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Metabolic bone changes after bariatric surgery: 2020 update, American Society for Metabolic and Bariatric Surgery Clinical Issues Committee position statement

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The following position statement is issued by the American Society for Metabolic and Bariatric Surgery (ASMBS) for the purpose of enhancing quality of care in metabolic and bariatric surgery. The ASMBS published the first position statement addressing metabolic bone changes after bariatric surgery in 2015 [1]. In this updated statement, interval suggestions for management are presented, which are derived from available knowledge, peer-reviewed scientific literature, and expert opinion regarding monitoring and treatment of metabolic bone changes after metabolic and bariatric surgery procedures. The statement will continue to be revised in the future should additional evidence become available.

The issue

Obesity rates in adults have continued to increase over the last decade. According to the Centers for Disease Control and Prevention, the disease of obesity affects 39.8% of US adults, or about 93.3 million Americans [2]. The ASMBS estimates that more than 24 million Americans have severe obesity. Metabolic and bariatric surgery remains the most effective and durable treatment for severe obesity and obesity-related co-morbidities. Despite the large-scale and far-ranging health benefits of these procedures, there are anatomic and metabolic consequences that necessitate adherence to life-long micronutrient supplementation and monitoring, as well as potential unintended adverse effects, including those on bone health. Metabolic and bariatric surgery is associated with bone metabolism disorders, acceleration of bone remodeling, bone turnover, and bone loss, with decreased bone mineral density (BMD) [1]. The intent of this updated statement is to review the current evidence regarding bone loss after bariatric surgery and to provide interval recommendations.

Bone changes in obesity

It is understood that any protective benefits of obesity against osteoporosis secondary to increased BMD (attributed to increases in mechanical loading, larger bone size, and increased aromatization of androgens from adipose tissue and adipokines [3,4]) may be limited by the prevalence of high levels of preexisting vitamin D deficiencies—namely, 25-hydroxyvitamin D (25-OHD) and elevated parathyroid hormone (PTH) levels—with additional variations based on race, sex, and age [5]. Preexisting vitamin D

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deficiencies and elevated PTH levels in patients being evaluated for bariatric surgery have been found to be as high as 60%–84% and 42.2%–49%, respectively [6–8]. It has been identified that leptin, a hormone secreted by adipocytes which is increased in individuals with a higher fat mass, regulates bone mass directly and indirectly via PTH in animal models with leptin-deficient mice. Leptin increases cortical bone mass but may have an adverse effect on trabecular bone mass [9]. Case control data suggest that leptin plays a role in elevating PTH levels. In patients with obesity, serum leptin levels were the highest predictive variable for an elevated serum PTH level. The mechanism is unknown, but it is theorized that leptin may increase parathyroid mass directly through a mitogenic effect [5]. Lower serum levels of vitamin D in patients with obesity can also be due to a dilutional effect of distribution into fat in these patients. In addition, patients with obesity typically need a higher dose of vitamin D replacement to achieve the same serum level as normal-weight patients.

Preoperative assessment

Because of the high prevalence of vitamin D deficiency and secondary hyperparathyroidism (despite normal calcium) in the obese population, obtaining a baseline preoperative assessment of bone health continues to remain standard. The specific recommendations remain unchanged from the prior statement, and consist of laboratory testing of 25-OHD, intact PTH levels, and serum alkaline phosphatase, as well as consideration of 24-hour urinary calcium in relationship to dietary intake, before bariatric surgery, with the initiation of treatment for deficiencies and documentation of improvement before surgery when possible. These recommendations are consistent with the ASMBS Integrated Health Nutritional Guidelines for the Surgical Weight Loss Patient 2016 [10].

