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# The Skeletal Consequences of Bariatric Surgery

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## Abstract

**Purpose of Review** This review outlines the recent findings regarding the impact of bariatric surgery on bone. It explores potential mechanisms for skeletal changes following bariatric surgery and strategies for management.

**Recent Findings** Bone loss following bariatric surgery is multifactorial. Probable mechanisms include skeletal unloading, abnormalities in calciotropic hormones, and changes in gut hormones. Skeletal changes that occur after bariatric surgery are specific to procedure type and persist for several years post-operatively. Studies suggest that while bone loss begins early, fracture risk may be increased later in the post-operative course, particularly after Roux-en-Y gastric bypass (RYGB).

**Summary** Further research is needed to assess the extent to which skeletal changes following bariatric surgery result in fragility. Current management should be geared toward prevention of bone loss, correction of nutritional deficiencies, and incorporation of weight bearing exercise. Pharmacologic treatment should be considered for high-risk patients.

**Keywords** Bariatric surgery · DXA · Bone microarchitecture · Biochemical markers of bone turnover · Fracture risk assessment

## Introduction

Bariatric surgery is a common and effective treatment for severe obesity [1–3], with many beneficial outcomes, including significant sustained weight loss [4], reversal of many comorbidities such as cardiovascular disease, obstructive sleep apnea, and diabetes [5–8], and decreased mortality [9, 10]. However, bariatric surgery may result in detrimental effects on bone and mineral metabolism, including vitamin D deficiency, hyperparathyroidism, and bone loss. Further, not only does the rate of weight loss and resolution of comorbidities vary by procedure, the skeletal effects are also procedure specific.

Bone loss following bariatric surgery is multifactorial. Proposed mechanisms include skeletal unloading, abnormalities in calciotropic hormones, and changes in gut hormones. An in-depth discussion of the association of gut hormones and adipokines with bone has been recently addressed elsewhere [11] and is beyond the scope of this review. Although research focusing on bone health following bariatric surgery has increased in the last decade, many important questions remain unanswered, namely, the extent to which the skeletal changes following bariatric surgery are pathological and increase fragility. This review will explore the current literature investigating the skeletal response to bariatric surgery, potential mechanisms, and strategies for management.

## Search Strategy

We performed a PubMed and Ovid MEDLINE search using the terms including "bariatric surgery," "bone," "obesity," and "microarchitecture." We primarily selected publications within the past 3 years, but did not exclude frequently referenced and highly regarded older publications. This review focused on prospective data and studies that reported bone mineral density (BMD) changes at sites used by the World Health Organization in their diagnostic criteria for osteoporosis, lumbar spine (LS), total hip (TH), femoral neck (FN), and 1/3 radius (1/3R) as well as those that investigated

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microarchitecture by high-resolution peripheral quantitative computed tomography (HR-pQCT).

## Abnormal Bone Metabolism in Obese Individuals

Obesity was initially thought to be protective of bone. However, it is now recognized that obese patients often have skeletal abnormalities. Vitamin D deficiency is well documented among this population [12–14] and may result in part from insufficient intake of foods and supplements containing vitamin D [15] and limited sunlight exposure [16]. While a prior report suggested decreased bioavailability of vitamin D secondary to sequestration of the fat-soluble vitamin in excess adipose tissue [17], other studies have not supported this hypothesis [18]. Our group found similar concentrations of vitamin D in both the subcutaneous and omental adipose compartments in both normal weight and obese women [19]. Moreover, the relationships between serum and adipose vitamin D did not differ according to obesity. These results suggest that the large amount of adipose tissue in obese individuals serves as reservoir for vitamin D. The increased amount of vitamin D required to saturate this large reservoir may predispose obese individuals to vitamin D deficiency. Translational studies have suggested that lower serum 25OHD may also be attributed to lower hepatic synthesis in the obese [20, 21]. Hyperparathyroidism is also prevalent among morbidly obese individuals [22]. While this may be a consequence of low vitamin D, it has been observed that there exists an independent relationship between parathyroid hormone (PTH) and obesity [23, 24].

