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Impact of Bariatric Surgery on Male Fertility and Semen Parameters: A Prospective Controlled Study

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Research Article

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Abstract

Background

Obesity is a well-established cause of male hypogonadism and impaired reproductive function. Bariatric surgery induces sustained weight loss and hormonal improvement; however, its effect on male fertility and semen quality remains poorly defined and controversial.

Objective

To evaluate the effects of bariatric surgery on serum reproductive hormone profiles and semen parameters in obese infertile men, and to compare outcomes between laparoscopic sleeve gastrectomy (LSG) and Roux-en-Y gastric bypass (RYGB).

Methods

This prospective cohort study enrolled 114 obese male patients with primary or secondary infertility who underwent LSG (n = 68) or RYGB (n = 46). Semen analysis was performed according to WHO 2010 guidelines, and serum reproductive hormones were measured by standardized chemiluminescent immunoassay, both preoperatively and at 12 months. Patients were stratified by baseline semen quality and magnitude of BMI reduction. Multivariate linear regression was conducted to identify independent predictors of postoperative changes in total motile sperm count (TMC).

Results

All patients achieved significant weight loss at 12 months (median BMI: 43.5 to 29.9 kg/m², $p < 0.001$). Bariatric surgery was associated with a marked increase in total testosterone (9.1 to 16.5 nmol/L, $p < 0.001$) and a significant reduction in estradiol ($p < 0.001$), with modest LH normalization. Despite these favorable hormonal changes, semen parameters deteriorated significantly: median sperm concentration declined from 15.2 to 10.1 million/mL ($p = 0.003$), total progressive motility fell from 40% to 31% ($p < 0.001$), and median TMC decreased from 13.4 to 7.2 million ($p < 0.001$). Among oligospermic patients, 61.4% showed further decline and five progressed to azoospermia. On multivariate analysis, greater BMI reduction was independently associated with testosterone recovery ($\beta = 0.46$, $p < 0.001$) and with TMC deterioration ($\beta = -0.39$, $p < 0.001$). No significant difference in reproductive outcomes was observed between LSG and RYGB.

Conclusion

Bariatric surgery produces reliable hormonal normalization but does not improve and may worsen semen quality in infertile men. Greater postoperative weight loss paradoxically associates with further spermatogenic deterioration. These findings underscore the need for comprehensive fertility counseling and consideration of sperm cryopreservation before bariatric surgery in men of reproductive age.

Introduction

Obesity has reached pandemic proportions, with more than 2.3 billion adults classified as overweight and over 700 million as obese globally [1, 2]. This trajectory has been accompanied by a well-documented decline in male reproductive potential, raising important questions about the biological relationship between adiposity and spermatogenesis [3, 4]. Male-factor infertility accounts for approximately 40% of infertile partnerships worldwide, and epidemiological data consistently show that obese men exhibit lower testosterone concentrations, impaired gonadotropin secretion, and inferior semen quality compared with normal-weight counterparts [4, 5].

The pathophysiology of obesity-related male reproductive dysfunction is multifactorial. Visceral adipose tissue functions as a metabolically active endocrine organ, driving peripheral aromatase hyperactivity and excess androgen-to-estrogen conversion. The resultant hyperestrogenism suppresses hypothalamic GnRH pulsatility and attenuates LH secretion, producing functional hypogonadotropic hypogonadism [6, 7]. Concurrently, obesity-associated systemic inflammation and excess reactive oxygen species impair germ cell maturation, increase sperm DNA fragmentation, and reduce motility [8]. Emerging evidence further suggests that paternal obesity induces epigenetic modifications in spermatozoa with potential intergenerational consequences [9, 10].

Bariatric surgery is the most durable treatment for morbid obesity and reliably reverses obesity-related metabolic comorbidities. Multiple studies have demonstrated postoperative normalization of testosterone levels and resolution of hypogonadism in the majority of affected men, primarily attributed to visceral fat reduction and attenuation of aromatase activity [11, 12]. However, the effect of bariatric surgery on spermatogenesis remains contentious. While some studies report modest improvements in sperm parameters, others document persistent or worsening semen quality despite hormonal recovery, with rapid weight loss, micronutrient depletion, and hypercatabolic metabolic states proposed as contributing mechanisms [13, 14, 15].

Prospective data evaluating the integrated reproductive consequences of bariatric surgery in infertile men are limited. Given the growing number of men of reproductive age undergoing these procedures, a clearer understanding of their fertility implications is urgently needed. This prospective study was therefore designed to assess the impact of bariatric surgery on male fertility by evaluating changes in semen parameters and serum reproductive hormones in 114 infertile men, comparing outcomes between laparoscopic sleeve gastrectomy and Roux-en-Y gastric bypass over a 12-month follow-up period.