A baseline dual-energy X-ray absorptiometry (DXA) scan is recommended by the National Osteoporosis Foundation Clinician's Guide 2014 for all women aged 65 and older and men aged 70 and older. It is also recommended in postmenopausal women and men above age 50–69, based on the risk factor profile, and in men aged 50 and older who have had an adult age fracture, to diagnose and determine the degree of osteoporosis [11]. Preoperative DXA can also be considered in estrogen-deficient women and in premenopausal women and men who have risk factors or conditions associated with bone loss or low bone mass, to establish a baseline before bariatric surgery. There remain insufficient data to support universal screening [12].

Since the previous statement, high-resolution peripheral quantitative computed tomography (QCT) has emerged as a way to evaluate BMD that may be more accurate than DXA in patients with obesity. QCT is less subject to magnification errors and extraosseous tissue changes than DXA [13]. It is also capable of distinguishing microarchitectural changes of bone [14]. QCT and DXA were found to produce discordant results at the proximal femur site of 30 patients who underwent Roux-en-Y gastric bypass (RYGB) versus 20 nonsurgical controls [13]. Similarly, in a group of 24 patients at 1-year post-RYGB, DXA at the spine and hip showed a significant decline. However, QCT of these same patients revealed no change in the bone geometry or bone density at the radius and tibia. QCT in a group of 22 patients at 1-year post bariatric surgery (majority RYGB) found within-bone microarchitecture cortical bone loss, but trabecular bone increases occurred, and these findings were predicted by a rise in PTH levels. Results were most significant in weight-bearing areas (tibia versus radius) [14]. QCT studied in 21 postoperative patients showed that bone geometry, volumetric density, and bone strength at the tibia and radius were unchanged 1 year after surgery [15]. Currently, indications for QCT in preference to DXA are limited, and include extremely high obesity and history of RYGB in practice guidelines from the American College of Radiology [16]. The limitations of QCT over DXA include higher amounts of radiation exposure and a significantly higher cost. Given the existing access and reimbursement concerns compared with DXA, QCT has not yet become standard screening practice.

Bone loss after bariatric surgery

Bone loss occurs when there is a greater ratio of bone removal to replacement. Age, hypogonadism, menopause, steroid dependence, lifestyle choices (smoking, alcohol consumption), and changes in gastrointestinal anatomy that occur with some bariatric procedures, as well as nonbariatric procedures, such as partial gastrectomy for ulcer disease, can contribute to bone loss [17].

As previously reported, the deleterious effects of bariatric surgery on bone metabolism and bone health appear to be multifactorial and procedure-dependent; related to the degree of weight loss are the potential for micronutrient deficiency and gut hormonal changes. Gastric bypass results in duodenal exclusion of nutrients and reduction of gastric acid, and may have a greater risk of micronutrient deficiency than the laparoscopic adjustable gastric band (AGB) or other purely restrictive procedures. The creation of a long intestinal bypass with added macronutrient malabsorption, as in biliopancreatic diversion with duodenal switch (BPD-DS) and single anastomosis duodenal switch procedures, may result in additional risks to bone health. Numerous changes in gut hormones, such as peptide YY (PYY), glucagon-like peptide-1, and ghrelin are found after RYGB, sleeve gastrectomy (SG), and BPD-DS. Although these hormonal changes are thought to impart many of the positive metabolic benefits of bariatric surgery, they may also contribute to bone loss. Hence, it remains essential to monitor levels of calcium, vitamin D, and parathyroid hormone both before and in the long term after bariatric surgery [18–22].

Adjustable gastric band

Bone loss after AGB is similar to that of changes that can occur with weight loss alone or other purely restrictive procedures, such as the vertical banded gastroplasty (VBG), and is not necessarily related to any additional micronutrient malabsorption [23–25].

