ENDOCRINOLOGY

Weight Loss Through Bariatric Surgery in Men Presents Beneficial Effects on Sexual Function, Symptoms of Testosterone Deficiency, and Hormonal Profile

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ABSTRACT

Introduction: Male obesity has a negative correlation with plasma testosterone (T) levels and sexual function (SF).

Aim: To evaluate the effect of weight loss through bariatric surgery (BS) on SF, low T symptoms, and hormonal profiles in obese men.

Methods: Thirty-three men who underwent BS participated in this cohort study. Before surgery, all participants underwent clinical examinations, including anthropometric, lipid, glycemic, and hormonal evaluations. SF was evaluated using the International Index of Erectile Function (IIEF) questionnaire; low T symptoms were evaluated using the Aging Males' Symptoms (AMS) and Androgen Deficiency in the Aging Male (ADAM) questionnaires. The participants were reevaluated 6 months post-surgery.

Main outcome measures: Sex hormone profile, SF, and low T symptoms

Results: After BS, a significant increase in mean total T (201 ± 111−548 ± 190 ng/dL, $P < .001$), free T (5.8 ± 2.8−9.3 ± 3.4 ng/dL, $P < .001$), bioavailable T (110.3 ± 57.8−198.6 ± 74.3 ng/dL, $P < .001$), and sexual hormone-binding globulin (19.8 ± 13.7−54.6 ± 23.2 nmol/L, $P < .001$) levels. There was a significant decrease in estradiol (64.6 ± 27.4−29.2 ± 20.0 [pg/mL], $P < .001$). SF significantly improved. The total IIEF score increased 5.2 points (62.3 ± 7.4−67.5 ± 7.4, $P = .004$), erectile function subdomain increased 2.4 points (25.7 ± 4.1−28.1 ± 3.9, $P = .011$), desire subdomain increased 1.0 points (8.3 ± 1.5−9.3 ± 1.6, $P = .006$), and intercourse satisfaction subdomain increased 1.2 points (11.4 ± 1.9−12.6 ± 1.8, $P = .012$). Post-surgery, a 44% reduction ($P = .001$) was observed in the positive ADAM questionnaire, and improvements in all domains of the AMS questionnaire were found ($P < .001$).

Conclusion: Significant weight loss through BS improves erectile function, hormonal profile, and symptoms of T deficiency. **Machado FP, Rhoden EL, Pioner SR, et al. Weight Loss Through Bariatric Surgery in Men Presents Beneficial Effects on Sexual Function, Symptoms of Testosterone Deficiency, and Hormonal Profile. Sex Med 2021;9:100400.**

Abbreviations: ADAM, Androgen Deficiency in the Aging Male; AMS, Aging Males' Symptoms; BS, bariatric surgery; BT, bioavailable testosterone; CRP, C-reactive protein; ED, erectile dysfunction; E2, estradiol; FSH, follicle-stimulating hormone; FT, free testosterone; GBP, gastric bypass; HbA1c, glycated hemoglobin; IIEF, International Index of Erectile Function; IR, insulin resistance; LH, luteinizing hormone; MD, mean difference; MetS, metabolic syndrome; MOSH, male obesity-related secondary hypogonadism; NO, nitric oxide; PDE5, phosphodiesterase type 5; SD, standard deviation; SF, sexual function; SG, sleeve gastrectomy; SHBG, sexual hormone-binding globulin; T, testosterone; TT, total testosterone

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Key Words: Bariatric Surgery; Weight Loss; Sexual Function; Erectile Function; Testosterone; Low T Symptoms

INTRODUCTION

According to the World Health Organization, in 2016, there were 650 million obese people worldwide, corresponding to 11% of men and 15% of women. The worldwide prevalence of obesity has almost tripled between 1975 and 2016.[1] Obesity poses a serious health risk because it predisposes to the appearance of numerous chronic diseases such as type 2 diabetes mellitus, hypertension, dyslipidemia, cardiovascular diseases, sleep apnea, liver disease, osteoarthritis, and cancer, as well as social and psychological problems.[2,3] In this context, it is also known that male obesity is negatively correlated with plasma total testosterone (TT) levels and sexual performance.[4-7]

