

## Case Report

# Severe Hypocalcemia due to Vitamin D Deficiency after Extended Roux-en-Y Gastric Bypass

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Vitamin D deficiency is a well-known comorbidity of obesity that can be exacerbated after bariatric surgery and can predispose the patient for hypocalcemia. Vitamin D and calcium doses to prevent and treat vitamin D deficiency after weight loss surgery are not well defined. We describe a patient who developed severe hypocalcemia due to vitamin D deficiency 5 years after an extended Roux-en-Y gastric bypass for a type II obesity. No precipitating factors were present and malabsorption induced by the bypass was considered to be the main causative factor. High doses of vitamin D and calcium were needed to reach and maintain normal calcium and vitamin D concentrations. This case emphasises the importance of routine screening for vitamin D deficiency in obese individuals and reflects that while consensus does not exist regarding optimal dosage, vitamin D replacement should be titrated based on calcidiol levels.

## 1. Introduction

Hypovitaminosis D and secondary hyperparathyroidism are associated with morbid obesity. In contrast with many obesity-related comorbid conditions, this vitamin D deficiency is not corrected by obesity surgery and may even be exacerbated, especially when the obesity surgery involves malabsorption [1, 2]. We report a patient who underwent a Roux-en-Y gastric bypass (RYGB) and who later developed severe, symptomatic hypocalcemia due to severe vitamin D deficiency.

## 2. Case Report

A 69-year-old woman was referred to our hospital with severe hypocalcemia detected during routine analyses for anemia.

Five years earlier she had had a RYGB (common channel 80 cm length and Roux limb 250 cm length) in another centre as treatment for type II obesity associated to type 2 diabetes mellitus and hypertension. No postsurgical follow-up was performed.

After surgery, her body mass index decreased from 37 Kg/m<sup>2</sup> to 24 Kg/m<sup>2</sup> and her diabetes remitted. She began to pass 4-5 stools per day and her primary care doctor diagnosed an iron deficiency anemia refractory to oral supplements.

Four months before she was first visited at our centre, she began to experience general weakness, muscle pain, and cramps. Her family doctor performed an ambulatory densitometry which showed a bone pattern compatible with osteomalacia. Perioral paraesthesias appeared some weeks later but she denied tetania or hand cramping.

At first visit at our centre she was taking irbesartan, iron, and potassium supplements and a multivitamin complex containing 500 IU of cholecalciferol and 125 mg of calcium, all prescribed in the previous months by her family doctor. The patient did not smoke nor drink.

Physical examination revealed positive Chvostek's and Trousseau's signs. Serum total calcium was 1.47 mmol/L (normal range 2,15–2,55 mmol/L), serum phosphate was 1.21 mmol/L (normal range 0,87–1,45 mmol/L), serum creatinine was 82  $\mu$ mol/L (normal <80  $\mu$ mol/L), and alkaline phosphatase was 239 U/L (normal range 35–110 U/L) with

normal levels of AST, ALT, and GGT. Calcidiol level was 19.6 nmol/L (normal range 80–275 nmol/L) and parathyroid hormone (PTH) level was 9.1 pmol/L (normal range 1.3–1.8 pmol/L). We prescribed 1500 mg/day calcium carbonate and 16,000 IU of 25-hydroxvitamin D per week.

Four days later, the patient required hospitalization as symptoms did not improve and blood tests showed persistent hypocalcemia with calcium levels of 1.37 mmol/L. Oral calcium intake was increased to 3 gr per day and 25-hydroxyvitamin D was prescribed every 48 hours. A calcium gluconate infusion was started. We also administered 1500 mg of oral magnesium as serum levels were 0.65 mmol/L (normal range 0.65–1.05 mmol/L). Hypocalcemic symptoms improved over the next 2–3 days but the calcium gluconate infusion was required for a further 7 days before calcium levels became stable with oral supplementation alone. At discharge, ten days after admission, her serum calcium level was 1.86 mmol/L and she was taking calcium carbonate 3 gr per day, 25-hydroxvitamin D 16,000 IU per day and magnesium sulphate 1,500 mg per day.