Gastric bypass

It continues to be recognized that RYGB can result in calcium deficiency and metabolic bone disease, with reduction of BMD. This has been attributed to decreased dietary calcium intake; decreased absorption due to bypassing the proximal bowel, where calcium is preferentially absorbed; decreased absorption secondary to reduced gastric acid; malabsorption of vitamin D; and decreased mechanical loading on bones [1,26,27]. As mentioned, patients with obesity have a high prevalence of vitamin D deficiency at baseline (60%–80%), and the prevalence may not change over time [22]. In addition, rates of secondary hyperparathyroidism are high in post-RYGB patients (23.7%–42%), although lower than in post–BPD-DS patients (72.5%). Furthermore, secondary hyperparathyroidism may develop in patients despite normal circulating levels of calcium and vitamin D, so other factors, such as calcium malabsorption, age, and menopausal status, may play a role [18,28,29]. Another contributing factor is decreased mechanical loading related to weight loss after RYGB, given that mechanical loading under normal circumstances is the principal mechanism in maintaining bone mass, strength, and size. A recent randomized controlled trial evaluated whether bone loss after RYGB could be prevented or decreased by resistance training. Although the study had small patient numbers and a short follow-up of 6 months, the authors showed that compared with RYGB alone (n = 25), patients undergoing resistance exercise training (n = 24) were able to mitigate the percent loss of BMD, measured using QCT as the estimated mean difference (EMD) of the femoral neck (EMD, −2.91%; P = .007), total hip (EMD, −2.26%; P = .009), and distal radius (EMD, −1.87%; P = .038), with attenuation of several bone turnover markers [30]. In addition, changes in gut hormones that are produced in fat tissue, such as adipokines, leptin, and adiponectin, are reduced after RYGB, while there are increases in other gut hormones, like PYY, glucagon-like peptide-1, and ghrelin, which has been shown in vitro to increase osteoclastic cell proliferation, although this finding has not been confirmed in patients using BMD and DXA [28,31]. Gastric bypass, in contrast to AGB, leads to elevation of markers of bone remodeling, such as C-terminal telopeptide of type 1 collagen (CTX), independently of weight loss or hyperparathyroidism, and this could possibly be linked to the increased levels of PYY seen after RYGB [32]. Finally, poor nutrition can be another reason for decreased BMD after RYGB, as patients may consume less protein per day than recommended [33].

RYGB patients develop higher bone turnover, more osteoporosis, and lower BMD in the lumbar spine and hip than patients who lose weight after exercise or comprehensive lifestyle interventions [34,35]. Measurements of bone markers can be utilized to assess bone turnover in RYGB patients, as increased bone turnover has been reported to occur as early as 3 months after surgery and may still be present for years [36]. Bone-specific alkaline phosphatase, osteocalcin, and procollagen type I N-terminal propeptide are markers of osteoblast activity and bone formation [13,37]. Additionally, CTX and N-telopeptide have been used as markers for bone resorption (related to rapid weight loss) after bariatric surgery [15]. Sclerostin is another regulator that reduces osteoblastic bone formation and has been shown to be increased after both RYGB and SG [38]. Numerous studies continue to document that bone turnover increases after RYGB, with increases in CTX, procollagen type I N-terminal propeptide, and osteocalcin [13,15,37,39]. The increases in bone resorption markers are steady and up to 200%, while the increases in bone formation markers have been less uniformly reported [14]. It is not clear whether the increase in bone turnover is an adverse effect of the surgery or a physiologic adjustment to the weight loss and skeletal loading [36].

Sleeve gastrectomy

Since the last statement, there has been a continued increase in the popularity of SG, with a decline in the numbers of RYGB performed annually. Although SG leads to slightly less weight loss and continued nutrient flow across the duodenum compared with RYGB, bone loss is still observed, as evidenced by elevated markers of bone turnover detected several years postoperatively [9,21]. Crawford et al. [40] compared levels of CTX and osteocalcin between 33 patients with type 2 diabetes (T2D) who underwent SG and 25 patients with T2D who underwent intensive medical therapy. At a 5-year follow-up, CTX levels were increased by an average of 61.1% and osteocalcin levels by an average of 71% from baseline in SG patients, compared with 29.8% and 43.8%, respectively, for patients in the intensive medical therapy arm [40].

