

The Relationship Between Insulin Resistance and Obesity and Serum Anti-mullerian Hormone Level in Chinese Women With Polycystic Ovary Syndrome: A Case-control Study.

Han Zhao

Shengjing Hospital of China Medical University <https://orcid.org/0000-0002-8907-3913>

Dexin Zhou

Dalian City Third People's Hospital

Cong Liu

Shengjing Hospital of China Medical University

Le Zhang (✉ zhangle198626@163.com)

China Medical University

Research

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Abstract

Background

Anti-Mullerian Hormone (AMH) has an important role in the pathophysiological process of polycystic ovary syndrome (PCOS) by regulating follicular development and is closely related to the severity of PCOS. Previous studies have suggested that AMH levels in PCOS is related to hyperandrogenemia levels and are affected by obesity and insulin resistance. However, the exact relationship between AMH levels and obesity and insulin resistance remains unclear. We aimed to elucidate the relationship between insulin resistance and obesity and serum AMH levels in women with PCOS.

Methods

We conducted a retrospective study of 220 women with PCOS who had undergone an assortment of physical, endocrine, and metabolic assessments. AMH levels and various other indicators of PCOS in patients with different body mass indices (BMI) and Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) levels were compared. Independent sample t-tests were performed to compare two groups. Pearson correlation analysis was performed to study the correlation between AMH and age, obesity, IR, and other indicators of PCOS, and multiple linear regression analysis was performed to determine the factors influencing AMH. Bilateral tests were performed for all statistical tests. The data were analysed using SPSS v25.0. Statistical significance was defined as a two-sided P-value of less than 0.05.

Results

We found that >50% of patients with PCOS had insulin resistance, obesity, hyperandrogenemia, and abnormal glucose tolerance. AMH, testosterone (T), and HOMA-IR levels were affected by age, and older participants had lower AMH, HOMA-IR, and androgen levels ($P < 0.05$). Glycated hemoglobin levels were higher and AMH, luteinizing hormone (LH)/follicle-stimulating hormone (FSH), and LH levels were lower in non-obese individuals than in obese individuals (both $P < 0.05$). Participants in the non-insulin resistant (IR; NIR) group were older than those in the IR group ($P < 0.05$). AMH, LH, LH/FSH, and T levels in the IR group were significantly higher than those in the NIR group ($P < 0.05$). AMH levels were positively correlated with LH, LH/FSH, T, fasting insulin (FINS), and HOMA-IR levels as well as the free androgen index and negatively correlated with age, BMI, and sex hormone binding globulin levels ($P < 0.05$). Through multiple linear regression, we found that AMH levels could be explained by T, LH/FSH, FINS, sex hormone binding globulin, LH levels, and BMI.

Conclusions

Serum AMH levels were closely related to metabolic abnormalities in PCOS. In patients with PCOS, AMH levels were positively correlated with HOMA-IR levels and negatively correlated with BMI. Thus, AMH combined with BMI and HOMA-IR levels could help determine the severity of PCOS.

Plain English Summary

AMH has an important role in the pathophysiological process of PCOS by regulating follicular development and is closely related to the severity of PCOS. Serum AMH levels are independent of the menstrual cycle and are more sensitive and specific than ultrasound as a way of the detection of PCOS. Previous studies have suggested that AMH levels in PCOS is related to HA levels and are affected by obesity and IR. Obesity and IR can aggravate the ovulation disorder associated with PCOS, increase the risk of pregnancy, significantly affect the quality of life, and lead to metabolic and reproductive abnormalities in offspring, resulting in a huge economic burden to the society; however, the exact relationship between AMH levels and obesity and IR remains unclear. This study aimed to explore the relationship between serum AMH levels and IR and obesity in PCOS and provide insight into optimal clinical treatment.

