Section: Obstetrics and Gynaecology

Original Research Article

EVALUATION OF THE RELATIONSHIP IN BMI (BONE MASS INDEX) AND SERUM AMH (ANTI-MULLERIAN HORMONE) LEVELS IN INFERTILE FEMALES WITH AND WITHOUT PCOD (POLYCYSTIC OVARIAN DISEASE)

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ABSTRACT

Background: AMH levels are usually high in subjects with high BMI and obesity, which is a peculiar feature of PCOD. Hence, higher AMH levels in females with PCOD can be attributed to increased AMH secretion from an increased number of small antral follicles. **Aim:** The present study aimed to evaluate the relationship between BMI (bone mass index) and serum AMH (anti-mullerian hormone) levels in infertile females with and without PCOD (polycystic ovarian disease).
Materials and Methods: The present study assessed 400 females who visited the Institute within the defined study period. All subjects underwent gynecological assessment and basic infertility tests, including BMI. All females' AMH levels were assessed, and subjects were divided into two groups: subjects with PCOD and subjects without PCOD.
Results: The study results showed an overall AMH level of 4.84±4.42 ng/ml. No significant correlation was seen in AMH and BMI levels in females without PCOD with $p>0.05$. However, in subjects with PCOD, a significant inverse correlation was seen in AMH and BMI levels with $p<0.05$.
Conclusions: The present study concludes that there is no significant correlation between AMH and BMI levels in infertile females without PCOD. However, in females with PCOD, there is a significant inverse correlation between serum AMH levels to BMI.
Keywords: Antimullerian hormone, body mass index, Infertility, polycystic ovarian disease.

INTRODUCTION

Presently, there has been an increased incidence of concerns in females from reproductive age group. In developing nations such as India, dire steps are being implemented for population control, on the other hand, concerns such as infertility are also increasing, particularly in females from higher socioeconomic backgrounds. Following the WHO study, primary infertility prevalence in India is 4-17% which is mainly reported in urban residents. It can be attributed to the various factors leading to an increase in infertility including obesity unhealthy food habits, unhealthy lifestyle, advanced age of conception, advanced age of marriage, and stress.[1]
Obesity is one of the major contributors to infertility which considers excessive BMI (body mass index) and body fat as the gold standard of assessing obesity. BMI calculation is done by evaluating the weight and height of the subject. Females having

BMI ≥25kg/m2 are considered overweight, whereas, females with BMI ≥30kg/m2 are considered obese. Categorization and assessment of obesity is vital as obesity poses detrimental effect on the whole body and predisposes subjects to various medical conditions where diabetes mellitus and hypertension are the most common, Considering the reproductive system in females, obesity is seen to affect oocyte development and folliculogenesis in the ovary.[2]

PCOD (Polycystic ovarian disease) is a heterogenous endocrine disease in females of reproductive age group having a wide range of clinical features such as hyperandrogenism, menstrual disorders, and obesity. It is one of the most common endocrine disorders that affect nearly 5-10% of females from the reproductive age range. The characteristic pattern is an increase in LH (luteinizing hormone) levels and an increase in androgen levels causing subsequent anovulation.[3]

To assess ovarian reserve and ovarian function, assessment of AMH (anti-mullerian hormone) levels is a useful test. Estimating AMH levels is a popular test in subjects with infertility as it depicts the ovarian reserve, which is further an indirect index assessing the reproductive capacity of a female. AMH is a glycoprotein that is secreted from the granulosa cells of the ovary following puberty. AMH helps in oocyte maturation and ovarian follicle development. The main function of AMH is inhibition of the FSH (follicle stimulating hormone) dependent selection process in the early folliculogenesis stage. It also decreases LH receptor number in granulosa cells which is also an FSH-induced process. As AMH levels are fairly constant during the menstrual cycle, its assessment is not time-dependent on the menstrual cycle making it more convenient to use.[4]

Normal AMH level is considered as 2-6.8 ng/ml and it decreases with an increase in age. At menopause, AMH levels are not detected. Fertility in a female is generally graded depending on AMH levels and females with AMH levels ≤ 1 ng/ml are considered to have poor ovarian reserve. AMH levels are high in obese females and high BMI is usually a feature of PCOD. High AMH levels are expected in females with PCOD where an increased antral small follicles number can increase AMH secretion.[5] The present study aimed to assess any relationship between serum AMH levels and BMI in females presenting with infertility keeping a special focus on subjects with PCOD.

MATERIALS AND METHODS

The present cross-sectional clinical study aimed to evaluate the relationship between BMI (bone mass index) and serum AMH (anti-mullerian hormone) levels in infertile females with and without PCOD (polycystic ovarian disease). The study subjects were from the Department of Obstetrics and Gynecology of the Institute. Verbal and written informed consent were taken from all the subjects before study participation.

