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Effects of Roux-en-Y gastric bypass and sleeve gastrectomy on bone mineral density and marrow adipose tissue

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Abstract

Bariatric surgery is associated with bone loss but skeletal consequences may differ between Roux-en-Y gastric bypass (RYGB) and sleeve gastrectomy (SG), the two most commonly performed bariatric procedures. Furthermore, severe weight loss is associated with high marrow adipose tissue (MAT); however, MAT is also increased in visceral adiposity. The purpose of our study was to determine the effects of RYGB and SG on BMD and MAT. We hypothesized that both bariatric procedures would lead to a decrease in BMD and MAT. We studied 21 adults with morbid obesity (mean BMI 44.1 ± 5.1 kg/m^2) prior to and 12 months after RYGB (n=11) and SG (n=10). All subjects underwent DXA and QCT of the lumbar spine and hip to assess aBMD and vBMD. Visceral (VAT) and subcutaneous (SAT) adipose tissue was quantified at L1-2. MAT of the lumbar spine and femur was assessed by 1H-MR spectroscopy. Calcitropic hormones and bone turnover markers were determined. At 12 months after surgery, mean weight and abdominal fat loss was similar between the RYGB and SG groups. Mean serum calcium, 25(OH)-vitamin D, and PTH levels were unchanged after surgery and within the normal range in both groups. Bone turnover markers P1NP and CTX increased within both groups and P1NP increased to a greater extent in the RYGB group (p=0.03). There were significant declines from baseline in spine aBMD and vBMD within the RYGB and SG groups, although the changes were not significantly different between groups (p=0.3). Total hip and femoral neck aBMD by DXA decreased to a greater extent in the RYGB than the SG group (p<0.04) although the change in femoral vBMD by QCT was not significantly different between groups (p>0.2). MAT content of the lumbar spine and femoral diaphysis did not change from baseline in the RYGB group but increased after SG (p=0.03). Within the SG group, 12-month change in weight and VAT were positively associated with 12-month change in MAT (p<0.04), suggesting that subjects with less weight and VAT loss had higher MAT. In conclusion, RYGB and SG are associated with declines in lumbar spine BMD, however, the changes are not significantly different between the groups. RYGB may be associated with

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greater decline of aBMD at the total hip and femoral neck compared to SG. MAT content increased after SG and this was associated with lower weight and VAT loss.

Keywords

bone mineral density (BMD); quantitative computed tomography (QCT); dual-energy x-ray absorptiometry; marrow adipose tissue (MAT); proton MR spectroscopy; bariatric surgery

1. Introduction

Bariatric procedures are increasingly used in patients with morbid obesity to reduce weight and to treat comorbidities. Roux-en-Y gastric bypass (RYGB) is the most commonly performed bariatric procedure followed by sleeve gastrectomy (SG) (1). Although RYGB and SG are highly effective for reduction of weight and metabolic comorbidities, their effects on the skeleton appear harmful. Studies have shown detrimental effects of bariatric surgery on bone and mineral metabolism (2–4) and an increase in fracture risk (5, 6).

Skeletal consequences may differ between RYGB and SG. SG involves resection of the gastric fundus, which secretes ghrelin, known to be stimulatory to osteoblasts, whereas bypassing of the small bowel in RYGB leads to malabsorption and other hormonal disturbances that may cause direct and deleterious effects on bone (7, 8). Patients typically lose more weight at one year following RYGB compared to patients who undergo SG (9) and data from animal studies suggest that bone loss after RYGB is greater than after SG (10). Only a few studies have compared the effects of RYGB vs SG on bone in humans (11–16), and these have reported conflicting results, with some suggesting that RYGB leads to greater bone loss than SG, whereas others find similar rates of bone loss after both procedures. Of note, all studies used dual-energy x-ray absorptiometry (DXA) to determine bone mineral density (BMD). DXA assesses areal bone mineral density (aBMD), which is susceptible to artifactual changes following extreme weight loss (17), whereas quantitative computed tomography (QCT) measures volumetric bone mineral density (vBMD), which is less susceptible to changes in body size (18). No studies have assessed the effects of RYGB and SG on vBMD by QCT.

