SYMPOSIUM OBESITY AND REPRODUCTIVE HEALTH

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Conflict of interest: The author declare no conflict of interest

Funding: Self-funded

Statement on the use of artificial intelligence: No artificial intelligence-related technology was used in preparation of this article

Received: 10 February 2025

Accepted: 25 March 2025

Online publication: 31 March 2025

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Cite as: Rechkemmer A. Obesity and its impact on reproductive medicine. Rev peru ginecol obstet. 2025;71(1). DOI: https://doi.org/10.31403/rpgo.v71i2745

Obesity and its impact on reproductive medicine

Obesidad y su impacto en medicina reproductiva

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DOI: https://doi.org/10.31403/rpgo.v71i2745

ABSTRACT

Obesity has a negative impact on reproductive health, with a higher incidence of anovulation, menstrual disorders and infertility. The increase in adipose tissue is associated with insulin resistance and hyperinsulinemia, reduction in sex hormone binding globulin levels, and hyperandrogenemia. There is an increase in pro-inflammatory adipokines and a decrease in adiponectins. Female obesity is associated with ovulatory dysfunction, lower ovarian response to ovulation inducers, decreased oocyte quantity and quality, and alteration in endometrial receptivity. Likewise, a decrease in the live birth rate has been reported in in vitro fertilization treatments, and an increase in the frequency of abortions. Male obesity increases the incidence of erectile dysfunction and alterations in sperm quality. Diet, exercise, and medical therapy have been shown to be effective. Bariatric surgery is an alternative in cases of body mass index greater than 40, and achieves a rapid and effective weight reduction, with improvement in metabolic parameters; it is recommended to postpone pregnancy in women for at least one year after surgery. Bariatric surgery significantly reduces the risk of fetal macrosomia, pregnancy-associated hypertension, and gestational diabetes. However, it increases the risk of maternal anemia and small-for-gestational-age newborns.

Keywords: Obesity, Infertility, Anovulation, Menstruation Disturbances, Reproductive medicine (source: MeSH NLM).

RESUMEN

La obesidad tiene un impacto negativo sobre la salud reproductiva, con mayor incidencia de anovulación, trastornos menstruales e infertilidad. El incremento en el tejido adiposo se asocia a resistencia a la insulina e hiperinsulinemia, reducción en los niveles de globulina trasportadora de hormonas sexuales e hiperandrogenemia. Existe un aumento en las adipokinas pro-inflamatorias y una disminución en las adiponectinas. La obesidad femenina se asocia con disfunción ovulatoria, menor respuesta ovárica a los inductores de ovulación, disminución en la cantidad y calidad oocitaria, y alteración en la receptividad endometrial. Asimismo, se ha reportado una disminución en la tasa de nacidos vivos en los tratamientos de fertilización in vitro, y un aumento en la frecuencia de abortos. La obesidad masculina aumenta la incidencia de disfunción eréctil y alteraciones en la calidad espermática. La dieta, los ejercicios y la terapia médica han mostrado ser efectivos. La cirugía bariátrica es una alternativa en casos de índice de masa corporal mayor de 40, y consigue una reducción rápida y efectiva de peso, con mejoría de los parámetros metabólicos; se recomienda postergar el embarazo en mujeres al menos un año post cirugía. La cirugía bariátrica reduce de manera significativa el riesgo de macrosomía fetal, hipertensión asociada al embarazo y diabetes gestacional. Sin embargo, aumenta el riesgo de anemia materna y recién nacidos pequeños para edad gestacional.

Palabras clave: Obesidad, Infertilidad, Anovulación, Trastornos de la Menstruación, Medicina reproductiva (fuente: DeCS BIREME).

Introduction

Currently, overweight and obesity are considered a public health problem and a global epidemic. Obesity is associated with ovulatory dysfunction and menstrual disorders, reduction in natural fertility, and other adverse reproductive effects such as reduction in pregnancy rate and safety in infertility treatments[(1)].