There is a growing body of literature on the complex relationship between adipose tissue and bone [25–27]. Increased marrow fat, common in obese individuals, may contribute to low BMD and fragility [28]. Patients with increased visceral fat have lower bone formation, lower bone volume, and worse biomechanical properties of bone [29, 30]. Moreover, visceral fat secretes increased levels of tumor necrosis factor alpha (TNF-α) and interleukin 6 (IL-6) [31–33], proinflammatory cytokines, which increase bone resorption by upregulating a receptor activator of nuclear factor k ligands (RANKL) and stimulating osteoclastogenesis [29]. Obese women may differ in their expression of calciotropic hormones as well as adipokines. One study reported higher levels of PTH, bone-specific alkaline phosphatase (BSAP), leptin, fibroblast growth factor-23 (FGF-23), and lower 1,25-dihydroxyvitamin D in obese women [23]. Further, leptin levels predicted both PTH and FGF-23 [23]. In a recent study of obese adult patients with type 2 diabetes, FGF23 concentrations decreased following significant weight loss after undergoing SG, highlighting that increased FGF23 concentrations may be a by-product rather than a causative factor of obesity or other adiposity related parameters [34].

The relationship between body mass index (BMI) and fracture risk is complex and may differ across skeletal sites [35]. While obese individuals have traditionally been considered protected against osteoporotic fractures, several current studies report that obese patients may in fact be at increased fracture risk, particularly at peripheral sites [36–39], although the sites most likely to fracture may differ in obese men and women [40]. Increased intramuscular adipose tissue in obese adults may result in impaired mobility and muscle strength [41], leading to an increased risk of falls [36]. Due to mobility restraints, obese individuals may experience different patterns of weight bearing and falling, resulting in increased risks of extremity fractures. They may have an increased propensity toward backward and sideways falls. One study found that obese women had a 70% increase in fracture risk, predominantly at the lower limbs, as compared with women with a normal BMI. Further, fracture risk increased by 15% with every 5-kg/m$^2$ increase in BMI [42].

## Skeletal Consequences of Bariatric Procedures

Bariatric surgical procedures result in weight loss via several different mechanisms. Some reduce the size of the stomach, restricting the amount of food that a patient can comfortably consume; others delay in the mixing of food with bile salts and pancreatic juices resulting in malabsorption. Some procedures utilize a combination of both approaches. The procedures vary according to the extent of weight loss as well as resolution of comorbidities. The skeletal effects also vary by procedure. The majority of the available data on changes in bone following bariatric surgery focuses on Roux-en-Y gastric bypass (RYGB), as it was once the most common bariatric procedure performed. However, sleeve gastrectomy (SG) has surpassed RYGB as the most commonly performed bariatric procedure in the USA [43] and worldwide [44].

## Gastric Banding

Gastric banding (GB) is an exclusively restrictive procedure in which a silicone band placed around the proximal stomach creates a pouch that holds only a limited amount of food. This procedure results in modest weight loss, typically 41–45% loss of excess body weight, and has a high incidence of weight regain [45–48]. Post-operative vitamin D and PTH remains stable up to 1 year following GB [49, 50]. However, increased bone resorption measured by C-Telopeptide (CTX) has been observed as early as 6 months following GB and may persist for at least 2 years [50, 51]. One year following GB, areal BMD (aBMD) has been reported to decrease slightly at the hip, but not at the spine [51, 52]. The bone loss that occurs after GB is far less than with other procedures.

### Sleeve Gastrectomy

In SG, more than 80% of the stomach is transected and metabolism is altered as nutrients rapidly pass through the new gastric conduit [1]. This results in effective long-term weight loss of > 50% excess weight [53]. In conjunction with the rise in SG to treat morbid obesity, recent studies have investigated bone metabolism and skeletal outcomes. However, the data is limited by small sample size and follow-up duration. The findings remain inconsistent with regard to post-operative changes in 25-hydroxyvitamin D (25OHD) and PTH levels, likely reflecting disparate supplementation strategies among the different institutions and over time. A recent meta-analysis of 22 studies including 1905 adult obese patients who underwent SG, with a median follow-up of 12 months, revealed significant increases in serum calcium, serum 25OHD, and a significant decrease in PTH [54]. Additionally, significant decreases in the TH and FN, but not the LS BMD were observed following SG [54]. The few studies that have observed changes in bone turnover markers after SG have noted a significant increase in CTX and osteocalcin (OC) at 1 and 5 years after surgery [55, 56].

