Reproductive Complications After Bariatric Surgery in Males and Females

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The reproduction of mankind is a great marvel and mystery. Had God consulted me in the matter, I should have advised him to continue the generation of the species by fashioning them of clay.

—Martin Luther

15.1 Female Reproductive System

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15.1.1 Introduction

According to the World Health Organization, obesity has tripled since 1975. In 2016, more than 1.9 billion adults, 18 years and older, were overweight including more than 650 million obese adults. Furthermore; over 340 million children and adolescents aged 5–19 were overweight or obese in the same year [1].

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A. G. Bhasker et al. (eds.), *Management of Nutritional and Metabolic Complications of Bariatric Surgery*,
https://doi.org/10.1007/978-981-33-4702-1_15

The number of bariatric & metabolic surgeries (BMS) is on the rise worldwide [2]. It is conceivable that physicians of all specialties will be encountering young females of child bearing age with previous BMS. Therefore, it is of uttermost importance that we understand the long-term hormonal and reproductive implications of BMS.

15.1.2 Obesity and Infertility

Infertility affects one in seven couples [3], and in a significant proportion of these affected couples obesity is directly or indirectly associated with anovulation, subfertility, or infertility [3].

In addition, just being overweight with a body mass index (BMI) > 25 kg/m^2 has a negative impact on the hypothalamic-pituitary-gonadal axis, on gametogenesis, and the outcomes of assisted reproductive technology [4].

Gonadal dysfunction can be one of the most prevalent co-morbidities in morbidly obese and is present in up to 36% of women undergoing BMS [5]. Obesity also increases the rate of miscarriage regardless of the mode of conception [3].

The term "obesity associated gonadal dysfunction" OAGD was introduced in 2014 to describe patients in which the normal adipose tissue distribution and function (dictated by low androgen and high estrogen concentrations) is lost due to an imbalance favoring the increase in androgen levels leading to a change in adipose tissue distribution. This imbalance occurs due to obesity and visceral adiposity [6]. This leads to a vicious cycle of androgen excess, abdominal deposition of body and visceral fat. Further androgen production by the peripheral fat leads to gonadal dysfunction which can be ameliorated by weight loss [7]. However, the literature on the effect of BMS on womens' reproductive health outcomes is still limited [8].

In this chapter, we will discuss important topics pertaining to the reproductive health of young women undergoing BMS including postoperative hormonal changes, pregnancy after bariatric surgery, nutritional deficiencies, sexual functions, and pelvic floor disorders.

15.1.3 Sexual Dysfunction and Pelvic Floor Disorders

15.1.3.1 Effect of Surgery on Sexual Function

Obesity and its associated comorbidities increase the probability of sexual disorders [9]. Female sexual dysfunction can be widespread and is affected by biological, social, as well as psychological factors. In addition, female Functional Sexual Index (FSFI) is a validated self-report questionnaire to assess the level of sexual function. It has six domains which include desire, arousal, lubrication, orgasm, satisfaction, and pain [10]. The Sexual Quality of Life-Female scores is another questionnaire that assesses the impact of sexual dysfunction on quality of life [11]. These tools are used to measure sexual dysfunction in females.

Female sexual dysfunction is more commonly diagnosed in the obese population [9]. FSFI has been shown to strongly correlate with BMI. Both obesity and BMI have been shown to affect multiple sexual function parameters from FSFI including arousal, orgasm, satisfaction, and lubrication. Obesity and BMI did not affect desire and pain [12].

However; sexual dysfunction in females with obesity appears to be improved by marked weight reduction [12]. This improvement has been demonstrated in women after BMS as well [9]. Women who lose weight after surgery will feel more attractive, have higher self-esteem, and have decreased anxiety which will translate into better Sexual Quality of Life-Female scores that describe the emotional aspects of their sexual life. The desire and arousal domains of FSFI are significantly improved after BMS as well [13]. A key issue is the excessive hanging skin after BMS, which the majority of patients develop, and its negative effect on the benefits of surgery. Excessive skin can cause overall impairment in daily life and affect sexual function and intimacy as well. The associated depression and concerns regarding body image can contribute to weight regain. Thus; it is important to optimize body image issues to optimize the psychological and weight outcomes of BMS [14].

