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Severe Refractory Hypocalcemia after Parathyroidectomy for Hyperparathyroidism and Parathyroid Adenoma in a Patient with Prior Bariatric Surgery: A Rare Case Report

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Abstract Hypocalcaemia is a common but transient complication post Parathyroid surgery. In patients who have gone through bariatric surgery including Roux-En-Y the risk increases further. We present a challenging case report of a patient with history of RYGB who underwent parathyroidectomy and consequently developed refractory hypocalcaemia. Hypocalcaemia was managed conservatively initially but subsequently required higher doses of both intravenous and oral calcium along with teriparatide. Patients post Bariatric surgery including those who have undergone Roux-en-Y who are proceeding with Parathyroid procedures should be followed up closely for hypocalcaemia.

Keywords: *Roux-en-Y, post parathyroidectomy, hypocalcaemia, refractory, teriparatide, bariatric surgery*

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1. Introduction

Obesity affects over 300 million individuals worldwide, and many of them are surgically treated. Roux-en-Y gastric bypass (RYGB) is a bariatric procedure aimed at weight reduction in morbidly obese patients. It bypasses the anatomical sites for calcium and vitamin D absorption, thus placing patients at risk for altered calcium homeostasis. [1] Gastric surgery reduces gastric acid therefore calcium may not be effectively absorbed. Hypothalamus-pituitary-parathyroid axis is affected by the changes in gastrointestinal tract, and the secondary hyperparathyroidism (SHPT) caused by the deficiency of vitamin D, is an important issue after bariatric surgery. In most RYGB patients, normocalcemia is maintained with a compensatory secondary hyperparathyroidism [2,3]. Long-term hypocalcaemia may cause development of parathyroid adenoma as a result of parathyroid gland stimulation [4]. We present a case study of a patient with history of RYGB who developed a parathyroid adenoma and consequently underwent a parathyroidectomy. We shall describe challenges we encountered in the clinical management of this case with refractory hypocalcaemia.

2. Case Summary

A 37-year-old female with a history of sleeve gastrectomy and RYGB presented to our endocrine clinic with recurrent carpopedal spasm and perioral numbness due to symptomatic hypocalcaemia (1.2 mmol/L) one month post parathyroidectomy for second opinion and further management.

Her background history was significant for a sleeve gastrectomy and RYGB for weight loss performed in 2011 and 2015 respectively. She had also undergone a laparoscopic cholecystectomy in 2017. She was on levothyroxine for hypothyroidism.

During Her follow up for hypothyroidism, she was incidentally found to have persistently elevated PTH level throughout 2019 and 2020 (PTH ranging from 101 pg/ml to 133.4 pg/ml) her calcium levels were ranging from 8.2 mg/dl to 8.55 mg/dl, her phosphorus levels were normal (4.3 mg/dl) and her vitamin D level was insufficient (24.2 ng/ml), however throughout these two years she remained asymptomatic. Prior to this her calcium levels were always within normal range.

Due to the persistently elevated PTH she had a parathyroid MIBI scan which showed a left parathyroid

adenoma and a dexascan which revealed severe Osteoporosis (Z-scores: lumbar spine -3.8, right hip -1.8, left hip -2.0, right forearm -3.1) in october 2020. For Her osteoporosis she received one dose of denusumab and was continued on teriparatide 20 mcg daily.

Following the results of the MIBI the patient had a parathyroidectomy on the 25th of november 2020. Post operatively she was started on calcium tablets and vitamin D however she was requiring large doses of Calcium carbonate tablets (up to 12 tablets per day) which she was unable to tolerate. Following which she continued to develop hypocalcaemia for which she was repeatedly given IV calcium gluconate multiple times per week after which her Symptoms would improve but would relapse two days later.

On presentation one month post parathyroidectomy the patient continued to have perioral numbness and carpopedal spasms every two days, she was unable to tolerate the large amount of calcium carbonate tablets due to nausea and vomiting. On examination she had a positive chvosteck sign.