Chronic anovulation and ovulatory disorders are responsible for 25 to 50% of the causes of female infertility, and are associated with a higher risk of long-term complications, such as endometrial hyperplasia, endometrial cancer and breast cancer. Polycystic ovary syndrome (PCOS) is a common cause of anovulatory infertility, and almost 50% of them are obese[(2)]. However, obesity with or without PCOS affects reproduc-

tion, through a series of mechanisms that we will describe below. It is associated with menstrual disorders, anovulation, infertility, less effective assisted reproduction treatments, abortions and greater obstetric complications.

A recent systematic review and meta-analysis[3] found that overweight and obesity increases the risk of infertility by 60% (OR: 1.60, CI: 1.31-1.94). Proper management of preconception body weight improves fertility.

Mechanisms of obesity in female infertility

1. Adipose tissue and insulin

Excessive adipose tissue in obese women result in increased estrogen production through peripheral aromatization. The resulting hyperestrogenism causes alterations in endometrial receptivity and the hypothalamic-pituitary-ovarian axis, which alters the secretion of gonadotropins. This causes poor oocyte recruitment and alteration in follicular growth[4].

There is an increase in pancreatic insulin production in obese women, and an excessive production of hepatic lipids with alterations in liver function with consequent insulin resistance and hyperinsulinemia[5].

Insulin potentiates the effects of LH by increasing LH receptors and improving the binding capacity of LH to theca cells, which increases ovarian androgen production. This causes arrest in follicular development.

Insulin induces increased pituitary production of LH by increasing the sensitivity of gonadotropes to GnRH, and inhibits hepatic and ovarian production of insulin-like growth factor-binding protein 1 (IGF1-BP). The reduction of IGF1-BP increases circulating levels of IGF 1, which acts on theca cells, increasing androgen production and reducing Growth Hormone, which inhibits follicular growth[6].

2. Sexual hormone binding globulin (SHBG) y adipokines

In obese women, circulating levels of SHBG are decreased. SHBG hepatic production is reduced primarily due to high insulin levels, although hyperandrogenemia also contributes. This causes an increase in circulating levels of free androgens, which alters ovarian function and folliculogenesis[4].

Adipokines are produced by adipose tissue and include leptin, adiponectin, tumor necrosis factor alpha (TNF-α), resistin, interleukin-6 (IL-6), visfastin, among others. In obese people, pro-inflammatory adipokines, such as leptin, increase and anti-inflammatory adipokines, such as adiponectins, decrease (Table 1).

Elevated leptin levels are associated with leptin resistance in the central nervous system, affecting GnRH and LH production, resulting in poor follicular development. High levels of leptin have been found in the follicular fluid, and it is known that it inhibits ovarian steroidogenesis by reducing the activity and production of aromatase, which causes poor growth of dominant follicles, poor oocyte maturation and decreased endometrial receptivity.

Obese women have reduced levels of adiponectin, which is a hormone that sensitizes insulin action; this causes a greater tendency to develop insulin resistance[4].

Alterations in ovulation and menstrual cycle

The mechanism of anovulation in women with obesity is unclear. The prevalence of insulin resistance and androgen excess is higher in obesity, especially of the central type[6]. The real prevalence of menstrual irregularities in women with obesity is unknown, since most studies are retrospective and based on self-reports on the length of menstrual cycles; the reported prevalence ranges from less than 10% to more than 50%[1] and tends to be higher with higher body mass index (BMI).

Table 1. Pro-inflammatory and anti-inflammatory adipokines.

Pro-inflammatory	Anti-inflammatory
Leptin	Adiponectin
TNF-α	TGF-β
IL-6	Interleukin-4
Visfastin	Interleukin-10
Resistin	Interleukin-11

TNF-α (Tumor necrosis factor alpha); TGF-β (Tumor necrosis factor beta); IL-6 (Interleukin-6)

Ovulatory dysfunction is more common in obese women, and directly correlates with the degree of obesity, especially with subcutaneous abdominal fat[8]. Central obesity and visceral fat are associated with insulin resistance and hyperinsulinemia. Increased insulin reduces hepatic production of SHBG and increases circulating free androgens; it is frequently associated with polycystic ovary syndrome.

