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Effect of body mass index on ovarian reserve and ART outcomes in infertile women: a large retrospective study

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Abstract

Background Obesity poses a significant global health challenge, with profound implications for women's reproductive health. The relationship between ovarian reserve and body mass index (BMI) remains a subject of debate. While obesity is generally associated with poorer outcomes in assisted reproductive technology (ART), the evidence remains inconclusive. This study aimed to investigate the effect of pre-pregnancy BMI on ovarian reserve and ART outcomes in infertile patients.

Methods We conducted a retrospective cohort study involving women who underwent in vitro fertilization (IVF) or intracytoplasmic sperm injection (ICSI) procedures at Tongji Hospital between 2016 and 2023. The study included 30,746 initial fresh cycles and 5,721 singleton deliveries. Patients were stratified by age and further categorized into four BMI groups: lean (< 18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2), and obese (≥ 30.0 kg/m^2). The primary endpoints of the study were pregnancy and perinatal outcomes. To explore the association between BMI and these outcomes, we adjusted for relevant confounding factors and utilized multivariate linear regression models, complemented by multifactorial logistic regression analyses.

Results Anti-Müllerian hormone (AMH) levels were significantly lower in the overweight and obese groups compared to the normal weight group. After adjusting for age, a negative correlation was found between AMH and BMI in the age subgroups of 20–30 and 30–35 years. Among women aged 20–35 years, those in the overweight and obese groups had significantly fewer retrieved oocytes, mature oocytes, and two-pronuclear (2PN) embryos than their normal weight counterparts. Despite these differences, pregnancy outcomes in the overweight and obese groups were comparable to those in the normal weight group across all age categories. Additionally, obesity was linked to an increased risk of gestational diabetes mellitus, hypertensive disorders of pregnancy, and macrosomia.

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Conclusions An age-related decrease in AMH levels was evident with increasing BMI. Although being overweight or obese is associated with poorer embryo and perinatal outcomes, it does not seem to have a substantial impact on fertility.

Keywords Body mass index, AMH, Obesity, ART outcome, Ovarian reserve

Background

Obesity is a pervasive issue with a rising prevalence worldwide, posing a significant public health challenge. Epidemiological studies have consistently shown a strong association between obesity and chronic conditions such as diabetes and cardiovascular diseases [1]. Furthermore, obesity adversely affects reproductive health [2], disrupting sex hormone balance and causing dyslipidemia, which increases the risk of conditions such as polycystic ovary syndrome (PCOS), menstrual irregularities, diminished ovarian reserve, and insulin resistance [3].

Obesity has a direct and multifaceted impact on the outcomes of assisted reproductive technology (ART) in women experiencing infertility. This influence primarily arises from hormonal imbalances, reduced ovarian response, and impaired embryo implantation [3]. Numerous studies have consistently demonstrated that obesity is associated with lower rates of implantation, pregnancy, and live birth, as well as higher rates of miscarriage and adverse perinatal outcomes [4–7]. As a result, weight loss has been recommended to improve pregnancy outcomes in young, obese women, underscoring the potential benefits of managing body weight [5]. However, conclusions in the literature are not uniform, possibly due to variations in study designs and sample sizes. Some studies still suggest that a higher body mass index (BMI) in women does not have a negative impact on pregnancy outcomes following in vitro fertilization [8].

Excessive fat accumulation can disrupt the endocrine system, thereby interfering with normal ovarian function [3]. Ovarian reserve, which refers to the quantity and quality of oocytes, serves as a key indicator of reproductive potential and declines with age [9]. Clinically, markers such as Anti-Müllerian hormone (AMH) levels and Antral Follicle Count (AFC) are commonly used to assess ovarian reserve function and predict the outcomes of ART [10]. Several studies have explored the relationship between ovarian reserve and BMI. While some research indicates a negative correlation between AMH and BMI [11, 12], others suggest a positive correlation [13], and still, other studies find no significant correlation [14]. The discrepancies among these findings may be attributed to the limited sample sizes.

Understanding how obesity affects ovarian reserve and ART outcomes in infertile women is crucial for developing individualized treatment plans and improving success rates. To address this, we conducted a retrospective study examining the impact of pre-pregnancy BMI on ovarian reserve, as well as pregnancy and perinatal outcomes, following assisted reproductive procedures in infertile patients.

Materials and methods

Study design and patients

In this retrospective study, we included 30,746 patients who underwent their first in vitro fertilization (IVF) or intracytoplasmic sperm injection (ICSI) cycle at Tongji Hospital between January 2016 and May 2023. Patients with polycystic ovary syndrome, previous ovarian surgery or endometriosis, ovarian cystadenoma, or missing core data such as BMI as well as those with a history of abortion due to cervical insufficiency in a previous single pregnancy, were excluded. For analysis, the participants were categorized into three age subgroups: 20–30 years, 30–35 years, and 35–45 years. Subsequently, they were classified into four BMI groups based on the WHO standard [15]: lean (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2), and obese (≥30.0 kg/m^2). This study adhered to the Declaration of Helsinki for Human Subjects in Medical Research and received approval from the Ethical Committee of the Reproductive Medicine Center, Tongji Hospital, Tongji Medicine College, Huazhong University of Science and Technology (TJ-IRB20230213).

ART treatment

The controlled ovarian stimulation (COS) protocols were performed as previously described [16]. Various protocols, including the gonadotropin-releasing hormone (GnRH) antagonist and GnRH agonist protocols, as well as other protocols like the mild stimulation and luteal phase stimulation protocols, were employed. The selection of each protocol was based on factors such as maternal age, body mass index (BMI), and ovarian reserve. The dosage of recombinant follicle-stimulating hormone (FSH) was tailored to each patient's ovarian response.

Follicular development was monitored through transvaginal ultrasound. Once the leading follicles reached an average diameter of at least 18 mm, an intramuscular injection of 10,000 IU of human chorionic gonadotropin (hCG) was administered to trigger oocyte maturation. Oocyte retrieval was performed 34–36 h after hCG administration. The retrieved oocytes were then fertilized either by conventional insemination or ICSI. The resulting zygotes were cultured until Day 3 or developed further to the blastocyst stage (Day 5 or Day 6). Embryos

were either transferred with ultrasonographic guidance or cryopreserved for future use.

Main outcomes measurements

AMH and AFC are key indicators of ovarian reserve. These test results are typically obtained within the first 12 months before initiating the IVF/ICSI program. Pregnancy outcomes mainly include clinical pregnancy rate, miscarriage rate, live birth rate, and other factors. A positive pregnancy was confirmed through repeated hCG testing two weeks after embryo transfer. Clinical pregnancy was defined as the presence of an intrauterine gestational sac documented by ultrasound. Biochemical pregnancy was defined as a positive pregnancy test without ultrasound evidence of an intrauterine gestational sac. Ongoing pregnancy was defined as an intrauterine pregnancy lasting beyond 12 weeks. Live birth was defined as the delivery of at least one live newborn after 24 weeks of gestation. Miscarriage was defined as the loss of pregnancy before 20 weeks of gestation. Ectopic pregnancy was identified when a gestational sac was located outside the uterine cavity, confirmed via ultrasonography or pathology.

The normal fertilization rate was defined as the number of two-pronucleus (2PN) embryos divided by the number of retrieved oocytes in IVF, or the number of 2PN embryos divided by the number of metaphase II (MII) oocytes in ICSI. The implantation rate was calculated as the ratio of fetal heartbeats to the number of embryos transferred. Good-quality cleavage stage embryos were defined as those with 7 or 8 blastomeres, a fragmentation rate of less than 20%, and no evidence of multinucleation. Blastocysts were graded morphologically according to the Gardner scoring system [17]. According to Chinese expert consensus, high-quality blastocysts are defined as those at stage 3–4 on day 5 or stage 4–6 on day 6, with A or B scores for both inner cell mass and trophectoderm.