Background

Polycystic ovary syndrome (PCOS) is a common reproductive, endocrine, and metabolic disease in women of reproductive age, with a prevalence rate of approximately 4-21% [1,2], including in mainland China. It is mainly characterized by hyperandrogenemia (HA), chronic anovulation, and polycystic ovaries and is accompanied by metabolic abnormalities such as insulin resistance (IR) and obesity [3,4]. Anti-Mullerian hormone (AMH) is a member of the transforming growth factor superfamily and is a reliable indicator of ovarian reserve. AMH has an important role in the pathophysiological process of PCOS by regulating follicular development and is closely related to the severity of PCOS. More, serum AMH levels are independent of the menstrual cycle and are more sensitive and specific than ultrasound. Recently, AMH have been reported as an independent predictor of PCOS [5-8]. Previous studies have suggested that AMH levels in PCOS is related to HA levels and are affected by obesity and IR[9,10]. Obesity and IR can aggravate the ovulation disorder associated with PCOS, increase the risk of pregnancy, significantly affect the quality of life, and lead to metabolic and reproductive abnormalities in offspring, resulting in a huge economic burden to the society. However, the exact relationship between AMH levels and obesity and IR remains unclear. This study aimed to explore the relationship between serum AMH levels and IR and obesity in PCOS and provide insight into optimal clinical treatment.

Materials And Methods

Participants

We enrolled 220 Chinese women with PCOS aged 20–39 years who visited the endocrinology clinic at Shengjing Hospital of China Medical University between January 2018 and July 2021. The diagnosis of PCOS was based on the diagnostic criteria of the 2018 Chinese PCOS Guidelines for women of reproductive age [11], which include the following: rare menstrual cycle, amenorrhea, irregular uterine bleeding, or irregular menstrual volume. The diagnosis is also based on at least one of the following criteria: hyperandrogen performance, hirsuteness, or HA; manifestations of HA, including acne and

hirsutism; and biochemical indexes of HA (i.e., testosterone [T] > 0.75 ng/mL) or a polycystic ovary on ultrasonography. The exclusion criteria were as follows: Cushing's syndrome, abnormal uterine bleeding, primary amenorrhea, hypothalamic amenorrhea, pituitary amenorrhea, uterine amenorrhea, hyperprolactinemia, ovarian premature aging, functional ovarian tumors, theca cell proliferation, adrenal cortex hyperplasia or tumors, thyroid dysfunction, autoimmune disease, malignant tumor, diseases of the central nervous system or other conditions caused by HA, ovulation disorders, and new prescriptions after enrollment. A complete medical history was required for all subjects, and no steroid of any kind was administered before the sample was taken.

This study was approved by the Institutional Review Board at China Medical University, and informed consent was obtained from each of the patients before the study.

Assessments

Data on the following demographic characteristics were collected: age, height, and weight. From these data, body mass index (BMI) was calculated. Venous blood levels were measured on days 2–5 of the menstrual cycle or when no dominant follicles were found on gynecological ultrasound. Serum AMH levels were determined by enzyme-linked immunosorbent assay. Levels of estradiol (E2), follicle-stimulating hormone (FSH), luteinizing hormone (LH), T, prolactin (PRL), sex hormone binding globulin (SHBG), and other sex hormones were measured using an electrochemical luminescence analyzer. Fasting plasma glucose (FPG) and fasting insulin (FINS) levels were measured using an automatic biochemical analyzer. Glycated hemoglobin (HbA1c) levels were determined using an automatic HbA1c detector (high pressure liquid chromatography). On the day of blood collection, the ovarian volume and number and size of ovarian follicles on each side were determined by ultrasound examination. Those who had never engaged in sexual activity underwent transabdominal ultrasound examination, and those who had engaged in sexual activity underwent transvaginal ultrasound examination.

The Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) level was calculated using the following formula [12]:

$$\text{HOMA-IR} = \frac{\text{FPG} \times \text{FINS}}{22.5}$$

HOMA-IR is currently the most commonly used clinical indicator to evaluate the degree of IR. China's diabetes cooperative group defines HOMA-IR ≥ 2.69 as IR, and obesity is defined as a BMI ≥ 25 kg/m² according to the World Health Organization (WHO)'s standard in Asia [13,14].

On the basis of age, participants were divided into the 20–29-year-old group (131 women) and 30–39-year-old group (89 women). The participants were also divided into two groups according to BMI: non-obese (BMI < 25 kg/m², 93 patients) and obese (BMI ≥ 25 kg/m², 127 patients). Finally, according to the

level of HOMA-IR, 126 women were categorized into the IR group (HOMA-IR \geq 2.69) and 94 women were categorized into the non-IR (NIR) group (HOMA-IR $<$ 2.69).

