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RELATIONSHIP BETWEEN OBESITY AND OVARIAN RESERVE MARKERS IN WOMEN

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ABSTRACT

Background: Obesity has a detrimental impact on most organ systems, including the reproductive system and leads to impairments in ovarian, follicular, and oocyte development, fertilization, and embryo development and implantation. The term "ovarian reserve" refers to the quantity and quality of a woman's current reservoir of oocytes and is closely associated with reproductive potential. It naturally declines with age from puberty to menopause.

Methods: 50 obese women and 50 non-obese women in the reproductive age group were selected in the study. Measured body mass index (BMI), anti-mullerian hormone (AMH), follicle-stimulating hormone (FSH), antral follicular count (AFC) and ovarian volume (OV) in both obese and non-obese women. The comparison of age and BMI, FSH and AMH, AFC and ovarian volume was done by using t test and P value less than 0.001 was considered statistically significant. Serum FSH and AMH levels were analyzed using radioimmunoassay (RIA) technique while AFC and ovarian volume were determined through transvaginal ultrasonography respectively**.**

Results: This study showed statistically significant lower values of Anti-Müllerian Hormone (AMH), Antral Follicle Count (AFC), and ovarian volume, alongside higher values of Follicle-Stimulating Hormone (FSH),

Vol. 2(2). DD. March-May, 2025
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PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

compared to the non-obese women of the same age group. Slightly elevated levels of serum FSH and lower levels of serum AMH, reduced Antral Follicle Count (AFC), and smaller ovarian volume, in obese women compared to non-obese women were shown.

Conclusion: This study concludes that obese women exhibit lower AMH levels, AFC and Ovarian volume, along with higher FSH levels, compared to non-obese women of the same age group. These findings suggest that obesity has a detrimental effect on ovarian reserve markers and may impact reproductive health, emphasizing the importance of weight management for preserving fertility.

KEYWORDS: Ovarian Reserve, Anti- Müllerian Hormone (AMH), Follicle-Stimulating Hormone (FSH), Antral Follicular Count (AFC), Ovarian Volume (OV), Body Mass Index (BMI).

INTRODUCTION

Obesity is a universal problem of health that adversely harms the reproductive system along with other organ systems. Over 890 million women globally struggled with obesity as of 2022, with the Americas holding the highest incidence (67%) and Southeast Asia and Africa having the lowest (31%). By 2030, about half of African women are expected to be overweight or obese (1). Compared to women of normal weight, obese women have a 30% lower In vitro fertilization (IVF) success rate, reduced anti-Müllerian hormone (AMH) levels, disturbed ovulation, and a 27% increased risk of infertility (2).

The total amount and quality of oocytes in a woman's ovaries, which dramatically impacts her potential for reproduction, is referred to as her ovarian reserve. Although the ovarian reserve certainly decreases with age, outside variables like obesity might hasten this process and have an undesirable impact on fertility (3). The correlation between body mass index (BMI) and ovarian reserve indicators, including ovarian volume (OV), anti-Müllerian hormone (AMH), follicle-stimulating hormone (FSH), and antral follicle count (AFC), has been the subject of numerous studies (4). In accordance to research, up to 40% of obese women suffer from impaired ovarian function, which raises their risk of infertility. Reproductive outcomes and ovarian reserve can be strengthened by weight loss and metabolic control (5). Obese women have considerably lower AMH levels than non-obese women, which suggests a reduced ovarian reserve, according to a thorough meta-analysis of 45 research. All subgroups, including PCOS and fertile non-PCOS women, showed the same inverse relationship between BMI and AMH. AFC and inhibin β levels did not substantially differ between obese and non-obese women, but the research also revealed that FSH levels were significantly lower in obese women with PCO (6).

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

In recent years, the association between ovarian reserve and obesity has gathered a lot of emphasis. Obesity is associated with hormonal imbalances, including elevated levels of estrogen, androgens, and insulin resistance, all of which can impair folliculogenesis and reduce ovarian reserve (7). Relative to their non-obese counterparts, obese women typically have decreased ovarian volumes, less antral follicles, and lower AMH levels, according to multiple research studies. Research found that women who were classified as obese had substantially reduced AMH levels (mean=2.5 ng/mL) than women who were not obese (mean=3.2 ng/mL), which was predictive of a decreased ovarian reserve. The same study also discovered that obese women had a lower average number of follicles (AFC), with an average of 10.3 against 15.2 for non-obese women. These results highlight the possible harm that obesity may do to ovarian function (8).

