259 POLYUNSATURATED FATTY ACID PROFILES IN THE RED BLOOD CELLS OF PREECLAMPTIC AND NORMAL PREGNANT PATIENTS

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OBJECTIVE: The importance of optimal levels of polyunsaturated fatty acid (PUFA) subsets such as omega-3, omega-6, and omega-9 fatty acids in various diseases has become clear. This descriptive, comparative study evaluates comprehensive PUFA profiles in the red blood cells (RBCs) of patients with preeclampsia (PE) compared to normal pregnant women. RBC profiles reflect long term PUFA status.

STUDY DESIGN: RBC samples from 93 pregnant women enrolled in a preeclampsia study were tested for fatty acid content using gas chromatography. Independent measurements of 33 different fatty acids in each patient were used to discriminate between control (n=58) and PE patients (n=35). A Bonferroni adjustment on alpha=0.05 was used and ROC curves were calculated. Differences between patients with mild and severe preeclampsia were also assessed.

RESULTS: Higher levels of 20:1n9 (AUC=0.77, $p<0.0001$) and lower levels of 20:3n3 (AUC=0.68, p=0.0004) were detected in PE patients compared to controls. An expression combining these two measurements further improved discrimination (AUC=0.84, $p<0.0001$). A subset of six omega-3 and omega-6 fatty acids was further evaluated in patients with mild (n=18) and severe (n=17) PE. Low levels of arachidonic acid, 20:4n6 (AUC=0.63, p=0.10) and docosapentaenoic acid, 22:5n3 (AUC=0.62, p=0.11) distinguished severe PE patients from those with mild disease.

CONCLUSION: This study showed that 20:1n9 and 20:3n3, two intermediary PUFAs, were highly discriminatory for PE. In patients with PE the omega-9 fat was higher and the omega-3 fat was lower. Further analysis of this clear difference is planned. Arachidonic acid was also lower in the RBCs of women with severe PE, suggesting depletion due to oxidative stress and/or metabolism into inflammatory mediators. Lower levels of the omega-3 fatty acid 22:5n3 in patients with severe PE also fits with some theories of etiology. While these latter two findings are not statistically significant, they suggest a relationship that may prove significant with a larger sample size.

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260 UTERINE ELECTROMYOGRAPHY BEFORE AND AFTER ADMINISTRATION OF EPIDURAL ANESTHESIA

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OBJECTIVE: Controversy exists regarding the effect of epidural anesthesia on progress of labor. We sought to determine if epidural anesthesia affects electrical uterine activity during active labor.

STUDY DESIGN: In a prospective double-blind study of women at term with normal labor (Friedman) curves, epidural anesthesia was placed during the active phase of labor. Electrial uterine activity and cervical characteristics were measured at least 30 minutes before administration and within one hour after completion of the epidural anesthesia. Data were recorded and processed by means of a uterine contractility algorithm. The system is comprised of a multi-channel surface electromyogram (EMG) operative that senses electromyographic activity, a three-dimensional position sensor, and a personal computer providing data analysis and a graphical user interface. The data were sent offsite to an individual blinded to the clinical data for analysis.

RESULTS: Fifteen women with low risk pregnancies participated in the study. The study revealed: (1) cervical dilation prior to administration of the epidural was 4.0 + 0.3 cm, whereas the second evaluation within one hour after placement of epidural was 6.0 + 0.6 cm. The difference between the first and second evaluations was significant, p=0.001. The study also revealed: (2) comparable measurements of electrical uterine activity between the initial and second evaluation (467±89 vs. 506±123) and (3) a positive association was found between the pre and post of electrical uterine activity measurements (r=0.47, p=0.05)

CONCLUSION: Our data suggests that epidural anesthesia during the active phase of labor does not affect the electrical uterine activity.

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261 TOCOLYTIC THERAPY IN PRETERM PREMATURE RUPTURE OF MEMBRANES

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OBJECTIVE: To determine if tocolytic administration will extend the latency period from preterm premature rupture of membranes (PPROM) to delivery.

STUDY DESIGN: Prospective, randomized, controlled trial

RESULTS: Sixty-nine patients were enrolled and randomized to the three study groups: 22 patient in Group A (tocolysis with MgSO4 until delivery), 26 patients in Group B (tocolysis with MgSO4 for 48 hours only), and 21 patients in Group C (no tocolysis). All three groups were similar in regard to age, race, and estimated gestational age at the time of enrollment. The latency period was found to be significantly different between the treatment groups, with 16.1 days in Group A, 14.4 days in Group B, and 7.2 days in Group C ($p = 0.002$). However, despite a significant difference in latency between the groups, there were no significant differences in maternal endometritis ($p = 0.931$) or neonatal outcomes (RDS: $p = 0.989$, days on the ventilator: $p = 0.763$, IVH: $p = 0.235$, sepsis: $p = 0.476$, NICU days: $p = 0.747$).

CONCLUSION: The utilization of MgSO4 tocolysis in patients with preterm premature rupture of membranes increases the latency period between rupture of membranes and delivery. In this study, MgSO4 for the 48 hours after admission during which antenatal corticosteroids were administered doubled the latency period when compared to the group receiving no tocolytics (14.4 days and 7.2 days, respectively), and prolonged usage of MgSO4 prolonged latency further. However, despite the significant difference in prolongation of latency, maternal and neonatal outcomes were not shown to be significantly different. A larger study may be necessary to demonstrate significant differences in these other outcomes.

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262 MOLECULAR SIGNATURES OF PREECLAMPSIA IN HUMAN TERM PLACENTAS

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OBJECTIVE: To generate genome-wide gene expression profiles of human placentas from normal, growth-restricted, and preeclamptic term pregnancies; to identify molecular signatures of these conditions.

STUDY DESIGN: Eighty-nine human placentas from normal (43), growth-restricted (27), and preeclamptic (19) term pregnancies were profiled using Illumina Human-6 Beadarrays interrogating a total of 48,701 probes across the human genome.

RESULTS: At a conservative statistical threshold (Bonferonni corrected $p < 0.05$), we identified a set of 90 differentially expressed genes between control and preeclamptic placentas as candidates for a molecular signature of the fetal contribution to this condition. Functional analysis of this molecular signature found significant enrichment for proteins associated with the cell membrane, signal transduction (in particular the Ras Homology A, RhoA associated signaling pathway), and nucleotide excision repair (a potential mechanism for active DNA demethylation). Significant enrichment was also observed for differentially expressed genes in sickle cell patients suggesting an overlapping molecular basis for these different vascular disorders.

CONCLUSION: The molecular signature identified here may serve as the basis for understanding the underlying etiology of this disorder, as well as developing the tools for early-term clinical diagnosis and intervention. DNA methylation states are known to affect gene expression; active DNA demethylation has been proposed to occur via a base excision repair pathway, although a specific DNA demethylase has yet to be identified. The observed enrichment of proteins in the nucleotide excision repair pathway suggests that perturbations in the epigenetic state may contribute to the onset of preeclampsia.

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