Review Article

Endocrine and Metabolic Complications After Bariatric Surgery

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ABSTRACT

Bariatric surgery is the most effective therapeutic option for obese patients; however, it carries substantial risks, including procedure-related complications, malabsorption, and hormonal disturbance. Recent years have seen an increase in the bariatric surgeries performed utilizing either an independent or a combination of restrictive and malabsorptive procedures. We review some complications of bariatric procedures more specifically, hypoglycemia and osteoporosis, the recommended preoperative assessment and then regular follow up, and the therapeutic options. Surgeon, internist, and the patient must be aware of the multiple risks of this kind of surgery and the needed assessment and follow up.

Key Words: Bariatric surgery, beta cell mass, dual energy X-ray absorptiometry, glucagon-like peptides, hypoglycemia, nesidioblastosis, osteoporosis

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The increase in the prevalence of overweight and obesity in recent years has seen an unprecedented rise so much so that they are now considered to have reached epidemic proportions in many parts of the world. The World Health Organization (WHO) projections[1] indicate that, by 2015, 2.3 billion adults will be overweight and 700 million will be obese.

In the United States alone, as per the National Health and Nutrition Examination Survey 2009–2010, about one third of US population approximately equal to 130 million adults[2] are obese. In Saudi Arabia itself the extent of the problem based on 2005 surveillance reported prevalence rates for overweight and obesity to be approximately 42.4% and 36%, respectively.[3] The body mass index (BMI) is a simple and effective method to define levels of obesity. The healthy weight ranges from 18.5 to 24.9 kg/m^2, overweight from 25 to 30 kg/m^2, whereas obesity is defined as BMI ≥30 kg/m^2 and classified into groups:[4]

- Class 1, BMI from 30 to 34.9 kg/m^2
- Class 2, BMI from 35 to 39.9 kg/m^2
- Class 3 (severe obesity), BMI ≥40 kg/m^2
- Class 4 (super obesity), BMI from 50 to 59.9 kg/m^2.

Obesity is a major health concern by itself and is associated with many fatal and nonfatal chronic conditions such as hypertension, hyperinsulinemia, diabetes mellitus, hypertriglyceridemia, low serum high-density lipoprotein (HDL) cholesterol concentration, hypercholesterolemia,[5] heart disease, hyperurecemia, gallstones, cancers, and stroke.[6] These obesity - associated comorbid conditions increase greatly with BMI >30 kg/m^2, increasing the risk of morbidity and mortality significantly and are estimated to increase the risk for premature death from all causes by 50%–100%.[7,8] The expected life span of a Caucasian male with a BMI >30 kg/m^2 is reduced by 9 years,[9] whereas that of individuals of BMI > 45 kg/m^2 is associated with a decrease of 13 years of life expectancy compared with a lean individuals (20–22 kg/m^2).[10] Although the increase in the number of cases of obese has levelled off, a marked increase in the number of cases with morbid obesity has been observed in recent years.[11]

The mainstay of treatment for obesity has been either medically through lifestyle modifications, dietary interventions, and pharmacotherapy or surgically through bariatric surgery. The poor short- and long-term outcomes of the presently applied medical modalities of treatment furthered the number of bariatric procedures. Bariatric surgery has become the most successful treatment for