In conclusion, obesity increases the probability of sexual disorders and dysfunction. Sexual dysfunction is improved by both marked weight loss and after BMS.

15.1.3.2 Effect of Surgery on Pelvic Floor Disorders

Obesity is an established risk factor for pelvic floor disorders. The risk of developing urinary incontinence for example increases with increasing BMI across all BMI classes [15].

BMS improves pelvic floor disorders through multiple mechanisms. The decrease in intra-abdominal pressure that accompanies weight loss has been suggested as one factor. The resolution of comorbidities like diabetes and hypertension is another contributing factor [16].

Furthermore, surgery has been shown in multiple studies to significantly improve urinary incontinence, pelvic organ prolapse, and colorectal/anal symptoms but not necessarily fecal incontinence [16–18].

BMS has been reported to result in resolution of urinary incontinence in 52% of preoperatively incontinent patients [18]. Relative risk reduction of urinary incontinence of 67% and pelvic organ prolapse of 52% postoperatively has been reported [19].

The risk of pelvic floor disorders including urinary incontinence increases with obesity. BMS and its associated weight loss along with resolution of comorbidities such as diabetes and hypertension lead to improvement or resolution of pelvic floor disorders.

15.1.4 Hormonal Changes in Women After BMS

Multiple complex hormonal mechanisms contribute to female obesity besides genetic predisposition. Abdominal obesity can perpetuate a vicious cycle where

abdominal adiposity leads to increased peripheral estrogen production through increased aromatase activity leading to androgen deficiency. This decreases muscle mass and increases abdominal adiposity. The fat cells cause a decrease in adiponectin levels and an increase in tumor necrosis factor alpha and interleukin 6 which will aggravate peripheral insulin resistance, obesity, and perpetuate the vicious cycle [5].

The exact hormonal changes and their effect on female reproductive function after BMS is not fully understood. However, few studies have described hormonal and menstrual changes after BMS.

A study assessing 4 years outcome of sex hormones after BMS revealed overall significant improvement in reproductive hormones. Testosterone and dehydroepiandosterone sulfate levels decreased over 4 years. Follicle stimulating hormone, leutinizing hormone, and sex hormone binding globulin levels all significantly increased after surgery. The exception was estradiol which did not change after BMS [20].

A second study following patients after RYGB with urinary progestin measurements at 12 months revealed that ovulatory frequency and luteal phase quality was not changed but the follicular phase was shorter. Biochemical hyperandrogenism improved with no change in the associated clinical features like sebum production, hirsutism, or acne. Thus ovulation rate and quality appears to be relatively unaffected by morbid obesity, body composition, caloric restriction after gastric bypass, or long-term weight loss [21].

One of the indicators of fertility and ovarian reserve is the Anti-Mullerian Hormone (AMH) and it represents the severity of polycystic ovary syndrome (PCOS). AMH is significantly higher in the PCOS patients in both women with or without obesity and is also positively related to testosterone, androstenedione, and dehydroepiandosterone [22]. After BMS, AMH levels decreases in patients whether or not they suffered from PCOS before surgery. However, those patients with preoperative PCOS had a higher chance of AMH levels normalizing postoperatively [22].

Obesity perpetuates a vicious cycle of hormonal imbalance and overall androgen deficiency. Postoperatively, BMS leads to improvement of reproductive hormones with no change in the ovulation frequency or ovum quality.

15.1.5 Pregnancy and Bariatric Surgery

15.1.5.1 Pregnancy Outcomes and Surgery-to-Conception Interval

BMS has positive effects on fertility. With these positive changes on fertility, inadvertent pregnancy despite adequate conventional contraception is possible after RYGB [23].