In spite of an extensive investigation, there are still a number of unresolved concerns regarding the connection between ovarian reserve and obesity. AMH levels and BMI have a well-established negative correlation, but it is unclear how obesity affects other ovarian reserve indicators as AFC and ovarian volume, with conflicting research findings (9). The majority of research has concentrated on women receiving ART, which restricts the findings' applicability to a larger population. To improve the results' relevance, future studies should try to cover several kinds of populations (10). It is essential to investigate how well weight-management programs can maintain ovarian reserve and enhance reproductive outcomes. Important information may be obtained from randomized controlled trials evaluating lifestyle changes (11). Although obesity is linked to a lower ovarian reserve, it is yet unknown how exactly these factors affect OV, AFC, FSH, and AMH. In order to gain a better understanding of how obesity affects reproductive health, our study intends to investigate these pathways.

Methodology

Study design

This comparative cross-sectional study was conducted at three THQ hospitals in Rawalpindi, Pakistan, from September 2024 to December 2024.

Study population

Ninety healthy menstruating female participants were recruited by using a stratified sampling technique. The participants were divided into two groups of 45 each based on their weight status: the non-obese group (BMI < 30 kg/m²) and the obese group (BMI > 30 kg/m²). All the participants filled the consent form. The women with a history of smoking, pregnancy, lactation, hysterectomy, endometriosis, previous ovarian surgery, polycystic ovary syndrome (PCOS) diagnosed by clinical or ultrasonographic criteria, using

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081
Journal of Medical & Health Sciences Review

hormone or medication that could affect ovarian function, or any other medical condition that may impact ovarian function were excluded from the study.

Data collection

Demographic variables, including age, weight, height, menstrual status, and overall health status, were collected through questionnaires. Body mass index (BMI) was calculated by dividing total body weight (in kilograms) by height (in meters squared).

Serum assay

Venous blood (5 mL) was collected from each participant into red-top vials after an overnight fast of 12 hours. Clear, non-hemolyzed serum was separated by centrifugation at 5000 rpms for five minutes and then loaded onto a Roche Cobas 6000 machine, which employs a radioimmunoassay technique to detect and quantify serum FSH and AMH levels.

Transvaginal sonography measurements

An unbiased and qualified technician was recruited to perform transvaginal ultrasonography in the early follicular phase of their menstrual cycle to measure their ovarian volume and antral follicle count (AFC). The study protocols were blinded to the technician to minimize the risk of bias. The volume of each ovary was calculated by measuring three perpendicular dimensions (length, width, height) and using the simplified formula:

$$\textbf{Volume} = 0.5 \times \textbf{length} \times \textbf{width} \times \textbf{height} \quad (1)$$

The total antral follicle count (AFC) was calculated as a sum of the numbers of follicles in both ovaries, varying in size from 2 to 10 mm.

Statistical Analysis

Before running statistical analysis, the collected data was checked for missing data of all variables, which did not exceed 2% per variable. The statistical outliers were identified using a box plot and were omitted. Statistical analysis was performed on SPSS Software Version 24.0 (Armonk, NY: IBM Corp.). Descriptive analysis was conducted for demographic variables (age, BMI), biochemical variables (AMH, FSH), and the clinical variables (AFC, ovarian volume) of both groups. The results were presented as mean, standard deviation, and frequency distribution. Graphs were plotted to reveal the relationships between age and BMI, AMH and FSH, as well as AFC and ovarian volume in obese and non-obese women. An independent sample t-test was employed to calculate the p-value to assess the statistical significance of the results, with an alpha value set at 0.05.

Results

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

Demographic and biochemical characteristics of obese and non-obese women:

Table 1 Demographic and biochemical characteristics of obese and non-obese women

Parameters	**Reference Ranges**	**Obese (n=50)**	**Non-obese (n=50)**
Age (Years)	Mid-reproductive age women 21-40 years	30.02 ± 5.69	30.09 ± 5.29
BMI (kg/m^2)	Obese (30-35 kg/m^2 obese (20-29 kg/m^2)	33.85 ± 3.17	23.2 ± 2.03
AMH (ng/mL)	2.3-6.8 ng/mL	2.04 ± 0.92	2.79 ± 0.96
FSH (mIU/mL)	3.85-8.78 mIU/mL	7.22 ± 3.56	6.31 ± 3.2
AFC (n)	5-8 follicles/ ovary	4.2 ± 1.79	7.62 ± 2.46
Ovarian Volume(mL)	4-7mL	4.97 ± 1.39	5.2 ± 1.42