To minimize bias from twin vanishing syndrome and multiple pregnancies, only patients with singleton pregnancies and live births were considered in the analysis of perinatal outcomes. These outcomes included gestational age, mode of delivery, sex, birth weight, preterm birth, very preterm birth, macrosomia, hypertensive disorders of pregnancy, placenta previa, and gestational diabetes mellitus. The diagnosis of gestational hypertension, including preeclampsia and gestational hypertension, was based on the consensus guidelines of the International Society for the Study of Hypertension in Pregnancy. The diagnosis of gestational diabetes mellitus followed established consensus criteria. Preterm birth (PTB) was defined as delivery occurring before 37 weeks of gestation, and very PTB was defined as delivery before 32 weeks.

Statistical analysis

Statistical analyses were performed using SPSS 26.0 (IBM, Chicago, IL) statistical software. Kolmogorov-Smirnov was used for the normality test and Levene's test was used for the homogeneity of variance test. Continuous variables were expressed as median values with corresponding first and third quartiles, and group comparisons were made using the Kruskal-Wallis one-way analysis of variance (ANOVA). Categorical variables were presented as proportions or rates (%), and between-group comparisons were performed using the chi-square test or Fisher's exact test. Bonferroni correction was applied to adjust for multiple comparisons. Pearson's correlation and multiple linear regression analyses were conducted to explore the relationship between BMI and AMH, including age as a covariate. AMH concentrations were log-transformed before analysis due to their non-normal distribution. Binary logistic regression and multiple linear regression analyses were employed to assess the impact of pre-pregnancy BMI on embryo, clinical and perinatal outcomes, adjusting for covariates such as maternal age, type of infertility, duration of infertility in years, cause of infertility, COS protocols, and fertilization methods. A P-value of less than 0.05 was considered statistically significant.

Results

Baseline and characteristics of patients

A total of 30,746 first fresh cycles were included at our center between January 2016 and May 2023. It was categorized into four groups based on pre-pregnancy BMI: 3,072 cycles in the lean group, 22,996 in the normal group, 4,272 in the overweight group, and 406 in the obese group. Table 1 displayed the baseline characteristics of infertile patients across the different BMI groups. Significant differences were observed among the groups in various characteristics, including age, BMI, duration of infertility, type of infertility, basal sex hormone levels, surgical procedures, ovarian stimulation protocols, and the dose and duration of gonadotropin treatment.

The relationship between ovarian reserve and BMI

Supplemental Fig. 1 shows AMH levels across different BMI groups within each age subgroup. Overall, AMH levels were significantly lower in the overweight and obese groups compared to the normal weight group ($P<0.001$). Across all age subgroups, AMH levels generally decreased with increasing BMI, reaching statistical significance only in the age subgroups of 20–30 and 30–35 years. In contrast, AMH levels did not differ significantly among the groups for individuals aged 35–45 years ($P=0.430$).

Figure 1 illustrates the distribution of AMH relative to BMI across various age subgroups. BMI and AMH

Table 1 Baseline characteristics of the study cycles (BMI categorization)

Parameter	Lean < 18.5 (kg/m^2)	Normal weight 18.5–24.9 (kg/m^2)	Overweight 25-29.9 (kg/m^2)	Obese ≥ 30 (kg/m^2)	*P* value
Number of patients	3072	22,996	4272	406	
Age (y)	30.0 (27.0, 33.0) *	31.0 (29.0, 35.0)	32.0 (29.0, 36.0) *	31.0 (28.0, 34.0)	< 0.001
BMI (kg/m2)	17.8 (17.2, 18.3) *	21.4 (20.1, 22.9)	26.4 (25.6, 27.5) *	31.2 (30.5, 32.5) *	< 0.001
Duration of infertility (y)	3.0 (2.0, 4.0)	3.0 (1.5, 4.0)	3.0 (2.0, 5.0) *	3.0 (2.0, 6.0) *	< 0.001
Type of infertility, n (%)					< 0.001
Primary infertility	2185 (71.1) *	14,087 (61.3)	2607 (61.0)	263 (64.8)	
Secondary infertility	887 (28.9) *	8908 (38.7)	1665 (39.0)	143 (35.2)	
Basal FSH (ng/mL)	7.8 (6.7, 9.3)	7.4 (6.3, 8.8)	7.1 (5.9, 8.4)	6.8 (5.8, 8.4)	< 0.001
Basal LH (mIU/mL)	2.6 (1.6, 4.2)	2.7 (1.6, 4.5)	2.9 (1.7, 4.8) *	3.1 (1.9, 5.7) *	< 0.001
Basal P (ng/mL)	0.9 (0.7, 1.2) *	0.8 (0.6, 1.1)	0.7 (0.5, 1.0) *	0.6 (0.4, 0.9) *	< 0.001
Basal E2 (pg/mL)	2736.0 (1759.0, 4325.0) *	2223.0(1434.0, 3609.0)	1802.0(1178.0, 2808.5) *	1654.0(1096.0, 2474.0) *	< 0.001
AMH level (ng/mL)	3.5 (2.0, 5.8) *	3.3 (1.8, 5.5)	3.0 (1.6, 5.0) *	2.6 (1.5, 4.4) *	< 0.001
Main infertility factor, n (%)					
Female factor	1506 (49.0) *	12,384 (53.9)	2298 (53.8)	201 (49.5)	< 0.001
Male factor	652 (21.2) *	3767 (16.4)	701 (16.4)	60 (14.8)	
Female and male factors	630 (20.5)	4791 (20.8)	880 (20.6)	97 (23.9)	
Unknown	284 (9.2)	2054 (8.9)	393 (9.2)	48 (11.8)	
Fertilization method, n (%)					< 0.001
IVF	2032 (66.1)	15,732 (68.4)	2927 (68.5)	287 (70.7)	
ICSI	1040 (33.9) *	7264 (31.6)	1345 (31.5)	119 (29.3)	
Dose of Gn (IU)	4710.0 (3300.0, 6450.0) *	5100.0 (3750.0, 6900.0)	6000.0 (4350.0, 7800.0) *	6900.0 (5250.0, 9300.0) *	< 0.001
Duration of Gn (d)	10.0 (9.0, 11.0)	10.0 (9.0, 11.0)	10.0 (9.0, 11.0) *	10.0 (9.0, 11.0) *	< 0.001
Ovarian stimulation protocols, n (%)					< 0.001
GnRH agonist	1709 (55.6) *	12,067 (52.5)	2032 (47.6) *	170 (41.9) *	
GnRH antagonist	1153 (37.5) *	8508 (37.0)	1766 (41.3) *	191 (47.0) *	
Others	210 (6.8) *	2421 (10.5)	474 (11.1)	45 (11.1)	
Endometrial thickness (mm)	11.1 (9.5, 12.8)	11.0 (9.3, 12.9)	11.2 (9.4, 13.1) *	11.0 (9.6, 12.8)	< 0.001
No. of embryos transferred, n (%)					< 0.001
1	1082 (63.3) *	8833 (67.6)	1758 (70.3)	197 (81.4) *	
2	626 (36.7) *	4228 (32.4)	744 (29.7)	45 (18.6) *	
Embryo type transferred, n (%)					0.401
Cleavage embryo	1510 (88.4)	11,534 (88.3)	2248 (89.8)	218 (90.1)	
Blastocyst	198 (11.6)	1527 (11.7)	254 (10.2)	24 (9.9)	

Note BMI body mass index; *LH* luteinizing hormone; *P* progesterone; *E2* estradiol; *Gn* gonadotropin; *IVF* in vivo fertilization; *ICSI* intracytoplasmic sperm injection; *AMH* Anti Mullerian hormone

*$P < 0.05$ as compared with the normal weight group

exhibited a significant negative correlation overall (r = -0.070, $P<0.001$), as well as within the age subgroups of 20–30 years (r = -0.037, $P<0.001$) and 30–35 years (r = -0.050, $P<0.001$).

A multiple linear regression model adjusted for age was used to further assess the relationship between BMI and AMH (Table 2). Regardless of whether BMI was treated as a continuous or categorical variable, a significant negative correlation between BMI and AMH was observed overall and for women aged 20–30 years or 30–35 years (all $P\leq0.001$). However, for individuals aged 35–45 years, no significant differences were found.

