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# Metabolic bone changes after bariatric surgery

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## Preamble

The following position statement is issued by the American Society for Metabolic and Bariatric Surgery for enhancing quality of care in bariatric surgery. In this statement, suggestions for management are presented that are derived from available knowledge, peer-reviewed scientific literature and expert opinion regarding monitoring and treatment of metabolic bone changes after bariatric surgery procedures. The statement may be revised in the future should additional evidence become available.

## The issue

Despite the undisputed health benefits, bariatric surgery requires not only an extensive preoperative evaluation but also a commitment from the patient to participate in life-long follow-up. Long-term follow-up after bariatric surgery focuses on weight loss maintenance and adherence to aftercare recommendations regarding micronutrient supplementation.

Weight loss outcomes, risks, and benefits and the potential for long-term complications vary among the different procedure types. In general, bypass procedures that result in duodenal exclusion of nutrients and reduction of gastric acid will have greater potential risk of micronutrient deficiency than purely restrictive procedures. Creating a long bypass with a malabsorptive component may add additional risk of both micronutrient and macronutrient deficiency.

No controversy exists regarding the need for life-long vitamin and micronutrient supplementation and screening after bariatric surgery. Guidelines regarding vitamin and micronutrient screening before and after bariatric surgery have been previously published in 2008 and recently updated in the 2013 AACE/TOS/ASMBS Guidelines for the Perioperative Nutritional, Metabolic, and Nonsurgical Support of the Bariatric Surgery Patient [1,2]. The intent of this statement is to provide an updated review of the current evidence regarding bone loss after the bariatric surgery and to provide recommendations regarding screening, surveillance, and replacement therapy for bone mineral micronutrients and hormones.

Obesity alone has been associated with altered levels of certain bone mineral homeostatic micronutrients and hormones, namely 25-hydroxyvitamin D (25-OHD) and parathyroid hormone (PTH) that can vary additionally with race and age [3]. Intentional or unintentional weight loss, without associated bariatric surgery, has been identified as a risk factor for both bone loss as well as increased hip fracture risk in middle aged to older women as well as older men [4–6].

There is controversy regarding the potential effect of extreme weight loss alone with or without the addition of potential micronutrient deficiencies on bone mineral density (BMD) and bone mass, both of which serve as indirect measures of osteoporosis and fracture risk. The utility of screening measures for BMD involving dual-energy X-ray absorptiometry (DXA) must consider technical limitations of both obesity as well as inaccuracies that may result during periods of weight loss that may affect the utility of such tests [7–9]. For very obese patients (over the weight limit for DXA table) as well as those patients who develop

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secondary hyperparathyroidism, which appears to be catabolic at cortical sites, the distal one third of the forearm BMD (which is a cortical site) should be measured [10].

## The data

### *Bone changes in obesity*

Obesity had previously been thought to be protective against osteoporosis secondary to increased BMD. This higher BMD in obese patients can occur despite the presence of secondary hyperparathyroidism due to vitamin D deficiency [11,12]. Vitamin D deficiency is defined as a 25(OH)D below 20 ng/mL (50 nmol/liter), and vitamin D insufficiency as a 25(OH)D of 21–29 ng/mL (525–725 nmol/L) [13]. Increased BMD is also affected by both ethnicity and gender differences. These findings are attributed to increases in mechanical loading, larger bone size, and increased aromatase from adipose tissue resulting in aromatization of androgens to estrogens as well as other hormonal factors such as adipokines [14,15]. Increases in BMD secondary to obesity are seen to varying degrees at different bone sites. Increases in the weight bearing femur have been reported to be up to 25% in morbidly obese individuals over healthy controls [16]. The protective benefits of obesity, however, may be limited by preexisting vitamin D deficiencies and elevated PTH levels, which have been found to be as high as 60–84% and 49% respectively [17,18]. Because renal 1-alpha-hydroxylase activity is increased due to elevated PTH levels, there are typically low levels of 25-OHD and normal or elevated 1,25-$OH_2$ D [11]. The increased PTH levels correlate with increasing BMI. This may be due to resistance to PTH by bone due to increased skeletal mass or an increasingly sedentary lifestyle with decreased sunlight exposure [11,19]. These deficiencies are significantly more prevalent in black obese patients [18–20] with some suggestion of gender differences with higher incidence seen in obese males [21].