There is a lot of debate regarding the “Ideal” surgery-to-conception timing. Early conception during the time of rapid weight loss after BMS has been speculated to cause preterm delivery, fetal growth restriction, low birth weight, and congenital anomalies. In addition the weight gain of conception during time of maximal weight loss can hypothetically limit the maternal benefit of postoperative weight loss [24].

Two studies reported the outcomes of surgery-to-conception-interval on pregnancy following BMS. The first study compared early (≤18 months) versus late (>18 months) conception after RYGB. There was no difference in adverse obstetrical outcomes (preterm premature rupture of membranes, gestational diabetes, oligohydramnios, intrauterine growth restriction, pre-term or post-term delivery) or adverse newborn outcomes (Apgar score < 7, intensive care admission, or birth defects) [24].

A second study used the same time interval criteria to compare outcomes after SG. The study revealed that the late pregnancy group had higher gestational weight gain (11 vs. 8 kg), lower hemoglobin in early pregnancy (10 vs. 10.4 gm/dl), and after delivery (12.3 vs. 12.6 gm/dl). The same study showed that the other maternal and perinatal outcomes were similar between the two groups [25].

Several studies compared pregnancy outcomes after BMS with matched controlled pregnancies. The first studies examined the Swedish Medical Birth Register. In the Swedish study, pregnancies post-surgery were associated with lower risk of gestational diabetes and large for gestational age infants. In contrast, the rates of small for gestational age and short gestation were higher (although the risk of preterm birth was not significantly different) [26]. Similarly, a study examining birth certificates and maternally linked discharge data in Washington State reported higher risk of small for gestational age, prematurity, neonatal intensive care admission, and lower Apgar score. This study however, reported that a long surgery-to-conception interval of more than 4 years (not 18 months like the aforementioned studies) is actually associated with lower risk of prematurity, small for gestational age, and neonatal intensive care admission [27].

Other reported outcomes of post bariatric surgery conception included lower risks of macrosomic infants, lower risk of prolonged labor, and lower risk of cesarean section which has been reported by some authors while others reported that mothers with prior BMS who remained obese were most likely to undergo a cesarean section [27–29] (Table 15.1).

15.1.5.2 Gestational Diabetes and Oral Glucose Tolerance Test

Gestational diabetes is frequently encountered during pregnancy, and BMS performed before pregnancy significantly reduces the risk of gestational diabetes but the risk is still higher than the normal weight population [30]. In addition, the oral

Table 15.1 Change of maternal and neonatal risks after bariatric surgery*

Increased risk	Decreased risk	No significant risk	Controversial
Small for gestational age	Gestational diabetes	Preterm birth	Cesarean section
Short gestation	Large for gestational age		
Neonatal intensive care admission	Macrosomia		
Lower Apgar score	Prolonged labor		

*Risks compared to general population

glucose tolerance test used to diagnose gestational diabetes is challenging in pregnant women after BMS due to hypoglycemia during the test in up to half of the patients with previous BMS and it is likely to induce symptoms of dumping syndrome. RYGB and short surgery to conception interval are associated with higher risk of developing hypoglycemic symptoms. Interestingly; hypoglycemia symptoms did not correlate with gestational diabetes and patients who developed hypoglycemia had lower rates of gestational diabetes [31] An alternative screening methods for gestational diabetes for this patient population includes a non-fasting 50 gm glucose challenge test which seems to be better tolerated or daily capillary blood glucose measurements before and after meals for 3–7 days to be done between the 24 to the 28-week of pregnancy [30].

15.1.5.3 Nutritional Deficiencies and Birth Defects

The risk of birth defects after BMS have been suggested to either be increased secondary to nutritional deficiencies or decreased due to the effect of decreased maternal BMI [32].