### Roux-en-Y Gastric Bypass

RYGB consists of a restrictive and malabsorptive component. During this procedure, a small gastric pouch from the proximal stomach is constructed and anastomosed to the proximal jejunum. This forms an alimentary tract in which food mixes with bile and pancreatic secretions in the distal jejunum. Similar to the biliopancreatic diversion with duodenal switch (BPD-DS), the intestinal surface area available for caloric absorption is reduced, leading to malabsorption of minerals and fat-soluble vitamins. Following RYGB, patients commonly lose 62–75% of excess body weight [57–60]. While vitamin D deficiency and secondary hyperparathyroidism are common after RYGB [61–63], this finding is not uniform [55, 64–66]. As a result of robust supplementation strategies, several recent studies have found that post-operative serum 25OHD and PTH remain stable [65, 66]. One study reported secondary hyperparathyroidism and elevated deoxypyridinoline (DPD) in 44 female RYGB female patients 4 years after surgery compared to 66 age and weight matched women without a history of bariatric surgery. A subset of the RYGB patients ($n = 13$) were then supplemented with modest calcium and vitamin D for 6 months. Despite the supplementation, PTH and DPD were not reduced [63].

There is an increase in bone formation and reabsorption markers following RYGB. There is a greater overall percent increase in bone resorption measured by CTX compared to bone formation, measured by Procollagen type 1 N-terminal propeptide (P1NP) [55, 66]. Elevations in CTX and P1NP persist for at least 2 years post-surgery [64–67], although data beyond that timepoint remain limited. A recent longitudinal prospective study reported that serum CTX remained elevated at 5 years following RYGB, yet P1NP peaked at 3.5 years and began to decline, but remained above baseline [68••].

Hip aBMD declines substantially in the first year following RYGB [55, 58, 65, 66, 69, 70•]. Reductions in aBMD by dual-energy X-ray absorptiometry (DXA) at the TH and FN range between 5 and 15% [26, 58–60, 65, 68, 71, 72]. Findings at the spine are less consistent [58, 59, 65, 66, 68, 71]. Only a few studies have investigated skeletal changes beyond 1 year. One study reported that while TH aBMD showed continued decreases, spine aBMD remained stable from 12 to 24 months following RYGB [66]. In contrast, another study reported that both TH and spine BMD by DXA and quantitative computed tomography (QCT) continued to decline between 12 and 24 months [65]. A more recent study reported declines in spine aBMD by DXA and confirmed by QCT 5 years following RYGB [68]. However, it was noted that the majority of the bone loss occurred during the first two post-operative years, [68] suggesting that this may be the optimal time for interventions to prevent bone loss. While some studies report that bone loss may be greater among post-menopausal women [68], others have not found this effect [58].

Recent studies have compared bone loss after RYGB with SG. Bone turnover markers increase to a lesser extent with SG than with RYGB [56]. Further, a more recent study noted a decline in TH, FN, and LS aBMD following SG, but to a lesser extent than RYGB [55].

### Biliopancreatic Diversion with Duodenal Switch

With BPD-DS, a gastric sleeve is anastomosed directly to the distal ileum where food mixes with digestive enzymes [73]. Thus, food bypasses the majority of the small intestine. This combined restrictive and malabsorptive procedure is not commonly performed and typically reserved for patients with a BMI > 50 kg/m$^2$ [3]. BPD-DS has been shown to result in a mean excess weight loss of 70–80% [74–76]. Very few studies have examined changes in bone after BPD-DS, and these have been very small. After BPD-DS, a very high percentage of patients develop vitamin D deficiency and secondary hyperparathyroidism despite supplementation, and bone turnover markers increase significantly [74, 76–78].