Furthermore, bone strength is determined not only by BMD but also its micro-environment, and it is hypothesized that marrow adipose tissue (MAT) negatively affects bone strength (19). Severe weight loss is associated with high MAT (20) which may contribute to impaired skeletal health after bariatric surgery. On the other hand, visceral adiposity is associated with high MAT (21) and loss of visceral adipose tissue following bariatric surgery might lead to a decrease in MAT. Furthermore, alterations in MAT may contribute to artifact in the assessment of BMD by both DXA and QCT modalities (22, 23). The non-invasive quantification of MAT using proton magnetic resonance spectroscopy (1H-MRS) has improved the feasibility of quantifying MAT content and allows longitudinal assessments *in vivo*. A recent pilot study of 11 women with morbid obesity undergoing RYGB showed a decrease in MAT in diabetic patients (n=6) while there was no change in non-diabetic patients (n=5) six months after surgery (24). However, no studies have compared the effects of RYGB vs SG on MAT. The purpose of our study was to determine the effects of RYGB

and SG on aBMD, vBMD and MAT. We hypothesized that both bariatric procedures would lead to a decrease in BMD and a decrease in MAT content 12 months after surgery.

2. Materials and Methods

Our study was IRB approved and Health Insurance Portability and Accountability Act compliant. Written informed consent was obtained from all subjects prior to performance of any study procedures.

2.1. Subjects

Subjects with morbid obesity were recruited from the MGH Weight Center. Inclusion criteria were age ≥ 18 years and plan to undergo RYGB or SG. Exclusion criteria were history of medical disorders known to affect bone metabolism, use of bone-active medication, pregnancy, weight >182 kg (due to limitations of the MRI scanner) and contraindications to MRI, such as the presence of a pacemaker or metallic implant. Subjects who were scheduled for bariatric surgery and met inclusion criteria were invited to participate in the study and were scheduled for the baseline visit. There were no drop-outs before or during the baseline visit.

Study visits were performed at baseline (prior to bariatric surgery) and 12 months after surgery. Each subject underwent a history and physical examination, fasting blood tests, DXA, QCT, and 1H-MRS at baseline and 12 months. Type 2 diabetic status was assessed by self-report and/or use of diabetic medications.

2.2. Calcitropic hormones and bone turnover markers

The following blood tests were obtained after an overnight fast: calcium, and 25-hydroxyvitamin D, parathyroid hormone (PTH) (intra- and interassay coefficient of variations (CV) ≤ 3%), procollagen type 1 N-terminal propeptide (PINP) (intra- and interassay CVs ≤ 6%) and serum type 1 cross-linked C-telopeptide (CTX) (intra- and interassay CVs ≤ 3%) as previously described (25).

2.3. Bone mineral density assessment

2.3.1. Dual-energy x-ray absorptiometry (DXA)—Areal bone mineral density (aBMD, g/cm^2) of the lumbar spine (L1-L4), total hip and femoral neck was assessed using DXA (QDR Discovery, Hologic, Inc, Bedford, MA). We have previously established that the same-day in vivo scanning precision at our center is 0.007, 0.008, and 0.012 g/cm^2 for PA spine, total hip, and femoral neck, respectively (26). If necessary, manual retraction of pannus overlying the proximal femur was performed during hip measurements.

2.3.2. Quantitative Computed Tomography (QCT)—Volumetric bone mineral density (vBMD) of the lumbar spine (L1-L2) and proximal femur was assessed using a 16-multidetector-row CT scanner (LightSpeed Pro, GE Healthcare, Waukesha, WI, USA). Subjects were placed supine in the CT scanner on a calibration phantom (Mindways Software, Inc., Austin, TX, USA), and helical scanning of L1-L2 and from the proximal articular surface of the femoral head to 1 cm below the lesser trochanter, was performed

using the following parameters: 120kV, 100mA (L1-2), 120 KV, 200 mA (proximal femur), slice thickness of 2.5 mm, FOV of 500 mm and table height of 144 mm (2-year CV ≤ 2%) (27).