All patients undergoing BMS should receive daily multivitamins with trace minerals as they are at risk for deficiency of vitamins including B12, B1, C, folate, A, D, K, irons, selenium, zinc, and copper [33]. These common postoperative deficiencies can be exacerbated by the effects of pregnancy like morning sickness, hyperemesis, reflux, and bloating [34]. It has been shown that BMS increases the risk of small for age infants and micro-nutrient deficiencies in the mother and the newborn [35, 36].

Compliance with vitamin supplementation after BMS is a significant concern. A single center study at a university in France followed 48 adult pregnant women who underwent BMS (AGB, SG & RYGB). In this French study, only 56.8% took vitamins preconception, 77.8% during the first trimester, 96.3% during the second trimester, and 100% during the last trimester. Nutritional deficiencies included Vitamins A, D, C, B1, B9, and selenium. Interestingly, and despite supplements vitamin A and C deficiencies increased throughout the third trimester [35].

Another study compared cord blood samples from newborns from RYGB mothers to newborns from non-obese mothers. The post RYGB neonates had higher chance of having lower concentrations of calcium, zinc, iron, and vitamin A. Currently, a comparison to the neonates of morbidly obese mother is lacking [36].

Apart from the aforementioned deficiencies, vitamin A levels seem to be negatively impacted by BMS, and when combined with pregnancy, the risk is greater [37]. This can lead to low retinol and b-carotene levels during pregnancy which might increase the risk for developing night blindness [37]. However, not all Vitamin A supplements are safe during pregnancy (retinol-based products are best avoided) [34].

It is advisable to wait at least 12 months or until the weight stabilizes postoperatively before pregnancy. In case of pregnancy, a multidisciplinary team approach is key to monitor nutrition. Adequate prenatal supplementation should start at the preconception phase as iron and vitamins A, B12, K, and folate deficiencies are associated with maternal and fetal complications especially because over-the-counter

supplements might not provide the required amounts of certain micronutrients in this high-risk population [33, 34].

The final key issue to be discussed is whether BMS increases the risk of birth defects. Two major studies assessed the risk of birth defects in pregnant mothers after BMS. The first examined the Swedish Medical Birth Register from 2006 to 2011 (matched controlled study) and the second examined birth certificates and linked discharge data in Washington State from 1980 to 2013 (population based retrospective cohort study). Both studies did not find any significant difference in the risk of congenital malformations and birth defects after BMS [26, 27].

In conclusion, fertility improves after BMS. The "Ideal" surgery-conception interval seems to be between 12 and 18 months post-surgery or after weight stabilization. BMS is associated with lower risk of gestational diabetes, large for gestational age, macrosomia, cesarean section, and prolonged labor. BMS was not shown to increase the risks of birth defects, Vitamin supplementation should start preconception and continue throughout the pregnancy.

Key Points

1. *Obesity is a significant cause of female infertility/subfertility*
2. *The "Ideal" surgery-conception interval seems to be between 12 and 18 months post-surgery or after weight stabilization.*
3. *Obesity causes hypogonadism secondary to increased oestrogen suppression of pituitary, decreasing the production of LH and FSH*
4. *The decreased fertility in obesity is due to hypogonadism, decreased sexual function, as well as effects on fertilisation, and conception.*
5. *Bariatric surgery improves frequency of ovulation, oocyte quality, fecundity, and decreases the risk of Gestational diabetes mellitus, miscarriages and macrosomia. There is also a lower risk of prolonged labor and emergency LSCS after bariatric surgery*
6. *Studies have not found any significant difference in the risk of congenital malformations and birth defects after BMS*
7. *Common preoperative deficiencies may be exacerbated by pregnancy.*