Among women aged 20–30 years, a modest yet significant difference in AFC was observed between the normal weight and lean groups(Supplemental Fig. 2). However, there were no significant differences in AFC between the overweight or obese group and the normal weight group across the overall cohort or within various age subgroups.

Embryo outcomes

Table 3 compared embryo outcomes across various BMI groups. In both the overall cohort and the age subgroups of 20–30 years and 30–35 years, we observed a decline in the number of oocytes retrieved, mature oocytes, and 2PNs with increasing BMI. However, for patients aged 35–45 years, no significant differences were found among the BMI groups. For patients aged 20–30 years, the overweight group showed a substantial reduction in the quality of transferred embryos compared to the normal weight group, which aligned with trends observed in

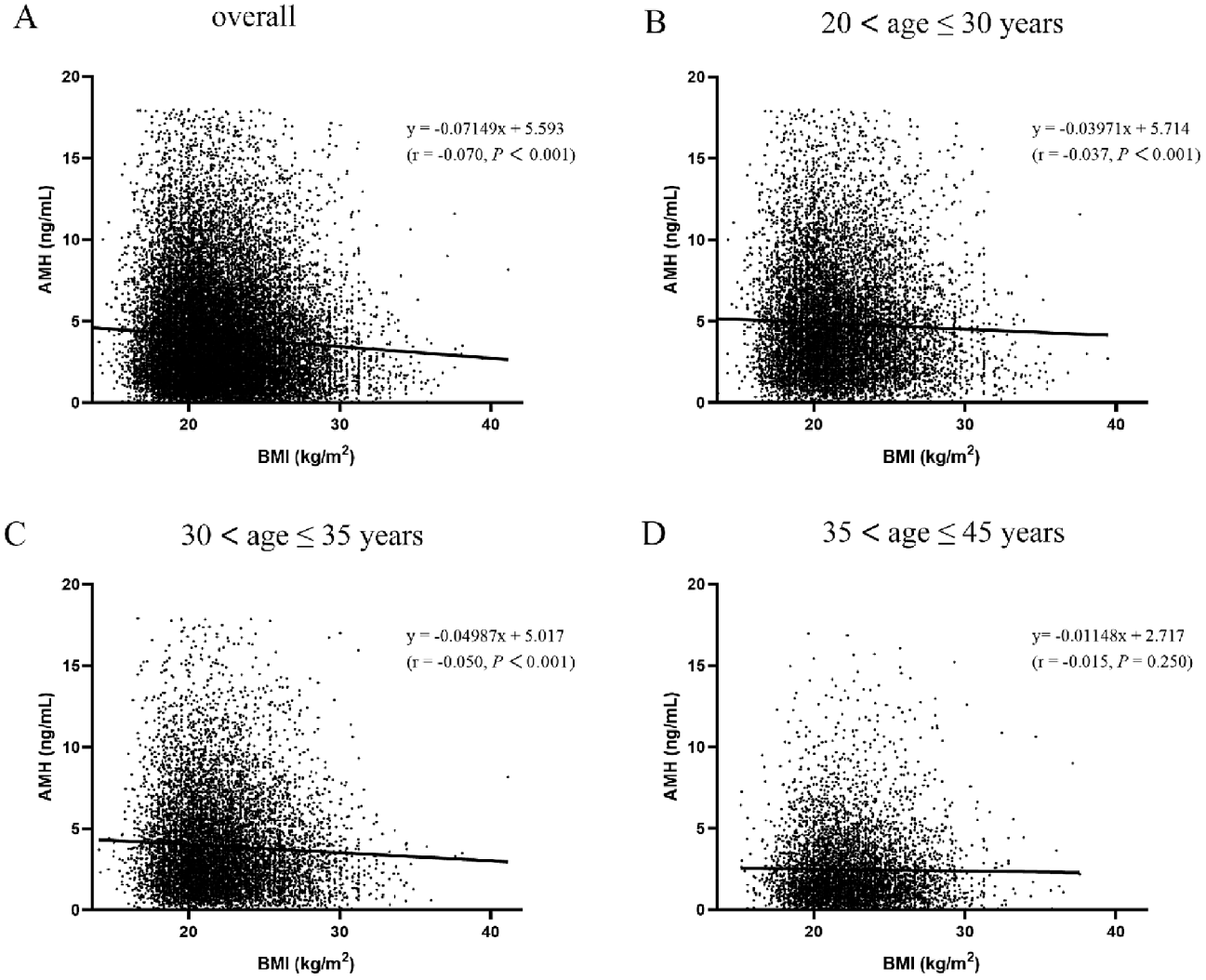

Fig. 1 Changes in Anti-Müllerian hormone (AMH) levels with increasing body mass index (BMI)

Table 2 Multivariate linear regression analysis of the association between BMI and AMH

	Overall		20–30 years		30–35 years		35–45 years	
Parameter	**β (95% CI)**	***P* value**	**β (95% CI)**	***P* value**	**β (95% CI)**	***P* value**	**β (95% CI)**	***P* value**
BMI (kg/m2) [a]	-0.011 (-0.014, -0.008)	< 0.001	-0.011 (-0.016, -0.007)	< 0.001	-0.013 (-0.018, -0.008)	< 0.001	-0.003 (-0.011, 0.005)	0.519
BMI category [b]								
Lean	-0.022 (-0.054, 0.009)	0.167	-0.034 (-0.074, 0.006)	0.098	0.003 (-0.051, 0.057)	0.920	-0.030 (-0.134, 0.075)	0.579
Normal weight	0 (Reference)		0 (Reference)		0 (Reference)		0 (Reference)	
Overweight	-0.063 (-0.090, -0.036)	< 0.001	-0.079 (-0.120, -0.037)	< 0.001	-0.075 (-0.120, -0.030)	0.001	-0.001 (-0.062, 0.060)	0.966
Obese	-0.257 (-0.338, -0.176)	< 0.001	-0.326 (-0.442, -0.211)	< 0.001	-0.219 (-0.349, -0.088)	0.001	-0.184 (-0.395, 0.027)	0.087

Note BMI body mass index; *CI* confidence interval; *AMH* Anti-Müllerian hormone

The results of the multivariate linear regression analysis with AMH as the dependent variable and age and BMI as independent variables

[a]BMI was analyzed as a continuous variable

[b]BMI was analyzed as a categorical variable

the overall population. Despite variations in fertilization and blastocyst formation rates in the overall population, there were no significant differences across BMI groups after age stratification.

The results of multiple linear regression analysis about embryo outcomes were shown in Supplemental Table 4. In the overall cohort, the number of oocytes retrieved, mature oocytes, and 2PNs was significantly lower in the overweight group compared to the normal weight group.

