**274 MATERNAL SERUM DYSLIPIDEMIA OCCURS EARLY IN PREGNANCY IN WOMEN WITH MILD BUT NOT SEVERE PREECLAMPSIA**

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**OBJECTIVE:** Pregnancy is an atherogenic state, and these changes occur early in pregnancy. Coupled with preeclampsia, this increases the risk for atherosclerosis later in life. Our goal was to compare maternal serum lipids in the second trimester among normal, mild, and severely preeclamptic women.

**STUDY DESIGN:** A case control study of 50 preeclamptic women (mild=26; severe=24) and 101 normotensive women with uncomplicated term deliveries was conducted. Demographic and medical data were chart abstracted. Preeclamptic women were categorized as mild or severe by ACOG criteria. Banked maternal serum collected for quad screening was accessed to measure lipid profiles. Mean lipid levels were compared between groups using student's t-test.

**RESULTS:** Women with preeclampsia were similar in age and parity, equally likely to be Caucasian, and were heavier in weight than controls. Women with mild preeclampsia had significantly higher triglycerides and a higher total cholesterol to HDL ratio than controls (199.9 vs. 164.1 mg/dL, $p=0.02$ and 3.31 vs. 2.91, $p=0.02$, respectively). Women with severe preeclampsia had significantly lower LDL and non-LDL cholesterol than controls (85.5 vs. 101.5 mg/dL, $p=0.04$ and 116.0 vs. 134.3 mg/dL, $p=0.03$, respectively). Women with severe preeclampsia had 13%, 27%, and 22% lower concentrations of total cholesterol, triglycerides, and non-LDL cholesterol than mild preeclamptics ($p<0.05$). The ratio of total cholesterol to HDL was also 21% lower in the severe preeclamptics ($p=0.02$).

**CONCLUSION:** Second trimester maternal dyslipidemia is a risk factor for developing mild but not severe preeclampsia. Women with severe preeclampsia have a less atherogenic lipid profile in the second trimester than both normotensive and mildly preeclamptic women. These findings may be significant in elucidating the different pathologic processes between mild and severe preeclampsia as well as risk for atherosclerosis later in life.

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**275 FURTHER EVIDENCE THAT TROPHOBLAST DERIVED MICROPARTICLES CAUSE ENDOTHELIAL ACTIVATION—A ROLE IN THE PATHOGENESIS OF PREECLAMPSIA**

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**OBJECTIVE:** Preeclampsia is characterized by damage to the maternal endothelium. It has been postulated that this damage is mediated by increased shedding of placental microparticles into the maternal circulation. We previously reported that trophoblast derived microparticles secrete Tissue Factor (TF), and we postulated that this TF in normal conditions may serve to allow the invading blastocyst to implant without causing hemorrhage at the implantation site. Follow up studies show a significant increase in TF identified in microvesicles isolated from first trimester trophoblast exposed to sera of preeclamptic patients. These findings suggest that a potential mechanism by which increased shedding of placental microvesicles mediates damage to the maternal endothelium in preeclampsia may be via Tissue Factor.

The aim of this study was to further investigate the role of trophoblast derived microparticles on endothelial activation.

**STUDY DESIGN:** First trimester trophoblast cells were cultured and grown to 80% confluency. The supernatants from the cell cultures were collected and microvesicles were isolated using an ultracentrifugation technique. P selectin and Flt 1 was identified using Flow cytometery. Simultaneously, endothelial cells (BMEC) were cultured and grown to 80% confluency in the presence of previously prepared trophoblast microvesicles. Tissue Factor and ICAM levels from the BMEC supernatants were determined by ELISA.

**RESULTS:** P selectin and Flt 1 was identified in microvesicles isolated from first trimester trophoblast. A statistical significant increase of TF and ICAM was identified in BMEC cells exposed to the trophoblast derived microparticles (9ng/ml; 8.5 ng/ml, respectively) compared to BMEC cells alone (3ng/ml; 6ng/ml).

**CONCLUSION:** These findings suggest that trophoblast derived microparticles not only express TF but also P selectin and Flt −1, which are known markers of endothelial activation. Moreover, these trophoblast derived microparticles appear to be biologically active as they are able to up regulate modulators of endothelial cell activation.

