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EMPIRIC ANTIBIOTIC TREATMENT OF SUSPECTED CHRONIC ENDOMETRITIS DOES NOT IMPROVE SUSTAINED IMPLANTATION RATES FOLLOWING FAILED EUPLOID FROZEN EMBRYO TRANSFER (FET).

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BACKGROUND: Chronic endometritis (CE) is associated with lower implantation rates in patients with recurrent implantation failure[1]. Some studies have demonstrated an improvement in pregnancy outcomes following antibiotic treatment for CE. Diagnosis of CE requires a procedure, typically an endometrial biopsy or dilation and curettage, in order to obtain a histologic specimen[2]. Some clinicians have moved towards empiric antibiotic treatment of suspected chronic endometritis for patients with a prior implantation failure.

OBJECTIVE: This study seeks to determine whether empiric antibiotic treatment of suspected chronic endometritis improves pregnancy outcomes following failed FET.

MATERIAL AND METHODS: All patients with one prior failed euploid FET (2011-2016) followed by a subsequent euploid FET were reviewed for inclusion. Patients without a documented histologic diagnosis of chronic endometritis were identified and divided into two groups. Group A consisted of patients treated with empiric antibiotics for suspected chronic endometritis throughout their subsequent FET cycle. Group B consisted of patients who were not given empiric antibiotics during their subsequent FET cycle. All patients underwent uterine cavity assessment with a saline sonogram prior to their second FET. Oocyte age, body mass index (BMI) and endometrial thickness were compared between the two groups using Student's t test. Sustained implantation rate and loss rate were compared between the two groups using Pearson's chi squared test. Significance was determined at a p-value of 0.05.

RESULTS: A total of 651 patients met inclusion criteria – Group A consisted of 67 (10.3%) patients who were treated empirically for suspected chronic endometritis during their second FET cycle and Group B consisted of 584 (89.7%) patients who were not treated. There was no difference in oocyte age, BMI or endometrial thickness between the two groups. There was no difference in sustained implantation rates (41.8% versus 50.0%, $P = 0.26$) or loss rates (17.9% versus 21.1%, $P = 0.55$) between the two groups.

CONCLUSIONS: Empiric antibiotic treatment of suspected chronic endometritis does not improve pregnancy outcomes for patients with a prior failed euploid transfer.

FINANCIAL SUPPORT: None

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ZINC: AN ESSENTIAL METAL FOR MAINTENANCE OF FEMALE FERTILITY.

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BACKGROUND: Zinc is one of the most abundant transition metals and plays an essential part in cells, including oocytes. It has a variety of roles such as catalytic, structural, and regulatory functions in every cell. Its deficiency is associated with over production of reactive oxygen species (ROS). These roles are firmly controlled and alteration from normal levels can be potentially toxic to cell function. For example, in oocytes, zinc accumulates during oocyte growth and is stored in lipoproteins for embryonic development. It can also play a vital role in the completion of meiosis I in oocytes for maturation thereby leading to ovulation failure if not present in sufficient quantities. However, intracellular mechanisms regulated by zinc, pertaining to female reproduction and ovarian function remain unclear. We hypothesize that exposure to toxins such as British Anti-Lewisite (BAL) and 2,3-Dimercapto-1-propanesulfonic acid (DMPS), a zinc scavenger, cyclophosphamide (CTX) and acrolein, both chemotherapy drugs, and styrene, a commonly used insulation material, deteriorate oocyte quality through a mechanism that involves zinc deficiency and overproduction of ROS.

OBJECTIVE: We sought to investigate the impact of zinc depletion in mouse oocytes after exposure to increasing concentrations of a series of toxins such as BAL, DMPS, CTX, acrolein and styrene.

MATERIALS AND METHODS: Metaphase II mice oocytes (n=90) were pretreated with increasing concentrations of toxins (25, 50, and 100 uM) that reach their peak plasma levels. In all cases, the oocytes were stained with 25 uM of Zinquin (a zinc sensitive and cell–permeable fluorescent probe) and incubated for 1 hour at 37°C, 5% CO_2 to determine intracellular zinc content. Confocal microscopy was utilized to measure intracellular zinc levels in oocytes exposed to the different concentrations of toxins. Oocytes were examined under confocal microscopy and images using a Zeiss LSM 510 META NLO microscope with emission wavelengths of 480 and 535nm, and 358 and 461 nm, respectively.

RESULTS: A decrease in intracellular zinc was noted as compared to the control as function a of increasing concentration of the various toxins as indicated by decreased zinc-mediated blue fluorescence. Florescent intensity was quantitated in terms of corrected total fluorescence (CTCF) and a statistically significant difference was found between the CTCF of the treated groups compared to control ($p<0.001$). The lowest CTCF was noted for oocytes treated with a zinc scavenger and to a lesser extent to styrene. In all cases exposed oocytes were noted to have an enhancement of ROS production. Collectively, these results demonstrate that the deterioration of oocyte quality is associated with intra-oocyte zinc depletion.

CONCLUSIONS: Exposure to these toxins is associated with deterioration of oocyte quality, zinc depletion and enhancement of ROS thereby leading to subfertility. Collectively, zinc is vital in female reproduction and therefore exposure to such toxins should be avoided in reproductive age women.

FINANCIAL SUPPORT: None

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THE EFFECT OF TOTAL GONADOTROPIN DOSE ON PLACENTAL WEIGHT (PW), BIRTH WEIGHT (BW) AND FETOPLACENTAL RATIO (FPR) IN GONADOTROPIN INDUCTION/INTRAUTERINE INSEMINATION (GN/IUI) CYCLES.

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