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Research Paper

The Correlation of Anti-Mullerian (AMH) Level with Anthropometric Measurements in a Sample of Iraqi Premenopausal Women

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ABSTRACT:

BACKGROUND:

Anti-Müllerian hormone (AMH) can serve as a good biomarker for the ovarian reserve that can predict female fertility. Studying the biological factors that impact AMH levels is necessary for predicting reproductive health outcomes. Obesity (general obesity by body mass index and central obesity by waist circumference), is associated with negative reproductive outcomes that could be related to its impact on AMH level. Female fecundity begins to decrease after women reach the age of 30 years due to multifactorial causes that could be hormonal as AMH levels changes.

OBJECTIVE:

The study aims to find an association between anti-mullerian hormone (AMH) level, as a biomarker for ovarian reserve & obesity (general and central), and other anthropometric measurements in premenopausal women.

SUBJECTS AND METHODS:

This is a descriptive cross sectional study that has enrolled a total of 270 Iraqi women in the reproductive age. Height ,weight and waist circumference were measured for all participants .Serum AMH has been analyzed using Enzyme Linked Fluorescent Immune Assay technique (ELFIA). Subjects were divided into three groups according to their body mass index (BMI), and into two groups depending on WHO criteria for central obesity, and into three groups according to WHO criteria for their susceptibility to metabolic risk, and into four groups according to their age. Statistical analyses were performed using SPSS software.

RESULTS:

AMH level was significantly lower in obese and overweight women than in normal weight women also in women with central obesity than those without it and in married women than in singles.

Women with increased and substantially increased risk for metabolic complications have significantly lower AMH level than those with no metabolic risk.

CONCLUSION:

AMH has a negative association with BMI , waist circumference , and age .

KEY WORDS: Anti-Müllerian hormone (AMH) , body mass index , central obesity .

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INTRODUCTION:

Anti-Müllerian hormone (AMH) is a glycoprotein hormone , its main roles are in growth , differentiation , and folliculogenesis. It is encoded by the AMH gene, on chromosome 19p13.3, and its receptor is encoded by the AMHR2 gene on chromosome 12 , in humans [1,2,3]. AMH is a molecular biomarker for relative size of the ovarian reserve in humans ,which can be used to predict timing of menopause and ovarian dysfunction as its main physiological role in the ovary seems to be targeted to the inhibition of primordial follicle recruitment, thus preventing too early depletion of the follicular reserve. Furthermore, the inhibition of sensitivity to FSH by AMH may occur due to the variable expression of AMH receptors among the recruited small pre-antral follicles. In such circumstances, only those with

lower AMH expression become sensitive to FSH, of which usually one is permitted for dominance [4,5].

In healthy females AMH is still fairly constant from mid-childhood to early adulthood and does not change significantly during puberty [6]. The rise during childhood and adolescence is likely due to different stages of follicle development [7]. From 25 years of age AMH declines to undetectable levels at menopause [6].

Anthropometric measurements are noninvasive quantitative measurements of the body. According to the Center for Disease Control and Prevention (CDC), anthropometry provides a valuable assessment of nutritional status and diagnosing obesity in children and adults. The core elements of anthropometry are height, weight, head circumference, body mass index (BMI), body circumferences to assess for adiposity (waist, hip, and limbs) [8].

According to WHO, Overweight and obesity are defined as abnormal or excessive fat accumulation that presents a risk to health. A body mass index (BMI) over 25 is considered overweight, and over 30 is obese [9]. Obesity has been associated with negative reproductive outcomes. Obese women are more prone to anovulation, infertility, miscarriage, and pregnancy complications, compared to normal-weight women [10,11]. Although infertility associated with obesity has been related to anovulation, it has been shown that the time to spontaneous pregnancy is much longer in obese women, even in those with regular menstrual cycles [12,13,14].

The effect of obesity on fertility is likely multi-faceted. It has been demonstrated that obese patients exhibit an altered ovarian follicular environment in multiple systems, including steroidogenic action, metabolism, and inflammation, which may contribute to these poorer outcomes [15].

Female fecundity begins to decrease after women reach the age of 30 years, it is difficult to predict the pace of reproductive decline in each individual. The age-related decline in fecundity is characterized by decreases in both egg quality and number, in addition to gradual increases in circulating FSH and decreases in circulating anti-Müllerian hormone (AMH) and inhibin B levels [16].