Scientific evidence demonstrates that male obesity is directly proportional to hypogonadism, as low TT levels favor weight gain, leading to persistent hypogonadism.[8] Although limited, studies have demonstrated the potential reversal of the hypogonadal state after weight loss induced by bariatric surgery (BS).[9]

Obesity is related to hypogonadism and is associated with metabolic syndrome (MetS), insulin resistance (IR), and erectile dysfunction (ED). Based on several large studies, obesity is an independent risk factor for ED.[10-19] Previous studies have attributed ED to low testosterone (T) levels and endothelial dysfunction associated with a proinflammatory state in obese individuals.[8,20]

Currently, BS is a well-established mainstay treatment option for obesity. It has low complication rates and provides consistent and long-lasting results. Surgery induces reversal and better control of several pathologies associated with weight gain. In this regard, the number of bariatric surgeries increases annually.[21-23]

Despite this physiological logic, few studies have evaluated the impact of BS on erectile function and sexual function (SF) in men.[9,24]

Does weight loss through BS improve the hormonal profile and SF of obese men?

AIMS

This study aimed to evaluate the effects of weight loss through BS on sexual dysfunction and low T symptoms affecting obese men.

METHODS

Study Design and Population

A prospective observational cohort study including 33 consecutive male BS candidates, recruited from the hospital's bariatric sector, was performed at a tertiary hospital in southern Brazil. The patients underwent surgery between February and December 2017. Twenty-five patients underwent standardized laparoscopic Roux-en-Y gastric bypass, and eight underwent laparoscopic sleeve gastrectomy. The inclusion criteria were male individuals qualified for BS[1] and did not present with any of the following exclusion criteria: previous diagnosis of hypogonadism, sexual inactivity, serious psychiatric illness, and use of T replacement therapy or medication interfering with T action or metabolism.[24] The Institutional Ethics Committee approved the study, and written informed consent was obtained from all the study participants.

In the week prior to the surgical procedure, all participants underwent medical, anthropometric, SF, low T symptoms, and biochemical and hormonal evaluations. After surgery, nutritional and medical counseling was provided by our multidisciplinary staff members according to published guidelines.[3] Medical counseling included basic information about nutrition, management of comorbidities, psychological issues, and weight regain prevention. Follow-up included routine evaluations at least 6 months after surgery, and these appointments were scheduled with the participants in the interview prior to surgery. The patients underwent the same clinical, biochemical, and hormonal evaluations as before surgery.

Clinical Examination

Anthropometric evaluation included assessment of body weight and height. Weight was measured with the subject barefoot and without clothing using an electronic scale to the nearest 0.1 kg. Height was determined using a wall-mounted stadiometer to the nearest 0.5 cm. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Excess body weight was calculated as the difference between the current body weight and body weight corresponding to a BMI of 25 kg/m^2. The weight loss percentage was calculated by dividing this value in kilograms by the initial weight.

SF was evaluated using the International Index of Erectile Function (IIEF).[25] The IIEF is a validated, self-administered test, including a series of 15 questions (maximum score: 75 points) on SF over the previous 4 weeks. The IIEF encompasses a spectrum of male SF domains: erectile function (maximum score: 30 points), orgasmic function (maximum score: 10 points), sexual desire (maximum score: 10 points), intercourse satisfaction (maximum score: 15 points), and overall satisfaction (maximum score: 10 points). The 5 sexual domains were categorized as previously described by Rosen et al.[25]

Low T symptoms were evaluated by both Aging Males' Symptoms (AMS)[26] and the Androgen Deficiency in the Aging Male (ADAM) questionnaires.[1]

The AMS scale was designed to evaluate the symptoms of aging among groups of men under different conditions, to assess the severity of symptoms over time, and to measure changes before and after T replacement. The AMS score increases point-by-point with increasing severity perceived in the complaints of the 17 items evaluated. By ticking 1 of the 5 possible severity alternatives for each item evaluated, the patient provides his or her personal perception. The total score is the sum of the scores for each dimension evaluated. The AMS questionnaire evaluates individuals in 3 dimensions: psychological, somatic, and sexual.[26]

The ADAM scale was developed from the experience at Saint Louis University. It is based on the symptoms of hypogonadism. We used questions related to ten symptoms commonly observed in elderly patients with low levels of bioavailable testosterone (BT). A positive result in the ADAM questionnaire is defined as a positive answer (yes) in questions 1 or 7 or in any other 3 questions. This questionnaire has high sensitivity and reasonable specificity, as demonstrated by Martínez-Jabaloyas et al.[1]

The patients' interviews were conducted by a trained medical professional on the day of the surgical procedure and during the follow-up evaluation in an appropriate environment.

