ASSOCIATION OF BODY MASS INDEX AND DIETARY HABITS WITH OVARIAN AND UTERINE MORPHOLOGY WITH SUBFERTILE POLYCYSTIC OVARIAN SYNDROME

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ABSTRACT

Objective: To correlate ovarian and uterine morphology in subfertile patients having polycystic ovarian syndrome with their body mass index (BMI) and eating habits

Methodology: One hundred subjects were included in this cross sectional study having ages between 20-40 years with history of subfertilty for the past two years. BMI (kg/m^2) was measured according to WHO protocol. Ovarian volume, follicle count and size, uterine area, endometrial thickness were measured sonographically and noted. Dietary habits were recorded by a structured questionnaire from all participants

Results: According to BMI two groups of 50 subjects each were formed; group I= 25.6 ± 4.7 kg/m^2 and group II= 28.6 ± 5.7 kg/m^2. The follicular count was 14.41± 2.18 and 12.75± 2.80; the follicular size (in mm) was 8.23±0.41 and 3.29±0.25 (p-value 0.023 & 0.001). The uterine area (in cm^2) and endometrial thickness (in cm) was 86.9±25.7 and 117.2±29 (p-value 0.001 and 0.05). The ovarian volume (OV) estimated by trans-abdominal scan (TAS) was 10.87±2.49 cm^3 and 14.33±3.17 cm^3 (p-value=0.022). The trans-vaginal scan (TVS) showed OV; 11.44±2.36 cm^3 and 14.79±2.19 cm^3 (p-value=0.034). A weak positive correlation of BMI with OV (TVS) was observed (r=0.05). The ovarian volume in those women who consumed fast food frequently was significantly raised as compared to those who rarely ate fast food; the difference was 14.57±3.75 vs. 9.62±2.43 (p value 0.00).

Conclusion: With increasing body mass index the ovarian volume, uterine area and endometrial thickness increases. However the follicular count and follicular size is reduced with higher BMI.

Key Words: Body Mass Index (BMI), Dietary habits, Ovarian morphology, Uterine morphology, Polycystic ovarian syndrome

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INTRODUCTION

Couples generally conceive as a result of fertilization of ovum by sperm, within a year after performance of regular unprotected intercourse. The biological inability to contribute to conception after more than 12 months of unprotected intercourse[1] can be referred to as infertility or subfertility. During embryonic intrauterine life the primordial follicle cells pool in the ovaries to be used later in child bearing age, when these follicles fail to mature the oocyte becomes incapable of being fertilized due to its abnormal morphology[2, 3]. One of the leading causes of infertility/subfertility is an endocrine disorder of indefinite cause, PCOs which has prevalence of 20% or even more[4]. It produces symptoms in approximately 5% to 10% in women of reproductive age (12–45 years old) which are oligomenorrhea, amenorrhea, acne, hirsuitism and excessive weight gain. Symptomless cases, on the other hand are diagnosed at the time of evaluation in infertility clinics with problems of ovulation-related infertility.

Obesity being another leading cause of infertility, decreases chance of spontaneous conception in normal ovulatory women by 5%[5] for each unit rise in body mass index (BMI). The epidemic of obesity has stretched out in due course of time especially in women of child bearing age with an incidence of 12% in Western Eu-

rope and 25% in North America[5]. Such issues were seen to be associated with the developed countries but with increasing exposure to fast food this epidemic has also touched the developing countries. The women of the developing countries like Pakistan are also becoming a part of this epidemic. According to World Health Organization (WHO) estimates, 26 % of women and 19% of men in Pakistan are obese (Body Mass index (BMI) > 25) but only 4% of women and 1 % of men are recognized as obese using the standard criteria (BMI > 30). The prevalence of obesity is even higher in urban areas (56% in men and 67% in women) when an Asian specific definition for obesity is used[6]. Increase in BMI causes collective impairment of a number of processes coupled with oocyte production, development, maturation, fertilization, endometrial development, uterine receptivity and implantation necessary for continuation of pregnancy[3]. A poor response to infertility treatment results in greater risk of miscarriages, gestational complications (diabetes and hypertension), neonatal morbidity and high neonatal mortality rates; these are further attributes to increased BMI[7].

