

552 EVALUATION OF PREGNANCY OUTCOME IN EUPLOID FETUSES WITH A SCREEN POSITIVE SECOND TRIMESTER MATERNAL SERUM SCREEN FOR TRISOMY 18 CALLA HOLMGREN¹, ALEXANDRA GROSVENOR¹, KRISTY NELSON², NANCY ROSE², ¹University of Utah, Obstetrics and Gynecology, Salt Lake City, Utah, ²Intermountain Healthcare, Obstetrics and Gynecology, Salt Lake City, Utah

OBJECTIVE: Second trimester maternal serum analyte markers in pregnancies that screen positive for trisomy 18 are all profoundly lower than expected. If these low levels of maternal serum analytes are identified in normal, euploid gestations, we hypothesize that this might be associated with adverse pregnancy outcome, and therefore this information might be important for further obstetrical management during gestation.

STUDY DESIGN: Between January, 2000 and August, 2004, we identified 65 patients at three tertiary care hospitals with a screen positive risk for trisomy 18 on second trimester serum analyte screening. Forty-three percent of these patients had amniocentesis in the second trimester. Charts from all patients were reviewed for both maternal and fetal outcomes. Statistical analysis was undertaken to evaluate these outcomes.

RESULTS: In total, 55 (84.6%) patients had known maternal and fetal outcomes, and, of these, 10 (18.0%) had trisomy 18 identified by amniocentesis. Of the women who had amniocentesis, 35% had trisomy 18. There were two triploidy fetuses with fetal deaths at 19-20 weeks gestation confirmed by autopsy. The remaining 43 patients had euploid gestations with no evidence of fetal abnormalities. There was 1 term stillbirth (2.2%), the cause of which was unknown.

For all patients, the mean gestational age at delivery was 37.1 weeks gestation (± 4.4 weeks) and mean Apgar scores were 7.5 at 1 minute and 8.6 at 5 minutes. Mean fetal weight at delivery was 3097 gm (± 727.8). 6.7% of the infants were delivered preterm secondary to preterm labor or preterm rupture of membranes. Of the total, 4.4% of the pregnancies were complicated by preeclampsia and 6.7% were complicated by IUGR.

CONCLUSION: There does not appear to be an increased risk of adverse pregnancy outcome in patients with a euploid gestation and a screen positive second trimester maternal serums screen for trisomy 18.

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553 CHRONIC HYPERTENSION AND RISK OF PLACENTAL ABRUPTION: IS THE ASSOCIATION MEDIATED THROUGH FETAL GROWTH? CANDE ANANTH¹, MORGAN PELTIER¹, JOHN SMULIAN¹, ANTHONY VINTZILEOS¹, ¹University of Medicine and Dentistry of New Jersey, Obstetrics, Gynecology and Reproductive Sciences, New Brunswick, New Jersey

OBJECTIVE: To assess the association between chronic hypertension in the risk of placental abruption and to evaluate if this association is modified by fetal growth status.

STUDY DESIGN: We utilized the US linked natality and fetal death data files (1995-2002), and limited the analysis to women that delivered a singleton birth at ≥ 22 weeks, and fetuses that weighed ≥ 500 g (n = 29,163,362). Fetal growth assessment was based on the estimated centile of birthweight for a given gestational age, and was categorized as < 1 , 1-2, 3-4, 5-9, 10-19, 20-29, ..., ≥ 90 centile. Women with a diagnosis of preexisting or gestational diabetes, or renal disorders were excluded. All analyses were adjusted for potential confounding factors through multivariable logistic regression.

RESULTS: Rates of abruption among women with and without chronic hypertension were 16.7 and 5.9 per 1,000 pregnancies, respectively. The association between chronic hypertension and abruption was strongest at term (OR 2.02, 95% CI 1.90, 2.15), with progressively declining risk at 32-36 weeks (OR 1.68, 95% CI 1.58, 1.79) and 22-31 weeks (OR 1.20, 95% CI 1.12, 1.28). At 22-31 and 32-36 weeks gestation, the association between chronic hypertension and abruption was present only among babies that were at ≥ 30 and ≥ 5 centiles for birthweight, respectively. However, the association at term gestation persisted at the entire spectrum of fetal growth centiles.

CONCLUSION: The association between chronic hypertension and abruption is strong, and differs based on fetal growth status. The absence of an association among chronic hypertension, placental abruption and fetal growth at very preterm gestations suggests that such pregnancies may have been delivered early.

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554 PROTEOMIC ANALYSIS OF THE AMNIOTIC FLUID (AF) PREDICTS FUNISTIS AND EARLY-ONSET NEONATAL SEPSIS CATALIN S. BUHIMSCHI¹, IRINA A. BUHIMSCHI¹, SONYA S. ABDEL-RAZED¹, VICTOR A. ROSENBERG¹, STEPHEN F. THUNG¹, GUOMAO ZHAO¹, ERICA WANG¹, VINEET BHANDARI², ¹Yale University, Ob.Gyn&Reprod. Sci, New Haven, Connecticut, ²Yale University, Pediatrics, New Haven, Connecticut

OBJECTIVE: Funistis and early-onset neonatal sepsis (EONS) are risk factors for neonatal mortality and poor neurodevelopmental impairment. Proteomics enables early recognition and targeted treatment of the appropriate candidates. Our purpose was to examine the relationship between 4 AF proteomic biomarkers (neutrophil defensins 1 and 2, calgranulins C and A)

characteristic of intra-amniotic inflammation, histological funistis and EONS in premature neonates.