Patients and Methods

Study Design and Setting. This prospective, single-centre, controlled cohort study was conducted at a tertiary bariatric and male infertility referral centre in Basrah, Iraq. The study was approved by the Institutional Scientific and Ethical Committee of the College of Medicine, University of Basrah, and all procedures were conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment.

Participants. Between February 2020 and December 2024, 114 consecutive obese male patients presenting with primary or secondary infertility were enrolled. Inclusion criteria were: age 18–55 years; BMI ≥ 40 kg/m² or ≥ 35 kg/m² with at least one obesity-related comorbidity; and documented infertility of ≥ 12 months' duration meeting NIH bariatric surgery criteria. Exclusion criteria comprised prior fertility treatments, known genetic or chromosomal causes of infertility, history of vasectomy, scrotal or inguinal surgery, testicular trauma, cryptorchidism, malignancy, and pre-existing primary endocrinopathies.

Sample Size. The sample size was calculated to detect a clinically significant 25% postoperative improvement in TMC (80% power, two-sided $\alpha = 0.05$), yielding a minimum requirement of 102 patients. A total of 114 were enrolled to account for anticipated loss to follow-up.

Surgical Procedures. Participants underwent LSG (n = 68, 59.6%) or RYGB (n = 46, 40.4%), selected by a multidisciplinary bariatric team based on individual patient characteristics and comorbidity profile. All procedures were performed by a single experienced bariatric surgeon using standardized techniques. Postoperative follow-up was conducted at 1, 3, 6, and 12 months. A 12-month interval was chosen to allow weight loss stabilization, resolution of early metabolic perturbations, and completion of several spermatogenic cycles (~ 74 days each).

Semen Analysis. Semen samples were collected after 3–5 days of sexual abstinence, preoperatively and at 12 months, and analysed within 60 minutes of ejaculation according to WHO 2010 guidelines, assessing volume, sperm concentration, total and progressive motility, and morphology (Kruger strict criteria). All samples were analysed in duplicate by the same blinded laboratory technician.

Hormonal Assessment. Fasting venous blood samples were collected between 07:00 and 09:00 h to minimize circadian variation. Total testosterone, estradiol, LH, FSH, and prolactin were measured by chemiluminescent immunoassay (Siemens ADVIA Centaur XP) at baseline and 12 months postoperatively.

Patient Stratification. Patients were stratified by baseline sperm concentration into azoospermia (0 million/mL), oligospermia (< 15 million/mL), and normozoospermia (≥ 15 million/mL). They were further classified by magnitude of BMI reduction using the cohort median as a cut-off: Group A (above median) and Group B (at or below median).

Statistical Analysis. Statistical analyses used SPSS version 24.0. Categorical variables are reported as frequencies and percentages; continuous variables as median (IQR). Between-group comparisons used

the chi-square or Fisher's exact test for categorical data and the Mann–Whitney U test for continuous variables. A two-tailed $p < 0.05$ was considered significant. Multivariate linear regression, adjusted for age, baseline BMI, preoperative testosterone, and surgical technique, identified independent predictors of postoperative TMC change.

Results

Baseline Characteristics. All 114 enrolled patients completed the study. Median age was 35 years (range 30–44). Primary infertility was present in 76 patients (66.7%) and secondary infertility in 38 (33.3%). Baseline demographic characteristics, BMI, hormonal profiles, and semen parameters were comparable between the LSG and RYGB groups (Table 1).

Table 1
Baseline patient demographic characteristics.

Variable	n or Median	Range or %
Total patients	114	100%
Age (years)	35	30–44
BMI (kg/m²)	43.5	40.1–47.2
Primary infertility	76	66.7%
Secondary infertility	38	33.3%
Laparoscopic sleeve gastrectomy	68	59.6%
Roux-en-Y gastric bypass	46	40.4%

BMI, body mass index.

Weight Loss. All patients achieved substantial weight loss. Median BMI decreased from 43.5 to 29.9 kg/m² ($p < 0.001$), with no significant difference in magnitude between LSG and RYGB.

Hormonal Outcomes. Weight reduction surgery produced a significant and clinically meaningful improvement in reproductive hormone profiles. Median total testosterone rose from 9.1 nmol/L (IQR 7.3–11.4) to 16.5 nmol/L (IQR 13.9–19.1) at 12 months ($p < 0.001$). Estradiol declined significantly ($p < 0.001$). LH showed modest normalization ($p = 0.02$), while FSH and prolactin remained unchanged (Table 2).