Table 3 Embryo and pregnancy outcomes of each BMI group

Age category	Parameter	Lean < 18.5 (kg/m^2)	Normal weight 18.5–24.9 (kg/m^2)	Overweight 25-29.9 (kg/m^2)	Obese ≥ 30 (kg/m^2)	*P* value
Overall	No. of oocytes retrieved	12.0 (7.0, 17.0) *	11.0 (7.0, 16.0)	10.0 (6.0, 15.0) *	9.0 (5.0, 14.0) *	< 0.001
	No. of mature oocytes	10.0 (6.0, 15.0) *	9.0 (6.0, 14.0)	9.0 (5.0, 13.0) *	8.0 (5.0, 12.0) *	< 0.001
	No. of 2PNs	7.0 (4.0, 11.0) *	6.0 (4.0, 10.0)	6.0 (3.0, 9.0) *	5.0 (3.0, 8.0) *	< 0.001
	Normal fertilization rate (%)	66.7 (52.9, 80.0)	66.7 (50.0, 80.0)	66.7 (50.0, 80.0) *	64.3 (50.0, 80.0)	< 0.001
	Blastocyst formation rate (%)	71.4 (50.0, 87.5)	68.4 (50.0, 86.7)	66.7 (42.9, 85.7) *	75.0 (50.0, 90.9)	< 0.001
	Embryo quality, n (%)					< 0.001
	High-quality embryos	1450 (84.9)	10,980 (84.1)	2019 (80.7) *	208 (86.0)	
	Low-quality embryos	258 (15.1)	2073 (15.9)	482 (19.3)	34 (14.0)	
	Implantation rate (%)	44.8 (1046/2334)	45.2 (7820/17290)	46.7 (1515/3246)	53.3 (153/287) *	0.020
	Clinical pregnancy rate (%)	52.0 (888/1708)	52.4 (6847/13061)	52.5 (1314/2502)	58.7 (142/242)	0.271
	Biochemical pregnancy rate (%)	4.8 (82/1708)	5.7 (739/13061)	6.1 (152/2502)	5.4 (13/242)	0.363
	Ongoing pregnancy rate (%)	46.4 (793/1708)	45.6 (5960/13061)	45.2 (1132/2502)	52.5 (127/242)	0.167
	Ectopic pregnancy rate (%)	0.8 (13/1708)	0.8 (104/13061)	0.9 (23/2502)	0 (0/242)	0.487
	Miscarriage rate (%)	11.1 (99/888)	13.4 (920/6847)	14.5 (191/1314)	13.4 (19/142)	0.148
	Live birth rate (%)	46.2 (721/1561)	45.0 (5289/11761)	44.0 (957/2174)	46.5 (93/200)	0.590
20–30 years	No. of oocytes retrieved	14.0 (9.0, 19.0)	13.0 (9.0, 18.0)	13.0 (9.0, 17.0) *	10.5 (7.0, 17.0) *	< 0.001
	No. of mature oocytes	11.0 (7.0, 16.0)	11.0 (8.0, 16.0)	11.0 (7.0, 15.0) *	9.0 (5.0, 13.0) *	< 0.001
	No. of 2PNs	8.0 (5.0, 12.0)	8.0 (5.0, 11.0)	7.0 (4.0, 10.0) *	6.0 (3.0, 9.0) *	< 0.001
	Normal fertilization rate (%)	66.7 (53.3, 80.0)	66.7 (50.0, 80.0)	65.0 (50.0, 78.6)	63.4 (50.0, 78.4)	0.006
	Blastocyst formation rate (%)	70.0 (50.0, 87.5)	70.0 (50.0, 85.7)	69.6 (50.0, 85.7)	72.7 (50.0, 90.9)	0.406
	Embryo quality, n (%)					< 0.001
	High-quality embryos	869 (85.6)	5207 (85.4)	867 (79.7) *	96 (86.5)	
	Low-quality embryos	146 (14.4)	892 (14.6)	221 (20.3)	15 (13.5)	
	Implantation rate (%)	47.3 (664/1403)	49.1 (4024/8188)	51.8 (739/1426)	54.7 (75/137)	0.057
	Clinical pregnancy rate (%)	55.2 (560/1015)	56.9 (3471/6101)	58.1 (632/1088)	61.3 (68/111)	0.436
	Biochemical pregnancy rate (%)	5.0 (51/1015)	5.5 (335/6101)	6.7 (73/1088)	7.2 (8/111)	0.280
	Ongoing pregnancy rate (%)	50.8 (516/1015)	51.2 (3126/6101)	50.5 (549/1088)	55.0 (61/111)	0.823
	Ectopic pregnancy rate (%)	0.5 (5/1015)	0.8 (47/6101)	1.3 (14/1088)	0 (0/111)	0.141
	Miscarriage rate (%)	8.8 (49/560)	10.7 (372/3471)	12.5 (79/632)	11.8 (8/68)	0.218
	Live birth rate (%)	50.8 (482/948)	50.2 (2822/5616)	49.3 (472/957)	48.9 (45/92)	0.915
30–35 years	No. of oocytes retrieved	11.0 (7.0, 16.0)	11.0 (7.0, 16.0)	11.0 (7.0, 15.0) *	9.0 (5.0, 13.0) *	< 0.001
	No. of mature oocytes	9.0 (6.0, 14.0)	10.0 (6.0, 14.0)	9.0 (6.0, 13.0) *	8.0 (5.0, 11.5) *	< 0.001
	No. of 2PNs	7.0 (4.0, 10.0)	7.0 (4.0, 10.0)	6.0 (4.0, 10.0)	5.0 (3.0, 8.0)	< 0.001
	Normal fertilization rate rate (%)	68.4 (53.8, 81.8)	66.7 (50.0, 80.0)	66.7 (50.0, 80.0) *	64.3 (50.0, 77.8) *	0.016
	Blastocyst formation rate (%)	75.0 (50.0, 88.9)	71.4 (50.0, 87.5)	66.7 (50.0, 85.7)	75.0 (51.3, 93.0)	0.015
	Embryo quality, n (%)					0.322
	High-quality embryos	472 (84.7)	4192 (83.0)	783 (81.6)	84 (86.6)	
	Low-quality embryos	85 (15.3)	857 (17.0)	177 (18.4)	13 (13.4)	
	Implantation rate (%)	43.7 (324/742)	45.8 (2986/6516)	49.4 (605/1224)	54.6 (59/108)	0.015
	Clinical pregnancy rate (%)	49.7 (277/557)	52.5 (2652/5052)	54.4 (522/960)	57.7 (56/97)	0.251
	Biochemical pregnancy rate (%)	5.0 (28/557)	6.0 (301/5052)	4.4 (42/960)	5.2 (5/97)	0.233
	Ongoing pregnancy rate (%)	43.6 (243/557)	45.1 (2278/5052)	47.2 (453/960)	53.6 (52/97)	0.189
	Ectopic pregnancy rate (%)	1.1 (6/557)	0.9 (44/5052)	0.7 (7/960)	0 (0/97)	0.716
	Miscarriage rate (%)	12.3 (34/277)	14.1 (374/2652)	14.6 (76/522)	10.7 (6/56)	0.718
	Live birth rate (%)	42.4 (208/491)	45.1 (1983/4399)	46.8 (379/809)	48.7 (38/78)	0.409
35–45 years	No. of oocytes retrieved	6.0 (4.0, 11.0)	7.0 (4.0, 11.0)	7.0 (4.0, 11.0)	7.0 (3.0, 12.0)	0.907
	No. of mature oocytes	5.0 (3.0, 9.0)	6.0 (3.0, 10.0)	6.0 (3.0, 9.0)	5.0 (3.0, 9.5)	0.960
	No. of 2PNs	3.0 (2.0, 7.0)	4.0 (2.0, 7.0)	4.0 (2.0, 7.0)	4.0 (2.0, 7.0)	0.788
	Normal fertilization rate (%)	66.7 (50.0, 83.3)	66.7 (50.0, 83.3)	66.7 (50.0, 81.8)	66.7 (50.0, 83.3)	0.158
	Blastocyst formation rate (%)	60.0 (40.0, 84.3)	62.5 (33.3, 85.7)	60.0 (33.3, 83.3)	75.0 (24.7, 83.8)	0.555
	Embryo quality, n (%)					0.758
	High-quality embryos	109 (80.1)	1581 (83.0)	369 (81.5)	28 (82.4)	

Table 3 (continued)

Age category	Parameter	Lean < 18.5 (kg/m^2)	Normal weight 18.5–24.9 (kg/m^2)	Overweight 25-29.9 (kg/m^2)	Obese ≥ 30 (kg/m^2)	*P* value
	Low-quality embryos	27 (19.9)	324 (17.0)	84 (18.5)	6 (17.6)	
	Implantation rate (%)	30.7 (58/189)	31.3 (810/2586)	28.7 (171/596)	45.2 (19/42)	0.133
	Clinical pregnancy rate (%)	37.5 (51/136)	37.9 (724/1908)	35.2 (160/454)	52.9 (18/34)	0.205
	Biochemical pregnancy rate (%)	2.2 (3/136)	5.4 (103/1908)	8.1 (37/454)	0 (0/34)	0.014
	Ongoing pregnancy rate (%)	25.0 (34/136)	29.1 (556/1908)	28.6 (130/454)	41.2 (14/34)	0.314
	Ectopic pregnancy rate (%)	1.5 (2/136)	0.7 (13/1908)	0.4 (2/454)	0 (0/34)	0.594
	Miscarriage rate (%)	31.4 (16/51)	24.0 (174/724)	22.5 (36/160)	27.8 (5/18)	0.611
	Live birth rate (%)	25.4 (31/122)	27.7 (484/1746)	26.0 (106/408)	33.3 (10/30)	0.735

Note BMI body mass index; *2PN* two pronuclei

*$P < .05$ as compared with the normal weight group

This trend persisted across the age subgroups of 20–30 and 30–35 years. However, in the 35–45 years age subgroup, only the number of 2PNs showed a significant decline in the overweight group when compared to their normal weight counterparts. Regarding blastocyst formation, a significant reduction was observed in the overall overweight and obese groups compared to the normal weight group. When analyzed by age strata, the obese group in the 20–30 years category showed a notably lower rate of blastocyst formation. In the 30–35 years subgroup, a significant decrease was observed only in the overweight group. Notably, among participants aged 35–45 years, there were no significant differences in blastocyst formation rates across all BMI categories.