Statistical analyses

SPSS (version 25.0; IBM, Armonk, NY, USA) was used to perform all statistical analyses. All data were tested for normality and homogeneity of variance. Normally distributed data are expressed as means \pm standard deviations, and non-normally distributed data as expressed as medians and interquartile ranges. Independent sample t-tests were performed to compare two groups. Pearson correlation analysis was performed to study the correlation between AMH and age, obesity, IR, and other indicators of PCOS, and multiple linear regression analysis was performed to determine the factors influencing AMH. Bilateral tests were performed for all statistical tests. $P < 0.05$ was considered statistically significant.

Results

Demographics

The mean age, AMH level, BMI, HOMA-IR level, and T level of our participants were 28.13 ± 4.29 years, 7.97 ± 5.10 ng/mL, 27.21 ± 4.85 kg/m², 4.31 ± 3.10 , and 0.79 ± 0.34 ng/mL, respectively. IR was present in 126 (57%) participants, 127 (57%) were obese, 110 (50%) had abnormal glucose tolerance, 167 (80%) had an LH/FSH >1 , and 119 (54.1%) had HA.

Differences in age groups

By comparing the two age groups, we found that women in the 20–29-year-old group showed significantly higher AMH, LH, T, E2, FINS, HOMA-IR, and LH/FSH levels and free androgen index (FAI) than women in the 30–39-year-old group (Table 1). This suggests that these hormone levels decrease with an increase in the age of patients with PCOS. Notably, there was no significant difference in BMI and HbA1c, FPG, PRL, FSH, progesterone (Prog), and SHBG levels between the two age groups.

Differences in BMI groups

We found that HbA1c was significantly higher in the obese group than in the non-obese group. AMH, LH/FSH, and LH levels in the obese group were significantly lower than those in the non-obese group, suggesting that BMI in PCOS has an inverse relationship with AMH, LH/FSH, and LH levels and a positive relationship with HbA1c. There were no differences in FAI, T, E2, FPG, PRL, FSH, Prog, SHBG, FINS, and HOMA-IR levels as well as age between the two groups (Table 2).

Correlation between AMH and other indicators in the HOMA-IR groups

We found that the ages of the participants in the NIR group were significantly higher than that of those in the IR group. AMH, LH, LH/FSH, and T levels were significantly higher in the IR group than in the NIR

group; a larger HOMA-IR suggests higher AMH, LH, LH/FSH, and T levels and a lower age. There were no differences in FAI, HbA1c, E2, PRL, FSH, Prog, and SHBG levels between the two groups (Table 3).

Correlation between AMH and other indicators

AMH was correlated with the following factors: BMI ($r = -0.208$, $P = 0.002$); age ($r = -0.315$, $P < .001$); and FINS ($r = 0.265$, $P = 0.000$), HOMA-IR ($r = 0.223$, $P < .001$), LH ($r = 0.223$, $P < .001$), LH ($r = 0.142$, $P = 0.049$), LH/FSH ($r = 0.311$, $P < .001$), FAI ($r = 0.169$, $P = 0.018$), T ($r = 0.356$, $P < .001$), and SHBG ($r = -0.142$, $P = 0.049$) levels. Notably, AMH was positively correlated with LH, LH/FSH, FAI, T, FINS, and HOMA-IR levels, whereas it was negatively correlated with age, BMI, and SHBG. In order of size of correlation, T showed the highest correlation, whereas BMI was the lowest ($T > \text{age} > \text{LH/FSH} > \text{FINS} > \text{HOMA-IR} = \text{LH} > \text{BMI}$). There was no correlation between AMH and HbA1c, E2, FPG, Prog, PRL, and FSH levels (Table 4).

Multiple linear regression analysis affecting AMH levels

Multiple linear regression analysis using AMH level as the dependent variable and LH, LH/FSH, FAI, T, FINS, and HOMA-IR levels; age; BMI; and SHBG as independent variables revealed statistically significant results ($F = 8.749$, $P < 0.01$). We found that AMH levels could be explained in terms of T, LH/FSH, FINS, SHBG, and LH levels and BMI using the following formula, indicating that for each 1 kg/m² increase in BMI, the AMH would decrease by 0.172 ng/mL. Similarly, with other factors unchanged, FINS and AMH increased by 1 mmol/L and 0.220 ng/mL, respectively ($P < 0.05$). The regression coefficients of the other variables are shown in Table 5.