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

Frequency Percentage Analysis:

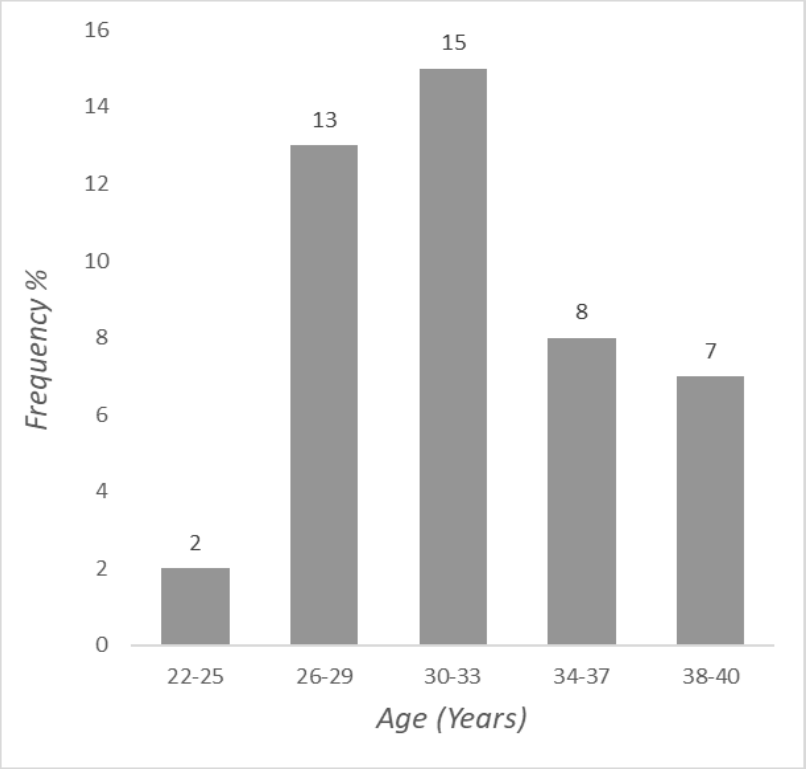


Figure 1 Age of obese women

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

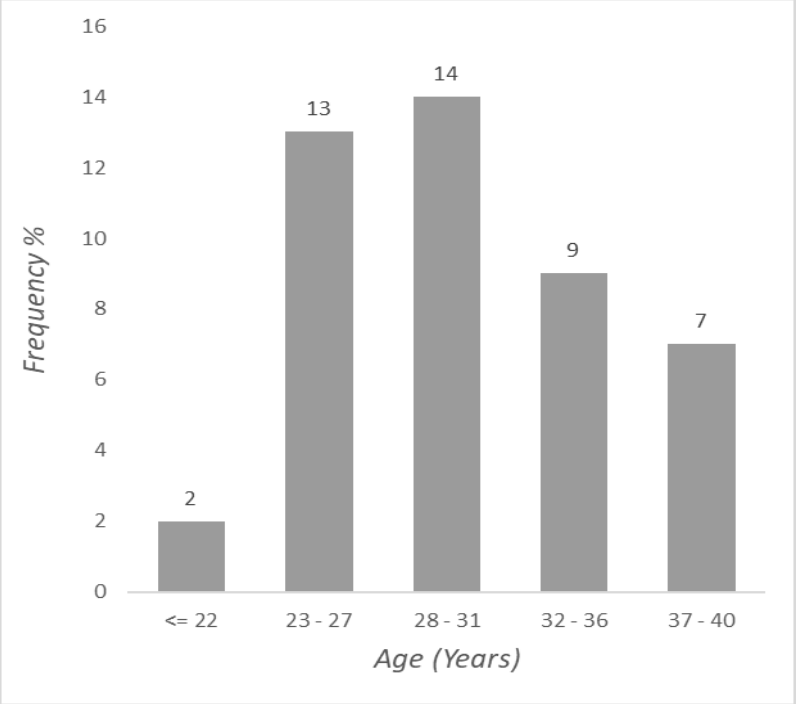


Figure 2 Age of non-obese women

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