The mechanisms thought to be associated with bone loss after SG are multifactorial and similar to those described following RYGB. The mechanical unloading of the peripheral skeleton described in RYGB also holds true for SG [9]. Available studies tend to point toward a steeper decline in BMD loss at the hip (femoral neck and total hip) than at the lumbar spine [31]. For SG, there is an overall reduction in nutrient intake, as well as decreased acid secretion and accelerated gastric emptying, leading to the decreased intake and absorption of calcium [41].

Recent studies have compared SG and RYGB in terms of BMD. Crawford et al. [40] reported on 7 patients who underwent SG and obtained DXA at the hip and lumbar spine at baseline, 1 year, and a mean of 6.7 years postoperatively. In that study, they described overall median bone losses of 17.2% at the total hip and 5.6% at the lumbar spine. Compared with patients who underwent RYGB, they found that the amount of BMD loss was not significantly different [40]. Similarly Bredella et al. [21] compared 11 patients undergoing RYGB and 10 patients undergoing SG at 1 year after surgery and found a greater BMD decrease at the total hip and femoral neck in RYGB compared with SG patients on a DXA scan but not on QCT, concluding that the observed changes were not significant between both groups.

Other authors have also reported similar BMD loss among RYGB and SG groups [20,41], including a recent meta-analysis by Tian et al. [42] that included 13 studies and found that although the RYGB cohort had lower mean difference (MD) levels of 25-OHD (MD = −1.85; 95% confidence interval [CI], −3.32 to −.39; P = .01) and calcium (MD = −.15; 95% CI, −.24 to −.07; P = .0006), as well as higher levels of PTH (MD = 3.58; 95% CI, .61–7.09; P = .02) and phosphorus (MD = .22; 95% CI, .10–.35; P = .0005), the body mass index (BMI) changes and BMD by DXA (femoral neck, lumbar spine, total hip, or total body) were comparable in both groups at 1 year. In contrast, Hsin et al. [43] conducted a 1-year observational study comparing RYGB, SG, and LAGB in patients and performed baseline and 1-year DXA scans. The mean BMD losses at the spine were similar in the SG group and the RYGB group, but the BMD loss at the hip was considerably higher in the RYGB group [43]. Some authors have found a significant decrease in BMD at the hip and femoral neck levels but not the lumbar spine [44], while others found a decrease in BMD at all levels following SG [45]. Interestingly, some authors have reported an increase in BMD at the lumbar spine level 2 years after SG [46].

Vitamin D deficiency and along with secondary hyperparathyroidism, as seen with RYGB, has been studied as contributors to the BMD loss. In the Bone Metabolism after Bariatric Surgery Study, Muschitz et al. [38] enrolled 220 patients who had undergone either RYGB or SG, and created an intervention and a nonintervention arm with follow-up for a period of 24 months. The intervention arm received vitamin D loading preoperatively and vitamin D, calcium, and protein supplementation postoperatively, along with obligatory physical exercise, and was compared with a nonintervention arm which received none of the above. The study found significant decreases in markers of bone resorption; declining levels of PTH; reduced declines of BMD at the lumbar spine (−1.2% versus −7.9%, respectively) and total hip (−3.9% versus −9.9%, respectively); and reduced total body BMD values (−2.0% versus −4.1%, respectively) in the intervention arm compared with the control arm. Weight loss was comparable in both groups up to 18 months, where the nonintervention group had a quicker decline in BMI [38]. However, bone loss despite adequate postoperative supplementation, as seen with RYGB, has also been described after SG by several authors [41,47,48].

Despite the growing body of evidence to support bone loss after both SG and RYGB, there are several limitations of the data. Most studies are small, averaging fewer than 30 patients, with short follow-ups. There is variable information regarding baseline and postoperative vitamin deficiencies and treatment. Most studies also use DXA to quantify BMD loss, even though QCT has been shown to be more accurate in patients with higher BMIs, as well as more accurate following weight loss [31]. Regardless, there is sufficient evidence to suggest that BMD loss after SG occurs to a similar degree as after RYGB, and therefore should continue to be evaluated. Further studies are needed to fully elucidate the mechanisms behind the BMD losses seen after SG.