Discussion

This study aims to lay a foundation for the future exploration of ovulation and metabolic abnormalities in PCOS patients in China. We found that >50% of patients with PCOS had IR, obesity, HA, and abnormal glucose tolerance. AMH, T, and HOMA-IR levels were affected by age, and older participants had lower AMH, HOMA-IR, and androgen levels. Glycated hemoglobin levels were higher and AMH, LH/FSH, and LH levels were lower in non-obese individuals than in obese individuals. Participants in the NIR group were older than those in the IR group. AMH, LH, LH/FSH, and T levels in the IR group were significantly higher than those in the NIR group. AMH levels were positively correlated with LH, LH/FSH, T, FINS, and HOMA-IR levels as well as the FAI and negatively correlated with age, BMI, and SHBG levels. Through multiple linear regression, we found that AMH levels could be explained by T, LH/FSH, FINS, SHBG, and LH levels and BMI.

PCOS is a complex reproductive, endocrine, and metabolic disease. HA and ovulation dysfunction are the main clinical difficulties in patients with PCOS. Importantly, > 50% of patients with PCOS also suffer from additional metabolic diseases, such as IR and obesity [15]. PCOS further aggravates the accompanying HA and ovulation disorder, severely affecting the physical and mental health of women of reproductive age. AMH is a dimeric glycoprotein synthesized by ovarian granulosa cells. Its secretion is primarily

influenced by the early follicular absorption rate in the follicular cysterna. It is independent of changes in the menstrual cycle and is a reliable indicator for the clinical evaluation of ovarian reserves. AMH levels in patients with PCOS are 2–3 times higher than that of those in healthy individuals [16] and are consistent with the increase in the number of intracavity follicles (AFCs). Previous studies have suggested that AMH measurements could be helpful in the diagnosis and assessment of the severity of PCOS; however, serum AMH levels are affected by multiple factors, such as the environment and heredity. Therefore, understanding the influencing factors of AMH in women with PCOS is advantageous to better understand the clinical significance of AMH level fluctuations.

IR and compensatory hyperinsulinemia (HI) are some of the causes of HA and ovulation dysfunction in women with PCOS. In women with PCOS, IR was present in 50–70% of patients regardless of obesity. Researchers believe that the inability of insulin to bind to its receptor and the change in insulin signal transduction can lead to IR. IR and HI can increase the free T level by stimulating the production of ovarian androgen and inhibiting the synthesis of SHBG in the liver. Moreover, it can also increase adrenal androgen levels, thereby stimulating the production of ovarian steroids mediated by LH and preventing follicular development [17]. In the follicular fluid of women with PCOS, the imbalance between oxygen free radicals or reactive oxygen species and antioxidant factors can lead to cell damage, which prevents oocyte maturation and decreases embryo quality. Meanwhile, the inflammatory environment caused by oxidative stress in women with PCOS promotes the occurrence of IR and HA [18]. IR and HI can be further accompanied by a series of metabolic abnormalities, such as abnormal glucose tolerance, uric acid metabolism, dyslipidemia, hypertension, and endothelial dysfunction [19]. IR in women with PCOS who are not pregnant and in early pregnancy not only increases the incidence of hypertension, gestational diabetes, and preeclampsia but also aggravates neonatal complications such as congenital malformations in early progeny and the long-term risk of IR, obesity, and diabetes in later progeny [20]. IR and HA are mutually causal, which can further worsen the metabolic disorder.

Previous studies have suggested that AMH levels are positively correlated with HA and LH and significantly negatively correlated with age in patients with PCOS, which is consistent with our current results; however, the exact relationship between IR and AMH levels is unclear, and studies on the correlation between IR and AMH levels in patients with PCOS have been reported. A relevant analysis of AMH genotypes in PCOS found that there were significant differences in the distribution of AMH genotypes between women with PCOS with IR and healthy women, but there were no differences in the distribution of AMH genotypes between women with PCOS without IR and healthy women. When metformin, an insulin sensitizer, was used to treat PCOS for 2 months, the serum AMH level decreased and ovulation increased, suggesting that there is an etiological relationship between AMH levels and IR-PCOS, which may be mediated by LH and T levels. Previously, researchers believed that LH could cause a four-fold elevation in AMH production in ovarian granulosa cells of women with PCOS and elevate AMH expression with or without ovulation. Androgens can also stimulate FSH independent of follicular development and may increase AMH production [21]. Although the internal mechanism of the relationship between AMH levels and PCOS is currently unclear, it appears that LH and androgens are related to the correlation between IR and AMH levels in PCOS. Wiweko et al.'s study also revealed that