It has also been reported that the changes in ovarian and uterine morphology between fertile and infertile women showing that the ovarian volume, follicular count and size decrease significantly in the infertile group of women. The authors have shown a decrease in endometrial thickness in women suffering from infertility with a decrease in uterine size[8]. The PCOs is often associated with obesity, Type 2 diabetes, high cholesterol levels and insulin resistance. Among these, obesity and PCOs show cause-effect relationship which is debatable since obesity has been documented in 40–80% of PCOs women[9]. It has lately been reported that in PCOs patients the effect of the obesity-linked gene is mediated through the excess fat deposited due to imbalance in sex hormone. Insulin resistance, one of the important dysmetabolic factors of PCOs, is closely related to bodyweight. At the same time, obesity does exacerbate many aspects of the phenotype, especially cardiovascular risk factors such as glucose intolerance and dyslipidemia. The detrimental synergic effects in PCOs with obesity thus 9 decreases chances of conception at the level of the ovary as well as on endometrial lining more than lean patients with PCOs. The estimation of BMI appears to be one of the most dependable predictors of PCOs severity as well as reduced fecundity that is often observed in overweight women[7, 9] and both contribute to non-conception cycles. The morbid association of adenocarcinoma of endometrium with PCOs and infertility is reported to be 25% in incidence; these women are also at risk of developing carcinoma of the endometrium[10].

This study was conducted to correlate ovarian and uterine morphology in subfertile patients having PCOs with their BMI and dietary habits.

METHODOLOGY

Hundred subjects between ages 20 to 40 years were recruited from Karsaz Hospital Karachi from January 2011 to March 2012. The type of study was a cross sectional study in which diagnosed cases of PCOs were inducted. The subjects were diagnosed by sign and symptoms and then referred for ultrasound before being a part of the study. Written informed consent was sought. A structured questionnaire was distributed in which they were asked about their eating habits.

The patients were included according to the following criteria, a) primary subfertility with no history of abortion b) ages between 20-40 years c) Presence of 12 or more cystic ovarian follicles d) The woman should have had no conception in the last two years after regular unprotected intercourse e) Not using contraceptives for at least 2 months prior to the study.

The exclusion criteria was a) conception via ART (Abbreviation) b) surgical removal of ovary(ies) c) Any pathology of pelvic reproductive organs other than PCOs d)Husband suffering from infertility e) Any chronic illness e.g. hypertension, diabetes, cancer etc f) irregular menstrual cycle.

The women who were included in the study had history of normal menstrual cycle. Between day 2 to 7 of the menstrual cycle ultrasound was performed using Toshiba ultrasound machine. Transabdominal and transvaginal probes were used; these were of 3.75 MHz and 7.5 MHz frequency respectively. To exclude abnormalities other than PCOs e.g. cancers, adhesion, endometriosis, tubal ligation, absence of ovaries (one or both), fibroids, the transabdominal scan (TAS) was performed on a full urinary bladder. If the subjects had PCOs on TAS then transvaginal scan (TVS) was done which was performed on an empty urinary bladder.

Subjects were weighed on a digital weighing scale in kilogram having an accuracy of ±100g in their normal clothing without shoes. Standing body height (BH) was measured without shoes to the nearest 0.5 cm with the help of height scale (floor type ZT-120 EVERICH, China) with the shoulders in relaxed position and arms hanging freely. BMI was calculated as body weight in kilograms (kg) divided by the square of the body height in meters (m^2).

The following parameters were measured sonographically and noted a) ovarian volume b) follicle count and size (2-9mm) c) uterine area d) endometrial

thickness. The two ovaries were scanned in the longitudinal (D1), anteroposterior (D2) and transverse diameter (D3); the total volume was calculated by applying the ellipsoid equation which is D1XD2XD3X0.523cm^3 and the sum of the two ovaries was noted.

The uterine area was calculated by the formula uterine length X anteroposterior diameter in cm^2. This was performed by measuring the uterine length from the top of the fundus to the cervix and the anteroposterior diameter by TVS.

The endometrial thickness was measured in mm by TVS.

For validation, the readings were taken twice for all variables and their average was calculated which was considered as the final reading.

