

and prostaglandins (PGs) (metabolites of AA) in terms of PGV proliferation.

Design: Effects of phospholipase A2 (PLA2) inhibitor, PLC inhibitor, phosphatidic acid (PA) phosphohydrolase inhibitor, DAG lipase inhibitor, cyclooxygenase inhibitor with or without AA and PGs on the PGV proliferation.

Materials and Methods: Porcine GCs from medium-size follicles were infected by SV40, then cloned and cultured for more than 9 years. PGV were cultured in flasks with TCM199 containing 10% fetal bovine serum. After trypsin digestion, PGV (1×10^4 /well) were inoculated to 96 wells. After 2 days of culture, various doses (10^{-12} ~ 10^{-6} M) of PLA2 inhibitor (bromophenacyl bromide, BPB), PA phosphohydrolase inhibitor (propranolol, PPL), PLC inhibitor (U73122, U), DAG lipase inhibitor (RHC80267, RHC), cyclooxygenase inhibitor (indomethacin) with or without 10^{-12} ~ 10^{-6} of AA and PGs (PGE1, E2, F2 α , I2 and D2) were added to PGV with TCM199 containing 10% horse serum. After 2 days of culture with reagents, cell numbers were counted using absorptiometer with XTT.

Results: BPB, PPL, U, RHC and indomethacin decreased PGV cell numbers dose-dependently. AA reversed the inhibition of PGV cell replication by BPB, U, PPL, and RHC. PGE1, E2, and F2 α effectively increased PGV cell numbers to control levels when added with indomethacin. PG12 and D2 had no effects.

Conclusions: These results confirm the central role of DAG in the proliferation of PGV and suggest that DAG is synthesized not only through the phospholipase C pathway from phosphatidyl inositol but also through the PLD pathway from other phospholipids. AA is synthesized from phospholipids through PLA2 and also from DAG. Our data also suggest that, besides PKC, AA (DAG metabolite) and PGs (AA metabolites) play an important role in the PGV proliferation. The mechanisms of these reagents must be studied further.

O-075

Angiogenin: A New Factor in the Pathophysiology of Ovarian Hyperstimulation Syndrome (OHSS).

^{1,2}M. A. Aboulghar, ¹R. T. Mansour, ¹G. I. Serour, ²M. Shaarawy, ^{1,2}A. Ramzi, ¹B. E. Helw, ¹Y. Amin. ¹The Egyptian IVF-ET Center, Maadi, Cairo, and ²The Department of Obstetrics and Gynecology, Faculty of Medicine, Cairo University, Cairo, Egypt.

Objectives: Angiogenin is a non-glycosylated peptide known for its ability to induce neovascularization and angiogenesis. The aim was to assess the potential involvement of angiogenin in the pathophysiology of (OHSS) and the hyperpermeability leading to accumulation of fluid in the third space.

Design: A controlled clinical study which compared angiogenin level in the serum of patients with severe OHSS with serum of a control group. Angiogenin in the ascitic fluid of severe OHSS was also compared with angiogenin of peritoneal fluid aspirated just before ovum pickup from a control group of patients undergoing IVF. An age matched group of patients undergoing ovarian superstimulation for IVF constituted the control group.

Material and methods: Ten patients with severe OHSS were admitted to the hospital. Diagnosis was based on ovarian enlargement more than 10 cm in diameter with massive ascites and abdominal pain and dyspnea. Blood samples were obtained from each patient upon admission. Ascitic fluid was obtained from all OHSS patients during our routine transvaginal aspiration of the fluid as a treatment modality. In the control group, peritoneal fluid was aspirated from Douglas Pouch before introducing the needle in the ovary for ovum pickup and blood sample was collected one week later. All serum samples were assayed for angiogenin, E2 and hematocrite. Peritoneal fluid samples were assayed for angiogenin only. Angiogenin levels were assayed by the quantitative sandwich enzyme immunoassay technique. E2 was determined by RIA.

Results: The mean serum level of angiogenin in OHSS was 8390 ± 6837 ng/ml as compared to 234 ± 91 ng/ml in the control group, $P < 0.001$. The mean ascitic fluid angiogenin was 2794 ± 1024 ng/ml in OHSS as compared to 254 ± 105 ng/ml in the control group $P < 0.001$. The serum level ranged from 2400 to 20000 ng/ml in OHSS and 80 to 400 in the control. The ascitic fluid level ranged from 1240 to 5000 ng/ml in OHSS, and 120 to 480 ng/ml in the control group. The mean E2 level in OHSS was 6300 ± 2450 ng/ml as compared to 1850 ± 1100 ng/ml in the control group and the hematocrite was 46.4 ± 4.4 as compared to 36.8 ± 4.6 in the control group. The difference was statistically highly significant.

Conclusion: Currently, factors mediating vascular hyperpermeability in OHSS remain undefined. The data presented however, suggest that angiogenin may be a candidate in this respect. High plasma and ascitic fluid angiogenin were recorded in all patients accompanied by clinical features of capillary leakage and hemoconcentration. This is the first report in the world literature to suggest involvement of angiogenin in the pathophysiology of OHSS.

O-076

Video Mapping to Assess Efficacy of an Antiestrogen (Raloxifene) on Spontaneous Endometriosis in the Rhesus Monkey.

¹P. M. Fanning, ¹T. J. Kuehl, ²R. Lee, ³S. L. Pearson, ¹T. J. Wincek, ¹J. F. Pliego, ¹A. M. Spiekerman, ⁴H. U. Bryant, ⁴M. K. Rippey. ¹Depts of Ob/Gyn, Pathology, and Medical Biochemistry & Genetics, Scott & White Clinic, Texas A&M University Health Science Center College of Medicine, Temple, TX, ²MD Anderson Cancer Center, The University of Texas Health Science Center, Bastrop, TX, ³HRP, Inc., Alice, TX, ⁴Eli Lilly and Company, Indianapolis, IN.

Objective: This study tests the hypothesis that the extent and severity of spontaneous endometriosis in rhesus monkeys are altered by treatment with an antiestrogenic agent, raloxifene, when compared with either a baseline evaluation or treatment for a comparable interval with placebo. Secondary objectives included examining the effects of raloxifene on uterine size measured by ultrasound, frequency of menses, ovarian ovulatory structures visible at laparoscopy, and circulating levels of estradiol and progesterone.