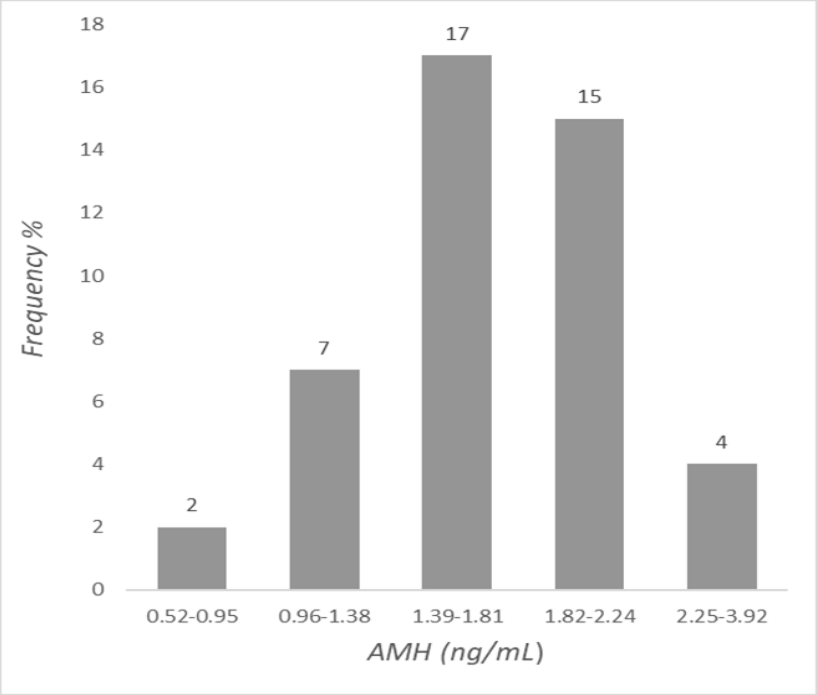
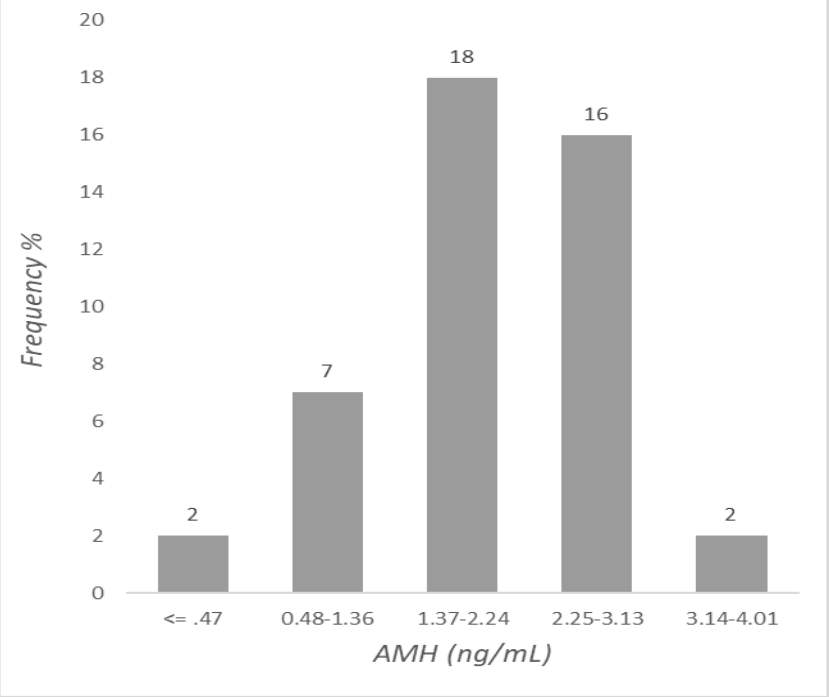


Figure 3 AMH levels in obese women



Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

Figure 4 AMH levels in non-obese women

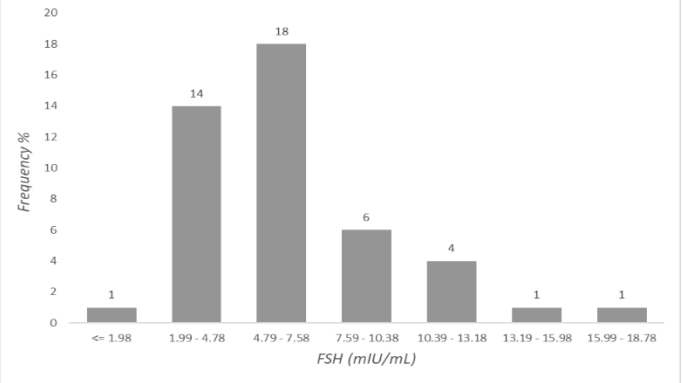


Figure .5 FSH levels in obese women.