Peak bone mass is usually achieved by age 18–25 years of age. Bone remodeling involves a continual process of bone removal and bone replacement. Bone loss occurs when there is greater bone removal than replacement. Age, hypogonadism and menopause, other risk factors such as steroid dependence, lifestyle choices (smoking, EtO H 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 [22]. Potential changes in bone metabolism after gastric surgery is not a new concern with publications that date back to the 1980s [23]. Despite changes that can occur in bone metabolism after bariatric surgery, published studies have failed to show any clear increased fracture risk after bariatric surgery. In a retrospective cohort study by Lalmohamed et al. [24], 2079 patients from the Clinical Practice Research Datalink who underwent bariatric surgery (laparoscopic adjustable gastric band [LAGB], Roux-en-Y gastric bypass, sleeve gastrectomy [SG], and other) were matched up to 6 controls (n = 10,442) and followed for a mean of 2.2 years with no significant difference in rates of fracture [24].

### *Preoperative assessment*

Because there is a high prevalence of vitamin D deficiency and secondary hyperparathyroidism (despite normal calcium) in the obese population preoperative assessment should include some routine screening as well as some focused testing for patients who are at higher risk for bone loss after surgery (patients requiring long-term steroid use, prior history of fracture, etc.). Serum calcium levels are rarely decreased before or after weight loss secondary to bariatric surgery. A normal serum calcium level does not imply adequate calcium intake or absorption as calcium homeostasis involves a complex interplay between gut absorption, bone resorption, and renal reabsorption and therefore cannot be interpreted outside of other bone homeostatic micronutrients, such as vitamin D and PTH [17]. Sometimes the PTH is found to be persistently elevated despite a 25 (OH)D level of >30 nl/mL, normal kidney function and compliant intake of vitamin D and calcium supplementation. A 24-hour urinary calcium level can be obtained to assess for hypocalciuria, which would support additional calcium intake or modification of the type of calcium ingested and/or adjust the dosing schedule (2–3 split doses) [25].

Preoperative dual-energy x-ray absorptiometry (DXA) in estrogen-deficient women and in premenopausal women and men who have conditions associated with bone loss or low bone density may be associated with lower T and *z* scores [26].

### *Postoperative management*

LAGB. There is currently no data to support LAGB altering calcium or vitamin D homeostasis beyond the weight loss effect achieved by the operation. Four studies that looked at bone mineral changes after LAGB and 1 comparison study with laparoscopic gastric bypass were reviewed [27–30]. One randomized controlled trial [24] assessed body composition changes after randomization to either laparoscopic adjustable gastric band or a very low energy diet and orlistat and found a small reduction in mean total bone mineral content for both groups (2.8% in the surgical group versus 2% in the diet group) but found no differences between the groups in total body bone mineral content at baseline and at 2 years [27,28]. Decreased bone mineral content and BMD without evidence of secondary hyperparathyroidism at 12 months [29] was confirmed in another prospective study at 24 months [30] both showing

increases in biochemical bone turnover markers C-telopeptides suggestive of increased bone resorption. These findings are consistent with changes in bone loss that can occur with weight loss alone or other purely restrictive procedures such as the vertical banded gastroplasty [31–33] that are not necessarily related to additional micronutrient malabsorption.

Gastric bypass. Gastric bypass can result in calcium deficiency and metabolic bone disease. This has been attributed to decreased dietary calcium intake, decrease absorption due to bypassing the proximal bowel where calcium is preferentially absorbed, decreased absorption secondary to reduced stomach acid, and malabsorption of vitamin D [23,29,34–38].