Laboratory Measurements

Venous blood samples were drawn between 7 and 11 AM, after a fasting period of 8 hours for biochemical and hormonal evaluations before and during the follow-up evaluation. All assays were performed in the same laboratory.

Biochemical parameters included lipid profile (total cholesterol, low-density lipoprotein, high-density lipoprotein cholesterol, and triglycerides), plasma glucose, and albumin levels, which were measured by a colorimetric assay. Glycosylated hemoglobin was measured by capillary electrophoresis. High-sensitivity C-reactive protein (CRP) levels were measured using an immunoturbidimetric assay. Gonadal function evaluation included the measurement of levels of TT (normal range: 300−1,000 ng/dL), sex hormone-binding protein (SHBG) (normal range: 10−57 nmol/L), estradiol (E2) (normal range: 10−40 pg/mL), luteinizing hormone (LH) (normal range: 1.3−9.6 mIU/mL), follicle-stimulating hormone (FSH) (normal range: 1.2−15.8 mIU/mL), and prolactin (normal range: 4.0−15.2 ng/mL) using chemiluminescent immunometric assay. Free testosterone (FT) and BT were calculated according to the method of Vermeulen et al.[27]

Statistical Analysis

All variables were tested for normality using the Shapiro-Wilk test. Quantitative variables are described as mean and standard deviation (SD)/standard error, and categorical variables as absolute and relative frequencies. To compare the parameters over time, a generalized estimating equation (GEE) model was applied. The linear model was used for variables with a symmetrical distribution, while the gamma model was used for those with an asymmetric distribution. Pearson's or Spearman's correlation tests were used to verify the correlation between the changes found after BS between continuous and categorical variables.

We chose to include all the variables described by the mean and SD to calculate the magnitude of the difference with a 95% confidence interval. As one of the models of GEE is gamma, which performs the logarithmic transformation of asymmetric data and readily provides the mean and standard error of the adjusted analysis, we chose to describe all variables in the same way.

A P value < .05 was considered as statistically significant, and statistical analysis was performed using SPSS software (version 21.0; IBM, Armonk, NY, USA).

Table 1. Demographic and clinical characteristics of studied sample

Characteristics	n = 33
Age (years)*	36.3 ± 8.1
Surgical technique†	
Roux-en-Y gastric bypass	25 (75.8)
Gastric sleeve	8 (24.2)
Race†	
Caucasian	31 (93.9)
African-American	1 (3.0)
Non-Caucasian Non-African	1 (3.0)
Marital status†	
Single	6 (18.2)
Married	26 (78.8)
Divorced	1 (3.0)
Level of schooling†	
Graduated	16 (48.5)
High school	14 (42.4)
Elementary school	3 (9.1)
Work status†	
Employed	30 (90.9)
Unemployed	3 (9.1)
Comorbidities†	
Hypertension	18 (54.5)
Diabetes	5 (15.2)
Dyslipidemia	13 (39.4)
Sleep apnea	17 (51.5)
Hepatic steatosis	26 (78.8)
Gastroesophageal reflux	12 (36.4)
Asthma	4 (12.1)
Arthritis	2 (6.1)

*Continuous data are expressed as mean ± SD.
†Categorical variables are expressed as absolute number of sample size and percentage (%).

RESULTS

Clinical Outcomes

Baseline anthropometric, sociocultural, and comorbidity data of the 33 volunteers are shown in Table 1. The average age of the participants was 36.3 years (range, 22−53). Only 1 patient reported using a phosphodiesterase type 5 inhibitor regularly, and 5 individuals reported the use of psychotropics. None of the participants were addicted to alcohol or tobacco.