## Limitations of Bone Studies in the Bariatric Population

As noted above, many of the studies regarding the effects on bone in bariatric patients have several limitations. For example, a majority of the studies were small and had significant drop-out rates, especially those that followed patients beyond 1 year post-operatively. Further, heterogeneity relating to age, sex, race, menopausal status, surgical approaches, and DXA

techniques, exist among the studies. Some of the studies lack non-surgical control groups. In addition, many of the studies do not address compliance with supplementation of calcium and vitamin D which is commonly prescribed as part of their clinical care. Many of the studies also do not address the level of physical activity of the patients in the post-operative period.

Accuracy and reproducibility of DXA is limited in morbidly obese patients, particularly during periods of weight change. There is artifact introduced by obesity itself, changes in fat mass, or in the case of GB, the location of the band [79–81]. Many of the older DXA machines can only accommodate patients of approximately 136 kg (300 pounds) thereby restricting the number of patients included in some studies that have axial DXA measurements. Further, even though the newer machines can support up to 450 pounds, morbidly obese patients may surpass the table dimensions, and therefore require either offset scanning or manual input for whole body measurement calculations [82]. Skeletal assessment with other modalities, such as QCT [64, 80] and HR-pQCT, may be less affected by changes in body fat [83] and avoid some of these limitations.

## Changes in Bone Quality and Microarchitecture

In addition to bone density, microarchitecture is an important factor that governs bone strength and fracture risk [84]. Evaluation of changes in skeletal microarchitecture may help to elucidate the mechanisms of bone loss following bariatric surgery. Of the few studies that have evaluated microarchitecture following gastric bypass, most only follow patients for 1–2 years following surgery. In our prospective study of 22 women who underwent either RYGB, SG, or GB, cortical area, density, thickness, and total density decreased at the tibia 1 year following surgery [58]. Declines in cortical bone were predicted by the increase in PTH (Fig. 1) [58]. Additionally, adults undergoing RYGB had more cortical bone loss than those with GB or SG and had declines in cortical load share estimated by finite element analysis [58]. Total volumetric BMD (vBMD) at the radius and tibia have been reported to decline 2 years following RYGB [65, 66]. The decline observed at the radius was attributed to a decline in trabecular vBMD, as well as a decrease in trabecular number and an increase in trabecular heterogeneity [65, 66]. However, at the tibia, in addition to declines in trabecular vBMD, cortical vBMD significantly decreased at least 2 years following RYGB [65, 66]. Only one study has reported vBMD and microarchitecture 5 years following RYGB [68]. vBMD and microarchitecture continued to decline between 2 and 5 years at a similar rate as that observed in the initial two post-operative years following RYGB. Cumulative declines exceeded 14% in total vBMD at both the radius and tibia. Further, cortical and trabecular microarchitecture also continued to deteriorate at the tibia, but to a lesser extent than at the radius [68].

## Mechanisms of Bone Loss After Bariatric Surgery

### Unloading

Mechanical loading of bone is an important factor influencing bone size, mass, and biomechanical properties. Changes in loading increase localized bone remodeling [85], likely mediated through osteocytes and the sclerostin pathway. One prospective observational study of 90 pre-menopausal women following RYGB and SG procedures demonstrated a direct relationship between an increase in sclerostin levels and bone loss [70]. Skeletal unloading has been shown to induce bone loss in other populations, including patients with spinal cord injury [86] and limited weight bearing following orthopedic surgery [87], as well as bed rest [88]. Hip bone loss has been documented in individuals who lose even small amounts of weight from caloric restriction [89, 90]. The hip ordinarily carries a load approximately 2–3 times body weight [91], and so sustains a multiplicative effect of unloading from weight loss after surgery. It is important to note that bone loss associated with unloading may be adaptive rather than pathological, and that the resulting skeleton may be well-suited to the new body habitus.

Most studies have noted a strong association between the amount of weight loss after bariatric surgery and extent of bone loss [51, 58, 59, 92]. It has been reported that following RYGB, the amount of weight loss was strongly correlated with bone loss at the TH ($r = 0.65$, $p = 0.02$) and FN ($r = 0.90$, $p < 0.0001$) (Fig. 2) [59]. Using HR-pQCT, we demonstrated significant changes at the tibia, but not the radius [58]. This suggests that there may be an early interaction between PTH and weight bearing. A longer longitudinal study reported greater declines in total vBMD at the radius than at the tibia 2 and 5 years following RYGB, despite weight remaining stable after the first post-operative year [68]. Thus, later declines may not be a direct consequence of unloading.