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obesity in individuals who have failed at supervised medical weight loss, which achieves a significant and durable weight loss. The incidence of diabetes in the obese has been noted in many studies to have either reduced after surgery or become completely resolved in 77% of cases. The incidence of hyperlipidemia improved in 70% or more of patients, hypertension in 62%, and obstructive sleep apnea resolved in 86%. Gastroesophageal reflux symptoms demonstrated improvements with a complete or partial regression of Barrett's esophagus, and urinary stress incontinence episodes decreased by 47% in women who achieved 8% weight loss.[12,13] These figures roughly translate into a 29% reduction of obesity-related morbidity and mortality following surgery. In another cohort, deaths from all causes were reduced by 40%, from diabetes by 92%, from coronary disease by 56%, and from cancers by 60%.[14-16] The beneficial effect of the common bariatric procedures has been shown to occur via increased glucose and lipid metabolism and due to alterations in hormonal mechanisms controlling the body's hunger and satiety responses subsequently leading to an overall change in metabolism. Based on these findings, there has been a paradigm shift in the concept and aim of performing the bariatric surgery from interventions primarily directed to obesity, to the broader category of treating metabolic disorders, for example, type2 diabetes. This has raised the possibility that these procedures should be considered "metabolic," instead of surgeries intended for just weight reduction.[17] Recent years have seen an increase in laparoscopically performed bariatric surgery utilizing either an independent or a combination of restrictive and malabsorptive procedures. The optimal choice for the type of bariatric procedure depends on many factors that include goals of therapy, available local–regional expertise (surgeon and institution), patient preferences, and preoperative risk stratification.[18] Routinely employed techniques include (1) adjustable gastric band (LAGB), (2) biliopancreatic diversion (BPD) with duodenal switch (BPDDS), (3) sleeve gastrectomy (LSG), and (4) Roux-en-Y gastric bypass (RYGB).[19-23] The loss of initial body weight and the equivalent excess body weight loss differ with the type of procedure. Gastric banding results in an average loss of 20%–30% of initial body weight[24] equivalent to of 41%–54% of excess body weight loss,[19] sleeve gastrectomy 20%–30%, with equivalent loss of 45%–64%,[19,21] and RYGB approximately 35% with equivalent loss of 62%–75%, this loss being maintained at 10–14 years following surgery.[19-22] RYGB, the more invasive procedure, is associated with greater long-term success but higher short-term morbidity in comparison with the less invasive ones, such as LAGB. In April 2013 various regulatory bodies including the American Association of Clinical Endocrinologists (AACE), The Obesity Society, and American Society for Metabolic and Bariatric surgery have recommended bariatric surgery for (1) All patients with a BMI ≥40 kg/m^2 without coexisting medical problems, and (2) patients with a BMI ≥35 kg/m^2 with presence of one or more obesity-related comorbidities, including type 2 diabetes, hypertension, hyperlipidemia, obstructive sleep apnea, hypoventilation syndrome, debilitating arthritis, severe urinary incontinence, venous stasis, fatty liver disease, gastroesophageal reflux disease (GERD), or with considerably impaired quality of life.[18]

The choice of procedure is dictated by the degree of obesity or BMI. LAGB is the procedure of choice in individuals with a BMI >30 kg/m^2, whereas RYGB in those with BMI >35 kg/m^2. Besides improving the quality of life, bariatric surgeries have also shown to improve the psychiatric dysfunction seen in obese patients when compared with only minor and sporadic improvement in medically treated patients.[25]

COMPLICATIONS OF BARIATRIC SURGERY

Although significant improvement is seen with weight loss along with decrease in comorbid conditions, there is growing concern that bariatric surgery exerts negative effects on individuals' health. Due to the high volume of bariatric surgeries being performed routinely, safety prior to and after the surgery has become an issue of utmost priority. Many guidelines and criteria related to the accreditation of obesity management centers and careful monitoring of outcomes have thus been released.[18,26-28] The US Bariatric Outcomes Longitudinal Database reported 1-year complication rates of 4.6%, 10.8%, 14.9%, and 25.7% respectively, following LAG placement, LSG, RYGB, and BPD.[27] In the long term, the contribution of nutritional factors is also of vital importance as growing evidence shows that all bariatric procedures are capable of producing significant nutritional and metabolic abnormalities.[28] Numerous factors are known to increase the risk of morbidity and mortality with different procedures in patients undergoing surgery. These factors include (1) older age, (2) male gender, (3) very high BMI, (4) presence of coexisting chronic diseases,[29,30] (5) the qualification and experience of the surgeons, (6) qualification and experience of the center and facilities available therein,[31,32] and (7) surgeries using the open approach instead of the laparoscopic one.[33]

Complications that may arise include the immediate intraoperative, late, and metabolic complications. We focus on the nonsurgical aspects of perioperative aspect of the bariatric surgery patient, with special emphasis on hormonal and metabolic problems and management.