Alterations in ovarian response and oocyte quality

Souter et al.[9] reported that obese women treated with gonadotropins to induce ovulation require higher doses of hormones and develop fewer follicles. In patients undergoing in vitro fertilization (IVF) treatments, a lower rate of live births has been found in obese women[10]. The dose of gonadotropins and the duration of treatment are increased, the cancellation rate increases and the number of recovered oocytes decreases.

In a retrospective cohort study that included 494 097 IVF cycles[11], a significant reduction in the live birth rate was found in obese women (aRR: 0.87, CI: 0.86-0.89).

An altered follicular environment has been found in obese women undergoing IVF, with increased levels of free fatty acids, markers of inflammation and insulin levels, affecting oocyte quality[1]. In a large study using data from the American Society for Assisted Reproduction (SART) that included 239 127 fresh IVF cycles[12], a negative impact of obesity on live birth rate (LBR) was found, directly proportional to body mass index; thus, LBR is reduced by 16% if BMI is between 30 and 34.9, and LBR is reduced by 27% if BMI is between 40 and 44.9 (Table 2).

In a meta-analysis published by Sermondade et al.[13] that included 21 studies, a significant reduction in LBR was found in women with BMI greater than 30, compared to women of normal weight, RR: 0.85 (CI: 0.82-0.87).

Endometrial receptivity

In obese women, a different expression of endometrial genes has been found during the implantation window, which is more pronounced if there is associated infertility. Furthermore, there is a significant elevation of inflammatory markers and reactive oxygen species (ROS) in the endometrial tissue of obese infertile women. This would be associated with a decrease in endometrial receptivity and greater implantation failure and compromise in decidualization[14].

Table 2. Impact of obesity on live birth rate in IVF, adapted from Provost et al.[12].

Live birth rate (%)	OR (CI)	Body mass index (Kg/m^2)
31.4		18.5 – 24.9
29.8	0.94 (0.91-0.96)	25 – 29.9
28	0.84 (0.81-0.87)	30 – 34.9
26.3	0.76 (0.72-0.79)	35 – 39.9
24.3	0.73 (0.67-0.77)	40 – 44.9
22.8	0.67 (0.58-00.77)	45 – 49.9
21.2	0.52 (0.41-0.66)	Over 50

One way to evaluate the impact of obesity on endometrial receptivity is to investigate the results of IVF in obese women undergoing an egg donation program. Various studies have found a significant reduction in the implantation rate in obese women undergoing IVF with donated eggs, which is proportional to higher BMI[15,16]. However, a meta-analysis with 4758 women undergoing IVF with egg donation found no association between obesity and pregnancy rate, although the number of studies included was small and with different inclusion and exclusion criteria[17].

Obesity and miscarriages

The association between obesity and spontaneous abortions has been reported in natural pregnancies and in pregnancies due to assisted fertilization treatments[18]. There is no consensus regarding the mechanisms that cause this increase in the abortion rate; it is postulated that obesity can affect the endocrine environment, the quality of the embryo and/or the quality of the endometrium. Some endocrinopathies, such as hypothyroidism, polycystic ovary syndrome, and insulin resistance, are more common in obese women, and the miscarriage rate is higher in women with these diseases.

Lee et al. found, after cytogenetic examination of the product of conception, that obese women had a higher frequency of euploid abortions compared to nonobese women[19].

There are studies that have shown an increase of up to 3 times in the abortion rate in obese women undergoing IVF with their own eggs and egg donation. However, the adjusted range of OR ranges between 1.2 and 1.9, which suggests that an association does exist, but it is modest and influenced by confounding factors[1,7].

Male obesity and infertility

Obesity in infertile men is associated with a higher frequency of erectile dysfunction. Likewise, there is an increase in insulin resistance, adipokines, leptin resistance and hyperglycemia. There is dysfunction of the hypothalamic-pituitary-testis axis, reduction in testosterone levels and alteration in sperm parameters. A recent review explains in detail the systemic inflammatory changes, metabolic syndrome and mitochondrial dysfunction, found in male obesity[20].