The mean weight loss after surgery was 45.4 ± 18.9 kg (range, 13−90). The median time between the evaluations (before and after BS) was 18 months (interquartile range: 8−34).

Of the 33 individuals analyzed before the procedure, 29 (87.9%) had TT levels below 300 ng/dL, 10 (30.3%) had an erectile function subdomain score below 25 points, suggesting some degree of ED, and 25 (75.8%) had a positive test for symptoms of hypogonadism on the ADAM questionnaire.

After BS, 20 patients were reevaluated using anthropometric measures and venous blood samples for questionnaire answers and biochemical and hormonal analyses. There was a loss of 13 participants. The reason for this loss was the patients' non-adherence to the established follow-up protocol. We tried to rescue this lost data without success.

Hormonal Profile

Table 2 depicts the mean ± SD of anthropometric, biochemical, and hormonal profiles before and after surgery. There were statistically significant increases in mean TT, FT, BT, and SHBG levels after surgery. In addition, there were statistically significant decreases in E2 and prolactin levels after BS. However, there were no statistically significant differences in mean LH and FSH levels.

There was a negative statistically significant correlation between BMI and TT variations (r = −0.637, P = .008) and FT variations (r = −0.561, P = .024), and individuals with the most reduced BMI had the highest TT and FT levels.

There was also a positive statistically significant correlation between the time between evaluations (before and after BS) and BT (r = 0.559, P = .020), with the ones that most increased BT being those with the longest time between evaluations.

Sexual Function

The evaluation of SF by questionnaires demonstrated that the total IIEF mean score increased by 5.2 points (P = .004). Analyses after surgery also demonstrated significant improvements in erectile function, sexual desire, and intercourse satisfaction scores. The erectile function subdomain increased by 2.4 points (P = .011), the sexual desire subdomain increased by 1.0 point (P = .006), and the intercourse satisfaction subdomain increased by 1.2 points (P = .012). There were no statistically significant differences in orgasm and overall satisfaction subdomains. The data are presented in Table 3.

Considering a significance level of 5%, a minimum difference of 2 points in the erectile function subdomain scores with an SD of 4 points, the power for the sample obtained was 79.6%. As the literature points to a minimum power of 80.0% for the

Table 2. Anthropometric, biochemical, and hormonal evaluations at baseline and after surgery

Variables*	Baseline (n = 33)	After surgery (n = 20)	Difference (95% CI)	P
BMI (kg/m^2)*	43.8 ± 7.8	27.7 ± 2.9	−16.1 (−18.9 to −13.3)	<.001
Total cholesterol (mg/dL)*	164 ± 45.8	152.6 ± 39.6	−11.4 (−37.3 to 14.6)	.391
HDL cholesterol (mg/dL)*	35.9 ± 16.1	47.2 ± 12.1	11.3 (2.6−20)	.011
LDL cholesterol (mg/dL)*	109.2 ± 42.5	86.8 ± 32.8	−22.4 (−43.5 to −1.3)	.037
Triglycerides (mg/dL)†	173.5 ± 121.9	120.3 ± 133.7	−53.2 (−129.5 to 23.1)	.171
Plasma glucose (mg/dL)*	98.8 ± 27.6	86.6 ± 18.2	−12.2 (−21.5 to −2.9)	.010
HbA1c (%)*	5.8 ± 0.9	4.9 ± 0.5	−0.8 (−1.2 to −0.4)	<.001
CRP (mg/L)†	6.0 ± 6.4	0.9 ± 1.7	−5.1 (−7.7 to −2.6)	<.001
Albumin (g/dL)	3.8 ± 0.6	4.3 ± 0.4	0.5 (0.2−0.7)	<.001
TT (ng/dL)†	201.3 ± 111.7	548.2 ± 190.1	346.9 (255.9−438)	<.001
FT (ng/dL)*	5.8 ± 2.8	9.3 ± 3.4	3.5 (1.8−5.2)	<.001
BT (ng/dL)†	110.3 ± 57.8	198.6 ± 74.3	88.3 (51.9−124.7)	<.001
SHBG (nmol/L)†	19.8 ± 13.7	54.8 ± 23.2	35.0 (25.2−44.7)	<.001
FSH (mIU/mL)*	3.2 ± 1.7	4.0 ± 2.3	0.8 (−0.3 to 1.9)	.168
LH (mIU/mL)*	3.4 ± 1.8	4.3 ± 1.7	0.9 (−0.1 to 1.8)	.064
Estradiol (pg/mL)†	64.6 ± 27.4	29.2 ± 20.0	−35.4 (−48.8 to −21.9)	<.001
Prolactin (ng/mL)†	51.1 ± 42.0	8.8 ± 6.5	−42.3 (−59.2 to −25.4)	<.001