Studies have demonstrated that with the decrease in the number of antral follicles with age, AMH serum levels also decline and will invariably become undetectable near menopause. Because AMH does not exhibit cyclic changes across the menstrual cycle, AMH levels have gained favor as a serum marker for the ovarian follicular reserve and may provide an index of age at menopause (17).

AIM OF THE STUDY:

The study aims to find an association of Anti-mullerian hormone (AMH) level as a biomarker for ovarian reserve and fertility, with general and central obesity, and other anthropometric measurements in premenopausal women.

SUBJECTS AND METHODS:

In this descriptive cross sectional study, a total of 270 women who met the inclusion criteria, were enrolled.

Inclusion criteria:

1. Female in the reproductive age (17– 45 year).
2. Regular menstrual cycle.
3. Not smoker.
4. Not pregnant.

Exclusion criteria:

1. History of amenorrhea or oligomenorhea.
2. History of polycystic ovarian syndrome or ovarian surgery.
3. History of chemotherapy, hormonal therapy or hormonal contraceptive methods.

Recruitment of participants occurred between September 2019 and January 2021. The participants who visited gynecological centers in Baghdad were referred to Central Public Health Laboratory (CPHL) after filling a request form by the referring physician. The form had been designed to get the target participants in our study.

A verbal permission was taken from each participant in this study. Data obtained from the participants were used without any referring to their personal information.

In the Central Public Health Laboratory, a blood sample had been taken from each enrolled participant according to the standard procedures and collected in gel plain tube and then serum had been separated using appropriate centrifuge. Separated serum was stored appropriately at -25 °C till AMH has been analyzed using ELFIA (Enzyme Linked Fluorescent Immunoassay) technique .

Height, weight and WC have been measured for all enrolled participants using standard methods for measurement. BMI has been calculated by dividing weight in kilograms on square of height in meters then the participants were divided into three groups by their BMI according to the World Health Organization (WHO) criteria as normal weight with BMI between 18.5-24.9 kg/m^2, overweight with their BMI ranging from 25-29.9 kg/m^2, and obese, with a BMI of ≥30 kg/m^2.

Women were divided into three groups depending on their susceptibility to metabolic risk using waist circumference. Low risk of metabolic complications when WC ≤ 80 cm, increased risk of metabolic complications when WC > 80 - 88 cm and substantially increased risk of metabolic complications with WC > 88 cm . Also we categorized the participants to four groups according to their age, less than 20 years, 20 - 29 years, 30 – 39 years and equal or more than 40 years. Finlay, Participants were divided according to their marital status into single and married.

Data has been reported as mean ± SD for continuous variables and as numbers or percentage for categorical variables. The studied parameters have been compared using the independent sample t-test or the chi-square test when the variables were continuous or categorical, respectively. The one-way analysis of variance [ANOVA] has been used to determine whether there are any statistically significant differences between the means of two or more independent groups. If p value was <0.05, the relation is considered significant. Statistical analyses have been performed using SPSS software [SPSS Inc., Chicago, IL, USA].

RESULTS:

In this study, a total of 270 women in the premenopausal age, who met the inclusion criteria, were enrolled, of which 118 (43.7%) were obese, with a BMI of ≥30 kg/m^2, and 108 (40%) were overweight with their BMI ranging from 25-29.9 kg/m^2, while women with normal weight with BMI between 18.5-24.9 kg/m^2 were only 44 (16.3%).

Women were divided into three groups depending on their susceptibility to metabolic risk using waist circumference. Those having substantially increased metabolic risk were 144 (53.5%), and those with increased metabolic risk were 65 (24.1%), while those with no metabolic risk were 61 in number (22.6%).

Table 1 shows the differences in the anthropometric measures and AMH levels of participants categorized by BMI as a measure of general obesity. Compared with AMH of women in normal weight group 1.96 ng/mL (0.82-4.28), subjects in the overweight group and obese group had significantly lower levels of AMH 0.94 ng/mL (0.28-2.34), and 0.66 ng/mL (0.17-2.43), respectively, p < 0.005.

Table 1: Comparison of anthropometric measures and AMH levels in participants categorized by BMI.