15.2 Male Reproductive Complications

Ahmad Bashir, Ashraf Haddad, and Abdelrahman Nimeri

15.2.1 Introduction

Although obesity rates were more common in females, the CDC reports that this gap has almost disappeared with more pronounced and visible effects on their reproductive system [38]. Nevertheless, more females undergo bariatric and metabolic surgery (BMS). Male Obesity-Secondary Hypogonadism (MOSH) is well known, and its negative effects on fertility and erectile function are established in

males [39] but may be understated in the bariatric patient population. There are very few reports on MOSH and its outcome after bariatric surgery. Most of the published studies report only the early outcomes. The number of studies reporting complications of bariatric surgery on reproductive system are scant, and generally, lack of improvement of preoperative sexual dysfunction in the presence of residual obesity or weight regain is more common than complications affecting the system directly after significant and maintained weight loss. In this segment of the chapter, we will discuss the effects of bariatric surgery on obesity related hypogonadism, sex hormone changes, sperm & seminal fluid, erectile dysfunction and sexual quality of life along with reported complications of bariatric surgery on reproductive system in males.

15.2.2 Male Obesity-Secondary Hypogonadism (MOSH)

MOSH in males is defined as a decrease in circulating Testosterone with low or reduced gonadotropin [40]. It affects 45–64% of patients suffering from obesity through direct and indirect effects [5, 39, 41]. We will look at the effects of obesity & bariatric surgery on sex hormones, sperm & seminal fluid, sexual dysfunction & quality of life secondary to these problems.

15.2.2.1 Sex Hormones

Obesity & age affect gonadal hormones as well as seminal quality in men [42]. In a large case-cohort study involving more than 2000 subjects, increasing BMI was associated with decrease in Testosterone, inhibin B & LH levels in males after adjusting for other comorbidities.

A recent systematic review on the effects of obesity and weight loss on sex hormone levels showed a decrease in total and free testosterone along with a decrease in Sex Hormone-Binding Globulin (SHBG) with increasing obesity [43]. It was previously thought that the decrease in SHBG was the reason for decrease in TT. However, FT is also reduced in males suffering from obesity. This meta-analysis included comparative studies analysing the effect of low-calorie diet (LCD) or bariatric surgery (BS) on sex hormones levels before and after weight loss, including 11 randomized controlled trials (RCTs) (9 LCD, 2 BS). Overall, with successful weight loss TT & FT, SHBG, and Gonadotropins (LH and FSH) increased. Circulating estrogen decreased with weight loss. The changes were proportional to the weight loss, especially in young non-diabetic patients. BS was associated with a greater improvement due to higher persistent weight loss, more so after Biliopancreatic Diversion (BPD). Decrease in fat mass leading to less estrogen production by adipose tissue and thus reducing the negative feedback on HPA axis has been suggested as the mechanism for the above changes. Other mechanisms suggested are: Central Leptin resistance, Insulin resistance, and decreased production of SHBG [39]. In a paper by Calderon et al., TT & FT levels were inversely related to both glucose and HOMA-IR, with glucose being the only variable inducing variability in both TT & FT.

Calderon et al. [41] studied the effect of three surgery types on the change in sex hormones: gastric bypass, sleeve gastrectomy and adjustable gastric banding. All types were successful in inducing improvement in sex hormones measured. Only SHBG was significantly higher in gastric bypass as opposed to other types. FT increase correlated with a decrease in fasting blood sugar. No statistical significance was found in other hormone changes, glycemic resolution or weight loss in a short follow-up period of 6 months.

When comparing the levels after bariatric surgery between the froup with obesity and a lean control group over 6–16 years, Rosenblatt et al. [44] demonstrated that changes in hormones after bariatric surgery (ORYGB and LRYGB) are sustained. TT, FT & SHBG were all significantly higher in bariatric group compared to obese controls and not statistically different from lean controls. LH & FSH were greater in bariatric group with normal LH/FSH ration. BMI correlated with levels of TT & FT in all three groups. Persistent or recurrent obesity led to decreased levels of hormones compared to patients maintaining weight loss.

15.2.2.2 Sperm and Seminal Fluid

It may expected that spermatogenesis and seminal fluid would be affected in obesity, given the changes in sex hormones.