*Data are described as mean ± SD.
†Data underwent logarithmic transformation through the gamma model.
BMI = body mass index; BT = bioavailable testosterone; CRP = C-reactive protein; FSH = follicle-stimulating hormone; FT = free testosterone; HbA1c = glycated hemoglobin; LH = luteinizing hormone; SHBG = sexual hormone-binding globulin; TT = total testosterone.

Table 3. International Index of Erectile Function scores at baseline and after surgery

Variables*	Baseline (n = 33)	After surgery (n = 20)	Difference (95% CI)	*P*
IIEF (subdomains)				
Erectile function	25.7 ± 4.1	28.1 ± 3.9	2.4 (0.5–4.2)	.011
Orgasm	9.1 ± 1.2	9.2 ± 1.7	0.1 (−0.6 to 0.7)	.922
Desire	8.3 ± 1.5	9.3 ± 1.6	1.0 (0.3–1.7)	.006
Intercourse satisfaction	11.4 ± 1.9	12.6 ± 1.8	1.2 (0.3–2.1)	.012
Overall satisfaction	7.8 ± 1.6	8.4 ± 1.4	0.6 (−0.2 to 1.3)	.158
Total	62.3 ± 7.4	67.5 ± 7.4	5.2 (1.7–8.6)	.004

*Data are described as mean ± SD.
IIEF = International Index of Erectile Function.

hypothesis test to be statistically relevant, the power found is very close to the minimum value indicated.

When weight loss, variations in BMI, and weight loss percentage were correlated with IIEF scores, there were statistically significant correlations between SF improvement (IIEF total score) and weight loss (r = −0.694, *P* = .001), BMI (r = 0.599, *P* = .007), and weight loss percentage (r = −0.607, *P* = .06), demonstrating that those who lost the least weight had the most improved SF. However, there was no statistically significant correlation among weight loss (r = 0.420, *P* = .074), BMI (r = 0.396, *P* = .093), weight loss percentage (r = 0.387, *P* = .101), or erectile function (erectile subdomain in IIEF).

Low T Symptoms

The symptoms of low T, based on the ADAM and AMS questionnaires, significantly improved after surgery. At baseline, 75.8% of the individuals tested positive based on the ADAM questionnaire, and 31.6% tested positive after surgery (*P* = .001). The AMS total score also improved (*P* < .001) as well as the 3 dimensions of psychological (*P* < .001), somatic (*P* < .001), and sexual (*P* = .013). These data are clearly shown in Table 4.

When weight loss was correlated with scores on the ADAM and AMS questionnaires, there was no statistically significant correlation between improvement in ADAM (r = 0.037, *P* = .881) and AMS (r = 0.323, *P* = .178) scores and weight loss after BS.

DISCUSSION

In the current study, weight loss through BS significantly improved the levels of sex hormones and SF and reduced the low T symptoms. Furthermore, a significant correlation between weight loss and increased TT and FT levels was observed. Thus, the results presented here add more body to the current evidence.

According to Cohen's theory, it is relatively well established that male hypogonadism and obesity are related in which weight gain promotes low T levels, inducing persistent hypogonadism.[8] In general, increased BMI is related to decreased androgen biosynthesis and increased peripheral aromatization of androgens to estrogens promoted by adipose tissue. This results in reduced SHBG and TT levels and increased E2 plasma levels, causing a disturbance in the negative feedback loop of the hypothalamic-pituitary-testicular axis, reducing Sertoli cell functioning, and leading to a persistent state of hypogonadism.[28-32] According to Fernandez et al, individuals with male obesity-related secondary hypogonadism (MOSH) exhibited a normal LH and FSH response to gonadotropin-releasing hormone, indicating a hypothalamic defect rather than a pituitary defect.[33] Although the actual prevalence of MOSH is still unclear, some studies suggest rates as high as 45%−57%.[33,34]

In the current study, the prevalence of low T levels in such a selected and specific population reached a rate of 87.87%, considering a TT cutoff below 300 ng/dL.