BPD-DS

BPD-DS may be associated with greater risks of vitamin D deficiency and secondary hyperparathyroidism compared to RYGB, secondary to greater protein calorie malnutrition. Low serum albumin is a strong predictor of severe protein malnutrition after BPD-DS, and may also predict bone loss in these individuals [49]. In the only procedure-specific interval publication, Tardio et al. [50] retrospectively reviewed the prevalences of calcium and vitamin D deficiencies and secondary hyperparathyroidism over a 5-year interval in a cohort of over 1400 BPD-DS patients, and reported significant preoperative and postoperative deficiencies, including in hypocalcemia, which is reported less commonly after RYGB or SG. The prevalence of vitamin D deficiency decreased up to 6–12 months after surgery (from 35.8% at baseline down to 6%–9%), then rose progressively, plateauing at 15.5% after 36 months. The prevalence of hyperparathyroidism was 28.5% before surgery and increased after surgery, reaching 68.6% at 5 years. Preoperatively, the prevalence of hypocalcemia was 7.3%, and the prevalence increased after 12 months, up to 26.9% at 48 months [50]. Bone loss, however, has also been described after BPD-DS, like after RYGB and SG, despite normal levels of vitamin D and PTH.

Fracture risk after bariatric surgery

Despite the recognized changes that occur in bone metabolism after bariatric surgery, published studies have historically failed to show a clear increased fracture risk after bariatric surgery [1]. The available interval evidence remains heterogeneous and somewhat conflicted, consisting mainly of observational studies with a small proportion of men and postmenopausal women, with mixed outcomes and limited randomized data gathered primarily from diabetic intervention trials comparing intensive medical

treatment (IMT) with bariatric surgery. Despite this, there appears to be a reasonable interval body of data to support an increased fracture risk after bariatric surgery, with the risk in BPD-DS greater than in RYGB, and the risk in RYGB greater than in SG, warranting ongoing evaluation and concern.

A meta-analysis from 2016 that included 10 articles with 241 surgical patients and 261 nonsurgical control patients, with follow-ups from 9.8 months to 10 years, revealed no difference in bone density at the lumbar spine. It did find a difference in bone density at the femoral neck, which was lower in patients who had surgery [51]. The BMD at the lumbar spine, as well as the z-score, has been found to decrease in postoperative patients in other studies [33].

A population-based study from Minnesota (The Rochester Epidemiology Project) showed that the risk of fracture after RYGB was 2.3-fold higher than in the general population, and the cumulative risk was as high as 58% in 79 patients experiencing 132 fractures over a 15-year period, and over 50% of these fractures were spontaneous vertebral fractures [52]. In addition, in a study comparing RYGB with AGB, patients who had RYGB had a 43% higher risk of nonvertebral fractures as compared with patients who had undergone AGB, and the higher risk remained for at least 2 years post surgery [53]. In contrast, a similar population-based study from the United Kingdom evaluated more than 2000 patients with obesity who were followed for a shorter period (2.2 years), and found no significant increases in the frequencies of fracture in RYGB and AGB [28,31]. Finally, the Surgical Treatment and Medications Potentially Eradicate Diabetes Efficiently (STAMPEDE) trial, in which patients with T2D were randomized to RYGB, SG, and IMT, showed that the rate of peripheral fractures did not differ across groups despite higher bone density loss in the surgical arms at 2 and 5 years. Extremity fractures at 2 years were reported in 4 IMT patients, 4 RYGB patients, and 2 SG patients. At 5 years, extremity fractures were reported in 4 (9%) IMT patients, 4 (8%) RYGB patients, and 3 (6%) SG patients [48,54].