## Changes in Calcium, Vitamin D, and PTH

Vitamin D deficiency in the bariatric surgery population may be multifactorial and vary with surgical procedure performed [93]. The high prevalence of vitamin D deficiency at baseline, as well as marked differences in repletion regimens also complicate our understanding of the impact of bariatric surgery on calciotropic hormones. Calcium absorption is impaired following malabsorptive procedures such as RYGB and BPD-DS as the majority is actively absorbed in the duodenum and jejunum. In addition, due to delayed mixing of ingested nutrients with bile acids and pancreatic enzymes, vitamin D

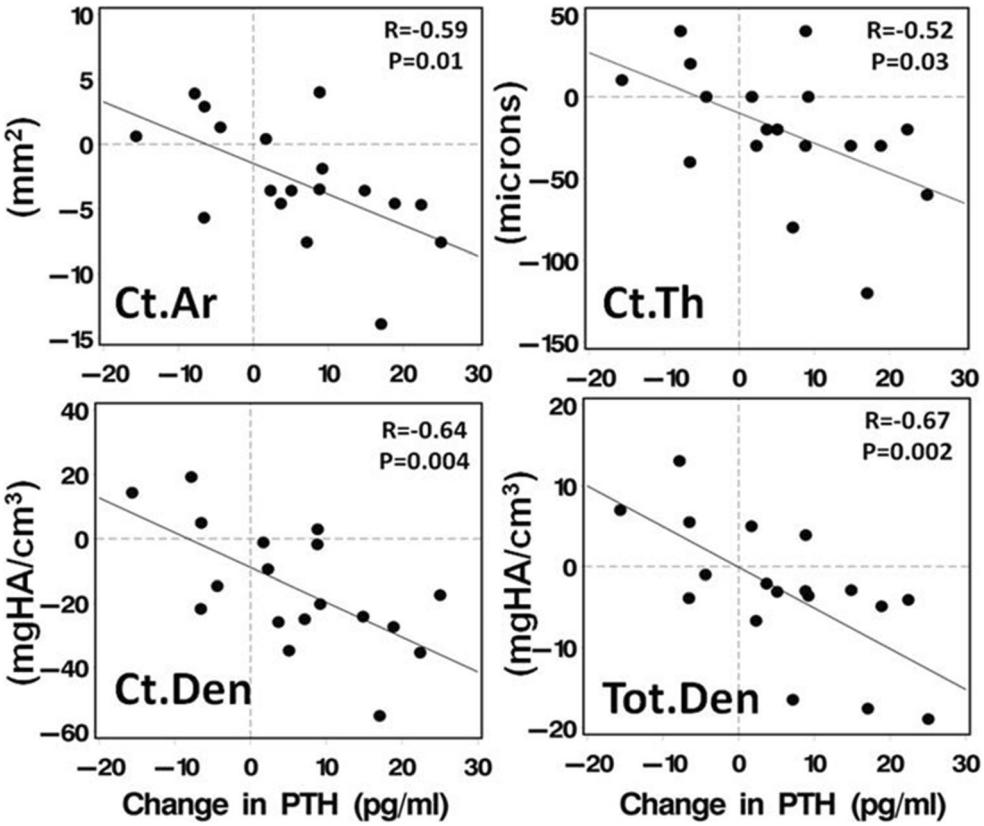


**Fig. 1** Association between change in parathyroid hormone (PTH) and cortical area (Ct.Ar), cortical thickness (Ct.Th), cortical density (Ct.Den), and total density (Tot.Den) by high-resolution peripheral quantitative computed tomography (HR-pQCT) at the tibia [58]. (Used with permission from Oxford University Press)