Short-term complications

The overall 30-day mortality postoperatively is shown to be less than 1% in general; however, it may range between 0.3% and 8%.[34-36] Wound infection, bleeding, deep venous thrombosis, and pulmonary embolism are the early

complications of the surgery,[32] whereas pulmonary embolism and surgical leak are the most common causes of death.[37,38]

Late complications

Late complications related to the surgical procedure include stomal stenosis, marginal ulcers, cholelithiasis, internal and incisional hernias, short bowel syndrome, nutritional deficiencies, and dumping syndrome.[19,21,22,24,39]

Metabolic complications

Metabolic complications of bariatric surgeries include metabolic acidosis, and/or alkalosis, electrolyte abnormalities including low calcium, potassium, magnesium, sodium, and phosphorus that may cause arrhythmias and/or myopathies. Nutritional abnormalities in the form of fat-soluble vitamin deficiencies involving A, D, E, and K, iron and folic acid deficiency, negative calcium balance, and vitamin D deficiency causing secondary hyperparathyroidism, oxalosis, kidney stones, thiamine deficiency, vitamin B12 deficiency, increased bacterial overgrowth causing nocturnal diarrhea and abdominal distension, have been documented.[37,40-45] Periodic measurement of electrolytes, minerals, and vitamins, and replacing deficiencies if any will result in better management of metabolic complications.

In this article, we will review more specifically the hypoglycemia and osteoporosis after bariatric surgery in greater details, as these are the most severe and difficult to manage.

Hypoglycemia

Hypoglycemia is increasingly being recognized as a complication of gastric bypass surgeries. Relative risk of hypoglycemia increases sevenfold in the postgastric bypass population,[46] and the frequency of asymptomatic documented hypoglycemia after oral glucose tolerance test reached 30% among postgastric bypass patients.[47] A median time from surgery to development of symptoms was observed to be 2.7 years (few weeks after surgery to 5 years).[48] The pathophysiology of hypoglycemia remains controversial; dumping syndrome, an increase in beta cell mass, alteration of beta cell function, and other factors not related to beta cell were all suggested as possible mechanisms.[34,47-52] Decreased storage capacity of the stomach and the lack of rate-limiting step to food delivery, directly into the small intestine results in an exaggerated release of insulin, eventually leading to reactive hypoglycemia. Dumping syndrome can occur in up to 50% of patients after surgery[22] along with an increase in insulin and C-peptide levels after meals.[53,54] Levels of other hormones and peptides, namely, glucagon-like peptides (GLP-1), glucose-dependent insulinotropic polypeptide (GIP), peptide YY (PYY), cholecystokinin (CCK), ghrelin, gastrin, somatostatin, pancreatic polypeptide (PP), amylin, and leptin were studied in relation to this. The studies found high concentrations of GLP-1 and PYY and a lower level of ghrelin, leptin, acylation-stimulating hormone, and visfatin.[54,55] Amplification of GLP-1 levels were attributed to the anatomical alteration of the gastrointestinal tract, resulting in dysfunction as well as hypertrophy of the beta cells causing overstimulation and prolonged secretion of insulin and a decrease in glucagon release. This proposed mechanism is one of the more accepted theories for the development of hypoglycemia in these patients. In addition, increased weight loss following surgery along with an increase in insulin sensitivity may additionally contribute to development of hypoglycemia. Service *et al.* and Patti *et al.* described cases of noninsulinoma pancreatogenous hypoglycemia syndrome (NIPHS) that presented with symptoms of postprandial neuroglycopenia secondary to hyperinsulinemic hypoglycemia. In their series, the diagnosis of NIPHS was confirmed pathologically by the presence of diffused islet hypertrophy or nesidioblastosis in pancreas.[56,57] Nevertheless, besides NIPHS other differential diagnosis of hypoglycemia, including insulinoma, factitious insulin or sulfonylurea administration, adrenal insufficiency, insulin autoimmune hypoglycemia, and tumor-producing insulin-like hormone or peptides must be considered.[58]