Her calcium level on admission was low (1.2 mmol/L), her ECG showed QT-prolongation (520ms), she was immediately started on IV calcium gluconate and her oral calcium was switched to suspension which she tolerated better than the tablets. She was started on hydrochlorothiazide 12.5 mg daily. She was also started on magnesium sulfate IV and oral Magnesium as her magnesium was low (0.68mmol/L). Her Urinary 24-hour Calcium was low (<1.4 mmol/24h). Her Phosphorus level was high (1.56mmol/L), PTH level was low (5.54ng/L), vitamin D level was sufficient (107.8 ng/ml)

During her hospital admission she required large doses of calcium gluconate IV (up to 11g IV Calcium) and calcium carbonate oral suspension (1200 mg elemental calcium TID), despite these large doses the patient continued to drop her calcium every two days and thus she was commenced on teriparatide 20 mcg twice daily for severe refractory hypocalcemia and her calcium carbonate was switched to calcium citrate suspension started at 5 ml (1250 mg of elemental calcium) QID for better absorption and her alfacalcidol was increased gradually to 4 mcg daily and her hydrochlorothiazide was also increased to 25 mg daily. Once initiated on this regiment her requirement for calcium infusions had reduced and eventually was no longer needed. Her oral calcium citrate supplement was increased gradually to 25 ml (6250 mg of elemental Calcium) QID.

On discharge, her corrected calcium was low (1.71 mmol), QT on ECG had improved to 434 ms, she was asymptomatic. She was discharged on the following regimen: oral calcium citrate 25 ml (6250 mg of elemental calcium) QID, hydrochlorothiazide 25 mg daily, teriparatide 20 mcg twice daily, alfacalcidol 4mcg daily, magnesium oxide 400mg TID. She was followed in the clinic on frequent basis. On follow up her calcium slightly reduced to 1.41mmol/L, corrected calcium 1.63mmol/L and thus her calcium citrate was increased to 30 ml (7500mg elemental Calcium) QID after which her calcium improved.

Six months later, on her latest follow up she remains on the same regiment stated above, she is asymptomatic and has not required any IV calcium gluconate. Her latest investigations show: Calcium level of 2.0 mmol/L, corrected calcium is Normal (2.1 mmol/L), Magnesium level was normal (0.8mmol/L).A summary of the lab investigations prior to parathyroidectomy (November 2020) and after initiation of treatment is illustrated in Table 1.

Table 1. Summary Of Lab investigations

Date	T.Ca(mmol/L)	Mg(mmol/L)	PTH(ng/L)	Vit.D(ng/ml)
December 2019	2.13	0.7	101	24
December 2020	1.2	0.68	5.54	49
July 2021	2.1	0.8	6.72	97

3. Discussion

Calcium and vitamin D malabsorption is a known result of RYGB. Active calcium transport occurs in the duodenum which is bypassed, disrupting absorption of enteric sources of calcium. Passive calcium absorption is facilitated by an acidic environment which is also impaired by the bypassed stomach. Moreover, vitamin D absorption in the proximal jejunum is altered. Most RYGB patients, however, maintain normocalcemia due to compensatory secondary hyperparathyroidism where physiologic normocalcemia is maintained. Transient disruption of increased parathyroid function following a thyroidectomy or removal of a hyperfunctioning parathyroid adenoma leads to increased calcium deposition into bone, leading to a decrease in serum calcium which can be symptomatic depending on its extent.

This so called hungry bone syndrome (HBS) is well described following parathyroidectomy and usually can be managed with aggressive oral and IV replacement of calcium and vitamin D [6]. Hungry bone syndrome was unlikely as her Magnesium and phosphate levels were within normal limits.

There are many reported cases of secondary hyperparathyroidism post RYGB patients. As per a retrospective study conducted in Turkey, long term hypocalcaemia in such patients may cause development of parathyroid adenoma due to parathyroid gland overstimulation [4,7].

Our patient developed parathyroid adenoma and consequently underwent a partial parathyroidectomy and had persistent refractory hypocalcaemia, clinical picture in keeping with secondary hyperparathyroidism.

Despite high doses of Intravenous, Oral calcium, our patient still remained hypocalcaemic. We decided to start her on teriparatide 20 mg once daily that was then increased to 20mg twice daily [5]. Furthermore, hydrochlorothiazide was used despite having normal urinary calcium in an attempt to help normalize her serum calcium levels.

This case highlights challenging management of rare, but serious, refractory hypocalcemia in patients undergoing parathyroid surgery with previous RYGB.

4. Conclusion

Here we discussed a challenging case of refractory hypocalemia post partial parathyroidectomy in a RYGB

treated with a twice daily regimen of teriparatide. In patients post bariatric surgery including those who have undergone Roux-en -Y it is essential that those who are proceeding with parathyroid procedures are followed up closely and regularly for hypocalcemia and to start on regular oral supplements of calcium citrate, vitamin D and teriparatide at an early stage if calcium level doesn't improve with oral supplements.

Abbreviations

RYGB: Roux-en-Y Gastric Bypass
HBS: Hungry bone syndrome
IV : Intravenous
ECG : Electrocardiogram
PTH: Parathyroid Hormone
T.Ca: Total calcium
Mg: Magnesium
Vit.D: Vitamin D

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