Pregnancy outcomes

Table 3 also presented the pregnancy outcomes across different BMI groups.No statistically significant differences were found in the rates of clinical pregnancy, biochemical pregnancy, ongoing pregnancy, miscarriage, or live birth among the BMI groups within each age stratum. After accounting for potential confounding factors, multivariate logistic regression analysis was conducted to explore the impact of BMI on pregnancy outcomes (Supplemental Table 5). In the overall cohort, the obese group exhibited higher rates of clinical pregnancy and ongoing pregnancy compared to those with normal weight (clinical pregnancy: aOR = 1.424; 95% CI, 1.093–1.856; ongoing pregnancy: aOR = 1.474; 95% CI, 1.133–1.916). However, in the age subgroup analysis, significant differences were observed only in the individuals aged 30–35 years (aOR = 1.654; 95% CI, 1.093–2.502). Additionally, the risk of ectopic pregnancy in the overweight group (aOR = 1.919; 95% CI, 1.042–3.536) was significantly higher compared to those with normal weight in the 20–30 years age subgroup. Whether in all cycles or subgroup cycles analysis, biochemical pregnancy, miscarriage, and live birth rates were not associated with BMI after adjustment for confounders.

Perinatal outcomes

The study analyzed a cohort of 5,721 patients with singleton pregnancies and live births to evaluate perinatal outcomes (Table 4). Singleton pregnancies among overweight or obese women showed a higher likelihood of cesarean delivery. Additionally, birth weight increased significantly across all overweight categories, leading to higher prevalence of macrosomia. Overweight and obese individuals had a greater incidence of poor maternal outcomes, such as gestational diabetes mellitus and gestational hypertension, compared to individuals with normal weight across all age subgroups. Within the age subgroup of 30–35 years, the incidence of placenta previa was significantly lower in the overweight group compared to the normal weight group.

A multivariate logistic regression analysis was conducted to assess the influence of BMI on pregnancy outcomes, adjusting for confounding factors such as maternal age, years of infertility, number and type of embryos transferred, and cause of infertility (Supplemental Table 6). No significant differences in perinatal outcomes were observed between the lean and normal groups, except regarding the mode of delivery. In the overall cohort, both the overweight and obese groups showed higher rates of macrosomia (overweight group: aOR = 2.398; 95% CI, 1.762–3.262; obese group: aOR = 5.238; 95% CI, 2.806–9.779), gestational hypertension (overweight group: aOR = 3.873; 95% CI, 2.687–5.581; obese group: aOR = 5.633; 95% CI, 2.482–12.786), and gestational diabetes mellitus (overweight group: aOR = 2.025; 95% CI, 1.526–2.688; obese group: aOR = 2.315; 95% CI, 1.092–4.909) compared to the normal weight group. For individuals aged 20–30 years, both the overweight and obese groups had an increased risk of macrosomia (overweight group: aOR = 2.368; 95% CI, 1.537–3.649; obese group: aOR = 8.710; 95% CI, 3.885–19.529), gestational hypertension (overweight group: aOR = 2.729; 95% CI, 1.522–4.895; obese group: aOR = 4.591; 95% CI, 1.317- 16.000), and gestational diabetes mellitus (overweight group: aOR = 1.647;

Table 4 Perinatal outcomes of each BMI group

Age category	Parameter	Lean < 18.5 (kg/m^2)	Normal weight 18.5–24.9 (kg/m^2)	Overweight 25-29.9 (kg/m^2)	Obese ≥ 30 (kg/m^2)	*P* value
Overall	Number of patients	563	4313	763	82	
	Gestational age (w)	39.0 (38.3, 39.7)	39.0 (38.3, 39.6)	39.0 (38.0, 39.6)	38.8 (37.7, 39.1) *	0.002
	Mode of delivery, n (%)					< 0.001
	Vaginal	201 (35.7) *	1251 (29.0)	151 (19.8) *	7 (8.5) *	
	Cesarean section	362 (64.3) *	3062 (71.0)	612 (80.2) *	75 (91.5) *	
	PTB (< 37w)	38 (6.7)	271 (6.3)	58 (7.6)	10 (12.2)	0.105
	Very PTB (< 32w)	1 (0.2)	27 (0.6)	8 (1.0)	0	0.212
	Sex, n (%)					0.402
	Male	317 (56.3)	2286 (53.0)	416 (54.5)	41 (50.0)	
	Female	246 (43.7)	2027 (47.0)	347 (45.5)	41 (50.0)	
	Birthweight, g	3100.0 (2900.0, 3400.0) *	3250.0 (3000.0, 3550.0)	3400.0 (3100.0, 3700.0) *	3300.0 (3117.5, 3762.5)	< 0.001
	LBW (< 2500 g), n (%)	28 (5.0)	159 (3.7)	28 (3.7)	4 (4.9)	0.468
	Macrosomia (> 4000 g), n (%)	14 (2.5)	157 (3.6)	63 (8.3) *	13 (15.9) *	< 0.001
	Placenta previa, n (%)	17 (3.0)	113 (2.6)	14 (1.8)	3 (3.7)	0.469
	HDP, n (%)	5 (0.9)	78 (1.8)	51 (6.7) *	7 (8.5) *	< 0.001
	GDM, n (%)	16 (2.8)	208 (4.8)	72 (9.4) *	8 (9.8)	< 0.001
20–30 years	Number of patients	377	2265	366	37	
	Gestational age (w)	39.0 (38.3, 39.7)	39.0 (38.3, 39.7)	39.0 (38.0, 39.7)	38.6 (37.5, 39.1) *	0.010
	Mode of delivery, n (%)					< 0.001
	Vaginal	144 (38.2)	724 (32.0)	73 (19.9) *	2 (5.4) *	
	Cesarean section	233 (61.8)	1541 (68.0)	293 (80.1) *	35 (94.6) *	
	PTB (< 37w)	19 (5.0)	131 (5.8)	23 (6.3)	5 (13.5)	0.209
	Very PTB (< 32w)	1 (0.3)	13 (0.6)	2 (0.5)	0	0.852
	Sex, n (%)					0.657
	Male	213 (56.5)	1212 (53.5)	198 (54.1)	22 (59.5)	
	Female	164 (43.5)	1053 (46.5)	168 (45.9)	15 (40.5)	
	Birthweight, g	3100.0 (2900.0, 3400.0) *	3300.0 (3000.0, 3550.0)	3400.0 (3100.0, 3700.0) *	3300.0 (3200.0, 4025.0)	< 0.001
	LBW (< 2500 g), n (%)	14 (3.7)	76 (3.4)	12 (3.3)	1 (2.7)	0.978
	Macrosomia (> 4000 g), n (%)	8 (2.1)	86 (3.8)	32 (8.7) *	9 (24.3) *	< 0.001
	Placenta previa, n (%)	10 (2.7)	50 (2.2)	4 (1.1)	0	0.350
	HDP, n (%)	2 (0.5)	40 (1.8)	17 (4.6) *	3 (8.1) *	< 0.001
	GDM, n (%)	10 (2.7)	88 (3.9)	23 (6.3)	5 (13.5) *	0.002
30–35 years	Number of patients	161	1652	302	36	
	Gestational age (w)	39.0 (38.1, 39.7)	39.0 (38.3, 39.6)	39.0 (38.0, 39.6)	39.0 (38.0, 39.3)	0.344
	Mode of delivery, n (%)					0.001
	Vaginal	51 (31.7)	454 (27.5)	56 (18.5) *	4 (11.1)	
	Cesarean section	110 (68.3)	1198 (72.5)	246 (81.5) *	32 (88.9)	
	PTB (< 37w)	14 (8.7)	99 (6.0)	26 (8.6)	3 (8.3)	0.232
	Very PTB (< 32w)	0	11 (0.7)	6 (2.0)	0	0.060
	Sex, n (%)					0.337
	Male	93 (57.8)	871 (52.7)	159 (52.6)	15 (41.7)	
	Female	68 (42.2)	781 (47.3)	143 (47.4)	21 (58.3)	
	Birthweight, g	3150.0 (2900.0, 3400.0) *	3255.0 (3000.0, 3500.0)	3400.0 (3097.5, 3700.0) *	3345.0 (3000.0, 3637.5)	< 0.001
	LBW (< 2500 g), n (%)	9 (5.6)	64 (3.9)	15 (5.0)	2 (5.6)	0.611
	Macrosomia (> 4000 g), n (%)	6 (3.7)	57 (3.5)	24 (7.9) *	4 (11.1)	0.001
	Placenta previa, n (%)	4 (2.5)	50 (3.0)	6 (2.0)	3 (8.3)	0.186
	HDP, n (%)	3 (1.9)	28 (1.7)	22 (7.3) *	2 (5.6)	< 0.001
	GDM, n (%)	6 (3.7)	99 (6.0)	34 (11.3) *	3 (8.3)	0.003
35–45 years	Number of patients	25	396	95	9	
	Gestational age (w)	38.9 (37.7, 39.1)	38.7 (38.0, 39.1)	38.7 (38.0, 39.4)	38.0 (36.9, 39.1)	0.618