At follow-up, one month later, calcidiol and PTH had reached 123 nmol/L and 14.7 pmol/L, respectively, calcium level was 1.64 mmol/L and the patient was asymptomatic. Medications were titrated to calcium carbonate 3000 mg per day, 25-hydroxyvitamin D 16,000 IU every 72 hours, and magnesium sulphate 1000 mg per day. At five-month follow-up on this regimen, the patient was stable with a serum calcium of 2.16 mmol/L.

### 3. Discussion

This case report illustrates the risk of severe hypocalcemia and vitamin D deficiency following RYGB and the need for high doses of calcium and vitamin D to prevent and treat these deficiencies.

The risk of hypocalcemia in patients submitted to RYGB may be explained by two mechanisms, the first related to calcium absorption. Calcium is mainly absorbed in the duodenum but when a RYGB is performed it is absorbed through a less efficient mechanism in the distal small bowel. This leads to a certain degree of calcium malabsorption, especially if diet calcium content is low. The second mechanism involves fat soluble vitamin malabsorption due to impaired mixing with bile salts. It causes a vitamin D deficiency which further impairs calcium absorption, thereby increasing the risk for hypocalcemia [3–5].

Despite the changes in calcium and vitamin D metabolism induced by RYGB, few cases of hypocalcemia have been reported, and all of them have presented with a precipitating factor such as thyroidectomy with secondary hypoparathyroidism [6]. We did not find any precipitating factor in our patient. One possible explanation may be the presence of a long Roux limb (250 cm), which in contrast with the standard technique, bypasses a greater part of the small bowel. Extended bypasses have proven to be more effective in terms of weight loss in individuals with BMI > 50 Kg/m<sup>2</sup> and, although their effects on calcium metabolism have not been well documented, it is plausible that the

greater malabsorption they produce may increase the risk for hypocalcemia [7].

Taking into account the frequency of vitamin D deficiency after RYGB, most practitioners routinely prescribe vitamin D and calcium supplements after bariatric surgery. Current recommended doses are 400–800 IU of cholecalciferol and 500–2000 mg of calcium per day, [8–10]. In our patient, vitamin D and calcium supplements were not prescribed until several years after surgery, and the dose prescribed was clearly insufficient to prevent vitamin D deficiency. Furthermore, the calcium 1500 mg per day and 25-hydroxvitamin D 16,000 IU per week that we prescribed at first visit at our centre failed to control hypocalcemia. An infusion of calcium gluconate was needed over seven days until oral regimens were effective in controlling these deficiencies. Our case reflects the difficulty in finding the appropriate dose of vitamin D and calcium needed to prevent and treat vitamin D deficiency following bariatric surgery. It also shows that high doses are often necessary to achieve normal vitamin D and calcium levels. Doses of 5000 IU per day have been found to be safe and necessary in many patients following RYGB [11]. Nevertheless, when malabsorption is suspected, as in the case of extended by-passes, doses as high as 50,000 IU of cholecalciferol per day can be safely administered [8, 9]. In our patient, high doses were required to maintain an adequate vitamin D status, so 16,000 IU of 25-hydroxvitamin D every 72 h were maintained during follow-up and could not be reduced. Taking into account that 25-hydroxvitamin D is considered to have a biological potency 10 times higher than cholecalciferol, our patient finally needed the equivalent of 53,000 IU of cholecalciferol per day [12].

In conclusion, vitamin D deficiency is frequent in the setting of RYGB and can cause severe hypocalcemia. Screening for this comorbidity is therefore essential and high doses of vitamin D and calcium may be needed. As there is no consensus regarding optimal dosage, vitamin D replacement should be titrated based on calcidiol levels.

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