The study assessed all the females who presented with the complaint of infertility to the Institute within the defined study period. The inclusion criteria for the study were females presenting with infertility and aged 20-40 years who were willing to participate in the study. The exclusion criteria for the study were subjects with premature ovarian failure (FSH level > 15 IU/ml), ovarian surgery history, diabetes, congenital adrenal hyperplasia, Cushing's syndrome, hyperprolactinemia, thyroid dysfunction, and subjects that did not give consent for study participation. The study also excluded subjects with established male infertility.

All the subjects underwent comprehensive assessment at baseline including gynecological, physical, and general examination along with gynecological history along with basic infertility assessment including TVS (transvaginal ultrasound) on the second day of the menstrual cycle. Serum AMH (anti-mullerian hormone) was assessed at the first visit irrespective of the day of the cycle. Blood samples were collected from the cubital vein followed by waiting for lot retraction and centrifugation of the sample at 2000 rpm for 5 minutes and collected serum was stored at 2-80C. Levels of AMH were assessed in mg/ml using CLIA (chemiluminescent immunoassay). A similar assay was used for the collection of AMH samples.

Subjects were then divided into two groups Group I including subjects with PCOD and Group II subjects without PCOD. Subjects were diagnosed as having PCOD depending on Rotterdam criteria in subjects with two of the following three criteria hyperandrogenism, oligo/anovulation, and/or polycystic ovaries on ultrasound. Polycystic ovaries were taken as ≥ 12 small antral follicles per ovary. For all females, BMI was assessed by division of weight in kilograms by the height in square meters.

The primary outcome assessed was establishing a correlation between BMI and AMH levels in subjects with PCOD and also assessing the relationship in the non-PCOD infertility population in the Institute. SPSS (Statistical Package for the Social Sciences) software version 24.0 (IBM Corp., Armonk. NY, USA) was used for the assessment of descriptive measures, Student t-test, ANOVA (analysis of variance), and Spearman correlation test. The results were expressed as mean and standard deviation and frequency and percentages. The p-value of <0.05 was considered.

RESULTS

The present cross-sectional clinical study aimed to evaluate the relationship between BMI (bone mass index) and serum AMH (anti-mullerian hormone) levels in infertile females with and without PCOD (polycystic ovarian disease). The study assessed 400 females who presented with infertility to the

Institute within the defined study period. There were 100 subjects diagnosed with polycystic ovarian disease and 300 subjects having infertility for causes other than polycystic ovarian disease including unexplained infertility, poor ovarian responders, and tubal factors.

Among 400 study subjects, on assessing the age distribution, the majority of the study subjects were aged 25-30 years with 43% subjects followed by 32% subjects in 31-35 years of age, 20% subjects>35 years of age, and 5% subjects in the age range of <25 years.

The study results showed that for mean age, BMI, and AMH levels in the two groups of study subjects, it was seen that AMH levels were significantly higher in subjects with polycystic ovarian disease (Group I) compared to subjects without PCOD (group II) with p<0.05. The study results also showed an inverse relationship in AMH and BMI levels which was statistically significant in Group I with p=0.04. However, no association was seen between AMH levels and BMI in group II with p>0.05 (Table 1).

It was seen that concerning the proportion of lean PCOD subjects in the study (subjects with BMI <25) and in non-lean PCOD subjects (subjects with BMI >25kg/m2), there were significantly higher number of subjects with lean PCOD. In subjects with lean PCOD, mean AMH levels were 11.16±2.62 ng/ml compared to 10.09±4.24 ng/ml in subjects with non-lean PCOD. However, the difference was statistically non-significant with p>0.05.

Table 1: Age, BMI, and AMH levels in the two groups of study subjects

S. No	Variables	Group I (n=100)		Group II (n=300)		Overall (mean)
		Mean	p-value	Mean	p-value	
1.	Age	28.16±2.89	0.04	33.2±5.40	0.77	32.11±5.39
2.	BMI	24.30±4.60		23.15±3.15		23.44±3.58
3.	AMH (ng/ml)	10.76±3.38		2.86±2.60		4.84±4.42

DISCUSSION

The present study assessed 400 females who presented with infertility to the Institute within the defined study period. There were 100 subjects diagnosed with polycystic ovarian disease and 300 subjects having infertility for causes other than polycystic ovarian disease including unexplained infertility, poor ovarian responders, and tubal factors. These factors were comparable to the reports of Amer SA et al,[6] in 2013 and Siefer DB et al,[7] in 2011 where authors reported similar causes of PCOD in their study subjects as in the present study.