Paasch et al. [42], compared a large population from an infertility clinic to a set of controls for sex hormones and sperm analysis. The two groups were matched for hormonal imbalances, and differed only in weight. They found an inverse U-shaped curve distribution of Total Sperm Count (TSC) and Total Sperm Count with normal morphology (TCN). Both underweight and subjects with obesity had lower counts than normal BMI or BMI in the overweight range. Motility did not appear to be affected by BMI. Age affected motility greater than BMI. However, in the age group 20–30 years, BMI inversely correlated with motility and therefore impacted fertility.

In another study, Samavat et al. [45] matched patients with obesity seeking surgical weight loss, with lean controls. In addition to the semen analysis, they also looked at acrosome reaction (AR), zona pellucida (ZP) binding and their actual timings as these determine fertility more accurately. They found a statistically significant difference in the volume and immotile fraction (50.2% vs. 37.4% respectively in patients with obesity versus lean subjects). Azoospermia was only present in patients with obesity (0% in lean subjects vs. 13% in obese group). Oligospermia & asthenozoospermia were more common in the group with obesity. Due to above findings, only 57% of the patients in group with obesity were able to complete the AR & ZP testing, while 76% of lean subjects did it. Spermatozoa in group with obesity had reduced ability to respond to respond to proper stimuli, & instead had a higher percentage of spontaneous AR without ZP binding, lacking the proper timing to bind, thus the reduced fertility.

A meta-analysis by Campbell et al. [46] studied more than 30 papers with greater than 115 thousand participants. Infertility, assisted reproduction & non-viable pregnancy were more common in couples with a male suffering from obesity. Routine parameters in semen analysis did not differ significantly: Sperm concentration, morphology & ejaculate volume. Progressive motility was slightly reduced in subjects

with obesity that was only significant in the clinical setting using WHO criteria. However, on further testing of DNA fragmentation, low mitochondrial membrane potential (MMP), and change in seminal fluid factors: neoprotein, interleukin-8, alpha-glucosidase, were significantly more common in subjects with obesity compared to normal weight. These cause the increased paternal infertility in patients with obesity through higher concentration of reactive oxygen species (ROS). All these factors coupled together reduce fertilization, impair embryonic development & increase loss of viable pregnancy.

The number of studies looking at the effects of bariatric surgery on semen quality are few. di Frega et al. [47] have reported complete arrest of spermatogenesis proven by testicular biopsy on six patients who underwent RYGB despite normal hormonal assays & previous fertility, with all presenting within 1–2 years from the operation.

Sermondade et al. [48] reported three cases: 2 RYGB, 1 LSG, suffering from primary infertility with no abnormalities found in their partners. Despite successful weight loss, their semen analysis within the first year showed drastic worsening: reduction in concentration, progressive motility and percentage of spermatozoa. One normalized after 2 years of follow-up, rest had not yet reached 2 years at the time of publication.

In a case series of six patients, Lergo et al. [49] reported that semen concentration reduced in the first month, but continued to increase slowly till it normalized by 12 months.

El Baridis et al. [50] studied 46 patients who underwent LSG up to 1 year after surgery. Semen parameters improved only in patients with oligospermia or azoospermia. Azoospermia improved in 46% of patients, 10% of oligospermia worsened or remained unchanged, 58% improved but not significantly, with only 32% almost normalizing.

Samavat et al. [51] compared patients who underwent bariatric surgery (LRYGB 23 subjects) with matched controls with obesity (eight patients awaiting bariatric surgery) who were under lifestyle intervention. At 6 months, repeat analysis showed a decrease in the prevalence of hypogonadism from 74 to 9% on hormonal assay in the surgery group. Patients with normal seminal analysis increased from 26 to 39% in the surgery group. An increase in sperm count, improved progressive motility and decreased DNA fragmentation were observed after BS.