Clinical presentation varies from asymptomatic to mild neurogenic, to more severe neuroglycopenic symptoms. The neurogenic or adrenergic symptoms include tremor, tachycardia, diaphoresis, anxiety, and a sensation of hunger. The neuroglycopenic symptoms include weakness, tiredness, or dizziness, inappropriate behavior, difficulty with concentration, confusion, blurred vision, and in severe cases, it may lead to coma and death. Most of the patients tend to have mild adrenergic post-prandial symptoms, whereas a few may have severe and refractory symptoms that require pharmacological and/or surgical interventions.[59] The diagnosis is classically based on the presence of hypoglycemic symptoms that are ameliorated by taking glucose, and a laboratory-based documentation of low plasma glucose (<55 mg/dL) (Whipple's triad). Hypoglycemia associated with dumping syndrome presents soon after surgery with vasomotor symptoms followed by symptoms of hypoglycemia 2–3 h after the meal.[60] In contrast to dumping syndrome, patients with autonomous hyperinsulinemia, arising due to alterations in beta cell mass and/or function, present late, one year or more postsurgery, without the early vasomotor symptoms.[61]

Patients with mild adrenergic post-postprandial symptoms can be managed by dietary advises without any further workup.[59] AACE/TOS/ASMBS recommends that patients presenting with postprandial hypoglycemic symptoms severe or unresponsive to nutritional manipulation should undergo an evaluation to differentiate NIPHS from factitious or

iatrogenic causes, dumping syndrome, and insulinoma. Cases of insulinoma have also been reported to occur postbariatric surgeries[62] with 10% of cases presenting with symptoms of postprandial hypoglycemia.[63] Supervised prolonged fasting (72 h fasting test) is usually indicated to rule out insulinoma. Additionally, the use of continuous glucose monitoring can also be employed to determine low interstitial fluid glucose at the time of symptoms, whereas provocative testing to induce hypoglycemia using oral glucose tolerance test (OGTT) and mixed-meal studies are less commonly used techniques. Further confirmatory tests using imaging studies such as triple-phase spiral computed tomography, magnetic resonance imaging, and transabdominal ultrasound of the pancreas, should also be undertaken. Additionally, selective arterial calcium-stimulation test is employed as the last step in the evaluation of these patients to localize the area of hypersecretion.[64]

The modality of treating hypoglycemia postbariatric surgery depends on the severity of the symptoms and the response of the patient to the initial dietary recommendations. Patients with early postoperative mild adrenergic post-postprandial symptoms can be managed initially with dietary advises including frequent, small meals and low-carbohydrate diet.[59] For resistant cases, or presenting later postoperative and those with severe hypoglycemia require further evaluation and therapy. In patients who do not respond well to the dietary modification pharmacological therapy can be initiated with acarbose, diazoxide, verapamil, and somatostatin.[65-67]

Acarbose, which is an α-glucosidase inhibitor (AGI), acts by reducing the glucose load in the jejunum thereby inhibiting the main stimulus for GLP-1 secretion. Studies in patients with dumping syndrome showed that acarbose produced a fivefold decrease in postprandial GLP-1 levels,[68] whereas in post-RYGBP patients an ingestion of acarbose 15 mins after a meal reduced serum insulin and GLP-1 levels and ameliorated symptoms of hypoglycemia.[69] The common side effects of this treatment include diarrhea, abdominal pain, and increased frequency and intensity of flatulence.