A study from the Quebec Integrated Chronic Surveillance System studied more than 12,000 patients after bariatric surgery, including AGB, SG, RYGB, and BPD-DS, and compared them with a control group matched for age, sex, and BMI, who were followed up for 4.4 years. This study showed that patients undergoing bariatric surgery were more susceptible to fracture (514; 4.1%) than were obese (1013; 2.7%) and nonobese (3008; 2.4%) controls. The postoperative adjusted fracture risk was higher in the bariatric group than in the obese (relative risk, 1.38; 95% CI, 1.23–1.55) and nonobese (relative risk, 1.44; 95% CI, 1.29–1.59) groups. After surgery, the pattern of fractures shifted from that typical of obesity to a pattern typical for osteoporosis, with the risk of a distal lower limb fracture decreased (relative risk, .66; 95% CI, .56–.78), whereas the risks increased for upper limb (relative risk, 1.64; 95% CI, 1.40–1.93); spine (relative risk, 1.78; 95% CI, 1.08–2.93); and pelvic, hip, or femur (relative risk, 2.52; 95% CI, 1.78–3.59) fractures, but reached significance only for BPD-DS [55]. Another recent study from the Swedish National Database in 38,971 patients undergoing RYGB, matched against well-balanced controls in a 1:1 fashion, reported a 26% increased risk for any fracture after RYGB in patients with T2D and a 32% increased risk in control patients at the 3-year follow-up. They also noted an increased risk of fall injury seen after surgery as a possible contributing factor to fracture risk [56].

A recent update in 2020 from the Swedish Obesity Study compared 5335 weight loss–surgery patients with 2037 matched-control patients. The weight loss surgeries included RYGB, LAGB, and VBG. The median follow-ups for both groups were greater than 17 years. This study showed patients in the RYGB group had significantly higher incidences of all fractures, osteoporotic fractures, first-time fractures, and repeated fracture events, compared with both (1) matched, nonoperative patients, and (2) other weight loss–surgery patients (LAGB and VBG). Of note, this study does not include data on SG patients and how they compare [57].

With added micro- and macronutrient malabsorption, the highest increased fracture risk after bariatric surgery has been reported after BPD-DS. Compared with obese controls, the fracture risk increased by 60% after BPD-DS (relative risk, 1.6; 95% CI, 1.25–2.03) [55]. In a 12-year study from the National Health Insurance Research Database of Taiwan, 2064 post–bariatric surgery patients were matched to 5027 obese controls. Overall, there was a 1.21-fold increased risk of fracture in the surgical group, but a 1.47 adjusted hazard ratio after malabsorptive procedures [58].

PPI and fracture risk

Proton pump inhibitors (PPIs) work by decreasing gastric acid production. This raises concerns for decreasing calcium salt absorption in an achlorhydric environment. Consensus statements based on evidence-based review have determined that short-term PPI use does not require routine BMD monitoring, and has not been associated with increasing risks of micronutrient absorption or decreased BMD [59,60]. The Food and Drug Administration initially placed a safety warning on over-the-counter PPIs in 2010, concerned for an increased fracture risk associated with these medications. This warning was later removed in 2011, when further data became available [61].

Long-term use of PPI therapy (1 year or longer) has been found to increase the hip fracture risk. In patients older than 50 years, a study from 2006 showed that the fracture risk increases with the duration of therapy as well as the dose [62]. A meta-analysis from 2011 included 10 studies with over 220,000 fracture cases. The results from this study showed increased risks of hip and vertebral fractures in patients

taking PPIs. However, further analysis failed to show a significant increase in the hip fracture risk in long-term PPI use, attributed to study heterogeneity [63]. Overall, there is consistency in the evidence for PPI use increasing the risk of fracture, especially in patients who are at an older age and on high-dose PPIs. This association has not reached a level of significance to warrant radiographic BMD monitoring in patients on long-term PPI without other risk factors for osteoporosis [64].