Analysis of vBMD of L1-L2, total hip, and femoral neck was performed with QCTPro software (Mindways Software, Inc., Austin, TX) as previously described (25).

2.4. Marrow adipose tissue assessment

Subjects underwent proton MR spectroscopy (1H-MRS) of the 1st and 2nd lumbar vertebrae (L1-L2) and the left proximal femoral metaphysis and mid-diaphysis. All studies were performed on a 3.0-T MR imaging system (Siemens Trio; Siemens Medical Systems, Erlangen, Germany) after an overnight fast. Single-voxel 1H MR spectroscopy data were acquired by using a point-resolved spatially localized spectroscopy pulse sequence without water suppression (TR/TE 3000/30, eight acquisitions, 1024 data points, and receiver bandwidth of 1000 Hz). For each voxel placement, automated optimization of gradient shimming was performed (28)

Fitting of all 1H-MRS data was performed using LCModel (version 6.3-0K, Stephen Provencher, Oakville, Canada). Metabolite quantification was performed using eddy current correction and water scaling. A customized fitting algorithm for bone marrow analysis provided estimates for all lipid signals combined (0.9, 1.3, 1.6, 2.3, and 5.3 ppm). LCModel bone marrow lipid estimates were automatically scaled to unsuppressed water peak (4.7 ppm) and expressed as lipid to water ratio. Average MAT content of L1-L2 was assessed (6-month CV 12%) (29).

2.5. Abdominal fat assessment

Visceral adipose tissue (VAT) and abdominal subcutaneous adipose tissue (SAT) compartments were quantified at the level of L1-L2 using the CT performed for BMD assessment. Fat attenuation coefficients were set at −50 to −250 Hounsfield unit as described by Borkan et al. (30) and VAT and SAT cross sectional areas (CSA) (cm^2) were assessed based on offline analysis of tracings obtained utilizing commercial software (VITRAK; Merge/eFilm, Milwaukee, WI) (CV 2.5%).

All QCT and 1H-MRS acquisitions and analyses were performed blinded to the surgical procedure under the supervision of a musculoskeletal radiologist with 11 years of experience (M.A.B.).

2.6. Statistical Analysis

Statistical analyses were performed using SAS 9.3 (SAS Institute, Cary, NC). Baseline characteristics were assessed by independent t-tests or Fisher's exact tests. Twelve-month changes between the RYGB and SG groups were compared by ANOVA and 12-month changes within the groups were assessed using paired t-tests. We also performed exploratory analyses to determine whether gender or diabetic status modulated the outcomes. Nonparametric Spearman rank correlation coefficients are reported. $P < 0.05$ was used to denote significance. Data are presented as mean ± SD.

3. Results

3.1. Baseline Characteristics

Subject characteristics, body composition, BMD, and MAT in the RYGB and SG groups are shown in Table 1. The study group included 21 subjects (mean age 49 ± 9 years), 18 women, 3 men, with morbid obesity (mean BMI 44.1 ± 5.1 kg/m^2) who underwent RYGB (n=11) or SG (n=10). Two participants in the RYGB group did not complete the study due to participant loss to follow-up. Both groups were of comparable age and BMI, calcitropic hormones, bone markers, BMD and MAT.

3.2. Effects of bariatric surgery on weight and body composition

Mean weight loss was −30.1 ± 7.6% in the RYGB and −25.0 ± 10.1% in the SG group but the change was not significantly different between the groups. VAT and SAT decreased in both RYGB and SG groups to a similar degree (Table 2).

3.3. Effects of bariatric surgery on calcitropic hormones and bone turnover markers

Mean serum calcium, 25(OH)-vitamin D, and PTH did not change significantly after surgery and were within the normal range in both groups at 12-months. There was an increase in bone turnover markers P1NP and CTX from baseline in both groups, with a greater overall percent increase in CTX than P1NP. RYGB was associated with a greater increase in P1NP compared to SG (Table 2).

