No Link Seen Between PCOS and Small Fetal Size

BY JEFF EVANS

WASHINGTON — Female infants born to women with polycystic ovary syndrome do not appear to have high levels of androgens or to be small for gestational age, based on the results of a prospective, case-control study.

In fact, offspring born to mothers with polycystic ovary syndrome (PCOS) were more likely than controls to be large for gestational age.

Findings from clinical and animal-based studies suggest that PCOS may originate during fetal development. Prenatal exposure to androgens has been shown to induce a PCOS phenotype in sheep, monkeys, and rats. In humans, retrospective studies have demonstrated that girls with PCOS features and premature menarche had been significantly small for their gestational age, according to Helen Anderson of the division of endocrinology, metabolism, and molecular medicine at Northwestern University, Chicago.

Female offspring of mothers with PCOS were more likely than controls to be large for gestational age.

MS. ANDERSON

To determine if the intrauterine environment of women with PCOS alters fetal growth and androgen levels, Ms. Anderson and her associates compared singleton pregnancies in 39 women with PCOS and 31 healthy control women. The participants were non-Hispanic white women who met National Institute of Child Health and Human Development criteria for PCOS. Women with PCOS had less then six menses per year, whereas healthy control women had a history of regular menses. None of the participants had a history of gestational diabetes, preexisting medical conditions, or complications during pregnancy.

Compared with healthy controls, a larger percentage of women with PCOS were nulliparous (64% vs. 39%) or had undergone ovulation induction or in vitro fertilization (77% vs. 6%). Women with PCOS were slightly, but significantly, younger than the healthy control women (30 years vs. 32 years). Although PCOS women had a slightly higher mean body mass index than did control women, they had comparable maternal weight gains.

The birth cohort consisted of more females (43) than males (27) because the investigators were primarily interested in female offspring, and they excluded women known to be carrying a male fetus.

Overall, the gestational age and birth weight of infants did not differ between women with and without PCOS. However, when Ms. Anderson and her colleagues stratified the analysis according to size at gestational age, a significantly greater proportion of the infants born to women with PCOS were large for gestational age (greater than 90th percentile), compared with healthy controls (23% vs. 3%).

"This may be secondary to the increased nutritional flow across the placenta," as elevated levels of insulin and glucose have been demonstrated in pregnant women with PCOS, Ms. Anderson said at the annual meeting of the Endocrine Society.

Analyses of the steroid hormones in whole (mixed arterial and venous) cord blood showed that the female offspring of PCOS women had significantly lower levels of androstenedione and estradiol than did the female offspring of controls.

However, female offspring had no differences in levels of testosterone, dihydrotestosterone, and dehydroepiandrosterone, although Ms. Anderson said that many of the testosterone and dihydrotestosterone values were at the low end of detectability for the assays.

Female offspring from either group of women showed no differences in levels of 17-hydroxyprogesterone or in the ratio of testosterone to estradiol levels.

The National Institutes of Health funded the study. Ms. Anderson reported having no conflicts of interest to disclose. ■