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Case Reports

Immobilization Hypercalcemia in Critical Illness Following Bariatric Surgery

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Background: Immobilization hypercalcemia has been previously reported in a number of entities but not as a complication of bariatric surgery. We recognized this complication in two consecutive bariatric patients requiring intensive care unit (ICU) admission.

Methods: These two patients are reported in detail, and a review of our ICU database is also reported.

Results: Treatment of immobilization hypercalcemia in these two patients with pamidronate was successful.

Conclusion: Immobilization hypercalcemia complicating the course of bariatric patients requiring ICU admission is a newly recognized and treatable entity.

Key words: Immobilization, hypercalcemia, bariatric surgery, morbid obesity, intensive care unit

Introduction

Immobilization hypercalcemia complicating critical illness is a reported, but uncommonly recognized, entity. Immobilization hypercalcemia has been reported in chronically critically ill patients[1] and in the setting of hip fracture,[2] hemiplegic stroke,[3] spinal cord injury[4] and polyneuropathy.[5] Immobilization hypercalcemia has not been described as a distinct entity in morbidly obese patients.

Herein, we report two consecutive cases of immobilization hypercalcemia seen in patients suffering from complications of bariatric surgery. Both patients were successfully treated with intravenous pamidronate. We review their course in detail and report our overall experience with hypercalcemia seen in critically ill bariatric surgery patients.

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Case One:

The patient was a 42-year-old morbidly obese male (weight 120 kg, height 183 cm, Body Mass Index [BMI] 36 kg/m^2) with a history of hypertension, hypercholesterolemia, gastroesophageal reflux disease and nephrolithiasis, who underwent laparoscopic gastric banding. His postoperative course had been complicated by gastric perforation requiring exploratory laparotomy, partial gastrectomy and repair of perforation on the tenth post operative day, resulting in adult respiratory distress syndrome, septic shock, gastrointestinal hemorrhage and enterocutaneous fistula. He was transferred to our hospital on postoperative day 42 for further management.

His ionized calcium was followed as a part of the intensive care unit (ICU) panel and showed a gradual increase from 1.21 mmol/L to 1.38 mmol/L during his stay in the ICU. No exogenous calcium was administered, and there was no suspicion of malignancy. Work-up was consistent with the diagnosis of immobilization hypercalcemia (Table 1).

The patient received 90 mg of intravenous pamidronate over 24 hours, and appropriate laboratory tests were repeated 5 days later. The serum ionized calcium and 24-hour urinary calcium normalized and the N-telopeptide levels markedly

decreased and reached a near normal level (Table 1).

Case Two:

A 36-year-old morbidly obese male (weight 144 kg, height 169 cm, BMI 50 kg/m^2) with a history of deep venous thrombosis and morbid obesity underwent a laparoscopic Roux-en-Y gastric bypass. His postoperative course was complicated by staple-line dehiscence at the distal anastomotic site on the third postoperative day, resulting in intra-abdominal sepsis and respiratory failure requiring mechanical ventilation. He underwent multiple laparotomies and remained in the surgical ICU ventilator-dependent for 45 days. On admission, his serum ionized calcium was normal. His serum ionized calcium was followed daily as a part of our ICU panel and showed a steady rise from 1.01 mmol/L to 1.36 mmol/L during his ICU stay. No exogenous calcium was administered, and there was no suspicion of malignancy. Work-up was consistent with the diagnosis of immobilization hypercalcemia (Table 1).

The patient received 90 mg of intravenous pamidronate over 24 hours, and appropriate laboratory tests were repeated 5 days later. Serum ionized calcium, 24-hour urinary calcium and N-telopeptide levels all markedly decreased and reached normal levels (Table 1).

Database Review

We retrospectively reviewed our surgical ICU database for all admissions to the surgical ICU for complications of bariatric surgery. There were 17 other admissions, and review of their laboratory tests revealed three other patients who had hypercalcemia during their ICU stay. One of them was found to have hyperparathyroidism. The cause of hypercalcemia in the other two was not evaluated.