absorption is impaired [94, 95]. Following restrictive procedures such as SG and RYGB, the reduced gastric acid production may also affect calcium absorption [96]. Several observational [58, 59, 93, 97] and randomized trials [98] have shown that despite supplementation, calcium, and 25OHD levels are frequently below or in the lower end of the normal range. This suggests decreased absorption of vitamin D, or increased distribution to the adipose tissue. It should also be noted that many RYGB patients remain overweight 4–5 years following surgery with a mean BMI of greater than 31 kg/m$^2$. This persistent obesity may also contribute to prevalent vitamin D deficiency after bariatric surgery. Studies have reported decreased fractional calcium absorption (FCA) 6 months following RYGB [94, 97], despite maintenance of adequate vitamin D status and calcium intake [97]. Changes in 25OHD may affect bone density. Subjects with stable or increased 25OHD had less bone loss at the FN compared to those whose 25OHD declined [58]. Further, subjects randomized to high dose vitamin D had less hip bone loss than those who received only 800 IU daily [98]. Thus, post-operative maintenance of 25OHD may be important to hip bone preservation.

Many [58, 59, 69], but not all studies [55, 60, 65–67], have documented increased PTH following bariatric surgery. Changes in PTH may relate to changes in cancellous and cortical bone after, surgery [58, 59, 99]. In studies that reported post-operative increases in PTH levels, LS BMD was stable [58, 59]. However, some studies in which the PTH was stable or decreased, found that spine BMD declined [60], suggesting that increased PTH may be protective of the predominantly cancellous bone at the LS. Additionally, increased PTH may be associated with greater bone loss at the FN [100] as well as cortical bone loss at the tibia (Fig. 1) [58].

## Fracture Risk After Bariatric Surgery

Whether and to what extent bariatric surgery increases fracture risk remains the most important question regarding the long-term skeletal effects of these procedures. There are variable results from recent studies, which may reflect heterogeneity with regard to the type of bariatric procedures examined, the duration of follow-up and the demographics of the patient population, and its corresponding control group.

One study, a retrospective cohort study from the UK, with a mean follow-up of 2.2 years, found no significant increased risk of fracture in 2079 bariatric surgery patients compared to 10,442 matched controls [101]. However, a trend toward increased fracture risk was indicated in all patients 3–5 years post-surgery [101]. Additionally, the majority of subjects in this study underwent a GB procedure, and thus, the reported results may not be representative of the risk for patients who undergo different bariatric procedures, in particular ones associated with greater bone loss, such as RYGB [101]. Other

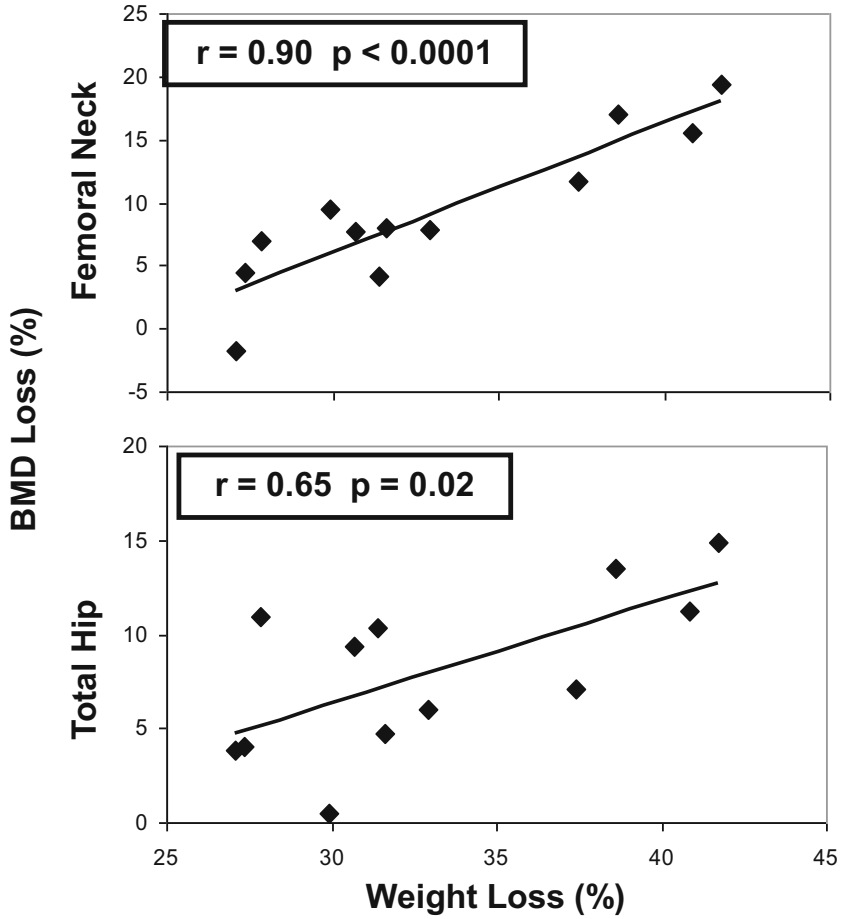


**Fig. 2** Relationship between decline in bone mineral density (BMD) at the hip, femoral neck (FN) and total hip (TH), and extent of weight loss at 1 year after Roux-en-Y gastric bypass (RYGB) [59]. (Used with permission from Oxford University Press)

reports also suggest that fracture risk begins to increase 2–5 years following bariatric surgery [102–104]. A historical cohort study consisting of 258 Olmsted County, Minnesota residents who underwent bariatric surgery, with 94% having had a bypass procedure, noted that the risk for any fracture was increased 2.3-fold [104]. This study consisted of a mean follow-up of 7.7 years and maximum follow-up of 25 years, which permitted a long-term fracture risk assessment. In addition to an increased risk of fracture within 5 years following surgery, the reported risk is even greater 5–10 and 10+ years after surgery. The risk of fracture was increased both at typical osteoporotic sites (hip, spine, wrist, and humerus) and all other sites [104].

Despite the emerging evidence of fracture risk following bariatric surgery, only a few studies have had the statistical power to report site specific fracture risks. A retrospective nested case-control study reported an increased risk of fracture at the upper limb, clinical spine, pelvis, hip, and femur [103]. These fracture sites resemble those in osteoporotic patients. Further, a change in the pattern of fractures following bariatric surgery was noted, with early fractures resembling a pattern associated with obesity and latter fractures resembling a pattern typical of osteoporosis [103]. In contrast, another study reported an increased risk of fracture at the scapula, clavicle, feet, and toes following bariatric surgery [105•].

It has also been reported that the risk of fracture following bariatric surgery varies with regard to procedure type. A recent retrospective cohort study of 15,032 morbidly obese adults reported that in comparison to GB, RYGB was associated with a 43% increased risk of non-vertebral fracture, with risk increasing greater than 2 years after surgery [102]. Restrictive and malabsorptive procedures such as RYGB and BPD-DS have been reported to have an increased risk greater than 1.4-fold [102–106]. Even though SG is now the most common bariatric procedure performed, it remains inconclusive as to whether this procedure raises fracture risk.

## Clinical Management

There are few guidelines to direct management of bariatric patients pre- or post-operatively [1, 107–109]. In 2013, the American Association of Clinical Endocrinologists (AACE), The Obesity Society (TOS), and the American Society for Metabolic and Bariatric Surgery (ASMBS) issued guidelines for management of bone health in bariatric surgery patients [1]. The majority of recommendations are based upon expert

opinion. The guidelines advise that prior to surgery, 25OHD should be measured on all patients. Post-operatively, all patients should receive 1200–1500 mg of calcium citrate daily from combined diet and supplements, and 3000 IU of vitamin D daily (titrated to 25OHD levels > 30 ng/ml). Further, aBMD should be measured by DXA at 2 years and 24-h urinary calcium checked at 6 months and then annually. Guidelines specific to RYGB and BPD-DS include the additional measurement of PTH and aBMD at the spine and hip prior to surgery. In addition, after surgery, 25OHD and PTH should be measured every 6–12 months. The 2016 ASBMS update suggests that peri- and post-menopausal women be screened for increased bone reabsorption [109]. The management strategy proposed in Fig. 3 is based upon published guidelines [1, 108, 109] and personal clinical opinion.