	Normal weight BMI (18.5 – 24.9) kg/m^2	Overweight BMI (25 – 29.9) kg/m^2	Obese BMI ≥(30) kg/m^2	p value
N	44 (16.3%)	108 (40%)	118 (43.7%)	
Age (years)	31.0 (24.0-37.2)	37.0 (28.2-40.0)	38.0 (32.0-41.0)**	0.000 [b]
BMI (kg/m^2)	23.4 (22.2-24.2)	27.2 (26.1-28.6)	33.4 (31.2-36.6)**	0.000 [b]
WC (cm)	81.9 ± 8.2	91.2 ± 8.2	105.2 ± 9.3*	0.000 [b]
Weight (Kg)	59.3 ± 6.8	69.2 ± 5.9	88 ± 13.2*	0.000 [b]
Height (cm)	160.0 (156.2-164.7)	160.0 (155.0-162.2)	158.0 (155.0-162.7)**	0.139
AMH (ng/mL)	1.96 (0.82-4.28)	0.94 (0.28-2.34)	0.66 (0.17-2.43)**	0.004 [a]

[a] for p< 0.05, [b] for p< 0.001
*Values are expressed as mean ± S.D
**Values (not normally distributed) are expressed as median (Interquartile range, IQR).
AMH, anti-Müllerian hormone; BMI, Body mass index; WC, waist circumference; N, number; S.D, Standard deviation.

In **table 2**, the subjects were stratified by WC according to their susceptibility to the risk for metabolic complications (9). AMH concentration was significantly lower (p < 0.05) in women with increased risk for metabolic complications, 0.96 ng/mL (0.34-3.32), and substantially increased risk for metabolic complications, 0.89 ng/mL (0.26-2.92) than in those with no metabolic risk, 2.82 ng/mL (1.13-5.73).

The two groups with increased and substantially increased risk for metabolic complications also have significantly higher BMI (p <0.001), and have significantly higher age than females with no metabolic risk, with p<0.001.

Table 2: Comparison of anthropometric measures and AMH levels in participants categorized by risk for metabolic complications depending on WC.

	WC ≤ 80 cm [c]	WC > 80 - 88 cm [d]	WC > 88 cm [e]	p value
N	61 (22.6%)	65 (24.1%)	144 (53.3%)	
Age (years)	27.1 ± 7.7	34.5 ± 8.7	34.0 ± 8.2	0.000 [b]
BMI (kg/m^2)	24.2 (20.30-26.07)	25.1 (23.72-27.52)	30.9 (28.0-34.25)	0.000 [b]
WC (cm)	77.0 (71.5-80.0)	85.0 (83.0-87.0)	100.0 (95.0-107.0)	0.000 [b]
Weight (Kg)	61.5 (52.75-64.25)	65.0 (60.75-70.00)	79.0 (72.0-89.0)	0.000 [b]
Height (cm)	157.5 ± 4.9	160.1 ± 5.9	160.2 ± 5.0	0.088
AMH (ng/mL)	2.82 (1.13-5.73)	0.96 (0.34-3.32)	0.89 (0.26-2.92)	0.010 [a]

[a] p< 0.05, [b] p< 0.001; [c] Low risk of metabolic complications; [d] Increased risk of metabolic complications; [e] substantially increased risk of metabolic complications. Values are expressed as mean ± S.D, or as median (IQ range). AMH, anti-Müllerian hormone; BMI, Body mass index; WC, waist circumference; N, number; S.D, standard deviation.

In **table 3**, the subjects were divided on the bases of central obesity. Women with central obesity had significantly lower levels of AMH, 0.90 (0.33-2.87) than those without it, 2.94 (1.53-6.09) (p <0.005). The age of subjects with central obesity were significantly higher 36.5 (29.75-40.0) than those without central obesity 25 (20.5-34.5), with p value of < 0.001.

Table 3: Comparison of anthropometric measures and AMH level in participants categorized by WHO criteria for central obesity according to WC.

	WC ≤ 80 cm [c]	WC > 80 cm [d]	p value
N	80 (29.6%)	190 (70.4%)	
Age (years)	25 (20.5-34.5)	36.5 (29.75-40.0)	0.000 [b]
BMI (kg/m^2)	24.2 (20.3-25.4)	29.7 (26.7-33.27)	0.000 [b]
WC (cm)	77 (71.5-80.0)	97 (90-106)	0.000 [b]
Weight (Kg)	61.0 (52.5-64.0)	78.0 (69.0-84.0)	0.000 [b]
Height (cm)	157.6 ± 5.1	160.2 ± 5.2	0.039 [a]
AMH (ng/mL)	2.94 (1.53-6.09)	0.90 (0.33-2.87)	0.002 [a]

[a] p< 0.05, [b] p< 0.001; [c] no central obesity; [d] central obesity. Values are expressed as mean ± S.D, or as median (IQ range).
AMH, anti-Müllerian hormone; BMI, body mass index; WC, waist circumference; N, number; S.D, Standard deviation.

