MALE FACTOR INFERTILITY IS ASSOCIATED WITH DECREASED PROGRESSION TO CLINICAL PREGNANCY IN EUPLOID EMBRYO TRANSFERS

Devora Aharon, MD¹, Dmitry Gounko, MA², Tamar Alkon, MD, MS, PhD², Joseph A. Lee, BA², Natan Bar-Chama, M.D.², Erkan Buyuk, MD¹ and Alan B Copperman, MD²

1. Icahn School of Medicine at Mount Sinai, New York, NY
2. Reproductive Medicine Associates of New York, New York, NY

OBJECTIVE:
The impact of male infertility on sustained implantation remains mostly unknown, with one prior study demonstrating an association between male infertility and recurrent pregnancy loss. This study aims to evaluate any relationship between male infertility and embryo implantation and subsequent progression to clinical pregnancy.

MATERIALS AND METHODS:
This study included patients undergoing single euploid frozen-thawed embryo transfer (FET) from September 2016-February 2021. ICSI was performed in all cycles. Couples with male factor infertility, defined as less than 15.6 million total motile sperm, were compared to those with normal total motile sperm count. Pregnancy (HCG ≥2.5 IU), clinical pregnancy (gestational sac (GS) visualized on ultrasound), biochemical pregnancy (no GS seen, with declining HCG), and clinical pregnancy loss were compared between the groups. Primary outcome was progression to clinical versus biochemical pregnancy after confirmation of a positive pregnancy. Student’s t test, chi-square test, and multivariable logistic regression were used for analysis.

RESULTS:
A total of 6712 cycles were identified, including 1111 couples (16.6%) with male factor infertility and 5601 couples (83.4%) without male factor. Pregnancy occurred in 848 (76.3%) of those with male factor and 4138 (73.9%) of those without. Biochemical loss occurred in 159 (18.8%) of those with male factor who achieved pregnancy and 663 (16.0%) of those without male factor. Controlling for female partner age, BMI, AMH, and endometrial thickness as well as embryo quality showed that male factor infertility was not associated with likelihood of pregnancy (aOR=1.09, 95% CI 0.94-1.28, p=.23) but was associated with an elevated odds of biochemical pregnancy loss (aOR=1.23, 95% CI 1.01-1.51, p=.04). Once clinical pregnancy was achieved, male factor infertility was not associated with an increased risk of clinical pregnancy loss (aOR=0.92, 95% CI 0.73-1.15, p=.46).

CONCLUSIONS:
Male factor infertility was associated with decreased progression to clinical pregnancy following successful implantation of a euploid embryo, but once clinical pregnancy was established had no impact on miscarriage.

IMPACT STATEMENT:
Low sperm motility may reflect an underlying defect in spermatogenesis, perhaps related to DNA methylation or other epigenetic changes, that impacts embryo growth and development despite successful initiating of implantation. Further investigation into the downstream effects of male infertility beyond fertilization will confirm and elucidate the pathophysiology of these findings.

REFERENCES: