

SUSAN E. WILLIAMS, MD, MS, RD, CNSP, CCD

Director, Center for Nutrition and Metabolic Medicine, Greene Memorial Hospital Advanced Medical Group, Xenia, OH; Assistant Professor of Clinical Medicine, Department of Internal Medicine, Wright State University Boonshoft School of Medicine, Dayton, OH; Chair-elect, Certification Council, International Society of Clinical Densitometry

KAREN COOPER, DO

Bariatric and Metabolic Institute, Cleveland Clinic

BRADFORD RICHMOND, MD*

Director, Musculoskeletal Radiology Fellowship Program, Department of Diagnostic Radiology, Cleveland Clinic; Associate Professor of Radiology, Cleveland Clinic Lerner College of Medicine of Case Western Reserve University

PHILIP SCHAUER, MD*

Director, Advanced Laparoscopic and Bariatric Surgery, Bariatric and Metabolic Institute, Cleveland Clinic; Professor of Surgery, Cleveland Clinic Lerner College of Medicine of Case Western Reserve University

Perioperative management of bariatric surgery patients: Focus on metabolic bone disease

ABSTRACT

Chronic vitamin D deficiency, inadequate calcium intake, and secondary hyperparathyroidism are common in obese individuals, placing them at risk for low bone mass and metabolic bone disease. After bariatric surgery, they are at even higher risk, owing to malabsorption and decreased oral intake. Meticulous preoperative screening, judicious use of vitamin and mineral supplements, addressing modifiable risk factors, and monitoring the absorption of key nutrients postoperatively are essential in preventing metabolic bone disease in bariatric surgery patients.

KEY POINTS

Metabolic bone disease in obese patients is multifactorial: causes include sequestration of vitamin D in the adipocytes, inadequate nutrition due to chronic dieting, and lack of physical activity.

Before bariatric surgery, one must look for and treat preexisting nutritional deficiencies.

In the immediate postoperative period, aggressive strategies (ie, giving multivitamins and minerals intravenously and orally) can prevent nutritional deficiencies and secondary bone disease.

Postoperatively, many bariatric patients require chewable or liquid supplements to facilitate adequate absorption.

Clinical suspicion, timely interventions, and lifelong monitoring can prevent metabolic bone disease in bariatric surgery patients.

A 56-YEAR-OLD WOMAN who underwent Roux-en-y bariatric surgery because of morbid obesity 6 years ago presents to her primary care physician with vague complaints of fatigue, myalgias, arthralgias, and weakness that have slowly been getting worse. Before surgery she weighed 340 pounds (154 kg), and in the first 2 years afterward she lost 160 pounds (72.5 kg). She is postmenopausal, has no history of fractures, nephrolithiasis, or thyroid disease, and does not smoke or consume alcohol. She gives herself monthly intramuscular vitamin B₁₂ injections, and takes a multivitamin tablet, calcium carbonate 500 mg, and vitamin D 400 IU daily.

After her surgery she returned for her first two postoperative appointments, but because she was feeling well, was losing weight, and had returned to work full-time, she cancelled all subsequent appointments with the surgeon, bariatrician, and dietitian.

On physical examination, the patient's weight is stable at 187 pounds (84.5 kg), her height is 165.1 cm, and her body mass index is 31. Her head, eyes, ears, nose, throat, heart, lungs, and abdomen are normal. Her upper legs are weak, requiring her to use her arms in rising from a chair, and she feels discomfort when the proximal muscles of her arms and legs are palpated. She has mild osteoarthritis of the hands and knees. Her neurologic examination is normal.

*Dr. Richmond has disclosed that he has received honoraria from Procter and Gamble for teaching and speaking. Dr. Schauer has disclosed that he is a consultant for or on the scientific advisory boards of Bard-Davol, Barosense, Baxter, Ethicon Endosurgery, Gore, and Stryker Endoscopy; is on the board of directors and is a partner in Remedy MD; and has received grant support from Invacare.

Pertinent laboratory data:

- Calcium 8.1 mg/dL (reference range 8.5–10.5)
- Albumin 3.7 g/dL (3.4–4.7)
- Magnesium 1.9 mg/dL (1.7–2.6)
- Phosphorus 2.7 mg/dL (2.4–4.5)
- Alkaline phosphatase 240 U/L (40–150)
- Intact parathyroid hormone 215 pg/mL (10–60)
- 25-hydroxyvitamin D < 7.0 ng/mL (31–80)
- 24-hour urine volume 2,310 mL
- Urine creatinine normal
- Urine calcium 25.4 mg/24 hours (100–300).

Dual-energy x-ray absorptiometry (DXA) data, lumbar spine:

- Bone mineral density 0.933 g/cm²
- T score –2.0
- Z score –0.8.

Left total hip:

- Bone mineral density 0.628 g/cm²
- T score –2.6
- Z score –2.4.

**■ METABOLIC BONE DISEASE:
A CASE IN POINT**

Many have nonspecific symptoms that are incorrectly diagnosed as fibromyalgia, arthritis, Paget disease, or depression

This is a classic presentation of metabolic bone disease in a bariatric surgery patient lost to follow-up. Many patients have nonspecific and vague symptoms for many months or years that are often incorrectly diagnosed as fibromyalgia, rheumatoid arthritis, polymyalgia rheumatica, Paget disease, or depression.¹ They typically have low serum and urine calcium levels, very low or undetectable 25-hydroxyvitamin D levels, high alkaline phosphatase levels, secondary hyperparathyroidism, and a clinical picture consistent with both osteomalacia and osteoporosis.¹

This case underscores the importance of monitoring nutrients and biochemical markers at baseline and on an ongoing basis to detect early indicators of malabsorption and ultimately prevent the development of metabolic bone disease and fragility fracture, with its risks of disability and even death. It also illustrates the essential role that primary care physicians play in the continuing care of these patients.