The association of obesity with MetS and ED is the subject of significant attention and debate in the literature.[7,35-38] The high

Table 4. Aging males' symptoms and androgen deficiency in the aging male scores at baseline and after surgery

Variables	Baseline (n = 33)	After surgery (n = 20)	Difference (95% CI)	*P*
ADAM*	25(75.8)	6(31.6)	−44.2 (−70 to −17)	.001
AMS† (dimensions)				
Psychological	11.0 ± 3.4	7.8 ± 2.7	−3.2 (−4.5 to −1.8)	<.001
Somatic	19.1 ± 5.1	10.9 ± 3.8	−8.2 (−10.2 to −6.1)	<.001
Sexual	8.8 ± 3.5	6.8 ± 2.6	−2.0 (−3.7 to −0.4)	<.013
Total	38.9 ± 10.2	25.6 ± 7.4	−13.4 (−17.8 to −9.0)	<.001

*ADAM results are described as n (%).
†AMS scores are described as mean ± SD.
ADAM = Androgen Deficiency in the Aging Male; AMS = Aging Males' Symptoms.

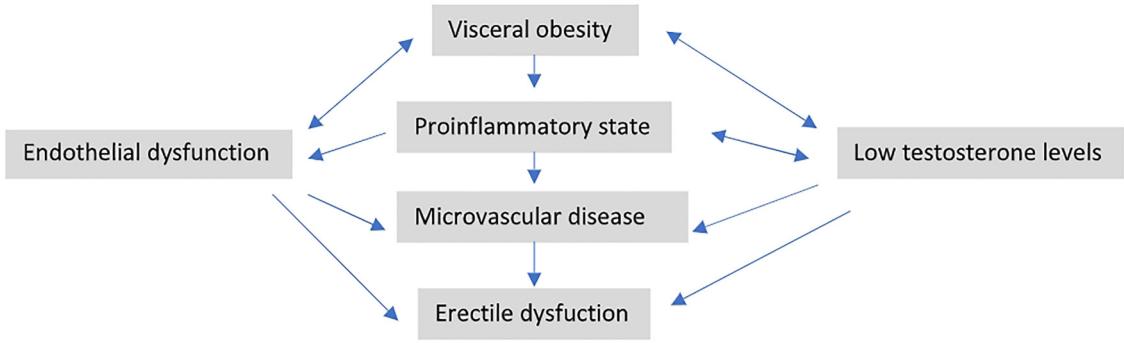

Figure 1. Relationship between visceral obesity and erectile dysfunction This model suggests that visceral obesity contributes to ED through 3 interdependent pathways: (a) a proinflammatory state promoted by an imbalance of cytokines and adipokines results in microvascular disease and endothelial dysfunction; (b) this endothelial dysfunction reduced nitric oxide (NO) synthase activity and NO production, resulting in an abnormal penile hemodynamic state and ED; and (c) obesity promotes an endocrine milieu disruption represented by a decrease in T levels and an increase in E2 levels, which contribute to a dysfunction in the veno-occlusive mechanism of the penis and ED. Adapted from Traish et al.[38]

incidence of ED in obese individuals is explained by several common coexisting clinical conditions associated with excess adipose tissue, such as diabetes, cardiovascular diseases, and dyslipidemia. In our study, 30.3% of obese patients who underwent BS had some degree of ED based on a cutoff point of 25 in the IIEF questionnaire.

The mechanism by which endothelial function contributes to penile erection physiology is well-documented. Thus, any element that leads to endothelial dysfunction may promote ED.[38] In addition, there is evidence that excess adipose tissue found in obese individuals increases the risk of cardiometabolic events due to endothelial dysfunction promoted by proinflammatory factors such as plasminogen activator inhibitor-1, tumor necrosis factor alpha, leptin, angiotensinogen, and reduced adiponectin levels, which are associated with the increased oxidative stress generated by adipocytes. In addition, increased serum levels of CRP also cause endothelial dysfunction, and obesity is associated with increased CRP concentrations.[39-41] This imbalance in cytokines and adipokines results in endothelial dysfunction.[38]

The imbalanced mechanism mentioned earlier leads to a pathophysiological state that reduces peripheral tissue hemodynamics. The inflammatory status promoted by obesity can damage the endothelium and contribute to ED.[38] Based on several large studies, it can be stated that obesity represents an independent risk factor for ED[10-19] (Figure 1).