Table 2
Serum reproductive hormone profiles before and 12 months after bariatric surgery.

Hormone	Preoperative Median (IQR)	Postoperative Median (IQR)	p-value
Total testosterone (nmol/L)	9.1 (7.3–11.4)	16.5 (13.9–19.1)	< 0.001
LH (IU/L)	4.9 (3.7–6.3)	5.6 (4.2–6.3)	0.02
FSH (IU/L)	6.2 (5.0–7.9)	6.5 (5.3–8.4)	0.19
Estradiol (pmol/L)	185 (152–226)	121 (94–147)	< 0.001
Prolactin (mIU/L)	218 (180–265)	211 (177–262)	0.48

IQR, interquartile range; LH, luteinizing hormone; FSH, follicle-stimulating hormone .

Baseline Semen Parameters. At baseline, 15 patients (13.2%) were azoospermic, 44 (38.6%) oligospermic, and 55 (48.2%) normozoospermic. Median sperm concentration was 15.2 million/mL and median TMC was 13.4 million.

Postoperative Semen Parameters. In contrast to the hormonal findings, semen parameters deteriorated significantly. Median sperm concentration fell from 15.2 to 10.1 million/mL ($p = 0.003$), total progressive motility declined from 40% to 31% ($p < 0.001$), and median TMC decreased from 13.4 to 7.2 million ($p < 0.001$). Patients with TMC < 5 million increased from 34.2% to 49.1%, while those with TMC > 20 million fell from 35.1% to 18.4% ($p < 0.05$) (Table 3). Among oligospermic patients, 61.4% deteriorated further and five progressed to azoospermia.

Table 3
Semen parameters before and 12 months after bariatric surgery.

Parameter	Preoperative Median (IQR)	Postoperative Median (IQR)	p-value
Sperm concentration (million/mL)	15.2 (6.9–33.9)	10.1 (4.2–22.1)	0.003
Total progressive motility (%)	40 (32–48)	31 (24–42)	< 0.001
Total motile sperm count (million)	13.4 (5.6–30.4)	7.2 (2.4–18.5)	< 0.001

IQR, interquartile range. p-values derived from Wilcoxon signed-rank test.

Effect of Weight Loss Magnitude and Surgical Technique. Patients with greater BMI reduction showed larger testosterone increases but paradoxically more pronounced TMC decline compared with those with lesser weight loss ($p = 0.01$). Both LSG and RYGB produced equivalent hormonal and semen outcomes, with no significant between-procedure differences (Table 4).

Table 4
Postoperative reproductive changes stratified by surgical procedure.

Outcome	LSG (n = 68) Change, Median (IQR)	RYGB (n = 46) Change, Median (IQR)	p-value
Total testosterone (nmol/L)	+ 7.2 (5.5–8.9)	+ 7.6 (5.9–9.2)	0.48
Sperm concentration (million/mL)	−5.2 (− 2.1 to − 9.8)	−5.6 (− 2.4 to − 10.3)	0.61
Total motile sperm count (million)	−6.4 (− 3.4 to − 11.1)	−6.8 (− 3.9 to − 12.0)	0.53

LSG, laparoscopic sleeve gastrectomy; RYGB, Roux-en-Y gastric bypass; IQR, interquartile range .

Multivariate Regression Analysis. On multivariate analysis, magnitude of BMI reduction was independently associated with testosterone recovery (β = 0.46, p < 0.001) and with TMC decline (β=−0.39, p < 0.001). Age, baseline testosterone, and surgical technique were not independently predictive of postoperative TMC change (Table 5).

Table 5
Multivariate linear regression: predictors of postoperative change in total motile sperm count.

Variable	β Coefficient	95% CI	p-value
Age (years)	−0.06	−0.18 to 0.05	0.29
BMI reduction (kg/m²)	−0.39	−0.55 to − 0.23	< 0.001
Baseline testosterone (nmol/L)	0.08	−0.04 to 0.19	0.18
Surgical procedure (RYGB vs LSG)	0.04	−0.01 to 0.17	0.57

β, standardized regression coefficient; CI, confidence interval; BMI, body mass index. Model adjusted for age, baseline BMI, preoperative testosterone, and surgical technique .