Conclusion

1. Obesity is independently associated with vitamin and mineral deficiencies involved in bone homeostasis, which may compound the postoperative absorption of bone homeostatic micronutrients, depending on the type of procedure, degree of weight loss, nutritional intake, compliance with supplements, age, sex, race, estrogen status, and presence of any additional risk factors, such as smoking, alcohol, or long-term PPI intake. Routine preoperative screening for the presence of vitamin D deficiency and hyperparathyroidism, with treatment initiation, is recommended for all patients.
2. Ongoing lifetime screening and repletion of bone homeostatic micronutrients is recommended for all postoperative patients, with specific recommendations listed below.
3. There appears to be a reasonable interval body of data to support increased fracture risks after bariatric surgery, with the risk in BPD-DS greater than in RYGB, and the risk in RYGB greater than in SG, warranting ongoing evaluation. Additional prospective and randomized data, as well as intervention trials, are essential to better delineate the etiology, identify the highest-risk patients, and determine optimal monitoring strategies, interventions, and treatments.
4. Calcium citrate is preferable to calcium carbonate, due to better absorption in the absence or reduction of gastric acid [10,12].

Postoperative recommendations

1. Supplementation after LAGB, SG, and RYGB should include calcium at 1200–1500 mg/d, which can be taken in 2–3 split doses, 4–5 hours apart, for optimal absorption. The minimum vitamin D3 intake is 3000 IU/d, titrate to >30 ng/mL.
2. Supplementation after BPD and BPD-DS should include calcium at 1800–2400 mg/d and a minimum vitamin D3 intake of 3000 IU/d, titrate to >30 ng/mL [10,12].
3. Repletion of vitamin D deficiency after any bariatric procedure should include vitamin D3 of at least 3000 IU/d and as high as 6000 IU/d or 50,000 IU vitamin D2/D3 1–3 times weekly. Vitamin D3 is recommended as a more potent treatment than D2, but both forms can be efficacious [10,12].
4. Bone loss monitoring should include a minimum of annual albumin (to screen for protein malnutrition), alkaline phosphatase, calcium, PTH, and 25-OHD levels. In patients with renal compromise, 1,25-dihydroxy Vitamin D should be monitored. The 24-hour urinary calcium in relationship to dietary intake can also be considered [10,12].
5. Bone loss monitoring can also include markers for altered bone turnover. Peri- and postmenopausal women with lowered estrogen levels or patients identified at high risk for osteoporosis can be considered for screening for increased bone resorption by using urinary and/or serum CTX levels [10,12].
6. DXA after bariatric surgery in patients who have had RYGB or BPD-DS may be indicated to monitor for osteoporosis at baseline and at about 2 years, as per the recently updated 2019 American Association of Clinical Endocrinologists/American College of Endocrinology, The Obesity Society, American Society for Metabolic & Bariatric Surgery, Obesity Medicine Association, and American Society of Anesthesiologists Clinical Practice Guidelines for the Perioperative Nutrition, Metabolic, and Nonsurgical Support of Patients Undergoing Bariatric Procedures [12].
7. Exercise after bariatric surgery may help mitigate some of the adverse bone changes and, unless contraindicated, is also recommended in the recently updated 2019 American Association of Clinical Endocrinologists/American College of Endocrinology, The Obesity Society, American Society for Metabolic & Bariatric Surgery, Obesity Medicine Association, and American Society of Anesthesiologists Clinical Practice Guidelines for the Perioperative Nutrition, Metabolic, and Nonsurgical Support of Patients Undergoing Bariatric Procedures, for all patients with a target of moderate aerobic physical activity that includes a minimum of 150 minutes per week and a goal of 300 minutes per week, including strength training 2 to 3 times per week [12].

Disclosures

Drs. Kim, Khorgami, El Chaar, Galvez Lima, and Vosburg have nothing to disclose. Dr. Nimeri is a speaker for Medtronic.

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