■ THE OBESITY-BONE CONNECTION

Although we used to think that morbid obesity protected against metabolic bone disease, in fact, vitamin D and calcium deficiencies and elevated parathyroid hormone (PTH) levels are common in extremely obese people, placing them at risk of low bone mass.^{2–6} More than 60% of candidates for weight-loss surgery are deficient in vitamin D,^{5,7} and 25% to 48% have elevated PTH levels.^{5,6}

And that is *before* bariatric surgery: afterward, severely restricted oral intake and significant weight loss, coupled with a procedure that bypasses the major site of calcium absorption, place many patients at extremely high risk.^{5,8}

After combination restrictive and malabsorptive procedures (eg, the popular Roux-en-y procedure, in which the stomach is reduced in size—“restricted”—and the proximal duodenum is bypassed so that less food is absorbed), as patients lose weight their PTH levels rise and 25-hydroxyvitamin D levels decrease, although corrected calcium levels usually remain within normal limits.^{3,9} Secondary hyperparathyroidism has been documented as soon as 8 weeks after bariatric surgery, and osteomalacia after gastric bypass surgery is not uncommon.^{1,7,10–13}

Exclusively restrictive procedures such as gastric banding, formerly presumed not to alter bone metabolism, now also appear to place patients at risk of metabolic bone disease due to inadequate intake of calcium and vitamin D in the immediate postoperative period.¹⁰

Numerous reported cases further illustrate the ever-present risk of metabolic bone disease in this population if adequate supplementation of calcium and vitamin D is not given. In these cases, significant bone disease occurred from 8 weeks to 32 years after bariatric surgery, often with devastating consequences.^{1,4,8,11–14}

**Voluntary weight loss,
Involuntary bone loss**

When overweight or obese people lose weight—whether by dieting or by bariatric surgery—they also lose bone: a voluntary loss of approximately 10% of body weight results in a loss of 1% to 2% of bone at all sites. This loss appears to vary among populations: pre-

The faster weight is lost, the greater the percentage of bone loss

TABLE 1

Essential nutrients for preventing and treating metabolic bone disease after bariatric surgery

Protein

Consuming 1.0–1.5 g protein/kg body weight daily appears to be optimal for bone health
In obese patients, use the adjusted body weight^a when calculating protein needs
The literature remains unclear as to whether animal or vegetable sources are more beneficial

Vitamin D

Daily requirement: 800–1,000 IU
Casual sunlight exposure is the best source in warmer climates
Excellent food sources include butter, margarine, liver, and eggs
Additional sources include fortified milk, juices, and cereals
It is difficult to obtain adequate vitamin D from dietary sources alone
Patients who are deficient require substantially higher oral doses for repletion

Calcium

Choose a supplement from a respected manufacturer
Avoid supplements that contain dolomite, bone meal, or unrefined oyster shell
A chewable or liquid supplement may be easier to take and may be better absorbed
Dose: ages 19–50, 1,000 mg; ages 51 and up, 1,200 mg; during weight loss, 1,800 mg
Higher doses may be required, based on urine calcium studies

Vitamin B₁₂

Recommended dietary allowance (RDA): 2.4 µg/day
Postbariatric surgery patients should take a multivitamin that contains at least the RDA for B₁₂
Patients who are mildly deficient should be started on 350 µg/day and monitored
Patients who do not respond to oral replacement need monthly subcutaneous injections

Magnesium

Excellent food sources include legumes; good sources include grains, root and green vegetables, and nuts
RDA for men, 420 mg/day; RDA for women, 320 mg/day
A diet without legumes will tend to fall short of the magnesium recommended daily allowance;
after bariatric surgery, patients should take a multivitamin that contains magnesium until they can consume sufficient amounts of magnesium-rich foods

^aIf the actual body weight is more than 125% of the ideal body weight, calculate the adjusted body weight:
 $[(\text{actual body weight} - \text{ideal body weight}) \times 0.25] + \text{ideal body weight}$

menopausal women younger than 45 years may be able to lose a moderate amount of weight without a significant increase in fracture risk, while a study of overweight men found that a 7% weight loss resulted in a 1% bone loss.¹⁵

The percentage of bone lost correlates strongly with how fast the weight is lost. A recent study found that losing 0.7 kg/week was more detrimental to bone than a slower loss of 0.3 kg/week, due to the activation of the calcium-PTH axis.¹⁶

After bariatric surgery, many patients rapidly lose 50 kg—some even lose 100 kg or

more. This rapid weight loss, combined with severely restricted oral intake, decreased calcium absorption, and vitamin D deficiency places these patients at extremely high risk of rapidly developing metabolic bone disease.^{3,8,9} In one large study, metabolic bone disease developed in more than 70% of patients who underwent a malabsorptive procedure, while in a second study, markers of bone resorption were elevated as soon as 8 weeks after bariatric surgery, regardless of whether the patient underwent a malabsorptive or restrictive bariatric procedure.¹³ Yet another study found that 48% of patients had a statistically signifi-

cant bone mineral reduction of more than 3% 12 months after undergoing gastric banding.¹⁰

■ ESSENTIAL NUTRIENTS FOR BONE HEALTH

A number of nutrients are essential for bone health; we will limit our discussion to the major ones directly affected by obesity and bariatric surgery (TABLE 1). Other nutrients often affected by obesity and bariatric surgery will be discussed in a subsequent article.