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**276 CEREBRAL HEMODYNAMIC ALTERATIONS AFTER LASER SURGERY FOR TWIN-TWIN TRANSFUSION SYNDROME**

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**OBJECTIVE:** We hypothesized that laser surgery for twin-twin transfusion syndrome (TTTS) would result in fetal hemodynamic alterations reflected by changes in cerebral blood flow velocities. The study objective was to compare the middle cerebral artery (MCA) pulsatility index (PI) before and after laser surgery for TTTS.

**STUDY DESIGN:** A prospective observational study of TTTS patients was conducted. MCA Doppler examination was attempted within 24 hours before and after laser surgery for TTTS. Patients were excluded from analysis if MCA PI Doppler measurements were unavailable. The mean (standard deviation) pre- and postoperative MCA PI's of the recipient and donor fetuses were compared. Demographic and outcome data were analyzed in relation to the MCA PI's. Student t-tests were used for analysis.

**RESULTS:** 99 patients underwent laser surgery for TTTS during the study period, of which 61 had pre- and postoperative MCA Doppler data on both fetuses, 27 did not have all data recorded, and 11 had postoperative demise precluding Doppler analysis. The mean MCA PI for the recipients increased from 1.35 (0.38) preoperatively to 1.86 (0.86) postoperatively ($p<0.001$), while the mean MCA PI for the donors decreased from 1.67 (0.65) to 1.48 (0.42, $p=0.04$). Preoperatively, the recipients had a lower mean MCA PI compared to the donors ($p=0.001$), while postoperatively the recipients had a higher mean MCA PI compared to the donors ($p=0.003$). No significant correlations were identified between the MCA PI's and demographic factors (Quintero Stage, donor growth restriction) or outcomes (gestational age at delivery, survival).

**CONCLUSION:** Laser surgery for TTTS resulted in increased recipient and decreased donor MCA pulsatility. These opposing alterations in cerebral blood flow velocities may, in part, be attributable to a decrease in fetal arterial blood pressure and/or systemic and cerebral blood flow in the recipient and a corresponding increase in these parameters in the donor.

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**277 HOW WILL INCREASES IN CESAREAN RATES AFFECT THE INCIDENCE OF PLACENTA PREVIA, PLACENTA ACCRETA, AND MATERNAL DEATH IN FUTURE YEARS?**

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**OBJECTIVE:** To forecast the effect of rising primary and secondary cesarean rates on annual incidence of placenta previa, placenta accreta, and maternal mortality in future years.

**STUDY DESIGN:** A model was built using TreeAge Pro software to estimate the annual incidence of placenta previa, placenta accreta, and maternal mortality using existing data on national birthing order trends and cesarean and VBAC rates in recent years. Baseline assumptions were derived from the literature, including the likelihood of previa and accreta among women with multiple previous cesarean deliveries, and the likelihood of maternal mortality and cesarean hysterectomy in the setting of a previa or accreta. A 2-year inter-pregnancy interval and an annual birth rate of 4,000,000 were used in the model's creation.

The model was calibrated to 2004 data and used to forecast previas, accretas, and maternal mortality in 2010, 2015, and 2020 under scenarios in which the cesarean rate increases further, remains stable, and decreases.

**RESULTS:** If primary and secondary cesarean rates continue to rise as they have in recent years, by 2020 the cesarean delivery rate will be 46.2% and there will be an additional 3728 placenta previas, 2524 placenta accretas, and 52 maternal deaths annually. Furthermore, the rise in these complications will lag behind the rise in cesareans by approximately 6 years.

If primary cesarean and VBAC rates remain unchanged from 2004 onward, the rate of previas, accretas, and maternal mortality will reach a steady state by the year 2010.

**CONCLUSION:** If primary and secondary cesarean rates continue to increase at their current rate, the annual incidence of placenta previa, placenta accreta, and maternal death will also rise substantially in the future. The full effect of increasing cesarean rates on these complications is seen approximately 6 years later.

Increases in placenta previa, placenta accreta, and maternal death accompanying a rising cesarean rate

| | 2004 | 2010 | 2015 | 2020 |
|---|---|---|---|---|
| **Previas** | 12308 | 13860 | 14968 | 16036 |
| **Accretas** | 3468 | 4540 | 5280 | 5992 |
| **Deaths** | 404 | 424 | 440 | 456 |
| **Cesareans** | 1196000 | 1460000 | 1659600 | 1846800 |

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