A total of 159 women clinically diagnosed with signs and symptoms of PCO were initially included. Out of these 59 women were excluded. In this way 100 women of which 50 belonged to Group I (20-30 years) and 50 to Group II (31-40) were finally recruited. The BMI of the two groups was calculated showing group I with BMI of 25.6 ± 4.7 kg/m^2 and group II with BMI of 28.6 ± 5.7 kg/ m^2 (mean ± SD).

SPSS version 15 for windows was used to enter and analyze the measurements; the application used was unpaired t-test. Pearson correlation coefficient "r" was used for associating BMI with ovarian volume. The results were given as mean± standard deviation (SD). P-value of 0.05 or less was considered statistically significant.

All subjects were informed of the research and their consent was sought. The study was approved by the ethical review committee of Bahria University Medical and Dental College.

RESULTS

Subfertile female's age, included in the study ranged from 20-40 years (mean age 28±4.23). Body mass index (BMI) of age 20-30 years was found to be 25.6 ± 4.7 kg/m^2 whereas 28.6 ± 5.7 kg/ m^2 (mean ± SD) was calculated in females with age 31-40 years (p-value 0.04). The results of ovarian and uterine morphology were stratified on the basis of calculated BMI into two groups; group I ; 25.6 ± 4.7 kg/m^2 and group II; 28.6 ± 5.7 kg/ m^2 (Table1) which shows a significant decrease in follicular count and size (p value 0.023 & 0.001 respectively) with an increase in BMI. The uterine area in cm^2 and endometrial thickness in cm increased with an increase in BMI (p value 0.001 and 0.05 respectively).The ovarian volume (OV) estimated by trans abdominal scan was 10.87 ± 2.49 cm^3 and 14.33 ± 3.17 cm^3 in Group I & II respectively (P value 0.022). The trans vaginal scan showed OV; 11.44 ± 2.36 cm^3 and 14.79 ± 2.19 cm^3 in I & II groups (P value=0.034).

Of the 100 women, 63 women who were working females and college going students reported that they ate fast food everyday for lunch, 18 reported that they consumed such items once a week, 10 reported that they ate fast food every 10-15 days whereas 9 reported that they rarely consumed such food. The ovarian volume of those who consumed fast food everyday and once a week were grouped into one and compared with the rest. The ovarian volume of these two were 14.57± 3.75 vs. 9.62±2.43cm^3 (p value 0.00). No significant relation could be determined with uterine morphology.

A positive correlation of BMI with OV (TVS) is given by scatter plot in figure 1.

DISCUSSION

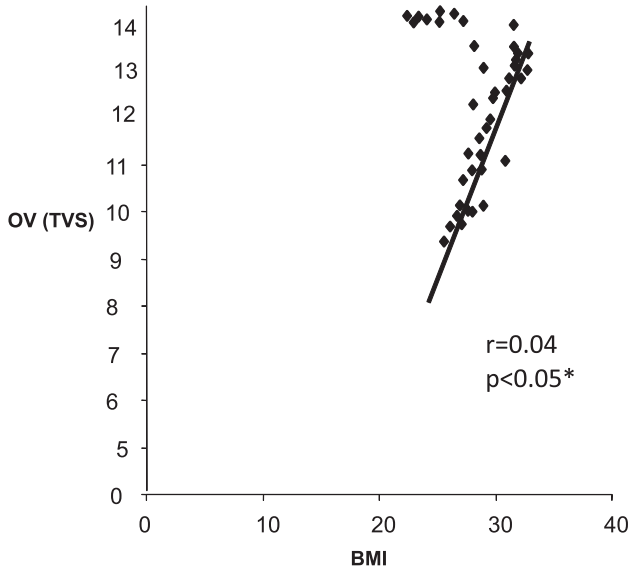
Body mass index BMI has been established as a predictor of fertility in polycystic ovary syndrome PCOS[11] which is among the most common cause ofanovulatory infertility in women[12]. On the other hand fertility status of a female has shown to be correlated with the morphology of pelvic reproductive organs significantly[13]. In light of this, our study attempts to establish an asso-

Table1: Comparison of ovarian morphology of PCO on the basis of their BMI

Subfertile group with PCO (BMI kg/m2)			
	Group I: 25.6 ± 4.7 (n=50)	Group II: 28.6 ± 5.7 (n=50)	**P-value**
FC	14.41 ± 2.18	12.75 ± 2.80	0.023
FS	8.23±0.41	3.29±0.25	0.001
UA	86.9 ± 25.7	117.2 ± 29.0	0.001
ENDO	0.47 ± 0.12	0.57 ± 0.17	0.052