3.4. Effects of bariatric surgery on bone mineral density (BMD)

Lumbar spine, total hip and femoral neck aBMD by DXA declined within the RYGB and SG groups compared to baseline (Figure 1). The decline in total hip and femoral neck aBMD was greater following RYGB compared to SG, while the change in lumbar spine aBMD was not significantly different between the groups. Despite the rapid bone loss, average bone density T-scores remained in the normal range 1 year after surgery in both groups (Table 3).

Lumbar spine vBMD by QCT declined within both groups while total hip vBMD only declined within the RYGB group compared to baseline. There was numerically larger bone loss after RYGB than SG but no statistically significant difference in lumbar spine or hip vBMD between the groups. No significant change in femoral neck vBMD was identified within or between groups.

3.5. Effects of bariatric surgery on marrow adipose tissue (MAT)

MAT content of the lumbar spine did not change from baseline in the RYGB group but increased after SG resulting in a significant difference between the groups. There was also a significant increase in MAT content at the femoral diaphysis after SG, but not RYGB (Figure 2).

3.6. Analyses by gender and diabetic status

The patterns of change in aBMD, vBMD, and MAT after surgery were similar among subjects with and without type 2 diabetes within the RYGB and SG groups. In addition, exclusion of the male study subjects did not change the overall pattern or statistical significance of the BMD or MAT outcomes after RYGB or SG.

3.7. Predictors of bone mineral density and marrow adipose tissue

Within the SG group, DXA-measured bone loss at the spine, total hip, and femoral neck over 12 months was significantly associated with both weight loss and VAT loss ($r = 0.68$ to 0.87, $p<0.05$ for all) (Supplementary Table 1). Within the RYGB group, 12-month weight loss was only associated with DXA-measured bone loss at the spine ($r=0.74$, $p=0.037$), and not at femoral sites. There were no significant associations between changes in body composition and QCT-measured bone loss in either surgical group.

Within the SG group, 12-month change in weight was positively associated with 12-month change in L1-L2 MAT ($r=0.66$, $p=0.038$), suggesting that subjects with less weight loss had higher MAT (Supplementary Table 2). A similar correlation was seen with 12-month change in VAT and L1-L2 MAT within the SG group ($r=0.72$, $p=0.030$), while no such association was observed between SAT and MAT. Within the RYGB group, there were no associations between change in L1-L2 MAT and body composition measures, although 12-month change in metaphyseal MAT was positively associated with changes in weight ($r=0.68$, $p=0.42$) and VAT (0.79, $p=0.021$). Finally, 12-month changes in MAT were not associated with changes in BMD in either surgical group.

Within the SG group 12-month changes in bone turnover markers were inversely associated with 12-month changes in aBMD of the spine and total hip ($p= -0.64$, $p=0.048$ for both) and P1NP was inversely associated with vBMD of the spine ($r= -0.79$, $p= 0.006$), while no such association was seen in the RYGB group (Supplementary Table 3). There were no associations between bone turnover markers and MAT (Supplementary Table 4).

4. Discussion

Obesity is now epidemic, and as a consequence, the use of bariatric surgery to manage morbid obesity is increasing (1, 31). Although RYGB and SG are highly effective in causing sustained weight loss, our results add to a body of literature that suggests that bone loss may in fact be the major metabolic complication of such surgeries (2, 4–6, 11–15, 32). In particular, our study shows that RYGB and SG, the two most popular forms of bariatric surgery, are both associated with declines in areal and volumetric BMD at the lumbar spine, and that RYGB is also associated with a decline in total hip areal and volumetric BMD at 12 months. DXA showed greater declines in total hip and femoral neck BMD after RYGB as compared to SG, although this difference was not detected by QCT. Declines in BMD were accompanied by significant increases in bone turnover markers CTX and P1NP, the latter being significantly higher after RYGB than SG. Vertebral and femoral diaphyseal MAT content was increased after SG but not RYGB and this was associated with less weight loss and visceral fat loss.