serum AMH was significantly correlated with the HOMA-IR level, and there were differences between different PCOS phenotypes [22]. In this study, 57% of our participants presented with IR, and the AMH, LH, LH/FSH, and T levels in women with IR-PCOS were significantly higher than those in women with NIR-PCOS; however, there was no difference in BMI. We determined that FINS and HOMA-IR were significantly positively correlated with AMH levels, which suggests that PCOS is independent of obesity and IR. This also shows that there is a difference in AMH levels among patients with PCOS with or without IR, suggesting that AMH levels can indirectly reflect the severity of PCOS. Our results are consistent with those of previous studies, suggesting that AMH levels are positively correlated with IR and HI levels in patients with PCOS.

In China, 34.1–43.3% of women with PCOS are obese [23]. The negative effects of obesity on reproductive health and fertility, such as ovulation dysfunction, infertility, abortion, and related pregnancy complications, are well documented [24]. Notably, obesity is one of the main contributors to the development of PCOS, can inhibit the production of gonadotropin through IR and produces circulating T, and can lead to IR and HI, thereby reducing the secretion of SHBG and resulting in HA. Both obesity and IR can disrupt the development of female antral follicles, interfere with the hypothalamic-pituitary-ovary axis, and lead to chronic ovulation failure [25]. Studies have also found that obese women with PCOS have higher infertility rates, poor response to ovulation induction drugs, poor embryo quality, a low success rate of in vitro fertilization, and significantly increased adverse pregnancy outcomes [20]. Moreover, a recent meta-analysis demonstrated that BMI was negatively correlated with AMH [26]. In obese women, changes in the ovarian follicular microenvironment, including steroidogenesis, metabolism, and inflammation, indirectly affect AMH levels. A decrease in AMH levels has been suggested to be the result of metabolism, storage, and clearance in obese individuals.

Piouka et al. demonstrated that the serum AMH levels of overweight and obese women with PCOS were significantly lower than those of lean women with PCOS [27]. Previous studies on the relationship between obesity biomarkers and AMH levels in women with PCOS have also revealed conflicting reports, which can vary depending on the definition of obesity and grouping based on BMI. In this study, participants were divided into obese and non-obese groups according to the WHO standard for obesity in Asia, and we found that AMH, LH/FSH, and LH levels in the obese group were lower than those in the non-obese group. Correlation analysis of AMH also showed that there was a significant negative correlation between AMH levels and BMI, and BMI could independently affect AMH levels, supporting the concept that follicular development may be impaired in women with PCOS with increased BMI. Obese women with PCOS primarily show abdominal obesity and large waist and hip circumferences, and other records of patients with PCOS were not included in our present study; therefore, there may be some bias in the determined relationship between obesity and AMH and HOMA-IR levels.

Normal ovarian development is affected by the factors inside and outside the ovary; factors inside the ovary include growth factors, cytokines, and inhibin in the follicular fluid, and the concentration is related to the plasma level. External factors include FSH deficiency, LH hypersecretion, high androgen levels in the ovaries and adrenal gland, IR, and HI. Unbalanced development of these factors will alter ovarian

development and the generation of mature oocytes, thus affecting the fertility of women with PCOS. Most women with PCOS are also afflicted by manifestations of metabolic syndrome such as obesity, hypertension, dyslipidemia, and IR. Up to 30–40% of women with PCOS have impaired glucose tolerance, and up to 10% develop type 2 diabetes before the age of 40 years [28]. In this study, AFC was not included because of the lack of specific values after their number exceeded 12 on ultrasound, and the large number of reports on the correlation between AMH and AFC; however, as the serum AMH level reflects small follicles that cannot be observed on ultrasound, the theoretical AMH level is more accurate than the AFC level, suggesting that the AMH level may play a role in the diagnosis of PCOS. Because of the correlation between AMH levels and obesity and IR, we believe that the clinical combination of AMH and HOMA-IR levels and BMI could be used to help determine the severity of endocrine and metabolic disorders in patients with PCOS. The exact mechanism of the relationship between AMH levels and IR and obesity in PCOS needs to be further elucidated, and there is no consistent serum AMH diagnostic threshold for PCOS. Moreover, because of the lack of a control group, we did not further elaborate on the diagnostic significance of AMH levels in PCOS. These are the limitations of the study. Therefore, we hope that more preclinical and clinical studies are conducted in the future to verify the role of AMH levels in the prediction, prevention, and treatment of PCOS and to provide more of a theoretical basis for the exploration of the etiology of PCOS.