Protein

Dietary protein is needed to maintain bone structure, and although there is a link between high protein intake, calciuria, and fracture risk, the potentially harmful effects appear to be ameliorated when high protein intake is coupled with adequate calcium.¹⁷⁻²⁰ This fact is of particular importance after bariatric surgery because once the patient can consume enough fluids to maintain hemodynamic stability, he or she is given a relatively high-protein diet to prevent protein malnutrition.²¹

Inadequate protein intake also has a detrimental effect on bone; therefore, it is essential to assess postoperative protein intake.²² Rizzoli and Bonjour²³ noted that markers of bone turnover were higher with a low-protein diet (0.7 g protein per kg body weight) than with a diet containing 2.1 g protein per kg. In two trials examining graded levels of protein ingestion (0.7, 0.8, 0.9, and 1.0 g protein per kg body weight), decreased calcium absorption and an acute rise in PTH were noted by day 4 of the 0.7- and 0.8-g/kg diets but not during the 0.9- or 1.0-g/kg diets.^{24,25} And a systematic review of protein and bone health concluded that diets containing 1.0 to 1.5 g protein/kg are best for bone health.²⁶ This is particularly worrisome, since the current recommended dietary allowance for protein is only 0.8 g/kg, which may be insufficient to promote calcium homeostasis.^{26,27}

Vitamin D

Vitamin D is essential for calcium absorption, stimulation of osteoblast activity, and normal bone mineralization throughout the life span.²⁷ Dietary vitamin D is mainly absorbed by passive diffusion in the proximal and mid

small intestine in a process that is highly dependent on bile salts.²⁸⁻³⁰ Dietary sources of vitamin D are clinically important because exposure to ultraviolet B radiation is often insufficient, especially in northern latitudes.²⁷

Up to 84% of morbidly obese patients have vitamin D deficiency.^{2,3,5,28,29,31} The mechanism of vitamin D deficiency and secondary hyperparathyroidism in the morbidly obese remains unclear, although one study concluded they were likely due to sequestration of vitamin D in adipose tissue and subsequent limited bioavailability.³²

Correction of vitamin D deficiency requires more than just an over-the-counter multivitamin, but standard multivitamins also contain vitamin A, so taking more than one tablet a day increases the risk of vitamin A excess.³³ Repletion can often be safely achieved orally by giving 50,000 IU of vitamin D weekly for 8 weeks, followed by a maintenance dose of one 50,000 IU tablet every 2 weeks. If a repeat serum level shows suboptimal repletion (less than 32 ng/mL), an additional 8-week course is recommended.²⁹ For patients who cannot tolerate or adequately absorb oral supplements, exposure to sunlight is still the best source of vitamin D and is an effective alternative.^{29,33}

Calcium

Dietary calcium deficiency is a well-established risk factor for osteoporosis and fragility fractures. Therefore, supplemental calcium should be prescribed for patients who do not meet their defined need.^{34,35} Of note: a normal serum calcium level does not imply adequate calcium intake or absorption. Calcium homeostasis is tightly regulated and is maintained by a combination of gut absorption, bone resorption, and renal reabsorption. If dietary intake is inadequate, calcium is resorbed from the bone.

The duodenum is the major site of active calcium uptake, while the rest of the small intestine and the colon appear to absorb some calcium passively. When the physiologic need for calcium is increased, active transport appears to take place throughout the duodenum, the ileum, and, to a lesser degree, the jejunum and the colon.³⁶

In the normal gastrointestinal tract, 20%

The current recommended dietary allowance of protein may be too low to promote calcium homeostasis

to 60% of dietary calcium is absorbed.^{36–38} Patients who have lost absorptive surface area (eg, after Roux-en-y bariatric surgery) need to have their calcium intake optimized. However, optimal dosing based on the type of surgical procedure is currently undefined.

Judicious monitoring for compliance and adequate absorption is recommended. Some patients will stop taking their calcium supplement due to gastrointestinal side effects such as gas, bloating, or constipation. And for some patients, a standard calcium supplement may be insufficient to promote adequate calcium absorption. Measuring urinary calcium in a 24-hour sample can help in assessing the adequacy of calcium intake: abnormally low urine calcium in the presence of normal renal function suggests inadequate absorption. For patients reporting gastrointestinal side effects or those with a history of calcium oxalate renal stones, calcium citrate supplements are better tolerated, alter urine acidity, and often prevent further stone formation.

Vitamin B₁₂ (cobalamin)

Vitamin B₁₂ deficiency is associated with increased fracture risk, and it may be an important modifiable risk factor for osteoporosis.^{39–41} After surgery, malabsorption of vitamin B₁₂ is commonly the result of altered gut function in the gastric pouch or sleeve, but malabsorption also occurs when more than 60 to 100 cm of terminal ileum has been bypassed.⁴²

Vitamin B₁₂ supplementation is recommended for all patients after bariatric surgery, because deficiency is common.⁴² Patients with relatively mild malabsorption can maintain their B₁₂ level by taking 350 µg orally; however, many patients require lifelong subcutaneous injections.^{7,39,42–45}

Magnesium

Magnesium appears to affect bone remodeling and strength, to have a positive association with hip bone mineral density, and to play an important role in calcium and bone metabolism.