It is important for informed clinical decision-making to understand whether RYGB and SG have differential effects on skeletal health. To date, studies comparing RYGB and SG and their effects on BMD by DXA have shown conflicting results. Two studies of patients undergoing RYGB and SG suggested accelerated bone loss at the lumbar spine and hip after RYGB compared to SG, although the differences were not significant (11, 15), while another study showed no change in lumbar spine BMD but a greater decline in hip BMD following RYGB compared to SG (12). These findings are consistent with our study, which found trends for greater femoral bone loss after RYGB than SG. In contrast, two recent studies found similar rates of vertebral and femoral bone loss in RYGB and SG patients (13, 14). However, artifacts associated with DXA-based BMD measurements in obesity and during weight loss (17, 18, 22, 33) may have confounded the interpretation of the true magnitude of bone loss and its determinants after bariatric surgery. We therefore used 3D imaging techniques to overcome limitations of DXA in the context of extreme soft tissue changes following bariatric surgery. We found declines in lumbar spine vBMD by QCT within the RYGB and SG groups 12 months after surgery, at rates that were numerically greater than the vertebral bone loss detected by DXA. Total hip vBMD by QCT was only decreased within the RYGB but not the SG group, and the overall magnitude of femoral bone loss by QCT was much less than as observed by DXA. These somewhat incongruous DXA and QCT results are similar to a study by Schafer et al (34) that documented a 6-month decline in lumbar spine vBMD by QCT after RYGB as compared with no significant decline in lumbar spine aBMD by DXA. Our current results are also similar to our previous study (25) that demonstrated greater declines in femoral BMD by DXA as compared to QCT 1 year after RYGB. In aggregate, these findings suggest that assessment of aBMD by DXA is associated with greater artifact from soft tissue changes following extreme weight loss. It should be noted, however, that DXA and QCT estimates of bone loss after RYGB were more concordant by 2 years after RYGB (32).

Mechanisms impacting bone after bariatric surgery are multifactorial and may depend on the surgical procedure used. Mechanical unloading from non-surgical weight loss and reductions in fat mass are associated with bone loss (35, 36). Although patients typically lose more weight at one year after RYGB compared to patients who undergo SG, we found no significant difference in loss of weight or abdominal adipose tissue between the groups. Changes in weight and body composition measures were more widely correlated with bone loss after SG than RYGB, which may suggest that mechanical unloading may play a more important role in the pathophysiology of bone loss after SG. Indeed, animal studies suggest that the mechanism of bone loss after RYGB is independent of weight loss (37, 38). RYGB, which bypasses the duodenum and proximal jejunum, is associated with impaired absorption of calcium and vitamin D, key determinants of bone health (34, 39). In addition, gastric acid production is reduced after RYGB and SG, which can further decrease calcium absorption (40). Patients undergoing bariatric surgery are now routinely supplemented with calcium and vitamin D and in our study calcium, vitamin D, and PTH levels remained within normal limits 12 months after RYGB and SG. Nevertheless, the maintenance of these metabolic bone labs within normal levels did not prevent the bone loss that we observed after both RYGB and SG. Therefore, other post-operative factors, such as alterations in gastrointestinal

and neuropeptide signaling, may contribute to the metabolic bone changes after RYGB and SG.

In addition, recent studies have suggested that MAT may contribute to impaired skeletal health in obesity and after bariatric surgery (21, 24, 28, 41). Interestingly, increased MAT has been found in chronic undernutrition (20, 42) but also in obesity (28), especially in visceral obesity (21). In these studies, MAT was inversely associated with BMD and a negative predictor of microarchitecture and mechanical properties of bone. We found increases in MAT content of the lumbar spine and femoral diaphysis after SG and this was directly associated with change in weight and visceral adipose tissue (VAT), suggesting that subjects who lost less weight and VAT had higher MAT. In contrast, we found no associations between MAT and SAT, suggesting that VAT has different effects on MAT compared to SAT. These results are opposite of our hypothesis that MAT would decline in setting of decreasing VAT, but could be consistent with the effects of relative undernutrition in SG playing a primarily role in increasing vertebral MAT. We found no association between change in MAT and BMD which supports the hypothesis that MAT is not acting as a space filler but can change independently of BMD, It is unclear, however, why MAT would change only after SG and not after RYGB. Furthermore, the impact of SG-associated increases in MAT on bone strength are unknown. Nevertheless, the lack of change in MAT after RYGB implies that there was no systematic artifact from MAT alterations on DXA or QCT-based BMD measurements within this group. Only one previous study assessed MAT content of the lumbar spine after bariatric surgery. In a study of 11 women undergoing RYGB, Schafer et al (24) showed decrease in MAT content in diabetic subjects while there was no change in non-diabetic subjects six months after surgery. We did not observe differences in 12-month change in MAT content between subjects with and without type 2 diabetes within the groups, which may be due to the small number of subjects with diabetes in each group. Larger studies are necessary to assess the effects of different bariatric surgery procedures on MAT content, taking into account diabetes status.

