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P1-IS-99 Sperm Motility Exsposed To Pentoxifylline On Peritoneal Fluid On Endometriosis Patient In Vitro

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Objective: comparing the sperm motility exposed by pentoxifylline on peritoneal fluid of endometriosis patient with peritoneal fluid of endometriosis patient without pentoxifylline exposure.

Materials and methods: This is an randomized clinical trial study. There were two samples of peritoneal fluid taken by laparoscopic procedure on endometriosis patients grade 1 and grade 2, and one sample of peritoneal fluid taken from women suffered from endometriosis grade 4 taken by minilaparotomy.

Results: The mean of sperm motility exposed to pentoxifylline $0.75\,\mathrm{mg/mL}$ on endometriosis grade 1.3 and 4 higher than the sperm motility without pentoxifylline. Almost all of samples show significant differences, only on endometriosis grade 1 incubated on 24th hours with no significant differences (p=0.32) and endometriosis grade 4 incubated on 24th hours, because there were no motile sperm. The exposure of pentoxifylline only significant on the sperm that incubated with peritoneal fluid of endometriosis patient with grade 1 in the 0 minute $\{1.3 \ (1.07-1.59)\}$ and the $\{0.25-2.56\}$ and on endometriosis grade $\{0.25-2.56\}$ and on endometriosis grade $\{0.273 \ (1.7-4.361)\}$.

Conclusion: generally, the mean of sperm motility is better on the sperm exposed to pentoxifylline compared to the sperm that wasn't exposed. The sperm motility didn't decrease with the lower grading of endometriosis the sperm motility has the special pattern on each grade of endometriosis.

P1-IS-100 Correlation between estrogen and serum adipocytokine in premenopause and postmenopause women

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Objective: Our purpose was to investigate the association between serum adipocytokines and endogenous estrogen levels in healthy premenopausal and postmenopausal women. Material & methods: We selected 53 healthy premenopausal women and 45 healthy postmenopausal women from general health examinations. Analysis was performed about adiponectin, resistin, leptin, TNFα, estrone (E1) and estradiol (E2). Results: When accounting for BMI in three groups, adiponectin and resistin levels did not change significantly in healthy postmenopausal women compared with healthy premenopausal women. Whereas leptin was decreased $(7.46 \pm 0.57 \text{ng/mL} \text{ vs } 5.07 \pm 0.57 \text{ng/mL})$, TNF α was increased significantly $(1.48 \pm 0.12 \text{ pg/mL vs } 2.02 \pm 0.12 \text{ pg/mL p} < 0.05)$ in healthy postmenopausal women compared with healthy premenopausal women. Adiponectin correlated negatively with insulin resistance measured as HOMA-IR (r = -0.325, p < 0.01) and leptin correlated positively with IR (r = 0.463, p < 0.01). When accounting for BMI, estrogen (E1, E2) only correlated with leptin positively in healthy premenopausal women, whereas estrogen did not correlate with any adipocytokine in healthy postmenopausal women. In the healthy premenopausal and postmenopausal women, E1 correlated negatively with adiponectin (r = -0.238, p<0.05) and with TNF α (r = -0.223, p<0.05) and positively with leptin (r = 0.352, p<0.01); and did not correlate with resistin. E2 correlated negatively with TNF α (r = -0.241, p<0.05) and positively with leptin (r = 0.336, p<0.01); and did not correlate with adiponectin, resistin. Conclusion: Estrogen deficiency resulted in increased serum inflammatory cytokine levels-TNFa. Serum resistin was not regulated by estrogen and serum leptin may stimulate the production of estrogen through the hypothalamic receptor in premenopausal women. In addition, BMI increase cause leptin to be increased in postmenopausal women.