Participants were divided according to their marital status in **Table 4**. Single women were 16, while married women were 254 in number. AMH level was significantly lower in married women (0.86 ng/mL) than in singles (3.05 ng/mL) with p value of < 0.05. Age and BMI were significantly lower in single women than in married group with a P value <0.001. WC was also lower in singles´ group with a P value of less than 0.05.

Table 4: Anthropometric measures and AMH levels in pre-menopausal women by marital status.

	Single	Married	p value
N (Percentage)	16 (4%)	254 (96%)	
Age (years)	24.0 (17.5-27.7)	37.0 (30.0-40.0)	0.000 [b]
BMI (kg/m^2)	25.2 (22.8-27.3)	29.3 (26.1-33.0)	0.000 [b]
WC (cm)	81.3 ± 8.8	96.0 ± 12.3	0.001 [a]
Weight (Kg)	63.5 (58.5-68.0)	74.0 (65.0-83.2)	0.002 [a]
Height (cm)	158.0 (154.2-165.0)	159.5 (155.0-163.0)	0.968
AMH (ng/mL)	3.05 (1.9-5.3)	0.86 (0.3-2.4)	0.001 [a]

[a] p< 0.05, [b] p< 0.001. values are expressed as mean ± S.D, or as median (IQ range).
AMH, anti-Müllerian hormone; BMI, body mass index; WC, waist circumference; N, number; S.D, standard deviation.

In **table 5**, we categorized the participants into four groups according to their age.

We found a significant difference in the AMH concentration between the four groups; with women in the age group of ≥ 40 years having the lowest AMH concentration. There were also significant differences in BMI and WC between the four groups with the highest mean for both in women with age of ≥ 40 years.

Table 5: Comparison of anthropometric measures and AMH levels in participants categorized by their age groups.

	Less than 20 years	20 - 29 years	30-39 years	Equal or more than 40	p value
N	12	59	103	94	
BMI (kg/m^2)	24.7 (20.8-28.9)	28.3 (25.0-32.9)	28.8 (26.1-34.2)	30.1 (27.0-32.4)	0.007 [a]
WC (cm)	81.2 ± 12.5	92.5 ± 12.2	97.1 ± 13.3	97.5 ± 10.1	0.001 [a]
AMH (ng/mL)	2.9 (1.6-4.9)	3.5 (1.9-6.8)	1.1 (0.5-2.6)	0.3 (0.1-0.7)	0.000 [b]

[a]p< 0.05, [b]p< 0.001. Values are expressed as mean ± S.D.

AMH, anti-Müllerian hormone; BMI, body mass index; WC, waist circumference; N, number; S.D, standard deviation.

DISCUSSION:

AMH and General Obesity

In the current study, AMH concentration was significantly lower in overweight and obesity groups than in normal weight group, with a p value of < 0.05. Although the relationship between AMH and obesity has been described earlier, findings of these studies were controversial.

Alexis L Oldfield and colleagues summarized 13 studies involving 210 obese and 550 non-obese healthy women, of which 5 reported decreased AMH levels in association with obesity that agreed with our study, whereas 8 showed comparable AMH levels between groups [(18)].