Diazoxide is the firstline drug for management of hyperinsulenimic hypoglycemia. It acts by opening the potassium ATP channels and preventing glucose-stimulated insulin secretion. Diazoxide responsiveness is determined by (1) appropriate fasting tolerance for age; (2) feed volume and frequency normal for age; and (3) normal blood sugar levels at the end of the fast. Patients can have hypotension, anxiety, dizziness, headache, insomnia, anorexia, and diarrhea as side effects of this medication.[66]

Verapamil is a calcium channel blocker that acts by inhibiting calcium ion from entering pancreatic cells and therefore inhibits insulin secretion. Common side effects include headache, gastrointestinal disturbance, peripheral edema, and hypotension.[66]

Octreotide, a somatostatin analog, is the other drug that can be used for the treatment of hypoglycemia. Besides its inhibiting action on growth hormone (GH), glucagon and insulin it also inhibits release of several gastrointestinal hormones, including serotonin, gastrin, vasoactive intestinal peptide, secretin, motilin, and pancreatic polypeptide. The criteria for responsiveness to octreotide are similar to those for diazoxide, and its common side effects include sinus bradycardia, fatigue, headache, abdominal pain, nausea, diarrhea, and cholelithiasis.[70]

In cases where medical treatment fails, surgical intervention is advocated through reversal of the bariatric procedure, employing postoperative feeding to the bypassed proximal gut by gastrostomy tube followed by partial pancreatic resection in refractory cases.[71]

Metabolic bone disease

Another complication of increasing concern arising as a consequence of bariatric surgery is metabolic bone disease that leads up to osteoporosis and osteoporotic fracture. Overweight and obesity have been considered in the past to exert a protective effect on the bones due to the loading effect. Both conditions in fact are associated with increased incidence of vitamin D (60%–86%) and calcium deficiencies with elevated parathyroid hormone (PTH) levels placing them at risk of low bone mass and metabolic bone disease, including osteoporosis.[72] In their study of 279 morbid obese Carli *et al*. found that 60% of cases were deficient in vitamin D and 48% had secondary hyperparathyroidism.[73] Increased weight due to mechanical loading effect, negatively affects the skeleton by accelerating bone loss, thereby increasing bone fragility.[74] The pathophysiology of bone disease in the obese patients is multifactorial ranging from inadequate nutrition due to chronic dieting practices, lack of physical activity, and increased sequestration of vitamin D within the adipocytes. Bone metabolism depends normally on an adequate dietary intake of calcium and phosphorus regulated by parathyroid hormone, vitamin D, and FGF23. It is also affected by a complex network of local neuronal and hormonal factors, including gastrointestinal hormones whose role has been proposed based on evidence that bone resorption dramatically falls after a meal.

The impact of major weight loss on bone metabolism after bariatric surgery was until recently considered to be the sole result of a combination of mechanical and nutritional effects. The notion changed with recent insights, which showed an intricate and complex interplay between the signaling factors of gut, bone, and fat tissue. This interplay

forms the basis of convergence of bone and energy metabolism utilizing a third neurohormonal mechanism regulating bone turnover through adipokines; leptin and adiponectin, gonadal steroids,[75] and gut-derived hormones; serotonin, ghrelin, PYY, GIP, and GLP-1, GLP-2.[76] GIP's receptor, glucose-dependent insulinotropic polypeptide receptor (GIPR), is expressed in bone tissue and its deficiency in animals reportedly led to increased bone resorption with a pronounced reduction in the degree of mineralization of bone matrix.[77]