The main limitation of our study is the small sample size. However, even with a limited number of subjects in each group, we were able to detect a difference in BMD and MAT 12 months after bariatric surgery. These changes in BMD and MAT persisted even after repeating analyses after excluding the male study subjects. Another limitation is that this observational study did not randomize participants to surgical type, and therefore there may have been bias in which subjects were referred to RYGB versus SG. Nevertheless, there were no significant differences in baseline characteristics between the two groups. Lastly, although not statistically different between groups, the racial composition of the two groups was imbalanced and might have contributed to our observed changes. It is not known whether MAT or skeletal responses to bariatric surgery are modified by race or ethnicity. Strengths of our study include the detailed assessments of areal and volumetric BMD by DXA and QCT and the assessment of lumbar and femoral MAT content using 1H-MRS.

In conclusion, our study shows that both RYGB and SG are associated with declines in areal BMD at the lumbar spine, total hip, and femoral neck at 12 months. Volumetric BMD at the lumbar spine declined after both RYGB and SG, and RYGB is also associated with decline in total hip volumetric BMD at 12 months. DXA showed a greater decline in total hip and

femoral neck BMD after RYGB compared to SG, although this difference between procedures was not detected by QCT. MAT content was higher after SG but not RYGB and this was associated with less weight loss and visceral adipose tissue loss. Larger longitudinal studies are necessary to determine the differential pathophysiologic effects of RYGB and SG on bone health. Clarifying these effects and underlying mechanisms will help identify therapeutic targets to optimize bone health after bariatric surgery.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Highlights

- RYGB and SG are each associated with declines in lumbar spine and hip BMD 12 months after surgery.
- There is greater loss of total hip and femoral neck aBMD after RYGB than SG at 12 months.
- There are no significant differences in lumbar spine BMD between RYGB and SG at 12 months.
- Vertebral and femoral MAT is increased after SG and is associated with lower weight loss and visceral fat loss.

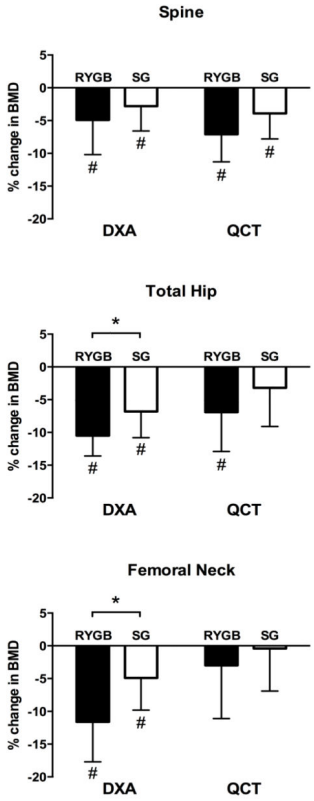

Figure 1.

Percent change in bone mineral density as assessed by DXA and QCT in the 12 months after either RYGB or SG. # indicates p-value <0.05 vs. baseline * indicates p-value <0.05 for RYGB vs. SG