The study results showed that among 400 study subjects, on assessing the age distribution, the majority of the study subjects were aged 25-30 years with 43% of subjects followed by 32% of subjects in 31-35 years of age, 20% of subjects in >35 years of age, and 5% subjects in the age range of <25 years. These data were comparable to the studies of Piouka A. et al,[8] in 2009 and Halawaty S et al,[9] in 2010 where demographic data comparable to the present study was also reported by the authors in their respective studies.

It was seen that for mean age, BMI, and AMH levels in the two groups of study subjects, it was seen that AMH levels were significantly higher in subjects with polycystic ovarian disease (Group I) compared to subjects without PCOD (group II) with p<0.05. The study results also showed an inverse relationship in AMH and BMI levels which was statistically significant in Group I with p=0.04. However, no association was seen between AMH levels and BMI in group II with p>0.05. These results were consistent with the findings of Freeman EW et al,[10] in 2007 and Jungheim ES et al,[11] in 2013 where results for age, BMI, and AMH levels reported by the authors in their studies were comparable to the results of the present study.

The study results also showed that concerning the proportion of lean PCOD subjects in the study (subjects with BMI <25) and in non-lean PCOD subjects (subjects with BMI >25kg/m2), there were significantly higher number of subjects with lean PCOD. In subjects with lean PCOD, mean AMH levels were 11.16±2.62 ng/ml compared to 10.09±4.24 ng/ml in subjects with non-lean PCOD. However, the difference was statistically non-significant with p>0.05. These findings were in agreement with the results of Su Hi,[12] et al in 2008 and Teixeria J et al,[13] in 2001 where the proportion of lean PCOD subjects in the study (subjects with BMI <25) and in non-lean PCOD subjects (subjects with BMI >25kg/m2) similar to the present study was also reported by the authors in their respective studies.

CONCLUSION

Within its limitations, the present study concludes that there is no significant correlation between AMH and BMI levels in infertile females without PCOD. However, in females with PCOD, there is a significant inverse correlation between serum AMH levels to BMI.

REFERENCES

1. Koninger A, Sauter L, EdimirisP, et al. Predictive markers for the FSH sensitivity of women with polycystic ovarian syndrome. Hum Reprod. 2014; 29:518-24.
2. Ciotta L, Stracquadanio M, Pagano I, Carbonaro A, Palumbo M, Gulino F. Effects of Myo-inositol supplementation on oocyte's quality in PCOS patients: a

double-blind trial. Eur Rev Med Pharmacol Sci. 2011; 15:509-14.
3. Jungheim ES, Travesio JL, Hopeman MM. Weighing the impact of obesity on female reproductive function and fertility. Nutr Rev. 2013;71: S3-S8.
4. World Health Organization. Report of the World Health Organization on Obesity 1997: Preventing and managing the global epidemic. Geneva: World Health Organization;1997.
5. ORC Macro and World Health Organization. DHS Comparative Reports No 9. Calverton, Maryland, USA: Infecundity, infertility, and childlessness in developing countries. Geneva: ORC Macro and the World Health Organization; 2004.
6. Amer SA, Mahran A, Abdelmaged A, EL-Adawy AR, Eissa MK, Shaw RW. The influence of circulating antiMullerian hormone on ovarian responsiveness to ovulation induction with gonadotrophins in women with polycystic ovarian syndrome: a pilot study. Reprod Biol Endocrinol. 2013; 11:115.
7. Siefer DB, Baker VL, Leader B. Age-specific serum anti-Mullerian hormone values for 17,120 women presenting to fertility centers within the United States. Fertil Steril. 2011; 95:747-50.
8. Piouka A, Farmakiotis D, Katsikis I, Macut D, Gerou S, Panidis D. Anti-Mullerian hormone levels reflect the severity of PCOS but are negatively influenced by obesity: relationship with increased luteinizing hormone levels. Am J Physiol Endocrinol Metab. 2009;296: E238-43.
9. Halawaty S, EL Kattan E, Azab H. Effect of obesity on parameters of ovarian reserve in premenopausal women. J Obstet Gynaecol Can. 2010; 32:687-90.
10. Freeman EW, Gracia CR, Sammel MD. Association of anti-mullerian hormone levels with obesity in late reproductive-age women. Fertil Steril. 2007; 87:101-6.
11. Jungheim ES, Traveiso JL, Hopeman MM. Weighing the impact of obesity on female reproductive function and fertility. Nutr Rev. 2013;71: S3-S8.
12. Su HI, Sammel MD, Freeman EW. Body size affects measures of ovarian reserve in late reproductive-age women. Menopause. 2008; 15:857-61.
13. Teixeria J, Maheshwaran S, Donahoe PK. Mullerian inhibiting substance: an instructive developmental hormone with diagnostic and possible therapeutic applications. Endocr Rev. 2001; 22:657-74.