Gastric bypass has been reported to result in an increase in bone turnover and a decrease in bone mass [23,35]. A comprehensive review of bone density studies was recently performed by Scibora et al. [39] and looked at both cross-sectional and retrospective studies as well as prospective studies analyzed by procedure type and bone site. The majority of cross sectional and retrospective studies determined that BMD up to 4 years at the femoral neck, lumbar spine, and radius was similar to or greater in postbariatric patients compared with BMI matched controls. One notable finding from these studies is that many patients in the postbariatric groups were still obese based on BMI [39].

Greater changes in weight have been associated with larger changes in BMD [40]. Four prospective studies of at least a 12-month duration dealing specifically with RYGB were reviewed, which showed an average BMI decrease of 32–34%, and a decrease in femoral neck BMD ranging from 9.2–10.9% [40–43]. Two of these studies reported development of osteoporosis of which 1 study had 1 patient developing osteoporosis after 1 year [43]. Confounding variables include routine calcium and vitamin D supplementation that varied both in dosing regimens and assessment of compliance.

Measurement of bone turnover markers can be utilized to assess RYGB patients. Bone-specific alkaline phosphatase and osteocalcin are markers of osteoblast activity and bone formation [23,36]. Additionally, C-telopeptides have been used as a marker for bone resorption (related to rapid weight loss) after bariatric surgery [29,44].

Biliopancreatic diversion (BPD) and biliopancreatic diversion with duodenal switch(BPD/DS). Two prospective studies of biliopancreatic diversion with at least 12-months follow-up were evaluated. Tsiftis et al. [45] reported decreases in lumbar spine BMD of 7% and 8% in 2 groups (n = 26 each) of BPD patients 12 months after surgery. Both groups received high calcium diets, 200 IU of vitamin D and 100 mg elemental calcium daily. One of the groups also received an additional 2 gm/d of calcium. PTH increased in both groups but this was not significant compared to preoperative values and neither group developed secondary hyperparathyroidism. Markers for both bone formation and resorption increased in both groups and bone density, which was increased before surgery, normalized in both groups at 1 year. The authors concluded that the bone turnover rate is attributed to weight loss and unloading of the bone, not malabsorption, after BPD [45]. Marceau et al. [46] followed BPD patients preoperatively and 4 and 10 years postsurgery to evaluate clinical, biochemical, and BMD measurements. Despite initial evidence of bone loss markers, overall bone mineral density was unchanged at the hip and was decreased by 4% at the lumbar spine at 10 years with no overall change in $z$ score [46]. Compston et al. [47] found an increased incidence of metabolic bone disease after BPD (50-cm common channel). This occurred with normal 25-OHD levels suggesting that protein malnutrition contributes to bone loss after malabsorptive operations [47]. Additionally, it was noted that general nutritional status and protein calorie malnutrition are important factors in calcium metabolism after malabsorptive procedures, particularly intestinal bypasses such as the jejunal-ileal bypasses and very malabsorptive BPDs, and low serum albumin is a strong predictor of severe protein malnutrition after such procedures and may also predict bone loss in these individuals [47]. Retrospective studies have shown improved absorption of micronutrients with BPD/DS, which preserves the duodenum and calls for a longer common channel over standard BPD (now less commonly performed) [48]. Comparative studies between gastric bypass and BPD/DS suggest a greater risk of vitamin D deficiency with BPD/DS [49].

