Iatrogenic Hypocalcemia With Treatment of Milk-Alkali Syndrome

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INTRODUCTION

- Hypercalcemia is commonly secondary to primary hyperparathyroidism or malignancy.
- With the emergence of acid reducing agents, milk-alkali syndrome (MAS) has become a rarity.
- We present a case of MAS overtreated with bisphosphonate therapy resulting in hypocalcemia.

CASE PRESENTATION

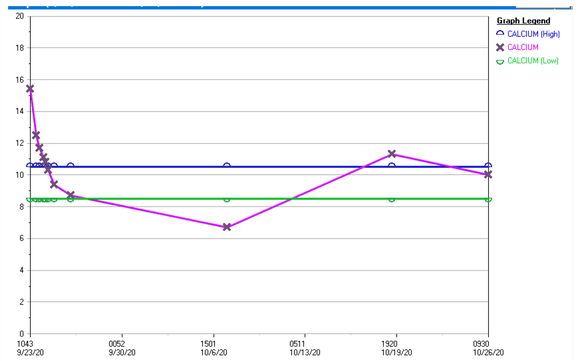
- A 77-year-old woman with a past medical history of HTN, GERD presented with slurring of speech for 2 days with nausea and vomiting.
- Initial Labs:

Calcium	15.4 mg/dL
Albumin	4.0 g/dL
Ionized calcium	7.3 mg/dL
PTH	15 pg/mL
PTHrP	9 pg/mL
Vitamin D 25-OH	16 ng/mL
TSH	2.16 IU/mL
Creatinine	1.16 mg/dL (AKI)
pH	7.49

CASE PRESENTATION

- Patient was started on intravenous fluids and given both calcitonin 300mg BID for 2 doses and pamidronate 60mg once by the admitting team.
- When seen in consultation, patient admitted to consuming more than eight calcium carbonate antacid tablets daily and was also taking hydrochlorothiazide.
- Within 48 hours, the calcium level decreased to 8.7 mg/dL.
- Given concern for potential hypocalcemia due to pamidronate, patient was advised to restart calcium carbonate 500 mg twice daily upon discharge, along with with close follow up.
- However, supplementation was not started and repeat calcium was 6.7 mg/dL twelve days later.
- The calcium normalized within a week after starting temporary calcium supplementation.

CALCIUM LEVELS



DISCUSSION

- MAS is often overlooked in the differential diagnosis resulting in overtreatment and potentially dangerous hypocalcemia.
- MAS causes hypocalcemia due to vicious cycle of PTH suppression, leading to enhanced bicarbonate retention, casing increased calcium resorption & decreased clearance, with resultant dehydration[1,2].
- Amount of calcium required to cause MAS is generally greater than 4g/day but there have been reports of 1g/day in those with pre-existing risk factors[4].

DISCUSSION

- Emergent management of intravenous hydration and bisphosphonate therapy is often immediately given by clinicians, however, patients with MAS generally improve with hydration and cessation of agent alone.
- Bisphosphonate therapy, which demonstrate calcium lowering effects by the second to fourth day, lead to hypocalcemia in MAS patients[3].
- This case demonstrates the importance of obtaining a proper history with a complete list of medications and over the counter supplementations prior to treatment.

REFERENCES

[1] Auchus, Richard, et al. “Chapter 29 - Hormones and Disorders of Mineral Metabolism.” Williams Textbook of Endocrinology, by Shlomo Melmed, 14th ed., Elsevier, 2020, pp. 1232–1233.

[2] Patel, Ami M., and Stanley Goldfarb. “Got Calcium? Welcome to the Calcium-Alkali Syndrome.” Journal of the American Society of Nephrology, vol. 21, no. 9, 2010, pp. 1440–1443., doi:10.1681/asn.2010030255.

[3] Picolos, Michalis K., et al. “Milk-Alkali Syndrome Is a Major Cause of Hypercalcaemia among Non-End-Stage Renal Disease (Non-ESRD) Inpatients.” Clinical Endocrinology, vol. 63, no. 5, 2005, pp. 566–576., doi:10.1111/j.1365-2265.2005.02383.x.

[4] Wang, Mawson, et al. “Milk-Alkali Syndrome: a ‘Quick Ease’ or a ‘Long-Lasting Problem.’” Endocrinology, Diabetes & Metabolism Case Reports, vol. 2020, 2020, doi:10.1530/edm-20-0028.