Obesity Research & Clinical Practice (2016) **10**, 344—347

CASE REPORT

Recalcitrant hypocalcaemia in a patient with post-thyroidectomy hypoparathyroidism and Roux-en-Y gastric bypass

Gonzalo Allo Miguel[a,*], **Elena García Fernández**[a], **Guillermo Martínez Díaz-Guerra**[a], **María Ángeles Valero Zanuy**[a], **Ana Pérez Zapata**[b], **Felipe de la Cruz Vigo**[b], **Federico Hawkins Carranza**[a]

[a] *Endocrinology Service, 12 de Octubre University Hospital, UCM, Spain*
[b] *General Surgery Service, 12 de Octubre University Hospital, UCM, Spain*

Received 18 March 2015; received in revised form 16 August 2015; accepted 4 September 2015

KEYWORDS
Hypocalcaemia;
Obesity;
Roux-en-Y gastric bypass;
Total thyroidectomy;
Hypoparathyroidism

Summary
Introduction: Roux-en-Y gastric bypass (RYGB) places patients at an increased risk of hypocalcaemia due to the reduction in calcium absorption (because the procedure bypasses the duodenum and jejunum) and vitamin D deficiency. Subsequent thyroid surgery increases the risk of severe hypocalcaemia due to potential post-operative hypoparathyroidism. Only a few cases have been published before of this type of treatment-challenging hypocalcaemia.
Clinical presentation: We report the case of a 31-year-old woman with a previous RYGB, who suffered severe and symptomatic chronic hypocalcaemia after total thyroidectomy. She required aggressive therapy with oral calcium and calcitriol and frequent calcium infusions, but there was no improvement in serum calcium level. Due to the lack of response to standard therapy, teriparatide treatment was started (first with subcutaneous injections and thereafter with a multipulse subcutaneous infusor) but the results were disappointing. As there was no response to different medical treatments, reversal of RYGB was performed with no complications and a subsequent sustained increase in serum calcium level.
Conclusions: This case shows that patients with postoperative hypoparathyroidism and RYGB have increased risk of severe recalcitrant symptomatic hypocalcaemia.

* Corresponding author at: Endocrinology Service, 12 de Octubre University Hospital, Avenida Córdoba S/N, 28026 Madrid, Spain. Tel.: +34 696092310.
E-mail address: gonzaloallo.endo@gmail.com (G. Allo Miguel).

http://dx.doi.org/10.1016/j.orcp.2015.09.001

In our case teriparatide was ineffective but, as this is the first patient reported, more results are needed to evaluate properly the effect of teriparatide in this multifactorial hypocalcaemia. Reversal of RYGB should be considered when medical therapy has failed, because surgery restores an adequate absorption of calcium and vitamin D from previously bypassed duodenum and proximal jejunum.

Introduction

High prevalence of obesity [1] and failure of conservative treatments have led to an increase of bariatric procedures [2]. Low vitamin D and secondary hyperparathyroidism are common findings in obese patients [3]. Vitamin D deficiency is not resolved after bariatric surgery and may even worsen [4,5], especially if the procedure induces malabsorption, as in RYGB. Also, in RYGB, calcium absorption is reduced because the procedure bypasses the duodenum and jejunum (main sites for calcium absorption), placing patients at increased risk of hypocalcaemia [6,7]. We report the case of a patient with a previous RYGB, who suffered chronic symptomatic hypocalcaemia after total thyroidectomy.

Case report

A 21-year-old woman with morbid obesity [body mass index (BMI) 49.5 kg/m^2] underwent RYGB surgery in May 2003. Eight years later her BMI was 27.3 kg/m^2. She was then diagnosed of Graves' disease, with severe orbitopathy that persisted active despite high-dose carbimazole and specific treatment (rituximab, prednisone and botulinum toxin). Therefore, she was referred to thyroidectomy. Before surgery, serum analysis showed: albumin-corrected calcium 9.1 mg/dL (8.4—10.2 mg/dL); parathyroid hormone (PTH) 63.3 pg/mL (15—51 pg/mL); 25(OH)-vitamin D 20.5 ng/mL (15—55 ng/mL). During the procedure, all parathyroid glands were preserved. Histological examination only revealed normal thyroid tissue. At discharge, corrected calcium level was 8.1 mg/dL, so a regimen of oral calcium carbonate and calcitriol was started.

One week later, she returned to the emergency room with muscle cramps (more pronounced in legs) and positive Chvostek sign. Corrected calcium level was 6.3 mg/dL. Intravenous calcium gluconate infusion was started, improving serum corrected calcium level to 7.8 mg/dL. At discharge oral calcium carbonate and calcitriol doses were increased. During the initial monitoring the patient was seen in several occasions because she had clinical evidence of hypocalcaemia recurrence. Oral treatment doses were repeatedly increased but even the highest dose (12 g of calcium carbonate and 8 μg of calcitriol per day) neither improved the symptoms nor adequately increased serum calcium levels.

Six months later she was referred to our bone and calcium metabolism unit because corrected calcium level had severely decreased (5.2 mg/dL). Intravenous calcium gluconate was started to correct calcium level (7.4 mg/dL). Physical examination only revealed positive Chvostek and Trousseau signs. The rest of the serum analysis showed: PTH 12.3 pg/mL; 25(OH)-vitamin D 23.5 ng/mL; phosphorus 4.5 mg/dL (2.3—4.6 mg/dL); magnesium 2.24 mg/dL (1.5—2.3 mg/dL). We supervised medication-intake confirming adequate compliance.

Our presumptive diagnosis was multifactorial hypocalcaemia: post-thyroidectomy hypoparathyroidism plus calcium and vitamin D malabsorption due to RYGB. We developed a non-standardised test (based on Pietras et al. [8]) to evaluate the absorption of oral calcium and calcitriol. A dose of 3 g of oral calcium carbonate and 1 mcg of calcitriol were given orally to the patient (baseline serum values: 7.4 mg/dL of corrected calcium and 47 pg/mL of calcitriol). Four hours later the increase in serum levels was lower than expected in both cases (corrected calcium was 7.5 mg/dL and calcitriol was 53 pg/mL). To evaluate fat malabsorption we use Van de Kamer's method, but it had to be stopped due to significant decrease of corrected calcium (from 7.3 to 6.4 mg/dL) after 48 h. Based on this, we started low-fat diet. Also, we changed half of calcium carbonate dose to calcium citrate, because its better absorption [9] (total dose: 12 g/day). We could not provide all the oral calcium treatment as citrate (the patient's refused because they were difficult to swallow). With these measures, corrected calcium remained stable around 7.5 mg/dL during 3 days so she was discharged.

Symptoms began to worsen after several weeks and corrected calcium got as low as 6.1 mg/dL so she was admitted. After patient's consent,