diagnosed with ASD. Controls were infants born without ASD in the same year.

The incidence of ASD reports in hospitals from eastern Washington seemed unusually high, so those hospitals were excluded and the analysis was limited to hospitals in western Washington. The analysis also excluded infants born at less than 32 weeks' gestation or less than 2,500 g. This left about 800 cases and 4,000 control infants who were included in a logistic regression analysis. The analysis controlled for several variables, including gestational age, birth weight, maternal age, maternal BMI, race, and hospital location.

The analysis showed that women with established diabetes were 10.6-fold more likely to give birth to an infant with an ASD than were mothers without diabetes, and that mothers who developed gestational diabetes were 3-fold more likely to have a child with ASD. The differences were statistically significant. ■

Term Neonates With CHD Show Brain Anomalies

Infants born at term with congenital heart disease show widespread brain abnormalities similar to those in preterm neonates, an imaging study of 57 newborns has shown.

These anomalies, are present well before the infants undergo cardiac surgery, reported Dr. Steven P. Miller of the University of California, San Francisco, and his associates. "Although most forms of congenital heart disease [CHD] are now amenable to early surgical repair, deficits that impair widespread neurodevelopmental domains are identified in up to half of childhood survivors: fine motor skills, visuospatial skills, and cognition, including memory, attention, and higher-order language skills," they noted.

Most studies on the issue have focused on acute brain injury related to delivery or to the surgery and its support procedures, such as cardiopulmonary bypass and hypothermic circulatory arrest. The investigators hypothesized that instead, much of this brain injury predates the surgery. They used MRI, three-dimensional magnetic resonance spectroscopic (MRS) techniques, and spectroscopic and diffusion tensor imaging to prospectively examine the brains of 29 term infants with transposition of the great arteries, 12 with single-ventricle physiology, and 16 normal term infants.

The newborns with CHD showed altered brain metabolism and microstructure shortly after birth, before they underwent surgery. These abnormalities were present even in areas that showed no visible injury on regular MRI. In some cases where the infants appeared to have discrete lesions, the abnormalities in metabolism and microstructure were evident even in areas that appeared to be uninvolved. For example, MRS showed that the ratio of *N*-acetylaspartate to choline, which increases with maturation, was significantly lower in the newborns with CHD than in the controls.

This impairment was widespread "and did not conform to the pattern of brain injury that is typical of hypoxia-ischemia in term newborns," so it was more likely due to longstanding abnormal brain development rather than to an acute injury, the investigators said (N. Engl. J. Med. 2007;357:1928-38).

—Mary Ann Moon