Magnesium is absorbed in the distal small intestine by carrier-mediated and paracellular routes.⁴⁶ When the distal small intestine is bypassed, magnesium deficiency occurs as a

result of reduced absorption and chelation with unabsorbed fatty acids in the bowel lumen.⁴² Chronic hypomagnesemia impairs PTH secretion, resulting in altered calcium metabolism, hypocalcemia, and vitamin D abnormalities, further decreasing jejunal magnesium absorption.^{26,42,47}

Few well-designed studies have investigated the effect of magnesium intake on bone health, and although there is evidence that postmenopausal women may benefit from magnesium supplementation, studies of magnesium supplementation after bariatric surgery are lacking.^{47,48}

A prevailing misconception promoted by manufacturers of calcium-magnesium supplements and others is that magnesium is necessary for calcium absorption and efficacy. In fact, magnesium deficiency typically must be severe to impair calcium absorption. With usual dietary intake of magnesium and normal serum magnesium levels, no such relationship exists.^{49–53}

■ THE ROLE OF DXA IN THE CARE OF THE BARIATRIC SURGERY PATIENT

DXA is the gold standard for measuring bone density. The results are reported as a T score and as a Z score.

The T score is the bone density in an area of interest expressed in standard deviations from the mean value of a reference database of young adults. The World Health Organization defines normal as a T score greater than or equal to -1 , low bone mass (previously called osteopenia) as a score between -1 and -2.5 , and osteoporosis as a score of less than or equal to -2.5 . (If a fragility fracture has occurred, “established” or “severe” osteoporosis is present.) Of note: these criteria only apply to DXA of the posterior-anterior spine, femoral neck, and the proximal (33%) radius in postmenopausal women and men over the age of 50 years.^{54,55} The International Society of Clinical Densitometry has extended the criteria to include total hip measurements.⁵⁶

The Z score should be used instead of the T score for premenopausal women and men younger than 50 years.⁵⁶ The Z score is the patient’s bone mineral density expressed in standard deviations from the mean in a refer-

Adequate vitamin D is essential for bone health throughout the life span

ence population matched for sex and age. A Z score greater than -2.0 is “within the expected range for age,” and -2.0 or lower is “below the expected range for age.” There are separate guidelines for DXA reporting in the diagnosis of metabolic bone disease in people younger than 20 years, and this topic is beyond the scope of this article.

If bone loss is exclusively due to the normal process of aging, the Z score will be near zero. However, if the Z score is less than -1.5 , secondary causes of bone disease should be considered. All patients with Z scores lower than -2.0 should be further evaluated, and therapy should be based on the results of that evaluation (TABLE 2).^{34,56,57}

Who should undergo DXA?

According to the International Society of Clinical Densitometry, bone density testing is indicated in the general population in women 65 years of age and older, postmenopausal women younger than 65 with risk factors, men 70 and older, adults with fragility fractures, adults taking a medication or having a disease or condition associated with low bone mass or bone loss, any patient being treated for low bone mass (to monitor the treatment effect), and any person in whom evidence of bone loss would affect treatment decisions.⁵⁸

The National Osteoporosis Foundation recommends initiating therapy to reduce fracture risk in postmenopausal women with a central DXA T score below -2 in the absence of risk factors, and in women with T scores below -1.5 if one or more risk factors is present.³⁴ Therefore, in view of the known risks, the likely need for interventions before surgery, and the ability to prevent the illness and death associated with metabolic bone disease, we recommend that all bariatric surgery patients undergo DXA at baseline as part of the preoperative evaluation.

Improvements in DXA technology

Newer DXA machines can accommodate patients weighing up to 450 pounds (the limit with older machines was 275 pounds for central measurements). In addition to measuring bone density, they also can map the distribution of fat in the body—patients with an android (apple-shaped) distribution are at higher risk of

TABLE 2

Risk factors for metabolic bone disease

Bariatric surgery

Family history

First-degree relative with fragility fracture

Medical history

Age > 65 (women) or > 70 (men)

Fragility fracture

Postmenopausal status

Early estrogen deficiency (< 45 years of age)

Hypogonadism

Chronic obstructive pulmonary disease

Type 1 diabetes mellitus

Significant weight loss

Medications (current or previous)

Antacids containing aluminum

Anticonvulsants (phenobarbital, phenytoin)

Cholestyramine

Gonadotropin-releasing hormone

Glucocorticoids (oral, used for > 3 months or in multiple courses)

Heparin (long-term use)

Lithium

Methotrexate

Thyroid hormones (in excess)

Morbid obesity

Social or lifestyle factors

Alcohol use (> 2 drinks daily)

Cigarette smoking

Eating disorder

Little or no physical activity

Low calcium intake (lifelong)

Poor dietary habits or chronic dieting

cardiovascular disease than those with a gynecoid (pear-shaped) distribution.^{59–63} For those patients who cannot be accommodated on a DXA table, DXA of the forearm can be used to assess bone density and fracture risk.

How often should DXA be repeated?

The estimated monitoring time interval is derived from the statistically defined least significant change divided by the anticipated change in bone density over time.⁶⁴ When estimating the monitoring time interval for changes in body composition, the rate of weight loss and the psychological impact on the patient must be taken into consideration.

