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Obesity-induced alterations in male reproductive hormones: A case-control study

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Abstract
Background: Obesity is a significant health issue that impacts various bodily systems, including male reproductive health. There is growing evidence that obesity is linked to male infertility, marked by hormonal abnormalities and reduced semen quality.
Methods: This case-control study assessed reproductive hormone levels and semen quality in obese guys relative to normal-weight controls. Hormonal assays quantified FSH, LH, testosterone, and prolactin, whereas semen analyses evaluated volume, sperm count, motility, morphology, DNA fragmentation, and protamine deficiency.
Results: Obese guys had significantly lower levels of serum FSH, LH, and testosterone, along with higher levels of prolactin. The volume, sperm count, total and progressive motility, and number of abnormal sperm morphologies all decreased down when the semen was looked at. Obese males exhibited a notable rise in sperm DNA fragmentation and a deficiency of protamine. There was a clear connection between BMI and difficulties with reproductive health.
Conclusion: Obesity negatively impacts male reproductive hormones and semen quality, leading to subfertility via endocrine disturbance, compromised spermatogenesis, and heightened sperm DNA damage. These results underscore the essential requirement for weight management therapies in males to enhance reproductive health. Addressing obesity presents significant promise for restoring reproductive problems.

Keywords: Obesity, male infertility, reproductive hormones, semen quality, sperm DNA fragmentation, prolactin, testosterone, sperm motility

Introduction
Obesity is now a major health problem all over the world, and the number of people who are obese has climbed to epidemic levels in the last few decades. This condition has a big impact on many body systems, including men's reproductive health. Obesity has long been connected to metabolic and cardiovascular illnesses, and it is now well recognized that it can impair the hypothalamic-pituitary-testicular (HPT) axis and male reproductive health. Obesity is a major changeable risk factor for male infertility that affects more than 10% of men and accounts for over 50% of all infertility cases worldwide. The extensive study conducted has shown that obese men have hormonal abnormalities, primarily due to increased aromatase activity in adipose tissue. These anomalies are characterized by diminished serum testosterone levels and increased estradiol concentrations. The identified hormonal disturbances lead to compromised spermatogenesis, causing a reduction in sperm count and motility, and an increase in morphological abnormalities, ultimately diminishing reproductive potential [1, 2]. Hormonal alterations, insulin resistance, chronic systemic inflammation, and oxidative stress are all associated to obesity. These things change the DNA in sperm and how well the testicles work. Men who were overweight had sperm DNA that was more damaged and less protamine. It would impact their children's health and prevent them from being able to have children. Many times, overweight men have a tremendous amount of prolactin in their bloodstream. It will prevent the body from producing testosterone and gonadotropins, making it worse for reproductive issues. Overweight persons are also more likely to experience erectile dysfunction and a decreased sex drive, further making it harder for them to produce offspring. There is great variability in the studies regarding the effect of obesity on male fertility and how severe its impact is. This also highlights the requirements for further case-control studies involving extensive hormone

profile and semen analysis in controlled settings. These studies are necessary for refining evidence-based targeted medication and lifestyle modifications to maintain and enhance fertility among the obese population. This case-control study aims to evaluate the distinctive clinical, biochemical, and hormonal changes which go hand-in-hand with obesity in men, with an emphasis on reproductive hormones and semen quality. The aim of this study is to enhance knowledge of reproductive issues secondary to obesity and make recommendations for the therapeutic treatment of male infertility in this expanding population.

Methodology

Study Design and Population

The case-control study aimed at examining obesity-associated changes in male reproductive hormones and semen characteristics. The study was conducted at a tertiary care hospital, and the study subjects were enrolled from the outpatient endocrinology and urology clinics during a period of twelve months. Informed consent was obtained from all the subjects according to the institution's ethical guidelines. The population of the study was males in the age group of 20-45 years, classified into two groups: cases, which include those with obesity, and controls, who include those of normal weight.

According to the World Health Organization (WHO), an individual is overweight if his or her body mass index (BMI) is 30 kg/m² or above and healthy if it ranges from 18.5 to 24.9 kg/m². To reduce confounding factors, participants with past chronic systemic diseases, endocrine disease, history of testicular surgery, varicocele, genetic or chromosomal anomalies, current medications influencing reproductive hormones, or active infections were all excluded from the research.

Data Collection Procedures

Participants received a thorough clinical assessment, encompassing an exhaustive medical history and physical examination. We recorded demographic details such as age, weight, height, BMI, and waist circumference. Blood samples were taken between 8:00 and 10:00 AM after a night of not eating to lower daily hormone changes. Standardized chemiluminescence immunoassays were used to measure the levels of "follicle-stimulating hormone (FSH), luteinizing hormone (LH), total testosterone, sex hormone-binding globulin (SHBG), and prolactin in the serum. Samples demonstrating hemolysis or undergoing processing delays were excluded. Semen samples were obtained through masturbation following a sexual abstinence interval of 2 to 7 days. Each participant provided two samples with a minimum interval of two weeks, and the mean values were employed for analysis to account for intra-individual variability. The sperm chromatin dispersion assay was employed to evaluate sperm DNA fragmentation, whereas Chromomycin A3 staining was applied to examine protamine deficiency. A semen analysis was conducted in accordance with the World Health Organization 6th Edition guidelines, including evaluations of volume, sperm concentration, total and progressive motility, percentages of non-progressive and non-motile sperm, and sperm morphology. Laboratory personnel were blinded to group allocation to mitigate observer bias.