SG. There is 1 prospective study looking at bone loss findings after the sleeve gastrectomy (n = 8) compared to gastric bypass (n = 7). Bone mass measurements were made on the lumbar spine, femur, and distal radius, and the bone remodeling markers N-telopeptide and bone alkaline phosphatase, as well as vitamin D levels before and 12 months after surgery. Both procedures resulted in bone loss of both femur and lumbar spine that were less in the sleeve but not significantly different from the gastric bypass. N-telopeptide increased in both groups and bone alkaline phosphatase increased in the sleeve group only [50]. In a study by Gehrer et al. [51] comparing micronutrient deficiencies after sleeve gastrectomy (n = 50) and gastric bypass (n = 86), 23% of patients overall had vitamin D deficiency preoperatively. At 1 year, postoperative deficiencies of vitamin D and PTH were found to be significantly higher in gastric bypass than SG despite the presence of pre-existing vitamin D deficiency in both groups. Calcium levels remained normal in both groups, reinforcing the role of PTH and vitamin D levels as more sensitive markers for disorders of calcium metabolism after bariatric surgery [51].

## Conclusion

1. Obesity appears to be independently associated with vitamin and mineral deficiencies involved in bone

homeostasis affected by race and potentially affected by gender. These pre-existing vitamin and mineral deficiencies may compound postoperative absorption of bone homeostatic micronutrients depending on the type of weight loss surgery and degree of weight loss. Patients preparing for bariatric surgery should be screened for the presence of vitamin D deficiency and hyperparathyroidism with treatment initiated.

2. Cross sectional, retrospective, and prospective studies do not conclusively support any increased incidence of osteoporosis or increased fracture risk after bariatric surgery. Accuracy of current methods of assessing BMD (DXA) in patients who have extreme obesity as well as after extreme weight loss should be evaluated with further research. The use of one third distal forearm to measure BMD can be considered in situations of extreme weight that exceeds the limits of conventional DXA tables as well as in cases of secondary hyperparathyroidism related to malabsorption of vitamin D and calcium.
3. The degree of bone turnover and BMD loss after bariatric surgery is related to the type of procedure performed, the amount and rate of weight loss, and the degree of malabsorption of other micronutrients and protein. Long-term follow-up monitoring and supplementation should be provided according the type of procedure and the individual patient's risk for bone loss.

Recommendations

1. Preoperative assessment:
   a). The high prevalence of vitamin D deficiency and secondary hyperparathyroidism in the obese population supports routine laboratory testing of 25-OHD and intact PTH levels before bariatric surgery, with initiation of treatment for deficiencies and documentation of improvement before surgery when possible.
   b). Preoperative DXA can be performed in estrogen-deficient women and in premenopausal women and men who have conditions associated with bone loss or low bone mass to establish a baseline before bariatric surgery. There is, however, no compelling data to support routine DXA for all obese adolescents, men or premenopausal women undergoing bariatric surgery. If low bone mass is diagnosed preoperatively, a thorough evaluation should be undertaken to identify secondary causes. This laboratory testing can include thyroid stimulating hormone and testosterone levels in men.
   c). A baseline DXA is recommended by the National Osteoporosis Foundation 2013 (http://www.nof.org/hcp/practice/practice-and-clinical-guidelines/clinicians-guide) for all women 65 years and older and for younger postmenopausal women, and men 70 years or older and men age 50–69 about whom you have concern based on their clinical risk factor profile patients such as those undergoing a malabsorptive procedure.
   d). The U.S. Preventative Services Task Force recommends a bone density test at least once for all women age 65 and older. This recommendation can be made in consideration with general medical optimization as indicated.
2. Procedure specific recommendations for monitoring bone loss are made below. The doses of recommended calcium supplementation and vitamin D supplementation are consistent with previously published 2013 AACE/TOS/ASMBS Guidelines for the Perioperative Nutritional, Metabolic, and Nonsurgical Support of the Bariatric Surgery Patient [1,2].

a). LAGB:
   i. Calcium supplementation after LAGB should include 1200–1500 mg/d, which can be taken in 2–3 split doses, 4–5 hours apart for optimal absorption [47]. In the early postoperative period, minimum vitamin D of at least 3000 IU/d, titrate to >30 ng/mL. Minimum vitamin D maintenance after LAGB should be consistent with established age-specific recommendations for patients at risk for vitamin D deficiency until nonobese. The Endocrine Society Clinical Practice Guideline currently recommends a minimum of 600 IU/d of vitamin D from age 19–70 years and 800 IU/d of vitamin D after 70+ years [1,13].
   ii. Bone loss monitoring should include annual albumin, calcium, PTH, and 25-OHD levels.
   iii. DXA should be used after LAGB according to the most recent established guidelines for the general patient population that a particular patient belongs to based on age, gender, and associated risk factors.