TABLE 3

Preventing and treating metabolic bone disease in bariatric surgery patients: Our recommendations

Preoperative evaluation

Dual-energy x-ray absorptiometry (DXA)

Serum levels of: albumin, calcium, magnesium, phosphorus, alkaline phosphatase, folate, vitamin B₁₂, thyroid-stimulating hormone, 25-hydroxyvitamin D, intact parathyroid hormone

Preoperative interventions

Give prophylactic doses (appropriate to the patient's age and sex) of multivitamins with minerals and of calcium and vitamin D

Give pharmacologic doses of vitamins, minerals, or both, if deficient

Immediate postoperative period

Intravenous multivitamin with minerals in the first liter of intravenous fluids daily × 3 days or until able to take oral supplements

Prescribe chewable or liquid form of multivitamin with minerals appropriate to the patient's age and sex, and 1,800 mg calcium with 800–1,000 IU vitamin D

Follow-up postoperative visits at 4 weeks; 3, 6, and 12 months; then annually

Repeat preoperative laboratory tests

Reinforce compliance with prescribed supplements

Give pharmacologic-dose vitamin and/or mineral replacement if deficient or if malabsorption is evident

DXA: as recommended based on examination findings and the patient's risk-factor profile

INTERVENTIONS BASED ON DXA^a

T score ≥ -1.0: normal bone density

Counsel all patients on risk factor reduction including:

Adequate consumption of calcium and vitamin D

Regular weight-bearing and muscle-strengthening exercise

Smoking cessation

Limiting alcohol intake to 2 drinks daily

Discontinue or substitute high-risk medications when clinically appropriate

Initiate therapy for patients on long-term steroids

T score between -1.0 and -2.5: low bone mass (osteopenia)

Counsel the patient on risk factor reduction

Start therapy for women with one or more risk factors and a T score below -1.5

Start therapy for women with T scores below -2.0

Start therapy for any patient with a history of fragility fracture

Start therapy for patients on long-term corticosteroid therapy

T score ≤ -2.5: osteoporosis

Counsel the patient on risk factor reduction

Start therapy

Schedule routine follow-up appointments to assess compliance and effectiveness of prescribed interventions

Requires lifelong management

^aWorld Health Organization definitions based on bone mass measurement at the spine, hip, and wrist in white, postmenopausal women. T scores are to be used to assist the clinician in determining the diagnosis and appropriate treatment options and can be misleading in younger women, men, and nonwhites.

A normal serum calcium level does not imply adequate calcium intake or absorption

In general, DXA testing more frequently than every 2 years remains controversial unless one is initiating, monitoring, or changing therapy or monitoring conditions associated with rapid bone loss such as glucocorticoid therapy. In the bariatric surgery population, however, there is convincing evidence that significant changes may be detected after 12 months that would influence clinical decisions, particularly in the year immediately after surgery.^{8,9,13,21,27,65}

Anabolic and antiresorptive bone drugs

Prescribed medications for the prevention and treatment of osteoporosis should also be an integral part of the treatment plan for at-risk morbidly obese patients. But the decision to prescribe an antiresorptive or bone-forming medication must take into consideration the patient's risk-benefit profile, including the likelihood of gastrointestinal side effects and his or her ability and willingness to follow specific dosing instructions. Intravenous preparations are now available for patients who cannot absorb or tolerate oral antiresorptive medications. However, specific recommendations about the use of anabolic or antiresorptive bone medications in perioperative bariatric patients have yet to be elucidated.

RECOMMENDATIONS

Although a variety of recommendations have been published, there are no established guidelines for perioperative screening, risk stratification, or management of metabolic bone disease in bariatric surgery patients.^{7,44,65-67} And the literature remains inconclusive on key issues such as when to start supplements, which biochemical indices should be checked before surgery, whether baseline and annual DXA should be done, and whether antiresorptive agents such as bisphosphonates should be used prophylactically during rapid weight loss.

However, numerous studies and case reports cited here and elsewhere further underscore the ever-present risk of metabolic bone disease in this patient population, and the need for meticulous perioperative and long-term monitoring.^{44,65-67}

Interventions should be individualized: we need to carefully consider the modifiable

risk factors, the severity of disease, and the patient's ability and willingness to participate in his or her care. Modifiable risk factors for progression of bone disease must be addressed in all patients at risk. It is also important to identify patients at high risk for falls due to poor visual acuity, frailty, neuropathy, or dementia and to implement risk management strategies to the degree possible.³⁴

With these caveats in mind, we offer our recommendations (TABLE 3).

Preoperative assessment

We recommend obtaining baseline biochemical indices, including albumin, 25-hydroxyvitamin D, calcium, magnesium, phosphorus, alkaline phosphatase, folate, vitamin B₁₂, thyroid-stimulating hormone, and PTH levels, and DXA in all bariatric surgery candidates. These indices should be used to assess for primary and secondary metabolic bone disease, to enable prompt presurgical interventions, and to guide the clinician in selecting appropriate postoperative interventions and surveillance.