Table 4 (continued)

Age category	Parameter	Lean < 18.5 (kg/m^2)	Normal weight 18.5–24.9 (kg/m^2)	Overweight 25-29.9 (kg/m^2)	Obese ≥ 30 (kg/m^2)	*P* value
	Mode of delivery, n (%)					0.609
	Vaginal	6 (24.0)	73 (18.4)	22 (23.2)	1 (11.1)	
	Cesarean section	19 (76.0)	323 (81.6)	73 (76.8)	8 (88.9)	
	PTB (< 37w)	5 (20.0)	41 (10.4)	9 (9.5)	2 (22.2)	0.302
	Very PTB (< 32w)	0	3 (0.8)	0	0	0.805
	Sex, n (%)					0.195
	Male	11 (44.0)	203 (51.3)	59 (62.1)	4 (44.4)	
	Female	14 (56.0)	193 (48.7)	36 (37.9)	5 (55.6)	
	Birthweight, g	3100.0 (2625.0, 3285.0)	3200.0 (2935.0, 3500.0)	3400.0 (3100.0, 3680.0) *	3150.0 (2975.0, 3445.0)	< 0.001
	LBW (< 2500 g), n (%)	5 (20.0) *	19 (4.8)	1 (1.1)	1 (11.1)	0.001
	Macrosomia (> 4000 g), n (%)	0	14 (3.5)	7 (7.4)	0	0.217
	Placenta previa, n (%)	3 (12.0)	13 (3.3)	4 (4.2)	0	0.153
	HDP, n (%)	0	10 (2.5)	12 (12.6) *	2 (22.2) *	< 0.001
	GDM, n (%)	0	21 (5.3)	15 (15.8) *	0	0.001

Note BMI body mass index; *PTB* preterm birth; *HDP* hypertensive disorders of pregnancy; *GDM* gestational diabetes mellitus; *LBW* low birth weight

**P* < .05 as compared with the normal weight group

95% CI, 1.020–2.659; obese group: aOR = 4.402; 95% CI, 1.627–11.914).Among individuals aged 30–35 years, both overweight and obese groups had an increased risk of macrosomia (overweight group: aOR = 2.487; 95% CI, 1.505–4.109; obese group: aOR = 3.752; 95% CI, 1.257–11.201) and gestational hypertension (overweight group: aOR = 4.682; 95% CI, 2.610–8.398; obese group: aOR = 4.545; 95% CI, 1.011–20.440), while only the overweight group demonstrated a significantly higher risk of gestational diabetes mellitus (aOR = 1.953; 95% CI, 1.286–2.964). For individuals aged 35–45 years, both the overweight and obese groups had a significantly higher prevalence of gestational hypertension (overweight group: aOR = 5.580; 95% CI, 2.395–14.291; obese group: aOR = 10.503; 95% CI, 1.669–66.107). However, the incidence of gestational diabetes mellitus was notably higher only in the overweight group (aOR = 3.525; 95% CI, 1.720–7.225) compared to individuals with normal weight. Additionally, the risk of very preterm birth in the overweight group (aOR = 2.808; 95% CI, 1.005–7.841) and the risk of placenta previa in the obese group (aOR = 3.854; 95% CI, 1.094–13.571) was significantly higher compared to those with normal weight in the age subgroup of 30–35 years.

Discussion

This retrospective study shows that in infertile women, BMI affects ovarian reserve in an age-related manner. Specifically, BMI does not significantly impact ovarian reserve in patients aged 35–45 years. Additionally, obesity influences embryo outcomes, including the number of oocytes retrieved, mature oocytes, and fertilized oocytes. Obesity also increases the risk of adverse perinatal outcomes such as gestational diabetes mellitus, gestational hypertension, and macrosomia. However, no significant association was found between obesity and poor pregnancy outcomes.

Current research on the relationship between BMI and ovarian reserve remains controversial. This study observed an inverse association between obesity and AMH levels. Specifically, AMH concentrations decreased as BMI increased, which is consistent with findings from some previous studies [11, 12, 18–20]. Jaswa et al. reported a reduction in AMH levels with higher BMI, which was not attributable to the dilutional effect of increased blood volume [11]. Bernardi et al. identified significant associations between AMH and various markers of obesity, including current BMI, late adolescent BMI, and leptin [12]. In contrast, some studies have found no correlation between BMI and ovarian reserve [14], while others have reported a positive correlation [13, 21]. Albu et al. found that in infertile women without severe obesity, an increase in BMI was positively correlated with AMH levels [13]. Halawaty et al. conducted a cross-sectional study and found no correlation between AMH levels and BMI [21]. Although the exact mechanism remains unclear, obesity may affect ovarian reserve through altered hormone levels. Elevated aromatase activity in adipose tissue promotes the peripheral conversion of androgens to estrogens, leading to negative feedback on the hypothalamic-pituitary-ovarian (HPO) axis, which can inhibit ovarian folliculogenesis [22]. Additionally, obesity affects ovarian reserve through specific adipokines. Obese patients exhibit elevated leptin levels in both serum and follicular fluid [23], which can downregulate AMH expression through the JAK2/STAT3 pathway [24].

The study revealed that the correlation between BMI and AMH significantly weakened in women aged 35–45 years. This may be due to the substantial decline in ovarian reserve that occurs with age [25]. In older women, age becomes the primary factor influencing ovarian reserve, while BMI has a lesser impact. The limited number of patients aged 35–45 years in this study could have contributed to this finding.

This study found no significant association between AFC and BMI. The accuracy of AFC results is compromised by the inability to distinguish between healthy and atretic follicles during transvaginal ultrasound [22]. Additionally, the predictive accuracy of AFC is limited in overweight and obese women due to greater inter-cycle and intra-cycle variability [26, 27]. In cases where AFC and AMH results are discordant, AMH is considered a more reliable predictor of ovarian reserve [28]. Therefore, it can still be assumed that overweight and obesity affect ovarian reserve in infertile women.