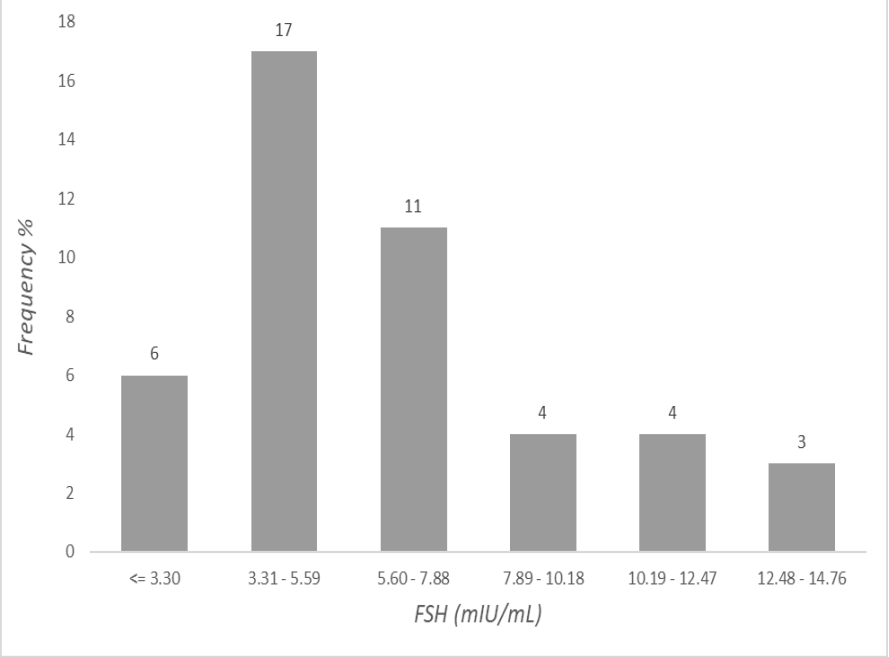


Figure 6 FSH levels in non-obese women.

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

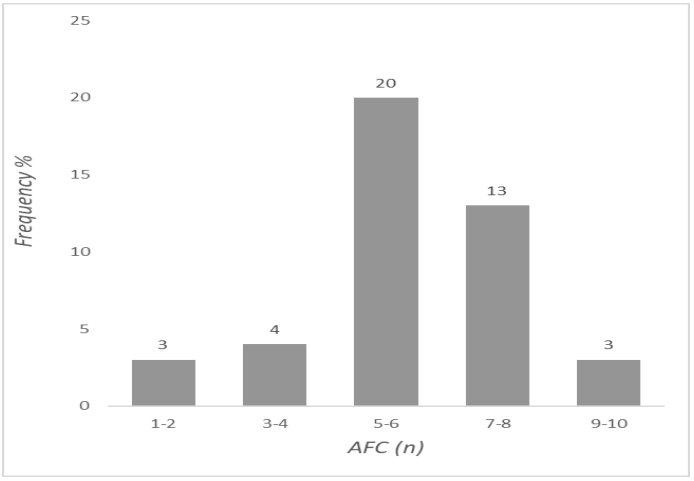


Figure 7 AFC of obese women.