We recommend starting a multivitamin with minerals at the first preoperative visit. A calcium supplement that provides calcium and vitamin D appropriate to the patient's age and sex is also recommended until surgery. After surgery, and while rapid weight loss is occurring, a minimum of 1,800 mg of calcium and 800 to 1,000 IU of vitamin D is recommended, keeping in mind that the required level of supplemental vitamin D during periods of rapid weight loss remains unclear.^{68,69}

However, before prescribing supplementation, one should thoroughly review the patient's nutrition history, including the use of homeopathic medications, herbal preparations, and supplements. Many over-the-counter and over-the-Internet supplements are touted as being good for bone health, and some may indeed be beneficial, but others can be detrimental and need to be discontinued.^{27,47} Furthermore, in a patient with a severely restricted stomach capacity, it is important to ensure that less efficacious supplements do not compromise the intake of essential fluids, protein, and prescribed medications.

We recommend DXA for all bariatric surgery candidates

Immediate postoperative period

Hospitalization and surgery result in nutrient deficiencies. In bariatric surgery patients, particularly those who have preoperative nutritional deficiencies, repletion in the immediate postoperative period is believed to be of benefit. Therefore, in the immediate postoperative period we recommend infusing a standard-dose multivitamin with minerals daily along with adequate intravenous hydration until the patient can resume oral feeding. Once the patient can tolerate liquids, presurgical supplementation needs to be resumed, preferably in a liquid or chewable form to facilitate tolerance and absorption.

Short-term and long-term follow-up

Follow-up visits with a bariatric specialist should start 4 weeks after surgery and should be repeated every 3 to 4 months for the first year. If the patient continues to do well, annual visits may be sufficient thereafter. Compliance with supplements should be

checked as indicated, as should nutritional indices. DXA should be repeated every 1 to 2 years, depending on the patient's risk profile.

■ WHAT SHOULD BE DONE FOR OUR PATIENT?

Initial treatment for the patient described at the beginning of this article should include vitamin D repletion with cholecalciferol 50,000 IU, calcium supplements of at least 1,200 mg daily, and addressing of modifiable risk factors for fracture, including the risk of falling due to her proximal weakness. Her laboratory studies should be repeated in 6 to 12 weeks, with calcium and vitamin D supplement dosages adjusted on the basis of her response. Once the serum calcium level has normalized, we would consider the use of a bisphosphonate. DXA should be repeated in 1 to 2 years to monitor the effectiveness of the prescribed interventions. ■

■ REFERENCES

1. De Prisco C, Levine SN. Metabolic bone disease after gastric bypass surgery for obesity. *Am J Med Sci* 2005; 329:57–61.
2. Ybarra J, Sanchez-Hernandez J, Gich I, et al. Unchanged hypovitaminosis D and secondary hyperparathyroidism in morbid obesity after bariatric surgery. *Obes Surg* 2005; 15:330–335.
3. Hamoui N, Anthonie G, Crookes F. Calcium metabolism in the morbidly obese. *Obes Surg* 2004; 14:9–12.
4. Parikh SJ, Edelman M, Uwaifo GI, et al. Gastric bypass surgery for morbid obesity leads to an increase in bone turnover and a decrease in bone mass. *J Clin Endocrinol Metab* 2004; 89:1196–1199.
5. Carlin AM, Rao DS, Mesleman AM, et al. Prevalence of vitamin D depletion among morbidly obese patients seeking gastric bypass surgery. *Surg Obes Rel Dis* 2006; 2:98–103.
6. Puzifferri N, Blankenship J, Wolfe BM. Surgical treatment of obesity. *Endocrine* 2006; 29:11–19.
7. Mason EM, Jalagani H, Vinik AI. Metabolic complications of bariatric surgery: diagnosis and management issues. *Gastroenterol Clin North Am* 2006 34:25–33.
8. Haria DM, Sibonga JD, Taylor HC. Hypocalcemia, hypovitaminosis D osteopathy, osteopenia, and secondary hyperparathyroidism 32 years after jejunoileal bypass. *Endocr Pract* 2005; 11:335–340.
9. Newbery L, Dolan K, Hatzifotis M, et al. Calcium and vitamin D depletion and elevated parathyroid hormone following biliopancreatic diversion. *Obes Surg* 2003; 13:893–895.
10. Pugnale N, Guisti V, Suter M, et al. Bone metabolism and risk of secondary hyperparathyroidism 12 months after gastric banding in obese pre-menopausal women. *Int J Obesity* 2003; 27:110–116.
11. Goldner WS, O'Dorisio TM, Dillon JS, Mason EE. Severe metabolic bone disease as a long-term complication of obesity surgery. *Obes Surg* 2002; 12:685–692.
12. Atreja A, Abacan C, Licata A. A 51-year-old woman with debilitating cramps 12 years after bariatric surgery. *Cleve Clin J Med* 2003; 70:417–426.
13. Collazo-Clavell ML, Jimenez A, Hodgson SF, et al. Osteomalacia after Roux-en-Y gastric bypass. *Endocr Pract* 2004; 10:287–288.
14. Dennison E. Osteoporosis. In: Pinchera A, Bertagna X, Fischer J, editors. *Endocrinology and Metabolism*. London: McGraw-Hill International (UK) Ltd.; 2001:271–282.
15. Shapses SA, Cifuentes M. Body weight/composition and weight change: effects on bone health. In: Holick MF, Dawson-Hughes B, editors. *Nutrition and Bone Health*. New Jersey: Humana Press; 2004:549–573.
16. Shapses SA, Cifuentes M, Sherrell R, et al. Rate of weight loss influences calcium absorption. *J Bone Miner Res* 2002; 17:5471.
17. Munger RG, Cerhan JR, Chiu BC. Prospective study of dietary protein intake and risk of hip fracture in postmenopausal women. *Am J Clin Nutr* 1999; 69:147–152.
18. Weikert C, Walter D, Hoffmann K, et al. The relation between dietary protein, calcium and bone health in women: results from the EPIC-Potsdam cohort. *Ann Nutr Metab* 2005; 49:312–318.
19. Whiting SJ, Boyle JL, Thompson A. Dietary protein, phosphorus and potassium are beneficial to bone mineral density in adult men consuming adequate dietary calcium. *J Am Col Nutr* 2002; 21:402–409.
20. Teegarden D, Lyle RM, McCabe GP, et al. Dietary calcium, protein, and phosphorus are related to bone mineral density and content in young women. *Am J Clin Nutr* 1998; 68:749–754.
21. Bloomberg RD, Fleishman A, Nalle JE, et al. Nutritional deficiencies following bariatric surgery: what have we learned? *Obes Surg* 2005; 15:145–154.
22. Greenspan SL, Resnick NM. Geriatric endocrinology. In: Greenspan FS, Gardner DG. *Basic and Clinical Endocrinology* (7th ed.). New York: McGraw-Hill; 2004:842–866.
23. Rizzoli R, Bonjour JP. Dietary protein and bone health. *J Bone Miner Res* 2004; 19:527–531.
24. Kerstetter J, Svastisalee C, Caseria D, et al. A threshold for low-protein-diet-induced elevations in parathyroid hormone. *Am J Clin Nutr* 2000; 72:168–173.
25. Giannini S, Nobile M, Sartori L, et al. Acute effects of moderate