Statistical Analysis: Descriptive statistics were generated for the first clinical and biochemical characteristics. Quantitative data were expressed as mean ± standard deviation (SD); categorical variables were represented as frequencies or percentages. The Shapiro-Wilk test was utilized to assess the normality of data distribution. Group disparities were evaluated using unpaired Student's t-test for continuous variables and chi-square test for categorical variables. Spearman's or Pearson's correlation analysis was utilized to ascertain the associations between BMI and reproductive parameters, contingent upon data distribution. A p-value less than 0.05 was deemed statistically significant. We used SPSS version 26.0 software to do all of the statistical analyses.

Ethical Considerations

The study followed the Declaration of Helsinki and got the go-ahead from the local Institutional Ethics Committee. Everyone who took part in the study knew what it was about, how it would work, and what risks it would represent. They also signed a paper saying they understood what they were doing and agreed to it. The entire process kept patients' identities and data safe. This all-encompassing effort made sure that the link between fat and reproductive problems was thoroughly examined, limiting outside influences and upholding high scientific standards.

Results

Clinical and Biochemical Characteristics

The effects of obesity on the male reproductive axis have become increasingly evident, with growing evidence substantiating its detrimental consequences in both clinical and physiological contexts. In this case-control study, the average age of obese males and controls did not differ considerably, so affirming the comparability between the groups. However, obese patients demonstrated a significantly elevated mean BMI, so affirming the role of excess adiposity as a crucial factor in reproductive failure. This rise in BMI is linked to several metabolic problems that often affect the male reproductive system, which may lead to hormonal changes and lower fertility.

Table 1: Clinical and Biochemical Characteristics of Obese and Normal-Weight Male Subjects

Parameter	Obese Males (Mean ± SD)	Controls (Mean ± SD)	P Value
Age (years)	31.5±6.2	32.1±7.1	0.430
BMI (kg/m²)	32.4±3.9	23.8±2.7	<0.001

Altered Semen Quality and Sperm Function

Obesity exerts a substantial detrimental effect on semen parameters and sperm function. The analysis revealed substantial decreases in semen volume, sperm count, and both total and progressive motility in obese guys relative to their normal-weight counterparts. Conversely, the obese group had a notable rise in the percentage of non-progressive and non-motile sperm, as well as an elevated proportion of sperm with aberrant morphology. Obese males demonstrated markedly increased sperm DNA fragmentation and protamine insufficiency, indicating impaired sperm integrity and a potential risk of infertility. The findings indicate that obesity affects both the number and quality of sperm, consequently undermining male reproductive capacity in multiple aspects.

Table 2: Comparison of Semen Parameters and Sperm Function Between Obese and Normal-Weight Males

Semen Parameter	Obese Males (Mean ± SD)	Controls (Mean ± SD)	P Value
Semen Volume (mL)	3.4±1.7	4.2±2.0	<0.001
Sperm Count (×10⁶)	25.2±23.9	67.0±57.9	<0.001
Total Motility (%)	38.6±18.3	58.3±18.3	<0.001
Progressive Motility (%)	14.8±13.7	44.2±16.7	<0.001
Non-progressive Motility (%)	23.5±12.3	14.1±6.7	<0.001
Non-motile Sperm (%)	61.7±18.4	41.7±18.3	<0.001
Abnormal Form (%)	70.9±25.4	54.6±26.0	<0.001
DNA Fragmentation (%)	55.2±30.7	17.5±20.1	<0.001
Protamine Deficiency (%)	70.7±16.5	25.1±20.8	<0.001

Hormonal Profile Alterations

In males, obesity is consistently linked to significant alterations in essential reproductive hormones. The levels of FSH, LH, and total testosterone were significantly reduced in the obese cohort, whereas prolactin levels exhibited a notable increase. The observed hormonal disruptions can be attributed primarily to dysfunction within the hypothalamic-pituitary-testicular axis, an increase in the aromatization of androgens occurring in expanded adipose tissue, and a reduction in the pulsatile secretion of gonadotropins. Decreased testosterone levels further facilitate adipogenesis, thereby sustaining the detrimental cycle of obesity and hypogonadism.

Table 3: Comparison of Reproductive Hormone Levels in Obese and Normal-Weight Male Groups

Hormonal Parameter	Obese Males (Mean ± SD)	Controls (Mean ± SD)	P Value
FSH (mIU/mL)	4.6±2.1	7.0±2.3	<0.001
LH (IU/L)	4.3±1.9	7.4±2.8	<0.001
Testosterone (ng/mL)	4.8±1.6	5.5±1.4	<0.001
Prolactin (ng/mL)	11.3±3.8	4.2±1.7	<0.001

Correlation of BMI with Reproductive Parameters

The study further evaluated the connections between body mass index and key reproductive indicators. Statistically significant negative correlations were identified between BMI and several parameters, including semen volume, sperm count, total motility, progressive motility, as well as key hormone levels such as testosterone, FSH, and LH. Conversely, positive correlations were observed between BMI and various factors, including impaired sperm motility, abnormal morphology, increased DNA fragmentation, protamine deficiency, and elevated prolactin levels. The observed relationships indicate that an increase in adiposity has a direct negative impact on both clinical outcomes and molecular indicators of male fertility.