Discussion

Hypercalcemia of immobilization is caused by increased efflux of calcium from bones due to enhanced osteoclastic activity. This results in a rise in serum calcium, decreased levels of parathyroid hormone (PTH) and vitamin D 1,25(OH)$_2$ associated with elevated levels of urinary calcium and N-telopeptide.[6-8] Measurement of urinary N-telopeptide, serum Vitamin D 1,25(OH)$_2$, vitamin D 25(OH) and PTH is helpful in differentiating this phenomenon from primary hyperparathyroidism and secondary bone hyper-resorption caused by high PTH levels secondary to a primary vitamin D deficiency.[6,8] In immobilization hypercalcemia, low levels of PTH are seen because of the inhibitory effect of high calcium levels on PTH secretion. Low levels of vitamin D 1,25(OH)$_2$ will be seen because PTH is necessary to convert vitamin D 25(OH) to vitamin D 1,25(OH)$_2$ (Figure 1). Underlying cancer, hyperparathyroidism, overzealous calcium administration and treatment with thiazide diuretics and lithium should always be considered in the differential diagnosis.

Treatment with bisphosphonates has been shown

Table 1. Pertinent Laboratory Values In Patients With Hypercalcemia

	Patient 1		Patient 2	
	Pre-treatment	Post-treatment	Pre-treatment	Post-treatment
iPTH (nml=10-70 pg/ml)	20	***330***	11	***136***
Urinary Ca (nml=50-300 mg/24hr)	***362.3***	20.8	***413.9***	<20
Urinary NTX (nml=3-51 nmol/24hr)	***398.0***	***92***	***110***	40
Vit D 25 (OH) (nml=8.9-46.7 ng/ml)	22.2	16.3	9.6	***8.0***
Vit D 1,25 (OH)$_2$ (nml=15.9-55.6 pg/ml)	***5.8***	***109.7***	***5.7***	38.0

Bold and *Italics* = ABNORMAL

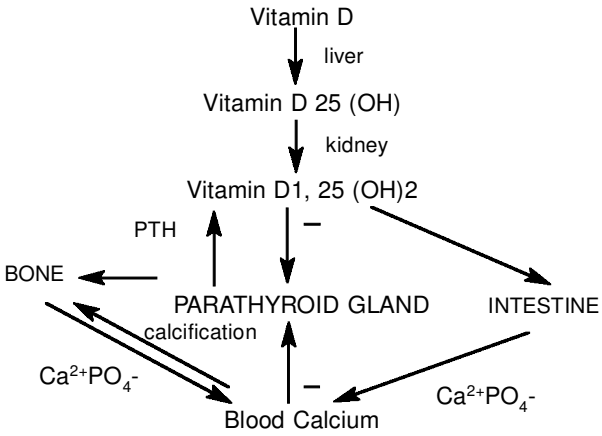

Figure 1. Vitamin D, calcium, and PTH homeostasis.

to be effective in treatment of immobilization hypercalcemia in a number of studies.[4,9,10] Treatment of our two patients with pamidronate resulted in a dramatic fall in serum ionized calcium, urinary calcium and urinary N-telopeptide, with an expected accompanying rebound in PTH and vitamin D 1,25$(OH)_2$. Patient 2 may have had a slight vitamin D deficiency unmasked which would explain the low vitamin D 25(OH) seen after treatment.

Immobilization hypercalcemia complicating critical illness is a known but uncommonly recognized entity. At our institution, since 1998 when an aggressive laparoscopic bariatric surgery program was established, approximately 1500 patients have undergone bariatric surgical intervention. Most of these patients are discharged home in under 1 week, while a very small number develop complications requiring admission to the surgical ICU, generally for management of intra-abdominal sepsis.

Given that we so rarely see immobilization hypercalcemia, it seems unlikely that at least two, and perhaps as many as of four, out of 19 bariatric surgery admissions developed this condition without being at genuinely higher risk. We hypothesize that the change from weight-bearing of such enormous weight to a condition of almost complete immobility due to acute critical illness combined with shear size, leads to an increased risk for immobilization hypercalcemia due to overwhelming disturbance of the calcium homeostatic axis, leading toward a marked increase in osteoclastic activity.

In conclusion, patients with morbid obesity suffering from complications of bariatric surgery requiring ICU admission, may be particularly prone to immobilization hypercalcemia that can be successfully treated with intravenous pamidronate.

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