STUDY DESIGN: The relationship between a proteomic fingerprint [Mass restricted (MR) score] generated from fresh AF, funistis and early-onset neonatal sepsis was examined prospectively in 123 consecutive women in preterm labor. The MR score ranged from 0-4 (none to all biomarkers present). Funistis was graded histologically. All neonates (n=97, GA at birth > 23 wks) were evaluated for EONS.

RESULTS: 1) Severity of AF inflammation correlated with the presence (r=0.50, p<0.001) and grades of funistis (r=0.52, p<0.001) (Table); 2) Funistis occurred independent of amniocentesis-to-delivery interval, membrane status, AF WBC count and was best predicted by the combination of an MR score 3-4 (OR: 9.3 [95% CI: 3.9-22.3], p<0.001) and an earlier GA at delivery (OR: 0.9 [95% CI: 0.8-1.0], p=0.039); 3) Neonates of women with MR scores 3-4 had increased incidence of EONS (OR: 4.0 [95% CI: 1.5-11.1], p=0.007) compared to those of women with MR scores 0 or 1-2, even after adjusting for GA at birth; 4) Calgranulin C had the highest association with funistis (OR: 16.7 [95% CI: 5.9-47.2], p<0.001); 5) Calgranulin A had the strongest association with EONS (OR: 4.8 [95% CI: 1.7-13.2], p=0.002).

MR score	Funistis Grades			
	Grade 0 n (%)	Grade 1 n (%)	Grade 2 n (%)	Grade 3 n (%)
MR = 0	24 (20)	1 (1)	1 (1)	2 (2)
MR = 1-2	34 (28)	4 (3)	3 (2)	1 (1)
MR = 3-4	12 (10)	7 (6)	10 (8)	17 (14)

CONCLUSION: Proteomic analysis of AF predicts histological funistis and identifies neonates at risk for EONS in utero.

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555 PRENATAL ALCOHOL EXPOSURE AND PUBERTY ONSET CATHERINE YURK¹, ROBERT SOKOL², LISA CHIODO¹, JOHN HANNIGAN², MARK GREENWALD³, JAMES JANISSE⁴, JOEL AGER⁴, CHANDICE COVINGTON⁵, VIRGINIA DELANEY-BLACK¹, ¹Wayne State University, Pediatrics, Detroit, Michigan, ²Wayne State University, Obstetrics/Gynecology, Detroit, Michigan, ³Wayne State University, Psychiatry and Behavioral Neurosciences, Detroit, Michigan, ⁴Wayne State University, Family Medicine, Detroit, Michigan, ⁵University of North Dakota, College of Nursing, Grand Forks, North Dakota

OBJECTIVE: The age of puberty has dramatically changed over the last hundred years, a developmental acceleration related, perhaps, to improved nutrition and reduced environmental stress. Animal models suggest that prenatal alcohol exposure may contribute to delayed puberty. In one 1979 study, heavy prenatal alcohol exposure was related to a delay in menarche (Robe et al.). Yet, a 1996 IOM report concluded that pubertal development in FAS children did not appear to be affected. The purpose of this research was to examine the relation between prenatal alcohol exposure and puberty onset.

STUDY DESIGN: Maternal alcohol consumption was assessed prospectively during pregnancy for African American gravidas in an urban antenatal clinic. At each prenatal visit, women were interviewed regarding alcohol and drug use during the preceding 2 week period. Exposure level was expressed as oz. of absolute alcohol per day. In addition, questions from three questionnaires re consequences of drinking (MAST, TWEAK & CAGE) were included. Puberty (Tanner Scales and age of 1st menses) was assessed at a 14-yr follow-up visit. Alcohol measures were z-scored and summed to obtain an exposure measure. Data were analyzed using multiple regression, controlling for teen Body Mass Index (BMI) and maternal age of 1st menses.

RESULTS: Data were available for 282 teens (56% girls). After controlling for BMI and maternal age at 1st menses, there was a significant positive relation between teen age at 1st menses and summary alcohol score ($\beta = .20$, p=.05). In addition, average alcohol per day at the 1st prenatal visit was related to puberty total scores in girls ($\beta = -.20$, p=.05) and boys ($\beta = -.19$, p<.05).

CONCLUSION: Prior research examining the relation between prenatal alcohol exposure and puberty onset has been inconsistent, perhaps related to methodological differences and lack of precision in the exposure measure. Here, using prospectively collected prenatal data, measures of both prenatal exposure and maternal alcohol abuse predicted delayed puberty in girls and boys.

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