Table 4: Correlation of Body Mass Index (BMI) with Hormonal and Semen Quality Parameters

Correlation Parameter	Pearson r	P Value
Semen Volume	-0.16	<0.001
Sperm Count	-0.62	<0.001
Total Motility	-0.40	<0.001
Progressive Motility	-0.53	<0.001
Testosterone	-0.17	<0.001
FSH	-0.42	<0.001
LH	-0.53	<0.001
PRL	+0.63	<0.001
DNA Fragmentation	+0.49	<0.001
Protamine Deficiency	+0.67	<0.001

Discussion

Obesity serves as a complex disruptor of male fertility, negatively impacting reproductive hormones and various parameters of semen quality. The findings of this study indicate a significant reduction in serum levels of follicle-stimulating hormone (FSH), luteinizing hormone (LH), and total testosterone in obese males, which is consistent with existing evidence that obesity leads to a state of hypogonadotropic hypogonadism. Aromatase activity is elevated in obese men due to the presence of increased adipose tissue, which enables the conversion of testosterone into estradiol. This process leads to an increase in the concentration of circulating estrogens, which disrupts the feedback loop of the hypothalamic-pituitary-gonadal (HPG) axis and reduces the release of gonadotropins. Endocrine disruptions adversely affect spermatogenesis by reducing the stimulation of Sertoli and Leydig cells, which in turn leads to a decline in both sperm production and quality [7]. Further to hormonal dysregulation, obesity-associated metabolic abnormalities, such as insulin resistance and chronic systemic inflammation, contribute to reproductive impairment. Hyperinsulinemia has been reported to suppress sex hormone-binding globulin (SHBG) synthesis, lowering testosterone bioavailability and exacerbating androgen deficiency. Besides hormonal effects, oxidative stress stemming from adiposity promotes sperm DNA fragmentation and protamine deficiency, which were markedly elevated in the obese cohort in this study. This molecular damage to sperm chromatin directly impedes fertilization capacity and is linked to poor embryo development and increased miscarriage rates [8, 9, 10, 11]. The prior reports that linked obesity with impaired semen parameters are corroborated by the decreased semen volume, sperm counts, and motility observed in this study. The elevated proportion of morphologically abnormal sperm is indicative of disruptions in spermatogenic processes and sperm maturation, which may involve epididymal dysfunction and alterations to the testicular microenvironment. Notably, the infertility risks are further exacerbated by the erectile dysfunction and reduced libido that have been reported in obese males, which are likely the result of compounded endocrine and psychological factors [12, 13, 14]. The negative impact of adiposity on fertility is highlighted by significant correlations between BMI and various reproductive parameters, demonstrating a dose-dependent relationship. Numerous studies indicate that weight loss achieved via lifestyle modifications or bariatric surgery can either partially or fully restore normal hormone levels and enhance semen quality, carrying important clinical implications. Improvements in testosterone and gonadotropin levels after weight reduction reaffirm the reversibility of obesity-related hypogonadism and support including fertility preservation counseling in obesity management programs [15]. Even if we know more, studies still show a lot of differences. This is probably because of differences in study design, cohort characteristics, and assessment procedures. Some inconsistencies are associated with the extent of alterations in sperm parameters and their direct effect on pregnancy outcomes, highlighting the complex, multifaceted nature of infertility influenced by both male and female variables. Recent findings emphasize the significance of novel modulators, including leptin, adipokines, and epigenetic pathways, in connection with reproductive failure associated with obesity, underscoring the need for additional research.

Conclusion
Obesity makes men less fertile by changing hormone levels, the quality of their semen, and how well their sperm works. Too much body fat throws off the balance of hormones by reducing testosterone levels and modifying how gonadotropins are released. This makes it tougher for sperm to form. Moreover, it has been noted that obese men often display reduced sperm count, impaired motility, and an increased prevalence of morphological defects in sperm, which may limit their fertility potential. Besides the physical changes already discussed, obesity is linked to higher oxidative stress, which damages DNA in sperm cells. This injury could affect both the processes of fertilization and the growth of embryos. Sexual dysfunction, which includes problems with getting an erection and a lower sex drive, makes reproductive results more complicated. The severity of these consequences shows a clear dose-response relationship with the level of obesity, meaning that the more obese a person is, the worse the effects will be. It is important to emphasize that many of the unfavorable reproductive effects of obesity can be reversed by losing weight, changing your lifestyle, or getting medical treatment. Addressing obesity as a changeable risk factor is very important for improving men's reproductive health and fertility rates. Adding fertility counseling to obesity treatment plans may be very helpful for people and couples who want to get pregnant.

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