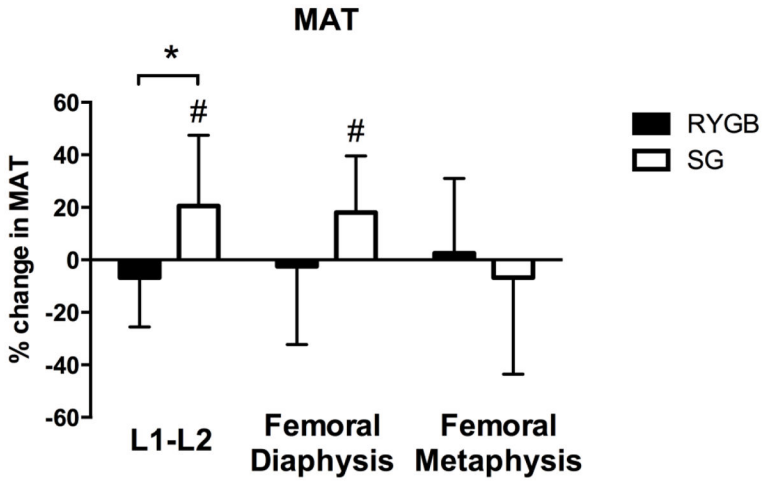

Figure 2.

Percent change in marrow adipose tissue as assessed by 1H-MRS in the 12 months after either RYGB or SG. # indicates p-value <0.05 vs. baseline * indicates p-value <0.05 for RYGB vs. SG

Table 1

Baseline characteristics.

	RYGB (n=11)	SG (n=10)	*p*
Age (years)	48.6 ± 8.9	49.5 ± 13.6	0.9
Female (n)	9	9	1.0
Weight (kg)	118.6 ± 15.2	113.7 ± 17.6	0.5
BMI (kg/m^2)	44.1 ± 5.1	43.7 ± 5.9	0.9
Race/Ethnicity			0.1
White non-hispanic	9	5	
White hispanic	1	1	
African American non-hispanic	-	4	
African American hispanic	1	-	
Type 2 Diabetes (n)	2	6	0.1
Serum calcium (mg/dl)	9.5 ± 0.5	9.6 ± 0.5	0.9
Serum 25-hydoxyvitamin D (ng/ml)	25.5 ± 5.7	31.2 ± 12.7	0.2
Serum PTH (pg/ml)	51.1 ± 16.1	62.9 ± 33.9	0.3
P1NP (ng/ml)	50.6 ± 21.9	61.1 ± 26.3	0.3
CTX (ng/ml)	0.38 ± 0.1	0.49 ± 0.2	0.1
DXA			
Spine aBMD (g/cm^2)	1.13 ± 0.11	1.19 ± 0.16	0.3
Spine T-Score	0.7 ± 1.0	1.0 ± 1.3	0.6
Total Hip aBMD (g/cm^2)	1.11 ± 0.15	1.08 ± 0.09	0.5
Total Hip T-Score	1.1 ± 0.8	0.7 ± 0.9	0.3
Femoral Neck aBMD (g/cm^2)	0.94 ± 0.16	0.94 ± 0.07	1.0
Femoral Neck T-Score	0.5 ± 1.0	0.4 ± 0.7	0.6
QCT (mg/cm^3)			
Spine vBMD	169.0 ± 18.3	167.0 ± 45.5	0.9
Total Hip vBMD	375.9 ± 51.9	355.2 ± 31.7	0.3
Femoral Neck vBMD	375.9 ± 66.8	375.6 ± 49.0	1.0
Visceral Adipose Tissue (cm^2)	207.8 ± 104.4	203.8 ± 111.9	0.9
Subcutaneous Adipose Tissue (cm^2)	463.9 ± 123.4	502.3 ± 103.5	0.5
Marrow Adipose Tissue (lipids/water)			
L1-L2	0.65 ± 0.25	0.86 ± 0.34	0.1
Femoral Metaphysis	2.3 ± 0.8	3.7 ± 2.4	0.1
Femoral Diaphysis	2.9 ± 1.6	3.1 ± 1.7	0.7

RYGB: Roux-en-Y gastric bypass, SG: sleeve gastrectomy; PTH: parathyroid hormone, P1NP: procollagen type 1 N-terminal propeptide; CTX: collagen type 1 cross-linked C-telopeptide; DXA: dual-energy x-ray absorptiometry; QCT: quantitative computed tomography; aBMD: areal bone mineral density; vBMD: volumetric bone mineral density