FC= follicle count, FS= follicle size in mm, BMI= body mass index, UA=uterine area in cm2, ENDO= endometrial thickness in cm. Values are expressed as; mean ± S.D

Figure 1: Correlation of BMI with ovarian volume (OV) in subfertile PCOS; TVS=transvaginal scan; BMI= body mass index; r=correlation, *significant p-value



ciation between ovarian and uterine morphology with BMI exclusively in subfertile PCOs females. The effect of fast food culture on the ovarian volume was also determined.

The assessment of both follicular size and count are essential for diagnosing polycystic ovaries which is defined as presence of 2–9 mm size follicles in 12 or more numbers in either ovary and/or a total ovarian volume of 10 cm^3 or greater[14]. In present study it was discovered that the follicular size and count both were significantly decreased with an increase in BMI. Zaidi et al have also reported a significant reduction in follicular count with reduced fertility which has been inversely related with BMI[15]. Other investigators have also shown an inverse relation of BMI with follicle size and count[16].

In this study the ovarian volume was noted to be directly related to BMI. Both follicular and stromal components contribute in establishing ovarian volume[17]. Mean ovarian volume increases from 0.7 cm^3 in pre pubertal age to 5.8 cm^3 at 17 years[18]. In absence of pathology there are no further major changes in ovarian volume during reproductive years until the premenopausal period[18]. Ovarian volume is observed to be significantly higher in PCOS females as compared to control groups[19]. Balen et al showed direct correlation of ovarian volume with body mass index[20].

Endometrial thickness undergoes changes according to different phases of menstrual cycle. At the time of menstruation the endometrium is about 1- 4 mm thick, becomes about 5- 7 mm during proliferative phase and reaches maximum thickness of 7-14 mm at the peak of secretary phase[21]. In present study, it was found that BMI was significantly correlated with endometrial thickness in subfertile PCOs women. Significant agreement is present with regard to the relationship between BMI and endometrial thickness in multiple studies. Douchi et al showed that the frequencies of thicker endometrium increased in relation to body mass index[22]. This study also agrees with multiple other researchers who demonstrated that endometrial thickness is positively correlated with BMI[23-25]. However Tsuda et al. reported no correlation of BMI with endometrial thickness[26].

The normal dimensions of adult nulliparous uterus on ultrasound are observed as 72.8 mm in longitudinal, 42.8 mm transverse, 32 mm anterioposterior axises[27]. Longitudinal and transverse diameters are used to determine the uterine area[28]. The results of present study concur with several other researchers who showed a significant correlation between BMI and uterine size in women independent of age and parity[29,30]. However, a great many conditions can influence the size of uterus like leiomyomas, race and hormone replacement therapy[30].

The association of BMI with PCOs and infertility has been well established[11,12]. By relating BMI with the morphology of pelvic reproductive organs the underlying mechanism for subfertility can be better understood especially in PCOs females who often suffer from obesity. The effect of BMI on endometrial thickness and uterine size can also help in predicting the risk factor for endometrial carcinoma and fibroids[30,31]. A study by Douglas CC et al showed that PCOS is related to consumption of

food. The authors have shown that certain type of food items like fried potatoes and white bread were taken in large amounts by the recruited subjects suffering from PCOS. The relation of PCOs and consumption of fast food was established in our study. Such similar findings have also been reported by other researches[32-34]. Further investigation is needed with larger sample size on morphology of reproductive organs in reference to BMI so that preventive measures can be taken to optimize BMI in order to control many associated diseases.

Limitation of the study was that it does not look into all BMI groups and represent only those BMI which were detected in our study however it highlights the influence of fast food and raised BMI on follicular and endometrial parameters that may lead to subfertility

CONCLUSION

A positive association was shown between ovarian volume and fast food consumption; this shows that with increasing trend of fast food there is increasing obesity in women especially working women and college students which can be a major cause of PCOs and related subfertility.

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CONTRIBUTORS

AU Formed research proposal, conducted research, compiled data and finally wrote the article. RR and ZA helped in manuscript writing and reviewed the article before final submission. All authors contributed significantly to the final manuscript.