Alterations of the adipokines and incretins after bariatric surgery has significant negative effect on bone metabolism and health[78] and further compounds the already existing metabolic bone disease in obese individuals. The degree of bone loss or disease varies with the type of surgery undertaken. Surgeries employing malabsorptive techniques such as RYGB, bypass the primary absorption sites for vitamins and minerals, that is, the duodenum and proximal jejunum resulting in deficiencies of key osteogenic factors such as calcium that is actively absorbed from the proximal foregut, and vitamin D that requires bile acids and pancreatic secretions for optimal absorption. Restrictive techniques, such as sleeve gastrectomy, on the other hand reduce the total amount of gastric acid required for lowering the pH for optimal absorption of other essential vitamins and minerals. RYGB and biliopancreatic diversion can thus cause malabsorption of calcium (25%–50%) and vitamin D (>50%) and other nutrients—protein, folate, iron, magnesium, thiamine, B12, and vitamin A—that are essential for bone health. In turn, the intensity or degree of these micronutrient deficiencies varies depending on the length of intestine bypassed. Numerous reported cases clearly show the risk of metabolic bone disease occurring from as early as 8 weeks until 32 years after bariatric surgery.[79] The percentage of bone lost also 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.[80] After bariatric surgery, many patients rapidly lose 50–100 kg of their weight; this weight loss combined with severely restricted oral intake of all the nutrients that includes proteins, calcium, and vitamins predisposes them to furthering the development of metabolic bone disease.[81]

Alterations in the levels of the adipokines also affect the outcomes of the surgical procedures. After RYGP, adiponectin levels were shown to increase while leptin levels decreased in proportion to loss of total body fat;[82] and GIP levels, fasting and postprandial, were found to be reduced only in diabetic patients. The levels of incretins, total GLP-1 and PYY, increased after RYGB, BPD,[83] LAG,[84] and LSG[85] as early as 1–6 weeks postsurgery and remained elevated for at least 1 year,[86,87] whereas the pancreatic polypeptide response decreased. Nannipieri *et al*. found that at 1-year, levels of PYY were increased, and pancreatic, amylin, ghrelin, and GLP-1 were reduced in RYGB and LSG except for PP and amylin, which were increased and unchanged in LSG group.[88] Ghrelin levels were shown to decrease at 1 and 12 months after RYGB surgery with subsequent improvements after 12 months postsurgery correlating strongly with weight loss and increased insulin sensitivity.[89] The effect of surgery on ghrelin levels remains uncertain, although few studies have reported postoperative decreases in its levels in RYGB and gastric sleeve.[90] In addition to gut hormones, locally synthesized hormones such as the estrogens, known to impact bone health were also altered post operatively. As weight decreases and adipose stores are depleted, the levels of estrogen decrease in both men and women resulting in decreased impact of estrogen on bone. Estrogen may also affect bone metabolism by direct effects on vitamin D and calcium metabolism.[91]

Von Mach *et al*. in their study reported a significant decrease in the bone mineral content of patients undergoing RYGB accompanied by significant increases in levels of markers of bone turnover, namely, urinary deoxypyridinoline and in

Table 1: Association of Clinical Endocrinologists, the Obesity Society, and American Society for Metabolic and Bariatric Surgery (AACE/TOS/ASMBS) recommendations of periodic laboratory tests postbariatric surgery

3-6 months postsurgery	Hematological: CBC, platelets; Biochemical: serum electrolytes, glucose, iron studies, ferritin phosphorus, vitamin B12, liver function, lipid profile, albumin, prealbumin, and RBC folate
6-12 months postsurgery	Biochemical: Vitamins A, 25-hydroxy vitamin D, K1, international normalized ratio, and intact parathyroid hormone and annually
Annually	Biochemical: 24-h urine calcium, citrate, uric acid, and oxalate. Selenium, Urine N-telopeptide, zinc and measurements of osteocalcin and carnitine can be done as needed

Table 2: American Association of Clinical Endocrinologists recommendation for replacement of calcium and vitamin D postbariatric surgery