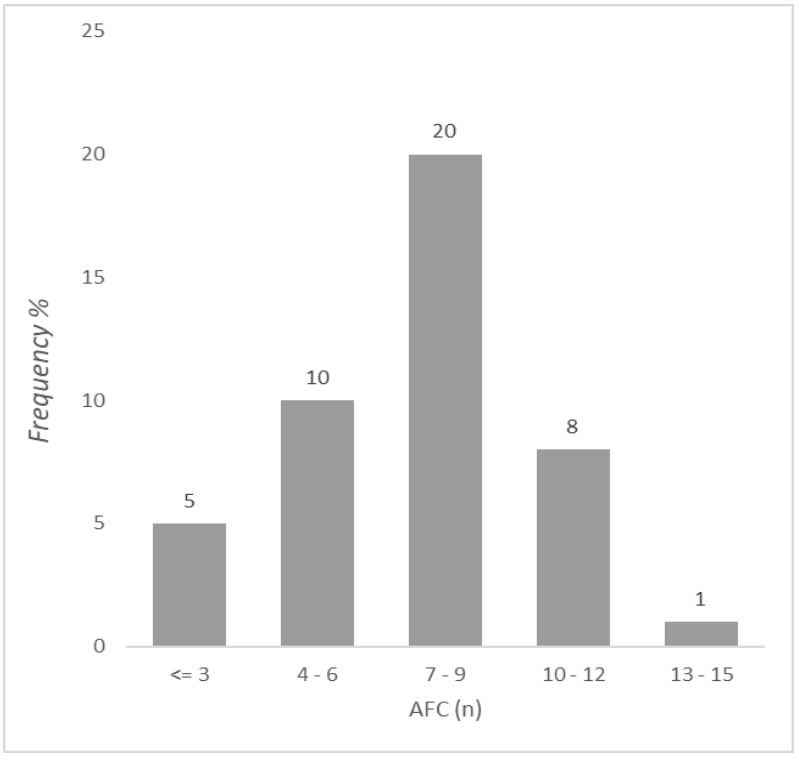


Figure 8 AFC of non-obese women.

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

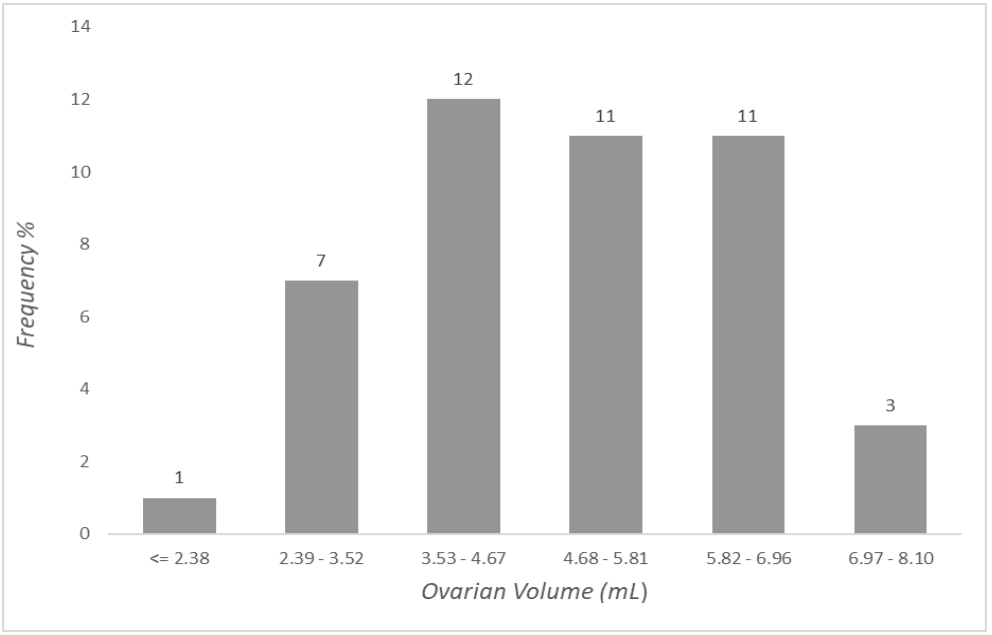
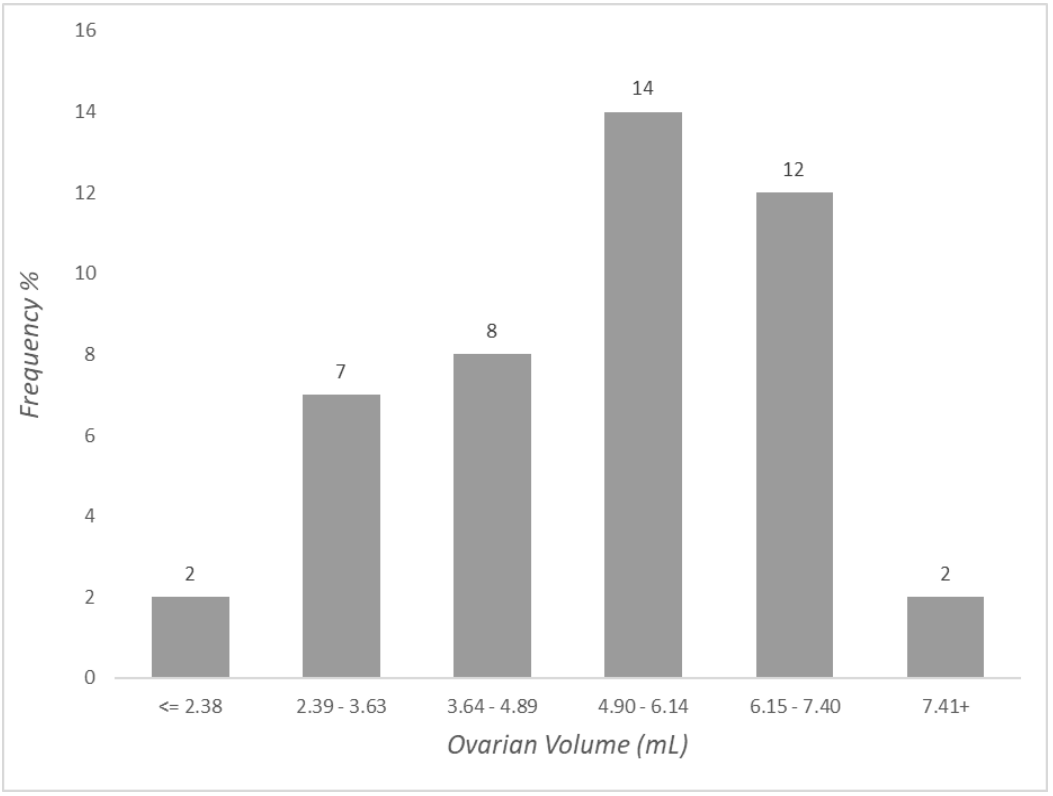