There are systematic reviews that find an association between male obesity and infertility. Campbell et al.[21] included 115 158 participants from 30 studies, finding that obese men have a higher prevalence of infertility than non-obese men, OR: 1.66 (CI: 1.53-1.79). An increase in the probability of alterations in semen quality has been reported in overweight and obese men. Lower sperm concentration, higher percentage of abnormal sperm morphology and lower semen volume have been found[22,23].

Regarding the impact of male obesity on assisted fertilization treatments, Mushtaq et al.[24], in a meta-analysis of 11 studies and 14 372 IVF cycles, found that an increase in BMI in men was associated with a significant decrease in the pregnancy rate and live birth rate, OR: 0.88 (CI: 0.82-0.95).

Management of obesity and its impact on fertility

Lifestyle modifications, such as a low-calorie diet and physical activity (at least 30 minutes a day) are important to lose weight. Physical activity is associated with a decrease in systemic inflammatory mediators, even in those who do not lose weight. The effectiveness of these non-pharmacological interventions on pregnancy rate was evaluated in a meta-analysis that included 21 randomized clinical trials[25]; an increase in the spontaneous pregnancy rate was found, but no significant effect was found in live birth rate. Further studies are required on the effects of non-pharmacological treatments on the psychological well-being of patients.

It is not clear whether it is appropriate to delay fertility treatment in order to lose weight, particularly in patients over 35 years of age or with low ovarian reserve, although the potential advantages in reducing obstetric complications at lower BMI are recognized. Several guidelines recommend lifestyle changes before starting fertility treatments such as IVF; however, the recommended percentage of weight loss to improve pregnancy rates has not yet been determined, and whether there is a direct favorable effect of healthy lifestyles on fertility independent of weight loss. Large, well-designed studies are required to answer these questions[23]. In obese women undergoing IVF, strict diet for 3 to 4 months has not shown improvement in live birth rate[1].

Weight loss drug therapies are available. Orlistat works by blocking fat absorption and glucose reabsorption; in obese women with polycystic ovary syndrome it reduces insulin resistance and androgen levels[4]. Analogs of glucagon-like peptide 1 (GLP-1), such as liraglutide, act by reducing appetite; they would have a greater impact on weight reduction than orlistat, at 20 weeks (5 to 7 kg with liraglutide vs 3 kg with orlistat). The impact of medical treatment on improving fertility in obese women and men is still unclear, and its use is contraindicated in pregnant women[22,26].

Bariatric surgery could be an option if the BMI is greater than 40, or if there are co-morbidities and BMI is greater than 35, or if other attempts to lose weight have failed. Improves hormonal balance and sexual function, both in men and women[27]. A weight reduction of 25 to 45 kg is achieved one year after surgery. There are no prospective randomized studies that investigate the time necessary to conceive after surgery, but it is recommended to postpone pregnancy for 12 to 18 months after surgery, although this time must be balanced with the risk of reduced ovarian reserve[28]. Bariatric surgery improves the metabolic profile and is very effective in preventing and treating type 2 diabetes in obese patients. Bariatric surgery significantly reduces the risk of fetal macrosomia, pregnancy-associated hypertension, and gestational diabetes. However, it increases the risk of maternal anemia and small-for-gestational-age newborns[1,26,29].

Genetic, clinical and experimental research on the mechanisms of obesity and the design of preventive measures, and efficient tools that include artificial intelligence, could play an important role in the future to establish precise treatments for obesity and thus reduce the complications associated with reproductive health[30].

Published works on obesity and infertility show that there are barriers to accessing fertility treatments and evidence-based protocols and multidisciplinary management are required in this group of patients[31].

Conclusions

Obesity, regardless of metabolic health status, is associated with an increased risk of infertility[32]. Female obesity is associated with ovulatory dysfunction, lower ovarian response to ovulation inducers, altered oocyte quality, endometrial dysfunction, lower live birth rate in IVF treatments, and an increased abortion rate. Male obesity can compromise reproductive function. Diet, exercise and medical therapy have been shown to be effective in losing weight. Bariatric surgery in women and men with morbid obesity achieves rapid and effective weight reduction, and it is recommended to postpone pregnancy in women for at least one year after surgery.

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