Recently, Carette et al. [52] in BARISPERM multicenter prospective study, added to the conflicting results. 46 patients (20 LRYGB, 26 LSG) with no history of infertility were followed up to 1 year. Total sperm counts continued to decrease from 6 months to 1 year, to the point that oligospermia increased from 17.4% preoperatively to 21.7%, with decrease in the total spermatozoa (15.4–12.4%). DNA fragmentation also worsened, with no change in motility or vitality of spermatozoa. No correlation was found between vitamin and pollutant levels in blood to these findings, as hypothesized by the previous studies to cause this temporary worsening.

Reis et al. [53] randomized 20 patients to Biliopancreatic diversion (BPD) versus lifestyle controls. At 20 months of follow up, they found no significant difference in all seminal fluid parameters between the surgical and control groups. This study represents the longest term follow up after bariatric surgery we currently have. The

parameters studied were sperm concentration, motility, morphology, volume, and vitality.

Given the evidence, bariatric surgery has shown some worrisome effects on sperm and seminal fluid in the early period after surgery. The reversibility of these early changes in the long-term remains unclear. There is a need for larger well designed trials with long term follow-up.

15.2.2.3 Erectile Dysfunction (ED) and Sexual Quality of Life

1. ED is generally defined as persistent inability to attain and maintain a penile erection adequate for satisfactory sexual performance. Sexual function in men encompasses several domains: desire or drive, erection, ejaculation and overall satisfaction. Erectile or sexual dysfunction diagnosis relies on the literature on patients' feedback from several questionnaires, which could pose a response bias. The questionnaires available to address sexual function are, The Brief Male Sexual Inventory (BSFI): developed by O'leary et al. [54], The International Index of Erectile Function (IIEF): developed by Rosen et al. [55], The Sexual Health Inventory for Men (SHIM): developed also by Rosen et al. [56], only for diagnosing the severity of erectile dysfunction (ED) and others like: Sexual Quality of Life-Male Questionnaire (SQOL-M): developed by Abraham et al. (J Sex Med 2008 referenced in [57]). In addition, sexual function questionnaire (SFQ), and impact of weight on quality of life-lite (IWQOL-Lite) have been used as well to study sexual function after bariatric surgery [58].

Depending on the tools applied to diagnose Ed and SD, the reported outcomes vary considerably.

Obesity increases the incidence of erectile dysfunction and decreases overall sexual health. In Massachusetts Male Aging Study [59], ED was present in 22% of individuals with BMI > 30 kg/m^2 as opposed to 13% in individuals with normal weight.

The theory behind the increased incidence is an endothelial dysfunction in the release of nitric oxide that would cause a decreased blood flow in the corpus cavernosum, decreasing the ability for erection. This dysfunction is not only caused by the hormonal disturbances mentioned above, but also by the increased pro-inflammatory markers present in obesity. In an OLETF rat model study by Choi et al. [60], obese rats who showed less mean intracavernous pressure, less endothelial Nitric Oxide Synthase (eNOS), & neuronal NOS (nNOS). Rats who underwent bariatric surgery showed more cavernosal smooth muscle/collagen ratio with increase in eNOS and nNOS that was statistically significant.

Weight loss in most studies appears to contribute to improving ED & sexual health in the short-term. In more than one RCT utilizing lifestyle and diet intervention, an increase in IIEF scores occurred [61, 62].

Dallal et al. [63], compared 97 patients who underwent RYGB with normal population controls. Using BSFI, they found that all domain scores of sexual function were significantly lower in the surgery group preoperatively, the higher the weight the less the scores were before the intervention. At a mean of 19 months follow up

(6–45 months) post RYGB, all domain scores improved with statistical significance. The amount of weight loss correlated with the improvement in scores. All scores approached those of reference controls except ejaculatory function and overall sexual satisfaction.

However, Ranasinghe et al. (BJUI 2010 [64]), studied 34 males who underwent Laparoscopic Gastric Banding (LGB), and found significant worsening in erectile index and orgasmic function utilizing IIEF, despite significant weight lost over a mean of 22 months follow up. The overall score did not differ over the period of follow up and more males were started on medical treatment for ED. Interestingly 83% of the subjects had ED on preoperative questionnaire, a percentage higher than any reported study.

