

Women with Obesity Display Defects in Follicular Recruitment, Selection, and Dominance Alongside Luteal Phase Dysfunction During Natural Ovulatory Cycles

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Although obesity is closely associated with reproductive dysfunction, many women with obesity remain eumenorrheic. The degree to which obesity affects menstrual cycle characteristics in this population is limited to endocrine assessments with few data available on how endocrine aberrations align with defects in antral follicle development. We have previously shown alterations in the distribution of recruitable (2-5mm) versus selectable (6-9mm) follicles in those with obesity that were associated with decreased gonadotropin and progesterone production despite normal menstrual cycle length. The objectives of the present study were to further contrast follicular growth and endocrine dynamics between obese and non-obese eumenorrheic women with the goal of determining any alterations in distinct events in folliculogenesis. 21 non-obese (total percent body fat <35%) and 21 obese (total percent body fat ≥35%) reproductive age females with regular menstrual cycles (21-35d) were evaluated by serial ovarian transvaginal ultrasonography and venipuncture every-other-day for one complete inter-ovulatory interval (IOI). The number and diameters of all follicles ≥2mm at each visit were documented offline using the Grid Method. Growth profiles of individual follicles which grew to >7mm were assessed using the Identity Method to characterize antral follicle recruitment, selection, and ovulation. Clinical, morphologic, and endocrine features were compared across groups using t-tests and mixed models. Women with obesity exhibited fewer recruitment events versus non-obese women (1±0.6 vs. 2±0.9; p=0.010). They also had fewer selectable follicles (4±3 vs. 8±6 follicles/participant, p=0.022) which grew faster but remained static longer and regressed slower (all growth rates, <0.05) compared to their non-obese counterparts. Anti-Mullerian hormone levels were lower in the obese versus non-obese group (4.40±3.01 vs. 5.94±2.49ng/mL, p=0.023). Of the selectable follicles, fewer progressed to >10mm in the obese versus non-obese group (1±0.7 vs. 2±0.8 follicles, p=0.030) with ultimately fewer anovulatory follicles achieving dominance (0±0.7 vs. 1±0.9 follicle, p=0.029). Ovulatory follicles in the obese group were selected at smaller diameters (7.5±1.6 vs. 9.5±1.9mm, p=0.001) versus their non-obese counterparts. However, the kinetics of dominant follicle growth were similar between groups. Post-ovulation, progesterone concentrations across the luteal phase were lower in women with obesity (4.95±3.34 vs. 7.85±5.74ng/mL, p=0.001) and the prevalence of luteal phase defects (LPDs) higher versus non-obese women, defined by either integrated (76 vs. 29%, p=0.002) or maximum (71 vs. 24%, p=0.002) progesterone levels. Eumenorrheic women with obesity display evidence of suppressed follicle dynamics as evidenced by fewer recruitment events, selectable follicles, and anovulatory dominant follicles. LPDs are more prevalent in women with obesity during natural ovulatory cycles which may underlie subfertility in this population. A smaller size at selection suggests premature responsiveness to luteinizing hormone which may result in suboptimal luteinization. Further studies are needed to elucidate the degree to which these defects impact fertility and fecundity in our increasingly obese demographic.

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