b). Gastric bypass and malabsorptive procedures (BPD and BPD/DS):
   i. Supplementation after gastric bypass should include calcium citrate 1,200–1,500 mg/d, which can be taken in 2–3 split doses, 4–5 hours apart for optimal absorption [25]. Minimum vitamin D intake of 3000 IU/d, titrate to >30 nl/mL. Calcium citrate is preferable to calcium carbonate due to better absorption in the absence or reduction of gastric acid. Supplementation after BPD and BPD/DS should include calcium of 1800–2400 mg/d and minimum vitamin D 3000 IU/d, titrate to >30 nl/mL [1,2].
   ii. Based on the most recent Endocrine Society clinical practice guidelines, vitamin D deficiencies should be treated with 50,000 IU of vitamin $D_2$ or vitamin $D_3$ once a week for 8 weeks or its equivalent of 6000 IU of vitamin $D_2$ or vitamin $D_3$, daily to achieve a blood level of 25 (OH)D above 30 ng/mL, followed by maintenance therapy of 1500–2000 IU/d. Severe deficiencies can be treated with higher doses up to 50,000 IU 3 times a day. Intramuscular injections of ergocalciferol 100,000 IU once a week can be used, but are rarely necessary [13].

iii. Bone loss monitoring should include a minimum of annual albumin, calcium, PTH, and 25-OHD levels. 1,25-$OH_2$ D should be monitored in patients with renal compromise.

iv. Bone loss monitoring can also include markers for altered bone turnover. Monitoring to include bone alkaline phosphatase, osteocalcin, serum C-telopeptide, serum propeptide of type I collagen, and urine N-telopeptides can be considered. The appropriate use of biochemical markers as a screening tool, however, has not been established and warrants additional investigation.

v. Routine DXA scan after RYGB is not supported by current data. A baseline DXA is recommended by the National Osteoporosis Foundation 2013 (http://www.nof.org/hcp/practice/practice-and-clinical-guidelines/clinicians-guide) for all women 65 years and older and for younger postmenopausal women, and men 70 years and older and men age 50 to 69 about whom you have concern based on their clinical risk factor profile patients such as those undergoing a malabsorptive procedure.

c). SG:

i. Given the current lack of procedure specific data, recommendations regarding supplementation and bone monitoring should at a minimum be consistent with that recommended for the LAGB although it is unlikely that recommendations for the gastric bypass would be harmful.

## Position statement and standard of care

This position statement is not intended to provide inflexible rules or requirements of practice and is not intended, nor should it be used, to state or establish a local, regional, or national legal standard of care. Ultimately, there are various appropriate treatment modalities for each patient, and surgeons must use their judgment in selecting from among the different feasible treatment options.

The American Society for Metabolic and Bariatric Surgery cautions against the use of this position statement in litigation in which the clinical decisions of a physician are called into question. The ultimate judgment regarding appropriateness of any specific procedure or course of action must be made by the physician in light of all the circumstances presented. Thus, an approach that differs from the position statement, standing alone, does not necessarily imply that the approach was below the standard of care. To the contrary, a conscientious physician may responsibly adopt a course of action different from that set forth in the position statement when, in the reasonable judgment of the physician, such course of action is indicated by the condition of the patient, limitations on available resources or advances in knowledge or technology. All that should be expected is that the physician will follow a reasonable course of action based on current knowledge, available resources, and the needs of the patient, to deliver effective and safe medical care. The sole purpose of this position statement is to assist practitioners in achieving this objective.

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