Kun et al. [65] confirmed the improvement of ED in 39 Chinese patients undergoing LRYGB over 1 year follow up after surgery utilizing IIEF. They also studied the cavernosal intima/media thickness with the cavernosal peak systolic velocity through doppler imaging and found the thickness to decrease by 45%, while PSV increased by 61%.

Rosenblatt et al. [44] provided the longest term (6–16 years) evidence available for sexual function after bariatric surgery (RYGB). Using IIEF, total scores in the 3 groups (BS, Obese controls, lean controls) did not differ. Bariatric surgery group had better ED scores compared to obesity controls, but not to lean controls. ED was still present in 54.5%, 78.6%, 35.7% of bariatric, obesity & lean groups respectively, while overall satisfaction was 31.8%, 7.1% and 42.9% in the same order, respectively. They attributed some of these results to relapsing obesity in some individuals.

Janik et al. [57] compared 30 patients undergoing eit'her LSG or LRYGB, followed up over 18 months, with 32 controls with obesity. 56% of the surgery group had ED. That number decreased to 20%, with none having severe ED scores compared to 16% preoperatively. Overall sexual satisfaction with sexual desire improved, while intercourse satisfaction worsened and orgasmic function remained unchanged. SQOL-M scores significantly improved postoperatively, and improvement occurred in 8/11 items, with strong correlation to IIEF scores. On multivariate analysis of the entire two groups, age and history of no bariatric surgery were the only two major risks for ED in their analysis.

One should also be aware of the effects of certain mineral deficiencies on sexual function. Mahawar et al. [66] reviewed Zinc deficiency after RYGB, and noted that impotence with skin rash, hair loss can be due this mineral deficiency. Zinc blood levels of patients with impotence should be investigated and replaced orally daily for up to 4 weeks, as they respond to such treatment in 83% of the time. Refractory deficiencies require intravenous replacement.

In the largest, most recent multicenter Longitudinal Assessment of Bariatric Surgery (LABS-2) trial published till present on sexual function, Steffen et al. [58], followed up 470 men with 59.9% follow up at 5 years after bariatric surgery. Overall, improvements were significant in overall satisfaction, sexual desire and physical limitations to sexual activity. Despite these improvements, and although better than the preoperative state, there was a deterioration in sexual desire and activity between

years 1 and 5. Lower depressive symptoms were associated with better improvements, however, use of antidepressant medication was associated with deterioration in one or more domains of sexual function.

15.2.3 Conclusion

Overall, there is a strong evidence that bariatric surgery improves the sexual hormonal homeostasis that seems to be sustained long term. However, it does seem to correlate with the degree of weight lost, and this improvement decreases with weight regain. Sperm and seminal fluid quality tends to worsen in the short term, without any solid mid or long-term evidence to suggest reversal or improvements of these changes. The overall quality of sexual function, especially erectile dysfunction, appears to improve. There is still a further need for larger and more long-term studies to know the sustainability of these improvements.

Key Points

1. There is limited literature on effect of bariatric surgery on male reproductive system.
2. *There is a high prevalence of MOSH in obesity attributed to decrease in SHBG, FT, TT, FSH and LH secondary to suppression of pituitary by increased circulating estrogen.*
3. *Most studies report improvement in MOSH after surgical and non-surgical weight loss proportional to the amount of weight loss. Weight regain corroborates with relapse of hypogonadism.*
4. *Some literature suggests worsening of semen and sperm quality in the form of oligospermia and azoospermia after bariatric surgery. Long-term literature evidence is not available to know for sure if this is a reversible complication.*
5. *ED is more prevalent in population with obsity. Increased inflammatory cytokines reducing the release of endothelial NO have been implicated.*
6. *Most studies report an improvement in sexual function and sexual disorders after BS. The conflicting reports may be attributed to multiple factors affecting sexual function (interpersonal relationships, attraction to the partner, stress, health, medications)*

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