Conclusion: A standard curve of hCG decline that characterizes sAB has not been previously defined. In our study, the rate of decrease in patients found to have complete resolution of pregnancy without the need for intervention is described by a log linear profile dependent on the initial hCG value. This information may enhance the clinician’s ability to predict outcomes and diagnoses in patients being followed with serial hCG levels. These data may facilitate the development of guidelines for the management of early pregnancies with declining hCG levels.

Wednesday, October 15, 2003
2:15 P.M.

O-229

Objective: To test the hypothesis that high (>20ng/ml) pretreatment plasma folic acid level significantly increases treatment failure of single-dose methotrexate for the management of ectopic pregnancy.

Study Design: A prospective study of 22 patients with ectopic pregnancy and measured pretreatment folic acid level. Patients were divided into two groups based on pretreatment plasma folic acid level (below or above 20.7ng/ml). All patients were candidates for single-dose methotrexate treatment and were followed to assess treatment success. Variables analyzed included: size of ectopic mass, presence of fetal heart tone and clinical outcomes.

Results: Twelve patients had plasma folic acid level below and ten above 20.7ng/ml. The two groups were similar in initial hCG level, size of the ectopic mass, presence of fetal heart tone and clinical outcomes. The failure rate was significantly higher in the group with pretreatment plasma folic acid level above 20.7ng/ml compared to the group below (40% vs. 0%, p = 0.02). In addition, significantly more patients required a second dose of MTX in the group with folic acid level above 20.7ng/ml compared to the group below (50% vs. 8.3%, p = 0.04).

Conclusion: High pretreatment folic acid level increases the risk for treatment failure with single-dose methotrexate.

Wednesday, October 15, 2003
2:30 P.M.

O-230
Trophoblast inclusions are rare in elective terminations and normal deliveries, but common in cases with karyotypic abnormalities. Harvey J. Kliman, Juliette C. McSweet, AnnMarie Franco, Xiaoyan Ying, Yulian Zhao, Gail Stetten. Yale Univ, New Haven, CT; Johns Hopkins, Baltimore, MD.

Objective: Defects in the genes that regulate developmental processes lead to a wide range of embryonic, fetal and neonatal defects, from minor cosmetic abnormalities, to disasters that terminate pregnancies within a few days to weeks after fertilization. We sought to determine if the presence of defects in placental villous development, specifically trophoblast inclusions (TIs: cross sections of deep invaginations of the villous trophoblast bilayer, Figure), is a marker of genetically abnormal pregnancies.

Design: Multicenter retrospective slide review with comparisons made between normal deliveries, elective terminations and spontaneous abortions.

Materials and Methods: Chorionic villi from 855 elective terminations (TABS), 303 spontaneous losses (SABs) and 820 normal term deliveries were examined microscopically for the presence of TIs (defined as at least one trophoblast inclusion identified microscopically in the available material). The failure rate was significantly more patients required a second dose of MTX in the Ontario Uterine Fibroid Embolization Trial involving the practices of eleven interventional radiologists at eight Ontario hospitals.

Results: Comparison of TABs to all SABs revealed that 24/855 TABs had TIs compared to 81/303 SABs with TIs (sens 27%, spec 97%, PPV 77%, NPV 79%). Comparison of these same TABs to karyotypically characterized SABs revealed that 32/48 karyotypically abnormal SABs had TIs (sens 67%, spec 97%, PPV 57%, NPV 98%). Comparison of normal term deliveries to karyotypically characterized SABs revealed that 21/820 term placentas had TIs compared to 32/48 karyotypically abnormal SABs with TIs (sens 67%, spec 97%, PPV 60%, NPV 98%). When only SABs were compared, 1/13 SABs with normal karyotypes had TIs compared to 32/48 karyotypically abnormal SABs with TIs (sens 67%, spec 97%, PPV 97%, NPV 43%).

Conclusions: We have observed that trophoblast inclusions are commonly found in the placentas of fetuses with known chromosomal abnormalities, are more commonly found in SABs compared to TABs, and are rare in normal term placentas. Abnormalities that affect the regulation of such basic cell processes as proliferation, cell movement and fusion are likely reflected in abnormal placental growth patterns, which can give rise to trophoblast inclusions. Since the fetus and placenta share the same genome (except in rare cases of confined placental mosaicism), the presence of trophoblast inclusions may serve as a marker for fetal genetic abnormalities.