Figure 9 ovarian volume of obese women.



Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081
Journal of Medical & Health Sciences Review

Figure 10 ovarian volume of non-obese women.

Comparison of Age and BMI of obese and non-obese women:

In our study, the age of both obese and non-obese women ranged from 21 to 40 years, representing mid-reproductive age. The mean age of obese women (30.02±5.69) and the mean age of non-obese (30.09±5.29) was observed to be nearly identical. According to the results of this study, there is no significant difference (p 0.863) in the mean age of both the groups. BMI as expected obese women (33.85±3.17) had a higher BMI as compared to non-obese (23.2±2.03). According to the results, this difference is highly significant (<0.001)

Table 2 Comparison of Age and BMI of Obese and non-obese women.

Parameters	**Obese (n=50)**	**Non-obese (n=50)**	**p value**
Age (Years)	30.02 ± 5.69	30.09 ± 5.29	0.863
BMI (kg/m2)	33.85 ± 3.17	23.2 ± 2.03	**< 0.001**

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

4.4. Comparison of hormonal profile of obese and non-obese women:

In comparing hormonal profiles among obese and non-obese women, significant differences were observed in Anti-Müllerian Hormone (AMH) levels between obese and non-obese women. The mean AMH level was significantly lower in obese women (2.04±0.92 ng/mL) compared to non-obese women (2.79±0.96 ng/mL), with a p-value of <0.001, indicating a statistically significant difference. However, the mean Follicle-Stimulating Hormone (FSH) levels were comparable between the two groups, with obese women having a mean FSH level of 7.2±3.56 mIU/mL and non-obese women having 6.31±3.2 mIU/mL. The p-value for FSH was 0.204, showing no significant difference between the groups as shown in table 3.

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

Table 2 Comparison of AMH and FSH of Obese and non-obese women.

Parameters	Obese (n=50)	Non-obese (n=50)	P value
AMH (ng/mL)	2.04±0.92	2.79±0.96	**<0.001**
FSH (mIU/mL)	7.22±3.56	6.31±3.2	0.204

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

4.3 Comparison of ultrasound parameters of obese and non-obese women:

This study also compared ultrasound parameters between obese and non-obese women, focusing on Antral Follicle Count (AFC) and ovarian volume. The mean AFC was notably lower in obese women (4.20±1.79) compared to non-obese women (6.60±2.49), with a p-value of <0.001, indicating statistical significance. However, the mean ovarian volume was comparable between the two groups with obese women having a mean ovarian volume of

4.97±1.39 mL and non-obese women having 5.20±1.42 mL. According to the results of this study, there is no significant difference (p 0.423) in the mean ovarian volume of both the groups as shown in table 4

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081

Journal of Medical & Health Sciences Review

Table 4 Comparison of AFC and ovarian volume in obese and non-obese women.