A few interventional studies have tested strategies to prevent bone loss after bariatric surgery. A two-arm prospective study of morbidly obese patients consisted of 220 pre-menopausal women and similarly aged men who underwent RYGB or SG [70]. The intervention group received 28,000 IU cholecalciferol/wk for 8 weeks pre-operatively, 16,000 IU/wk and 1000 mg calcium citrate/day post-operatively, as well as daily BMI-adjusted protein supplementation and aerobic exercise [70]. The non-intervention group received no supplementation or exercise. The intervention group had smaller increases of sclerostin and CTX levels, and normal intact PTH levels as well as a mitigated decline in aBMD at the spine, hip, and total body [70]. However, given the compound intervention, the relative importance of which supplementation and exercise individually contributed to these findings remains unclear. A 6-month randomized control trial investigated the role of weight bearing exercise training in severely obese women who underwent a RYGB procedure [110]. Those who did not receive exercise training had greater bone loss at the FN, TH, and radius, as well as cortical vBMD at the distal radius. The reduction in bone loss may be attributed to the relationship between exercise and suppression of bone turnover and sclerostin [110].

The optimal strategies for intervention vary according to patient risk factors for osteoporosis. For those who already have a diagnosis of osteoporosis based upon a T-score below −2.5 or the presence of a low trauma fracture, a metabolic work-up including serum PTH, calcium, phosphorus, 25OHD, and 24-h urine calcium is advised [1]. While sufficient calcium and vitamin D intake are necessary, compliance with supplementation commonly decreases over time. Non-compliance subsequently leads to an increased risk of inadequate calcium, vitamin D deficiency, and secondary hyperparathyroidism. Pharmacologic therapy, including bisphosphonates, should only be administered to bariatric surgery patients with osteoporosis after adequate restoration of calcium and vitamin D levels. Side effects of oral and intravenous bisphosphonates include the risk of reflux and anastomotic ulceration, and hypocalcemia and tetany in patients with low calcium or vitamin D, respectively. However, due to the risks associated with oral bisphosphonates and its insufficient absorption, parenteral therapies are favored. Although not addressed in the guidelines, other parenteral therapies may have particular risks in this population. Bariatric patients treated with denosumanb are at high risk for hypocalcemia. Teriparatide should only be used in patients who do not have secondary hyperparathyroidism.

## Conclusion

The skeletal status of bariatric patients is influenced by pre-operative and post-operative abnormalities in bone and mineral metabolism. Post-operative bone loss, most consistently

**Pre-Operative Management Recommendations**

| Nutrition | Biochemical Evaluation | BMD Evaluation |
|---|---|---|
| Calcium intake of 1000-1200 mg/day from combined diet and supplements<br><br>Titrate 25OHD level to 30 ng/ml | Measure 25OHD and PTH levels and replete calcium and vitamin D | Baseline DXA performed on candidates with risk factors for osteoporosis |

**Post-Operative Management Recommendations**

| Nutrition | Biochemical Evaluation | BMD Evaluation | Exercise |
|---|---|---|---|
| 1200-1500 mg/day following GB,SG,RYGB and 1800-2400 mg/day following BPD-DS of calcium citrate<br><br>Titrate 25OHD level to 30 ng/ml | Measure calcium, albumin, PTH, 25OHD levels initially and annually thereafter | Follow-up DXA measurements at 2 years | Moderate aerobic exercise for at least 150 minutes per week<br><br>Incorporation of strength training 2-3 times per week |

**Fig. 3** Recommendations for pre- and post-operative management of bariatric patients. Gastric banding (GB); sleeve gastrectomy (SG); Roux-en-Y gastric bypass (RYGB); biliopancreatic diversion with duodenal switch (BPD-DS)

found at the hip, is multifactorial. The potential mechanisms for bone loss include skeletal unloading, deficiencies in calcium and vitamin D, secondary hyperparathyroidism, and other hormonal changes. The risk of fracture may be increased following bariatric surgery, RYGB in particular. Risk of fracture appears greatest later in the post-operative period. At present, we recommend that treatment be geared toward correcting nutritional deficiencies. Pharmacologic therapy should be reserved for patients at high risk for fracture. Additional research is needed to identify the optimal strategies to mitigate the negative skeletal effects of bariatric surgery.

## Compliance with Ethical Standards

**Human and Animal Rights** All reported studies/experiments with human or animal subjects performed by the authors have been previously published and complied with all applicable ethical standards (including the Helsinki declaration and its amendments, institutional/national research committee standards, and international/national/institutional guidelines).

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- • Of importance
- •• Of major importance


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