Table 2

Effects of bariatric surgery on body composition, calcitropic hormones and bone turnover markers in the 12 months after either RYGB or SG

Variable	Treatment	Baseline	12 months	Delta 12 months % change	*p*-value within group	*p*-value between groups at 12 months
Weight (kg)	RYGB	118.6 ± 15.2	67.5 ± 35.8	−30.1 ± 7.6	**<0.001**	0.228
	SG	113.7 ± 17.6	85.7 ± 20.3	−25.0 ± 10.1	**<0.001**	
BMI (kg/m^2)	RYGB	44.1 ± 5.1	31.2 ± 4.8	−29.6 ± 7.6	**<0.001**	0.372
	SG	43.7 ± 5.9	32.6 ± 7.2	−25.5 ± 11.2	**<0.001**	
Visceral adipose tissue (cm^2)	RYGB	207.8 ± 104.4	121.7 ± 80.1	−44.8 ± 17.5	**<0.001**	0.895
	SG	203.8 ± 111.9	109.0 ± 70.4	−43.2 ± 29.5	**0.002**	
Subcutaneous adipose tissue (cm^2)	RYGB	463.9 ± 123.4	261.4 ± 117.0	−45.9 ± 19.0	**<0.001**	0.922
	SG	502.3 ± 103.5	344.1 ± 209.8	−47.1 ± 24.9	**0.002**	
Serum calcium (mg/dl)	RYGB	9.5 ± 0.5	9.4 ± 0.3	−1.8 ± 4.9	0.312	0.423
	SG	9.6 ± 0.5	9.6 ± 0.3	0.2 ± 5.5	0.911	
Serum 25-hydoxyvitamin D (ng/ml)	RYGB	25.5 ± 5.7	29.1 ± 11.2	12.3 ± 35.9	0.333	0.231
	SG	31.2 ± 12.7	41.7 ± 16.1	48.3 ± 82.1	0.096	
Serum PTH (pg/ml)	RYGB	51.1 ± 16.1	50.4 ± 14.4	−3.8 ± 20.8	0.595	0.909
	SG	62.9 ± 33.9	52.2 ± 19.6	−5.7 ± 42.9	0.687	
P1NP (ng/ml)	RYGB	50.6 ± 21.9	105.3 ± 33.2	117.0 ± 64.5	**<0.001**	**0.035**
	SG	61.1 ± 26.3	91.6 ± 36.9	56.9 ± 49.4	**0.005**	
CTX (ng/ml)	RYGB	0.38 ± 0.1	0.99 ± 0.31	158.9 ± 43.3	**<0.001**	0.211
	SG	0.49 ± 0.2	0.94 ± 0.4	106.8 ± 115.7	**0.017**	

RYGB: Roux-en-Y gastric bypass, SG: sleeve gastrectomy; PTH: parathyroid hormone, P1NP: procollagen type 1 N-terminal propeptide; CTX: collagen type 1 cross-linked C-telopeptide

Table 3

Effects of bariatric surgery on bone mineral density T-Score in the 12 months after either RYGB or SG

Variable	Treatment	Baseline	12 months	Absolute change 12 months	*p*-value within group	*p*-value between groups at 12 months
Spine T-Score	RYGB	0.7 ± 1.0	0.3 ± 1.2	−0.5 ± 0.5	**0.019**	0.301
	SG	1.0 ± 1.3	0.7 ± 1.5	−0.3 ± 0.4	**0.044**	
Total Hip T-Score	RYGB	1.1 ± 0.8	0.1 ± 0.7	−0.9 ± 0.3	**<0.001**	**0.021**
	SG	0.7 ± 0.9	0.2 ± 0.9	−0.5 ± 0.3	**<0.001**	
Femoral Neck Hip T-Score	RYGB	0.5 ± 1.0	−0.6 ± 0.6	−0.9 ± 0.5	**<0.001**	**0.013**
	SG	0.4 ± 0.7	0.0 ± 0.7	−0.4 ± 0.4	**0.017**	

RYGB: Roux-en-Y gastric bypass, SG: sleeve gastrectomy