Conclusions

We found that serum AMH levels were associated with metabolic abnormalities in women with PCOS. AMH levels were positively correlated with HOMA-IR levels and negatively correlated with BMI and that the relationship between AMH levels and IR was independent of BMI. AMH levels combined with BMI and HOMA-IR might help to estimate the severity of PCOS. Future studies are needed to explore the potential mechanism linking BMI, HOMA-IR, and AMH might lead to important insights into ovarian physiology in patients with PCOS.

Abbreviations

PCOS Polycystic ovary syndrome

AMH Anti-Mullerian Hormone

T Testosterone

BMI Body mass index

FINS Fasting insulin

HOMA-IR Homeostatic Model Assessment of Insulin Resistance

LH Luteinizing hormone

FSH	Follicle-stimulating hormone
IR	Insulin resistant
NIR	Non-insulin resistant
SHBG	Sex hormone binding globulin
FAI	Free testosterone index
PRL	Prolactin
Prog	Progesterone
ART	Assisted Reproductive Technology
AFC	Antral follicle count
HA	Hyperandrogenemia
TG	Triglyceride
TC	Total cholesterol
HDL-C	High density lipoprotein cholesterol
LDL-C	Low density lipoprotein cholesterol

Declarations

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Availability of data and materials

The datasets supporting the conclusions of this article are included within the article and its additional file.

Ethics approval and consent to participate

Not applicable.

Competing interests

The authors declare that they have no competing interests

Consent for publication

Not applicable.

Author Contributions

H.Z. conceived the study. C.L. and L.Z. designed the study. H.Z. and Z.D. wrote the study protocol, registered the study protocol. H.Z. and Z.D. developed the statistical methods and analyzed the data. H.Z. wrote the first draft of the manuscript. C.L. and L.Z. critically checked its content and approved its final version. All authors agreed with the results and conclusions of this article.

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Tables

Table 1: PCOS general information and comparison of PCOS general information among different age groups.

General indicators	Total PCOS [n=220]	20 to 29 years old [n=131]	30 to 39 years old [n=89]	Age group T	Age group P
AMH [ng/mL]	7.97±5.10	8.90±5.22	6.61±4.61	3.347	0.001#
LH/FSH	1.65±0.91	1.83±0.91	1.39±0.85	3.615	0.001#
LH[mIU/mL]	10.57±6.23	11.84±6.24	8.70±5.75	3.774	0.000#
FAI[%]	18.52±16.86	23.77±19.10	10.11±6.51	5.975	0.000#
T[ng/mL]	0.79±0.34	0.99±0.29	0.50±0.14	15.111	0.000#
E2[pg/mL]	65.48±54.77	73.11±63.34	54.12±36.12	2.547	0.012*
BMI[kg·m ⁻²]	27.21±4.85	26.85±4.99	27.75±4.60	-1.359	0.176
HbA1c[%]	5.87±1.19	5.93±1.28	5.78±1.09	0.599	0.550
FPG [mmol/L]	5.59±1.14	5.64±1.00	5.50±1.33	-1.090	0.277
FINS [mmol/L]	16.94±9.26	18.08±10.04	15.23±7.71	2.259	0.025*
HOMA-IR	4.31±3.10	4.67±3.33	3.78±2.66	2.110	0.036*
PRL [umol/L]	11.99±11.09	11.54±12.18	12.63±9.34	-0.707	0.132
FSH [mIU/mL]	6.53±1.89	6.67±2.02	6.32±1.68	1.364	0.174
Prog [ng/mL]	0.93±1.48	0.94±1.15	0.91±1.9	0.152	0.879
SHBG [nmol/L]	25.60±26.11	24.21±25.59	27.80±26.95	-0.926	0.356

Note: * $P < 0.05$, # $P < 0.01$.