Despite the biological plausibility, few studies have confirmed the potential reversal of the hypogonadal state and ED in obese men.

In a meta-analysis conducted by Lee et al, only 5 studies analyzed erectile function using the IIEF questionnaire in obese populations who underwent BS. In these studies, BS induced a small but statistically significant improvement in the IIEF results (mean difference [MD] 0.46, 95% CI 0.89 to −0.02, P = .04; I^2=70%).[9] Similarly, in the current study, it was also interesting to observe an improvement in the mean IIEF score of erectile function (25.7 ± 4.1 to 28.1 ± 3.9, P = .011). This improvement of 2.4 points can be described as modest, based on the study conducted by Rosen et al.[42] However, the crucial point is that the individuals included in our cohort were young men with minimum ED and a mean of 25.7/30 points in the IIEF-5, and therefore have only mild endothelial dysfunction, such that improvements of 5 points were not possible.

In the current study, it was interesting to observe a 2.72-, 1.6-, and 1.80-fold increase in the mean TT, FT, and BT levels, respectively, after weight loss induced by BS. Furthermore, we demonstrated a statistically significant correlation between weight loss through BS and an increase in TT (r = −0.637, P = .008) and FT (r = −0.561, P = .024) levels.

Lee et al, as mentioned above, conducted the largest systematic review and meta-analysis on the effects of BS on sex hormones. In this evaluation, 28 studies published between 1998 and 2018 filled out specific criteria for inclusion. The results demonstrated that BS increased TT and FT levels as well as LH, FSH, and SHBG levels in men who underwent this procedure. In addition, a reduction in E2 and prolactin levels has also been observed.[9]

Similarly, Wood et al demonstrated in a prospective study that the median TT more than doubled 6 months after BS (294.5 −604 ng/dL, P < .0001). Moreover, they demonstrated that patients who experienced greater weight loss had greater postoperative changes in TT levels, in a clear "dose-dependent" effect.[43]

LIMITATIONS

First, we had a significant loss of participants throughout the study, and although we tried to rescue this lost data, we were unsuccessful. Second, there was a large variation between the measurement times after the procedure.

In addition, we need to carefully analyze the data regarding the improvement in low T symptoms. Despite the ADAM and AMS questionnaires being widely used, they are not well-supported or validated scales. Therefore, our results may not be convincing.

STRENGTHS

To the best of our knowledge, this is the first study to evaluate SF and low T symptoms through the IIEF, ADAM, and AMS questionnaires simultaneously before and after BS. In addition, we evaluated the hormonal and biochemical profiles.

Despite the biological plausibility, few studies have confirmed the potential reversal of the hypogonadal state and ED in obese men. One of the reasons for this is the underrepresentation of the male population in BS since men represent only 20% of patients undergoing BS, which does not correspond to the prevalence ratio of obesity in the general population. In addition, social and cultural factors make it difficult to collect and analyze data in the sexual sphere.[44,45]

CONCLUSION

Our data showed that there was an improvement in SF, symptoms of T deficiency, and sex hormone profiles after weight loss induced by BS. Therefore, ED and T deficiency may be considered conditions with potential improvement after weight loss in obese men. Long-term studies with more specific evaluations are warranted to better understand the results observed in the current evaluation and to assess if these changes stabilize over time and if a possible future weight gain would affect these findings.

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STATEMENT OF AUTHORSHIP

Machado FP: Conceptualization, Investigation, Writing - Original Draft; Rhoden EL: Conceptualization, Supervision, Project administration; Pioner SR: Resources; Halmenschlager G: Formal analysis, Writing - Review & Editing; Souza LVB: Investigation; Lisot BC: Investigation; Drachler IP: Investigation.

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