- dietary protein restriction in patients with idiopathic hypercalcaemia and calcium nephrolithiasis. *Am J Clin Nutr* 1999; 69:267–271.
26. **Ilich JZ, Kerstetter JE.** Nutrition in bone health revisited: a story beyond calcium. *J Am Coll Nutr* 2000; 19:715–737.
 27. **Williams S, Seidner D.** Metabolic bone disease in gastrointestinal illness. *Gastroenterol Clin North Am* 2007; 36(1):161–190.
 28. **Shoback D, Marcus R, Bilke D.** Metabolic bone disease. In: Greenspan FS, Gardner DG, editors. *Basic and Clinical Endocrinology* (7th ed.). New York: McGraw-Hill; 2004:295–361.
 29. **Holick MF.** Vitamin D. In: Shils ME, Olsen JA, Shine M, et al, editors. *Modern Nutrition in Health and Disease* (9th ed.). Philadelphia: Lippincott Williams & Wilkins; 1999:329–346.
 30. **Rosen CJ.** Vitamin D and bone health in adults and the elderly. In: Holick MF. *Vitamin D: Physiology, Molecular Biology, and Clinical Applications*. New Jersey: Humana Press; 1999:287–306.
 31. **Buffington C, Walker B, Cowan GS, et al.** Vitamin D deficiency in the morbidly obese. *Obes Surg* 1993; 3:421–424.
 32. **Wortsman J, Matsuoka L, Chen TC, et al.** Decreased bioavailability of vitamin D in obesity. *Am J Clin Nutr* 2000; 72:690–693.
 33. **Holick MF, Garabedian M.** Vitamin D: photobiology, metabolism, mechanism of action, and clinical applications. In: *Primer on the Metabolic Bone Diseases and Disorders of Mineral Metabolism* (6th ed.). Washington DC: American Society for Bone and Mineral Research, 2006:106–114.
 34. **National Osteoporosis Foundation.** *Physician's Guide to Prevention and Treatment of Osteoporosis*. <http://www.nof.org/physguide>. Washington D.C. 1998.
 35. **National Academy of Sciences Executive Summary: Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride.** Washington DC. 1997.
 36. **Favus MJ, Bushinsky DA, Lemann J.** Regulation of calcium, magnesium and phosphate metabolism. In: *Primer on the Metabolic Bone Diseases and Disorders of Mineral Metabolism* (6th ed.). Washington DC: American Society for Bone and Mineral Research, 2006:76–83.
 37. **Weaver CM, Heaney RP.** Calcium. In: Shils ME, Olsen JA, Shine M, et al, editors. *Modern Nutrition in Health and Disease* (9th ed.). Philadelphia: Lippincott Williams & Wilkins, 1999:141–155.
 38. **Gueguen L, Oibuttukart A.** The bioavailability of dietary calcium. *J Am Coll Nutr* 2000; 19:1195–1365.
 39. **Tucker KL, Hannan MT, Qiao N, et al.** Low plasma vitamin B₁₂ is associated with lower BMD: the Framingham osteoporosis study. *J Bone Miner Res* 2005; 20:152–158.
 40. **Goerss JB, Kim CH, Atkinson EJ, et al.** Risk of fractures in patients with pernicious anemia. *J Bone Miner Res* 1992; 7:573–579.
 41. **Eastell R, Vieira NE, Yergey AL, et al.** Pernicious anemia is a risk factor for osteoporosis. *Clin Sci* 1992; 82:681–685.
 42. **Nightingale JMD, Woodward JM.** Guidelines for management of patients with a short bowel. *Gut* 2006; 55:1–12.
 43. **Rhode BM, Tamin H, Gilfix BM, et al.** Treatment of vitamin B₁₂ deficiency after gastric surgery for severe obesity. *Obes Surg* 1995; 5:154–158.
 44. **Brethauer SA, Chand C, Schauer PR.** Risks and benefits of bariatric surgery: current evidence. *Cleve Clin J Med* 2006; 73:993–1007.
 45. **Weir DG, Scott JW.** Vitamin B₁₂ “cobalamin”. In: Shils ME, Olsen JA, Shine M, et al. editors. *Modern Nutrition in Health and Disease* (9th ed.). Philadelphia: Lippincott Williams & Wilkins; 1999:447–458.
 46. **Shils ME.** Magnesium. In: Shils ME, Olsen JA, Shine M, et al. editors. *Modern Nutrition in Health and Disease* (9th ed.). Philadelphia: Lippincott Williams & Wilkins; 1999:169–192.
 47. **National Osteoporosis Foundation.** *Osteoporosis Clinical Updates: Over-the-counter products & osteoporosis: Case discussions*. 2002; Vol III Issue 2. Washington DC.