Table 2: Comparison of AMH level and other indexes under different BMI

General indicators	Non-obese group (n=93)	obese group (n=127)	T	P
AMH (ng/mL)	8.83±5.44	7.35±4.75	2.146	0.033*
LH/FSH	1.82±0.91	1.54±0.91	2.260	0.025*
LH (mIU/mL)	11.68±6.30	9.75±6.07	2.298	0.023*
FAI (%)	18.08±14.39	18.80±18.34	-0.290	0.772
T (ng/mL)	0.84±0.37	0.76±0.31	1.694	0.092
E2 (pg/mL)	72.97±66.92	60.06±43.42	1.729	0.085
Age (years)	27.61±4.15	28.50±4.36	-1.528	0.128
HbA1c (%)	5.50±0.52	6.04±1.37	-2.137	0.035*
FPG (mmol/L)	5.73±1.15	5.47±1.14	1.592	0.113
FINS (mmol/L)	18.61±10.61	15.70±7.94	-0.505	0.614
HOMA-IR	4.86±3.62	3.91±2.60	-0.172	0.863
PRL (umol/L)	12.81±14.57	11.42±7.81	0.907	0.365
FSH (mIU/mL)	6.58±2.19	6.49±1.65	0.335	0.738
Prog (ng/mL)	1.02±1.81	0.86±1.19	0.808	0.420
SHBG (nmol/L)	25.39±25.27	25.74±26.74	0.928	0.928

Note: * $P < 0.05$, # $P < 0.01$.

Table 3: Comparison of AMH levels and other indicators under different HOMA-IR conditions

General indicators	NIR [n=94]	IR[n=126]	T	P
AMH[ng/mL]	6.03±3.74	9.39±5.48	-5.121	0.000#
LH/FSH	1.47±0.77	1.79±0.98	-2.647	0.009#
LH[mIU/mL]	9.49±5.77	11.36±6.46	-2.215	0.028*
FAI[%]	18.51±19.92	18.51±14.26	-0.001	0.999
T[ng/mL]	0.73±0.31	0.83±0.35	-2.138	0.034*
E2[pg/mL]	60.81±44.05	68.93±61.44	-1.086	0.279
Age[years]	28.89±4.32	27.57±4.19	2.255	0.025*
HbA1c[%]	5.74±1.01	5.96±1.31	-0.905	0.368
BMI[kg·m ⁻²]	27.62±4.63	26.91±4.99	1.075	0.284
PRL[umol/L]	11.89±9.38	12.07±12.24	-0.114	0.909
FSH[mIU/mL]	6.61±2.23	6.47±1.61	0.521	0.603
Prog[ng/mL]	0.90±1.19	0.95±1.67	-0.228	0.820
SHBG[nmol/L]	26.59±31.07	24.86±21.78	0.454	0.651

Table 4: Correlation analysis of AMH and various indexes

Note: * $P < 0.05$, # $P < 0.01$.

Table 5: Multiple linear regression analysis of AMH influencing factors

Note: * $P < 0.05$, # $P < 0.01$.

indicators	AMH	
	R	P
BMI kg·m ⁻²	-0.208	0.002#
FINS mmol/L	0.265	0.000#
LH mIU/mL	0.223	0.001#
LH/FSH	0.311	0.000#
FSH mIU/mL	-0.126	0.062
HOMA-IR %	0.223	0.001#
T ng/mL	0.356	0.000#
Age years	-0.315	0.000#
PRL umol/L	-0.121	0.076
HbA1c %	-0.011	0.915
FPG mmol/L	-0.053	0.439
E2 pg/mL	0.003	0.971
Prog ng/mL	0.018	0.796
SHBG nmol/L	-0.142	0.049*

model	Nonstandardized coefficients		standardized coefficients Beta	t	p
	B	Std.Error			
Constant	0.541	7.505		0.072	0.943
T ng/mL	6.581	2.600	0.464	2.531	0.012*
SHBG nmol/L	-0.032	0.014	-0.174	-2.361	0.019*
LH/FSH	2.101	0.719	0.405	2.921	0.004#
LH mIU/mL	-0.214	0.103	-0.284	-2.078	0.039*
FINS mmol/L	0.220	0.092	0.315	2.389	0.018*
BMI kg·m ⁻²	-0.172	0.065	-0.172	-2.636	0.009*