Bariatric surgery and secondary hyperparathyroidism; a mini-review

Ali Azarpey1, Mahshid Imankhan2, Sina Neshat3

Abstract
Bariatric surgery is a type of weight loss surgery that is commonly used to treat obesity. However, this surgery can also affect the body's calcium and PTH metabolism, leading to the development of secondary hyperparathyroidism (SHPT). Several factors contribute to the development of SHPT after bariatric surgery. Malabsorption of calcium due to reduced intestinal surface area, decreased intake of calcium-rich foods, and altered vitamin D metabolism play a significant role. The loss of weight-bearing adipose tissue can also disrupt the balance between bone formation and resorption, leading to increased bone turnover and calcium release from the skeleton. The management of SHPT after bariatric surgery involves a multidisciplinary approach. Calcium and vitamin D supplementation is essential to correct deficiency and maintain bone health. However, achieving optimal calcium and vitamin D levels can be challenging due to malabsorption issues and the need for higher supplementation doses. In some cases, pharmacological interventions such as calcimimetics or PTH analogs may be required to control PTH levels. However, these medications should be used cautiously due to limited data on their safety and efficacy in the bariatric surgery population. Prevention of SHPT is an important aspect of managing patients undergoing bariatric surgery. Nutritional counseling and regular monitoring of calcium, vitamin D, and PTH levels can help identify and address deficiencies early on. Additionally, using procedures that preserve the duodenum and proximal jejunum, such as duodenal switch or biliopancreatic diversion with duodenal switch, may reduce the risk of developing SHPT.

Keywords: Bariatric surgery, Sleeve gastrectomy, Osteomalacia, Osteoporosis, Secondary hyperparathyroidism, Gastric bypass, Vitamin D, Bone health, Parathormone

Introduction
Bariatric surgery, which includes the procedures like gastric bypass, sleeve gastrectomy, and adjustable gastric banding, has become increasingly popular as a treatment for obesity (1). While bariatric surgery is effective in achieving weight loss and improving overall health outcomes, it can also have negative effects on calcium and parathyroid hormone (PTH; parathormone) metabolism, leading to the development of secondary hyperparathyroidism (SHPT) (2-4). SHPT is a common complication following bariatric surgery, especially in procedures that bypass the duodenum and proximal jejunum, where calcium absorption predominantly occurs. The exact prevalence of SHPT after bariatric surgery varies depending on the procedure and the duration of follow-up, but studies have reported rates ranging from 13% to 71% (3,4).

Search strategy
For this review, we searched PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ), and Embase, using different keywords such as bariatric surgery, sleeve gastrectomy, osteomalacia, osteoporosis, secondary hyperparathyroidism, gastric bypass, vitamin D, bone health, calcium, malabsorption, and parathormone.

Development of SHPT following bariatric surgery
Several factors contribute to developing SHPT after bariatric surgery. First, the surgery can cause a reduction in the surface area of the intestines, leading to decreased absorption of calcium (5,6). Additionally, patients may have decreased intake of calcium-rich foods after surgery. The surgery also alters vitamin D metabolism, which is important for calcium absorption. Lastly, losing weight-bearing adipose tissue can disrupt the bone formation and resorption balance, leading to increased bone turnover and calcium release from the skeleton (7,8). The symptoms of SHPT after bariatric surgery can include low levels of calcium in the blood, high levels of PTH, and increased markers of bone turnover. In severe cases,
Implication for health policy/practice/research/medical education

Secondary hyperparathyroidism (SHPT) is a common complication after bariatric surgery, particularly in procedures that bypass the duodenum and proximal jejunum. Managing SHPT requires a multidisciplinary approach, including appropriate supplementation, regular monitoring, and potential use of medications. Preventing SHPT through optimal surgical techniques and early identification of deficiencies is crucial for improving patient outcomes. Further research is needed to better understand the underlying mechanisms and optimal management strategies for SHPT after bariatric surgery.

Prevention of SHPT after bariatric surgery

Prevention is an important aspect of managing SHPT after bariatric surgery. This involves nutritional counseling and regular monitoring of calcium, vitamin D, and PTH levels to identify deficiencies early on. Additionally, the use of surgical techniques that preserve the duodenum and proximal jejunum may help reduce the risk of developing SHPT.

Conclusion

SHPT is a common complication following bariatric surgery, especially in procedures that bypass the duodenum and proximal jejunum. The management of SHPT requires a multidisciplinary approach, including appropriate nutritional supplementation, regular monitoring, and potential pharmacological interventions. Prevention of SHPT through optimal surgical techniques and early identification of deficiencies is crucial for improving patient outcomes. Further research is needed to better understand the pathophysiology and optimal management strategies for SHPT after bariatric surgery.