Discussion

This prospective study examined the reproductive consequences of bariatric surgery in 114 obese infertile men, systematically evaluating hormonal and spermatogenic outcomes over 12 months. The principal finding is a clear dissociation between hormonal normalization and spermatogenic recovery: despite significant increases in total testosterone and reductions in estradiol, sperm concentration, progressive motility, and TMC all deteriorated significantly postoperatively. This decline was most pronounced in men with pre-existing oligospermia and those achieving the greatest BMI reduction, and it occurred independently of the surgical technique employed.

Our hormonal findings are entirely consistent with the published literature. Both LSG and RYGB produced equivalent, substantial increases in total testosterone, corroborating the established concept that hormonal recovery after bariatric surgery is primarily weight loss-dependent rather than procedure-specific [20, 21, 22]. Testosterone normalization rates of 80–90% following bariatric surgery, irrespective of surgical technique, have been reported in large observational series and meta-analyses [23, 24, 25], findings our study fully corroborates.

The divergence between hormonal recovery and spermatogenic outcomes requires careful mechanistic consideration. Rapid and profound weight loss induces a transient hypercatabolic state characterized by elevated oxidative stress, mitochondrial energy imbalance, and elevated cortisol, all of which may impair germ cell development even as circulating testosterone normalizes [31, 32, 33]. Micronutrient deficiencies including zinc, selenium, folate, and vitamin B12 — are common after bariatric surgery and are directly implicated in impaired spermatogenesis, sperm DNA integrity, and chromatin remodeling [34, 35]. Emerging evidence further indicates that bariatric surgery induces global epigenetic reprogramming of spermatozoa, potentially impairing spermatogenic efficiency during active metabolic remodeling [36, 37, 38]. These mechanisms may explain our paradoxical observation that greater weight loss was independently associated with worse semen outcomes.

Our comparative analysis revealed no significant difference between LSG and RYGB in either hormonal or semen outcomes, suggesting that the negative spermatogenic impact reflects systemic metabolic consequences of rapid weight loss rather than the malabsorptive component of RYGB specifically. This is consistent with prior comparative studies [28, 42]. While some published series report improvement in semen parameters following bariatric surgery [43, 44, 45], these are typically limited by small sample sizes, inclusion of eugonadal men, and short follow-up. Prospective studies enrolling men with established infertility — the population most relevant to this question — have generally reported less favorable reproductive outcomes, consistent with our findings and corroborated by a recent systematic review [30].

The clinical implications are clear. Bariatric surgery should not be recommended or anticipated as a treatment for male infertility. Preoperative counseling must explicitly address the risk of postoperative semen deterioration, and sperm cryopreservation should be strongly considered for all men of reproductive age undergoing these procedures, particularly those with pre-existing oligospermia. Postoperative targeted nutritional supplementation, optimizing zinc, selenium, and folate should form part of routine reproductive follow-up care.

This study's strengths include its prospective design, WHO-standardized semen analysis, paired hormonal assessment, multivariate regression modelling, and head-to-head procedural comparison. Limitations include the absence of a non-surgical obese control group, lack of systematic assessment of sperm DNA fragmentation and micronutrient levels, and a 12-month follow-up horizon that may not capture late spermatogenic recovery. Longer-term prospective studies with comprehensive biological

characterization are required to determine whether the observed spermatogenic impairment is reversible.

Conclusion

In this cohort of 114 obese infertile men, bariatric surgery produced significant weight loss and reliable hormonal normalization, with near-doubling of serum testosterone and marked reduction in estradiol. However, these endocrine improvements did not translate into spermatogenic benefit. Sperm concentration, progressive motility, and total motile sperm count all declined significantly at 12 months, most notably in men with pre-existing oligospermia and those achieving the greatest weight loss. No difference in reproductive outcomes was observed between LSG and RYGB.

These findings establish a clinically important dissociation between endocrine recovery and spermatogenic function following bariatric surgery, likely driven by metabolic stress, micronutrient depletion, and epigenetic remodeling of the spermatogenic epithelium. Bariatric surgery should not be regarded as a treatment for male infertility. Comprehensive preoperative fertility counseling and sperm cryopreservation should be integral components of the bariatric surgical pathway for men of reproductive age.

Declarations

Conflicts of interest: The authors declare no conflicts of interest.

Funding: This research was self-funded. No external funding was received.

Ethical approval: Approved by the Institutional Scientific and Ethical Committee, College of Medicine, University of Basrah. All procedures complied with the Declaration of Helsinki.

Informed consent: Written informed consent was obtained from all participants prior to enrolment.

Data availability: All data supporting the findings of this study are available from the corresponding author upon reasonable request.

Provenance and peer review: Not commissioned; externally peer-reviewed.

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