Another study of Eleni et al. had the same result of ours they have observed that increasing BMI was correlated with reductions in AMH [(19)]. While Albu et al. had different result as reported that AMH is positively correlated with BMI, especially in infertile patients younger than 35 years of normal weight and with normal ovarian reserve [(20)]. Sahmay and Halawaty et al. could not find any relationship between AMH level and BMI [(21,22,23)]. The study of Zahra Heidar et al. showed no significant relationship between AMH and BMI [(24)]. At the level of our country, Reyam E. Ahmed and her colleague showed that a non-significant correlation was found between AMH levels and BMI in single women, while a significant negative correlation was found in married women [(25)]. Skałba, P. et.al found that there were no correlations between BMI and AMH levels in normal and overweight study groups [(26)]. Another study found that serum AMH levels were 33% lower in overweight and obese women compared with women with normal weight [(27)]. The physiological mechanism has not been clearly identified, while conflicting study results on the association between AMH levels and BMI have been reported.It has been reported that obese women have alterations in their ovarian follicular environment, which causes a negative impact on AMH production [(28,29)].The possible explanation of inverse relationship between BMI and AMH could be that obesity is associated with decreased ovarian reserves or that obesity is associated with follicular dysfunction [(30)]. Insulin Resistance (IR), that related to general obesity, might exert its negative effect on AMH directly or indirectly to decrease the inhibitory effect of AMH on follicular development, thus increasing the sensitivity of granulosa cells to follicle-stimulating hormone [(31)]. In addition, hyperinsulinemia resulting from IR stimulates ovarian steroidogenesis and inhibits sex hormone-binding globulin production in the liver, thereby increasing the availability of free androgens. The adipose tissue also provides storage and a metabolic site for various lipid-soluble steroids, such as androgens, which contributes further to hyperandrogenism and decease female fertility [(32)].

AMH and Central Obesity

The link between AMH and central obesity may be related to that central obesity is associated with greater insulin resistance (IR) likely mediated by free fatty acids and the paracrine actions of the abdominal depot than general obesity [(33)]. Moreover, central obesity also worsens the insulin-related metabolic and reproductive features of PCOS [(34)].

Our study showed that women with substantially increased risk (WC >88 cm), and increased risk for metabolic complications (WC >80-88 cm), have significantly lower AMH concentration than those with no metabolic risk (WC <80 cm).

Also when the subjects divided on the bases of WHO criteria for central obesity with WC ≤80 for no central obesity, and WC >80 cm for central obesity, we found in this study that women with central obesity had significantly lower levels of AMH than those without it ($p < 0.05$).

Similar results to ours were found in a study of 951 healthy reproductive-age women, when low AMH levels were associated with higher waist circumference [35].

Another study agreed with the result of our study, Eman H El-Adawy and her colleagues found that AMH levels in obese group were significantly lower than control group, and there were significant negative correlations between each of BMI, WC with AMH [36].

Xiying Zeng, et al. found that AMH was associated only with central obesity but not with general obesity in women with PCOS [37], a result that agree with the current finding.

The results of the study of Gorkem U et al. showed that there were no significant differences in BMI and WC in three groups of participants categorized by ovarian reserve pattern; low, normal, and high. Their comparisons of AMH and WC revealed no significant differences in women with all ovarian reserve patterns [38].

Athanasia Piouka and colleague found that AMH levels were negatively correlated with WC [39]. While a study of Leana Alaa Abbas et al. showed that there was no significant correlation between AMH level and waist circumference [40].

AMH and Marital Status

In this study, AMH level was significantly lower in married women when compared to single women. At the level of our country, Reyam E. Ahmed and her colleague showed that a non-significant correlation was found between AMH levels and BMI in single women, while a significant negative correlation was found in married women [25]. This finding could be related to increase BMI and WC in married women than singles as we found in our study or could be related to age factor .

AMH and Age

We found a significant difference in the AMH concentration between four age groups; with women in the age group of ≥ 40 years have the lowest AMH concentration, and women between 20-29 years have the highest concentration. This is consistent with the similar previous findings.

Scientists found that AMH level peaks at around 25 years of age and studies showed an inverse correlation with age and AMH, as AMH showed a longitudinal decline over time after peaking in the mid-twenties, suggesting that AMH reflects the decline in the ovarian follicular pool with age better than any other ovarian reserve markers. Because of the convenience of sampling regardless of menstrual cycle and known age-specific values, AMH is now preferred as a bio-marker to evaluate ovarian reserve in women[41].

CONCLUSION:

AMH level was significantly lower in obese and overweight women than in normal weight women and in women with increased and substantially increased risk for metabolic complication than with those without metabolic risk. Women with central obesity had significantly lower levels of AMH than those without it, Same for married women than in singles. There was also a significant difference in the AMH concentration between the four age groups; with women in the age group of ≥ 40 years have the lowest AMH concentration.

Recommendations:

1. We recommend to consider central obesity depending on WC as an important impact factor on female fertility in addition to BMI as a measure for general obesity .
2. We depended in this study on anthropometric measures for general and central obesity , further studies can be done with more accurate measurements for body composition (central and peripheral obesity), like CT or MRI
3. More studies can be performed to find if there is a correlation between AMH level and marital status .

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