Several studies have demonstrated that obesity significantly impacts oocyte quality [29, 30]. Obese women typically have fewer oocytes retrieved and matured compared to non-obese women [31, 32], a finding consistent with this study. This study observed significant differences in the number of retrieved oocytes, matured oocytes, and fertilized oocytes among patients with varying BMIs in the age subgroups of 20–30 and 30–35 years, as previously mentioned. Maternal metabolic changes can lead to abnormalities in the follicular fluid microenvironment [30]. Research indicates that increased inflammation and oxidative stress are associated with reduced oocyte developmental potential [29]. Additionally, altered mitochondrial activity has been identified as a possible mechanism for poor oocyte quality in obese women [33]. Mitochondrial dysfunction can impair oocyte maturation, fertilization, and subsequent embryonic development [34]. Furthermore, obesity's impact on oocyte quality appears to be age-related, similar to its effect on ovarian reserve.

Previous research has explored the effect of BMI on IVF/ICSI outcomes, but the results have been inconsistent. While many studies have shown that obese infertile women tend to have poorer outcomes with ART [4–7, 35–37], others have found no significant difference between obese and non-obese women [8, 38]. This study, however, did not find significant correlations between BMI and key pregnancy outcomes, including biochemical pregnancy, miscarriage, and live birth rates. Interestingly, the obese group in this study showed higher rates of clinical and ongoing pregnancies, which challenges the prevailing view that higher BMI negatively affects fertility. Notably, a higher incidence of ectopic pregnancy in the overweight group was observed in a subgroup analysis. This complicates the understanding of the relationship between BMI and pregnancy outcomes. The inconsistencies in findings across studies may stem from differences in study populations, methods of weight classification, sample sizes, exclusion criteria, number of cycles, and other factors contributing to heterogeneity. Moreover, it is well-documented that obesity primarily affects fertility by causing anovulation [39]. Treatments such as controlled ovarian stimulation and in vitro fertilization are generally effective in overcoming infertility issues related to ovulatory dysfunction. The unexpectedly higher pregnancy rates in obese patients may suggest a selection bias in the normal weight group. Within this group, there exists a higher prevalence of infertility causes not associated with ovulatory dysfunction, which might not respond as effectively to the interventions aimed primarily at inducing ovulation.

Obesity in women is associated with a high risk of perinatal complications, and similar results have been consistently reported s in infertile women [38, 40, 41]. In this study, obesity increased the risk of gestational diabetes mellitus, gestational hypertension, and macrosomia. Obesity exerts multifaceted effects on the oocyte, embryo, and endometrium, leading to an increased risk of multiple adverse outcomes [42]. These effects stem from altered endocrine and metabolic environments due to adipose tissue accumulation. Key mechanisms include insulin resistance, hyperinsulinemia, upregulation of proinflammatory factors, and oxidative stress [43].

This study has several limitations. As a retrospective analysis, there is a possibility of unmeasured confounders despite adjustments for known factors. Additionally, the sample size for age-stratified analyses and perinatal outcomes was relatively small, which may explain why some results did not show significant differences or diverged from previous findings. To reduce bias from multiple repeat cycles, we included only the first fresh cycles. However, earlier studies have demonstrated that obese patients often require more ART cycles than normal weight patients [7, 44], and the outcomes of the first cycle may not fully reflect their true outcomes.

The study's strength lies in its large sample size of over 30,000 cases, which enhances the generalizability of our findings. Data collection from a single center ensured uniformity in the evaluation of indicators across studies. Additionally, analyzing both ovarian reserve and ART outcomes within the same cohort provides a clear depiction of how BMI impacts these factors, thereby aiding in the management of obese infertile patients.

Conclusions

In conclusion, this large retrospective study reveals that a high BMI is associated with diminished ovarian reserve in infertile women, and this association is influenced by age. Although obesity negatively impact certain embryo

and perinatal outcomes, it does not significantly correlate with pregnancy outcomes. While weight control remains important for patients undergoing ART, it is crucial to consider the effects of age on treatment outcomes.

Abbreviations

BMI	Body mass index
ART	Assisted reproductive technology
hCG	Human Chorionic Gonadotropin
WHO	World Health Organization
PTB	Preterm birth
HDP	Hypertensive disorders of pregnancy
GDM	Gestational diabetes mellitus
LBW	Low birth weight
FSH	Follicle stimulating hormone
AFC	Antral follicle count
AMH	Anti Mullerian hormone
GnRH	Gonadotropin releasing hormone
Gn	Gonadotropin
IVF	In vivo fertilization
ICSI	Intracytoplasmic sperm injection
2PN	Two pronuclei
OR	Odds ratio
CI	Confidence interval

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13048-024-01521-1.

Supplemental Figure 1 Levels of Anti-Müllerian hormone (AMH) in different body mass index (BMI) groups.

Supplemental Figure 2 Levels of antral follicle count (AFC) in different body mass index (BMI) groups.

Supplemental Table 1 Baseline characteristics of the study cycles (20 < age ≤ 30)

Supplemental Table 2 Baseline characteristics of the study cycles (30 < age ≤ 35)

Supplemental Table 3 Baseline characteristics of the study cycles (35 < age ≤ 45)

Supplemental Table 4 Multivariate linear regression analysis of the association between BMI and embryo outcomes

Supplemental Table 5 Logistic regression of pregnancy outcomes by BMI categorization

Supplemental Table 6 Logistic regression of perinatal outcomes by BMI categorization

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Author contributions
G.Z., B.M., and L.J. conceived of the study and participated in its design and conceptualization. Y.L. and E.Y. collected and analyzed the data. Y.L. wrote the original draft. All co-workers reviewed and approved the manuscript.

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Data availability
The datasets utilized in the present study can be obtained from the corresponding author upon a reasonable request.

Declarations

Ethics approval and consent to participate
The study complied with the Declaration of Helsinki for Human Subjects in Medical Research and the Board of Institutional Review (TJ-IRB20230213) approval was given by the Ethical Committee of Reproductive Medicine Center, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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References

1. Haslam DW, James WPT, Obesity. Lancet Lond Engl. 2005;366:1197–209.
2. Sharpe RM, Franks S. Environment, lifestyle and infertility — an inter-generational issue. Nat Med. 2002;8:S33–40.
3. Broughton DE, Moley KH. Obesity and female infertility: potential mediators of obesity's impact. Fertil Steril. 2017;107:840–7.
4. Kluge L, Källén K, Thurin-Kjellberg A, Wennerholm U-B, Bergh C. The association between body mass index and live birth and maternal and perinatal outcomes after in-vitro fertilization: a national cohort study. Front Endocrinol. 2023;14:1239702.
5. Rafael F, Rodrigues MD, Bellver J, Canelas-Pais M, Garrido N, Garcia-Velasco JA, et al. The combined effect of BMI and age on ART outcomes. Hum Reprod Oxf Engl. 2023;38:886–94.
6. Provost MP, Acharya KS, Acharya CR, Yeh JS, Steward RG, Eaton JL, et al. Pregnancy outcomes decline with increasing body mass index: analysis of 239,127 fresh autologous in vitro fertilization cycles from the 2008–2010 society for assisted Reproductive Technology registry. Fertil Steril. 2016;105:663–9.
7. Moragianni VA, Jones S-ML, Ryley DA. The effect of body mass index on the outcomes of first assisted reproductive technology cycles. Fertil Steril. 2012;98:102–8.
8. Tao P, Yan X, Yao Y, Wang Z, Li Y. Pre-pregnancy obesity is not associated with poor outcomes in fresh transfer in vitro fertilization cycles: a retrospective study. BMC Pregnancy Childbirth. 2023;23:633.
9. Moolhuijsen LME, Visser JA. Anti-Müllerian hormone and Ovarian Reserve: update on assessing ovarian function. J Clin Endocrinol Metab. 2020;105:3361–73.
10. Broekmans FJ, Kwee J, Hendriks DJ, Mol BW, Lambalk CB. A systematic review of tests predicting ovarian reserve and IVF outcome. Hum Reprod Update. 2006;12:685–718.
11. Jaswa EG, Rios JS, Cedars MI, Santoro NF, Pavone MEG, Legro RS, et al. Increased body Mass Index is Associated with a nondilutional reduction in Antimüllerian hormone. J Clin Endocrinol Metab. 2020;105:3234–42.
12. Bernardi LA, Carnethon MR, de Chavez PJ, Ikhena DE, Neff LM, Baird DD, et al. Relationship between obesity and anti-Müllerian hormone in reproductive-aged African American women. Obes Silver Spring Md. 2017;25:229–35.
13. Albu D, Albu A. The relationship between anti-Müllerian hormone serum level and body mass index in a large cohort of infertile patients. Endocrine. 2019;63:157–63.
14. Simões-Pereira J, Nunes J, Aguiar A, Sousa S, Rodrigues C, Sampaio Matias J, et al. Influence of body mass index in anti-Müllerian hormone levels in