Vitamin D	
	Supplement if the level of 25-hydroxyvitamin D falls below 30 ng/mL, supplement with vitamin D3 or D2 as indicated by serum levels in oral doses of 50,000 IUs 1-3 times/week to reach sufficiency, followed by additional supplementation of 2000-10,000 IU/day as needed to attain and maintain serum levels of >30 ng/mL (≥75 nmol/L)
Calcium	
	Natural calcium from diet plus supplementation with oral calcium to reach total of 1500 to 2000 mg/day

serum osteocalcin 24 months after RYGB, suggesting both, increased bone resorption and a parallel decreased bone formation.[92]

The investigations generally should include serum and urine calcium, 25-hydroxyvitamin D, alkaline phosphatase, and serum intact PTH levels. An increase in serum intact PTH level is indicative of negative calcium balance or a vitamin D deficiency (or both), whereas elevations of bone-specific alkaline phosphatase level, which is indicative of increased osteoblastic activity and bone formation, are often the initial abnormalities. Complications may be seen as early as 8 weeks after bariatric surgery in the form of secondary hyperparathyroidism and osteomalacia.[93,94] 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. The American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic and Bariatric Surgery AACE/TOS/ASMBS guidelines[95] recommends performing certain laboratory tests every three months in the first year after surgery, and every 3–12 months thereafter, depending on symptoms [Table 1].

Dual-energy X-ray absorptiometry (DXA) is accepted as a marker of bone strength, fracture risk, and an important tool that can be used to assess the bone mineral density of individuals and to identify the presence of osteoporosis. DXA of the hip and spine is a common modality used to diagnose osteoporosis in men over age 50 years and postmenopausal women, whereas in younger patients the levels serve as baseline measurement for future comparison. DXA assessment should be carried out in both men and women prior to and after bariatric surgery. The markers of bone resorption also showed significant elevations in osteocalcin and bone alkaline phosphatase levels.[96] One year following the RYGB, bone mass density declined at femoral neck by 9.2%–10.9%, at total hip by 8%–10%, and in lumbar spine by 3.6%–7.4%.[92] Exclusively, restrictive procedures such as gastric banding formerly considered not to impact bone health and metabolism are now known to place patients at risk for metabolic bone disease due to the inadequate intake of calcium and vitamin D in the immediate postoperative period. Pugnale *et al*. found that 48% of patients had more than 3% statistically significant bone mineral reduction 12 months after undergoing gastric banding.[97] Seventy percent of patients undergoing malabsorptive procedure also developed osteomalacia, with elevations in markers of bone resorption as early as 8 weeks after bariatric surgery.[94]

Nakamuro *et al*. in their cohort with a majority of middle-aged morbidly obese (BMI was 49 ± 8 kg/m^2) female participants followed up for a median of 7.7 years, documented a 2.3-fold increase in the relative risk for any fracture at the common osteoporotic sites (hip, spine, wrist, and humerus). Incidences of fractures were seen early after surgery till as late as 5 years.[96]

Postoperatively, many bariatric patients require chewable or liquid supplements to facilitate adequate absorption. The AACE-recommended treatment guidelines for these patients include postsurgery replacement of calcium and vitamin D [Table 2].

In patients who require pharmacological therapy for osteoporosis, treatment can be initiated, if the vitamin D and calcium levels are normal or after therapeutic correction of insufficiency, with the use of bisphosphonates. In cases where therapy is indicated, intravenously administered bisphosphonates should be used, as there are concerns about inadequate oral absorption and potential anastomotic ulceration with orally administered bisphosphonates.[98]

CONCLUSION

Bariatric surgery is the most effective therapeutic option for obese patients; however, it carries substantial risks including procedure-related complications, malabsorption, and hormonal disturbance. Preoperative assessment and then regular follow up postoperatively by an endocrinologist or obesity-specialized internist is crucial. In addition, patients must be aware of the multiple risks and the need for regular assessment and follow up by specialized physicians. Dietary instructions and vitamins and mineral supplements must be initiated early on to prevent major consequences.

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