Parameters	Obese (n=50)	Non-obese (n=50)	p value
AFC (n)	4.2 ±1.79	6.6±2.49	**< 0.001**
Ovarian volume	4.97±1.39	5.20±1.42	0.423

DISCUSSION

Obesity significantly impacts reproductive health, particularly ovarian reserve, in women of reproductive age. Ovarian reserve refers to the number and quality of ovarian follicles available for ovulation, assessed through biomarkers like Anti-Müllerian Hormone (AMH), Follicle-Stimulating Hormone (FSH), Antral Follicle Count (AFC), and ovarian volume. Our study found that obese women had significantly lower AMH levels compared to non-obese counterparts, suggesting diminished ovarian reserve. This aligns with previous research indicating that obesity is associated with reduced AMH levels, which reflect a decrease in antral follicles essential for ovulation.

FSH levels varied between the groups, with obese women showing altered gonadotropin secretion patterns. Elevated FSH levels often indicate ovarian insufficiency, implying that follicular recruitment may be suboptimal in obese women. The relationship between obesity and FSH could be influenced by obesity-related disruptions in the hypothalamic-pituitary-ovarian axis due to insulin resistance and hyperinsulinemia, leading to imbalances in gonadotropin regulation.

AFC levels were also significantly reduced in obese women, indicating compromised ovarian reserve. This reduction may stem from metabolic disturbances related to excess adiposity, such as inflammation and altered gonadotropin secretion. Similarly, our study found that obese women had reduced ovarian volume, which signifies a lower number of healthy follicles. Visceral fat accumulation contributes to insulin resistance and hyperinsulinemia, negatively affecting ovarian function and reducing ovarian size.

Chronic low-grade inflammation associated with obesity can impair ovarian reserve by accelerating follicular

Vol. 2(2). DD. March-May, 2025
ONLINE ISSN: 3007-309X
PRINT ISSN : 3007-3081
Journal of Medical & Health Sciences Review

atresia. Insulin resistance, common in obesity, adversely affects follicle growth and maturation, leading to reduced ovarian reserve. Although our study did not specifically focus on polycystic ovary syndrome (PCOS), it is a known risk factor for obesity, characterized by hormonal imbalances that further impact ovarian function. Notably, not all obese women experience the same decline in ovarian reserve, as individual differences in adiposity distribution, metabolic profiles, and insulin sensitivity play a role. Our findings highlight the need for early intervention and lifestyle modifications in obese women of reproductive age to preserve ovarian reserve and enhance fertility potential. Weight loss through dietary changes, increased physical activity, and behavioral therapy, along with pharmacological interventions like insulin sensitizers, may improve ovarian function and fertility outcomes in this population.

Limitations of study:

The study is cross-sectional, collecting data at a single time point, which restricts the ability to determine causal relationships between obesity and ovarian reserve markers. The sample size was small, and there was no clear differentiation among normal, overweight, obese, and morbidly obese women. Future research should involve a larger sample and categorize participants by age groups for more comprehensive insights. Obesity was assessed solely through BMI, which does not consider variations in fat distribution. Utilizing metrics like waist-to-hip ratio or body fat percentage would provide a clearer understanding of how obesity influences ovarian reserve. Additionally, the study does not examine the long-term effects of obesity on fertility or ovarian function, indicating a need for longitudinal studies to assess how obesity impacts ovarian reserve and fertility over time.

Conclusions

Our findings reveal the link between obesity (BMI> 30 kg/m^2) in reproductive-aged women and diminished ovarian reserve markers, evident of lower AMH levels, reduced AFC and ovarian volume, and elevated FSH levels compared to non-obese women group (BMI < 30 kg/m2). These results underscore the potential detrimental effect of obesity on ovarian function and fertility, emphasizing the importance of weight management approaches in reproductive health care. However, further investigation on a large scale is needed to better understand underlying mechanisms.

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Vol. 2(2). DD. March-May, 2025
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Journal of Medical & Health Sciences Review

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Vol. 2(2). DD. March-May, 2025
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