951 non-polycystic ovarian syndrome women followed at a reproductive medicine unit. Endocrine. 2018;61:144–8.
15. Organization WH, Obesity. Preventing and managing the global epidemic: report of a WHO Consultation. World Health Organization; 2000.
16. Ma B-X, Zhang H, Jin L, Huang B. Neonatal outcomes of embryos cultured in a Time-Lapse incubation system: an analysis of more than 15,000 fresh transfer cycles. Reprod Sci. 2022;29:1524–30.
17. Schoolcraft WB, Gardner DK, Lane M, Schlenker T, Hamilton F, Meldrum DR. Blastocyst culture and transfer: analysis of results and parameters affecting outcome in two in vitro fertilization programs. Fertil Steril. 1999;72:604–9.
18. Buyuk E, Seifer DB, Illions E, Grazi RV, Lieman H. Elevated body mass index is associated with lower serum anti-mullerian hormone levels in infertile women with diminished ovarian reserve but not with normal ovarian reserve. Fertil Steril. 2011;95:2364–8.
19. Wang Y, Wu L, Yang Z, Xu R, Duan Y, Lin J, et al. Association of body mass index with serum anti-Müllerian hormone and inhibin B levels among 8323 women attending a reproductive medical center: a cross-sectional study. Endocrine. 2022;75:284–92.
20. Freeman EW, Gracia CR, Sammel MD, Lin H, Lim LC-L, Strauss JF. Association of anti-mullerian hormone levels with obesity in late reproductive-age women. Fertil Steril. 2007;87:101–6.
21. Halawaty S, ElKattan E, Azab H, ElGhamry N, Al-Inany H. Effect of obesity on parameters of ovarian reserve in premenopausal women. J Obstet Gynaecol Can JOGC J Obstet Gynecol Can JOGC. 2010;32:687–90.
22. Moslehi N, Shab-Bidar S, Ramezani Tehrani F, Mirmiran P, Azizi F. Is ovarian reserve associated with body mass index and obesity in reproductive aged women? A meta-analysis. Menopause N Y N. 2018;25:1046–55.
23. Yadav A, Kataria MA, Saini V, Yadav A. Role of leptin and adiponectin in insulin resistance. Clin Chim Acta. 2013;417:80–4.
24. Merhi Z, Buyuk E, Berger DS, Zapantis A, Israel DD, Chua S, et al. Leptin suppresses anti-mullerian hormone gene expression through the JAK2/STAT3 pathway in luteinized granulosa cells of women undergoing IVF. Hum Reprod Oxf Engl. 2013;28:1661–9.
25. Kelsey TW, Wright P, Nelson SM, Anderson RA, Wallace WHB. A validated model of serum Anti-Müllerian hormone from conception to Menopause. PLoS ONE. 2011;6:e22024.
26. La Marca A, Sighinolfi G, Radi D, Argento C, Baraldi E, Artenisio AC, et al. Anti-Müllerian hormone (AMH) as a predictive marker in assisted reproductive technology (ART). Hum Reprod Update. 2010;16:113–30.
27. Fleming R, Seifer DB, Frattarelli JL, Ruman J. Assessing ovarian response: antral follicle count versus anti-Müllerian hormone. Reprod Biomed Online. 2015;31:486–96.
28. Li HWR, Lee VCY, Lau EYL, Yeung WSB, Ho PC, Ng EHY. Ovarian response and cumulative live birth rate of women undergoing in-vitro fertilisation who had discordant anti-mullerian hormone and antral follicle count measurements: a retrospective study. PLoS ONE. 2014;9:e108493.
29. Gonzalez MB, Robker RL, Rose RD. Obesity and oocyte quality: significant implications for ART and emerging mechanistic insights. Biol Reprod. 2022;106:338–50.
30. Cardozo E, Pavone ME, Hirshfeld-Cytron JE. Metabolic syndrome and oocyte quality. Trends Endocrinol Metab. 2011;22:103–9.
31. Salih Joelsson L, Elenis E, Wanggren K, Berglund A, Iliadou AN, Cesta CE, et al. Investigating the effect of lifestyle risk factors upon number of aspirated and mature oocytes in in vitro fertilization cycles: Interaction with antral follicle count. PLoS ONE. 2019;14:e0221015.
32. Cardozo ER, Karmon AE, Gold J, Petrozza JC, Styer AK. Reproductive outcomes in oocyte donation cycles are associated with donor BMI. Hum Reprod Oxf Engl. 2016;31:385–92.
33. Igosheva N, Abramov AY, Poston L, Eckert JJ, Fleming TP, Duchen MR, et al. Maternal Diet-Induced obesity alters mitochondrial activity and Redox Status in Mouse oocytes and zygotes. PLoS ONE. 2010;5:e10074.
34. Grindler NM, Moley KH. Maternal obesity, infertility and mitochondrial dysfunction: potential mechanisms emerging from mouse model systems. Mol Hum Reprod. 2013;19:486–94.
35. Fedorcsák P, Dale PO, Storeng R, Ertzeid G, Bjercke S, Oldereid N, et al. Impact of overweight and underweight on assisted reproduction treatment. Hum Reprod Oxf Engl. 2004;19:2523–8.
36. Luke B, Brown MB, Stern JE, Missmer SA, Fujimoto VY, Leach R, et al. Female obesity adversely affects assisted reproductive technology (ART) pregnancy and live birth rates. Hum Reprod Oxf Engl. 2011;26:245–52.
37. Robker RL, Akison LK, Bennett BD, Thrupp PN, Chura LR, Russell DL, et al. Obese women exhibit differences in ovarian metabolites, hormones, and gene expression compared with moderate-weight women. J Clin Endocrinol Metab. 2009;94:1533–40.
38. Dokras A, Baredziak L, Blaine J, Syrop C, VanVoorhis BJ, Sparks A. Obstetric outcomes after in vitro fertilization in obese and morbidly obese women. Obstet Gynecol. 2006;108:61–9.
39. Sermondade N, Huberlant S, Bourhis-Lefebvre V, Arbo E, Gallot V, Colombani M, et al. Female obesity is negatively associated with live birth rate following IVF: a systematic review and meta-analysis. Hum Reprod Update. 2019;25:439–51.
40. Hynes JS, Weber JM, Truong T, Acharya KS, Eaton JL. Body mass index is negatively associated with a good perinatal outcome after in vitro fertilization among patients with polycystic ovary syndrome: a national study. FS Rep. 2023;4:77–84.
41. Kawwass JF, Kulkarni AD, Hipp HS, Crawford S, Kissin DM, Jamieson DJ. Extremities of body mass index and their association with pregnancy outcomes in women undergoing in vitro fertilization in the United States. Fertil Steril. 2016;106:1742–50.
42. Goldsammler M, Merhi Z, Buyuk E. Role of hormonal and inflammatory alterations in obesity-related reproductive dysfunction at the level of the hypothalamic-pituitary-ovarian axis. Reprod Biol Endocrinol. 2018;16:45.
43. Catalano PM, Shankar K. Obesity and pregnancy: mechanisms of short term and long term adverse consequences for mother and child. BMJ. 2017;356:j1.
44. Christensen MW, Ingerslev HJ, Degn B, Kesmodel US. Effect of female body Mass Index on Oocyte Quantity in Fertility treatments (IVF): treatment cycle number is a possible effect modifier. A Register-based Cohort Study. PLoS ONE. 2016;11:e0163393.

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