 48. **Stendig-Lindenberg G, Tepper R, Leicher I.** Trabecular bone density in a two year controlled trial of personal magnesium in osteoporosis. *Magnes Res* 1993:155–163.
 49. **Heaney RP.** Sodium, potassium, phosphorus, and magnesium. In: Holick MF, Dawson-Hughes B, editors. *Nutrition and Bone Health*. New Jersey: Humana Press; 2004:327–344.
 50. **Spencer H, Fuller H, Norris C, et al.** Effect of magnesium on the intestinal absorption of calcium in man. *J Am Coll Nutr* 1994; 13:483–492.
 51. **Chapuy MC, Arlot ME, Duboeuf F, et al.** Vitamin D and calcium to prevent hip fractures in elderly women. *N Engl J Med* 1992; 327:1637–1642.
 52. **Dawson-Hughes B, Harris SS, Krall EA, et al.** Effect of calcium and vitamin D supplementation on bone density in men and women 65 years of age and older. *N Engl J Med* 1997; 337:670–676.
 53. **Rude RK, Olerich M.** Magnesium deficiency: possible role in osteoporosis associated with gluten-sensitive enteropathy. *Osteoporos Int* 1996; 6:453–461.
 54. **Assessment of fracture risk and its application to screening for postmenopausal osteoporosis.** Report of a WHO Study Group. 1994; 843:1–129.
 55. **Kanis JA, Melton LJ, Christiansen C, et al.** The diagnosis of osteoporosis. *J Bone Miner Res* 1994; 9:1137–1141.
 56. **Official Positions of the International Society for Clinical Densitometry.** West Hartford, CT. September, 2005. <http://www.iscd.org/Visitors/positions/OfficialPositionsText.cfm>.
 57. **Licata A.** Diagnosing primary osteoporosis: It's more than a T score. *Cleve Clin J Med* 2006; 73:473–476.
 58. **Writing group for the ISCD Position Development Conference.** Indications and reporting for dual-energy x-ray absorptiometry. *J Clin Densitom* 2004; 7:37–44.
 59. **Park YW, Heymsfield SB, Gallagher D.** Are dual-energy X-ray absorptiometry regional estimates associated with visceral adipose tissue mass? *Int J Obes Relat Metab Disord* 2002; 26:978–983.
 60. **Glickman SG, Marn CS, Supiano MA, et al.** Validity and reliability of dual-energy x-ray absorptiometry for the assessment of abdominal adiposity. *J Appl Physiol* 2004; 97:509–514.
 61. **Hull HR, Hester CN, Fields DA.** The effect of the holiday season on body weight and composition in college students. *Nutr Metab (Lond)* 2006; 3:44–51.
 62. **Aubertin-Leheudre M, Goulet EDB, Khalil A, et al.** Effect of sarcopenia on cardiovascular disease risk factors in obese postmenopausal women. *Obesity* 2006; 14:2277–2283.
 63. **Lubrano C, Cornoldi A, Pili M, et al.** Reduction of risk factors for cardiovascular diseases in morbid-obese patients following biliary-intestinal bypass: 3 years' follow-up. *Int J Obes Relat Metab Disord* 2004; 28:1600–1606.
 64. **Bonnick SL, Johnston CC Jr, Kleerekoper M, et al.** Importance of precision in bone density measurements. *J Clin Densitom* 2001; 4:105–110.
 65. **Mason EM, Jalagani H, Vinik AI.** Metabolic complications of bariatric surgery: diagnosis and management issues. *Gastroenterol Clin North Am* 2006 34:25–33.
 66. **Hensrud DD, McMahon MM.** Bariatric surgery in adults with extreme (not morbid) obesity. *Mayo Clin Proc* 2006; 81(suppl):S3–S4.
 67. **McGlinch BP, Que FG, Nelson JL, et al.** Perioperative care of patients undergoing bariatric surgery. *Mayo Clin Proc* 2006; 81(10, suppl):S25–S33.
 68. **Ricci TA, Chowdhury HA, Heymsfield SB, et al.** Calcium supplementation suppresses bone turnover during weight reduction in postmenopausal women. *J Bone Miner Res* 1998; 13:1045–1050.
 69. **Jensen LB, Kollerup G, Quaade F, et al.** Bone mineral changes in obese women during moderate weight loss with and without calcium supplementation. *J Bone Miner Res* 2001; 16:141–147.
-
- ADDRESS:** Susan Williams, MD, Center for Nutrition & Metabolic Medicine, Greene Memorial Hospital Health Center, 50 North Progress Drive, Xenia, OH 45385; e-mail swilliams@greenehealth.org.