Two Truths and Alkali: When to Double-Think a Differential

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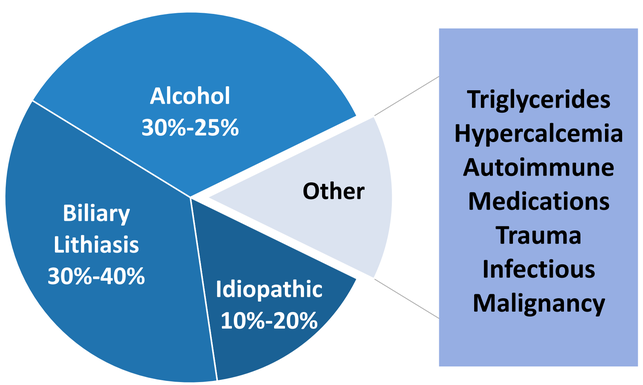
Learning Objectives

1) Outline the differential diagnosis for pancreatitis, focusing on less common etiologies.
2) Identifying Milk-Alkali Syndrome (MAS) as an infrequent manifestation of hypercalcemia and pancreatitis.
3) Recognize the dangers of anchoring and the importance of a thorough differential and medication reconciliation.

Background

- MAS presents with a classic triad of hypercalcemia, metabolic alkalosis, and acute renal failure due to excess calcium and alkali ingestion. There has been a resurgence of this disease, making it the 3rd leading cause of hypercalcemia.[1]
- In the absence of gallstones and alcohol abuse (which account for 80% of all cases[2]), the etiology of acute pancreatitis may be unclear.
- Hypercalcemia leads to a secretory block in the pancreatic duct, leading to inflammation of the pancreas.[3]



Clinical Case

Presentation

A 54-year-old woman with significant history of alcohol and cocaine use presented to the ED with 5 days of severe epigastric pain, nausea, vomiting.

- Her last drink was 2 days prior, and she was beginning to experience auditory and visual hallucinations.
- Laboratory evaluation and CT imaging confirmed pancreatitis and acute kidney injury.
- CT Head showed no evidence of hemorrhage, fracture, or intracranial abnormality. Urine tox screen was positive for cocaine and cannabinoids.
- The admission work-up also revealed an elevated calcium of 16.9 with appropriate suppression of PTH.
- Initial history revealed no medications, but upon direct questioning, it was revealed that the patient had been self-medicating with “handfuls of Tums multiple times daily” for her abdominal pain. Her agony had also prevented her from eating anything in the past 5 days.

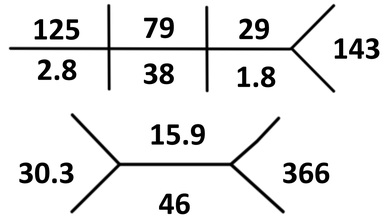
Hospital Course

Day 1: After MAS was identified as a potential contributor of her pancreatitis, a combination of IV fluids and antacid cessation resulted in a rapid correction in her calcium levels in a matter of hours. Serial metabolic panels were collected to guide IV therapy. Her hallucinations similarly resolved within hours of therapy.

Day 3: With hydration and calcium correction, she had resolving constipation and improved renal function. Refeeding syndrome labs were followed for 3 days as her nutrition intake increased and her electrolytes stabilized.

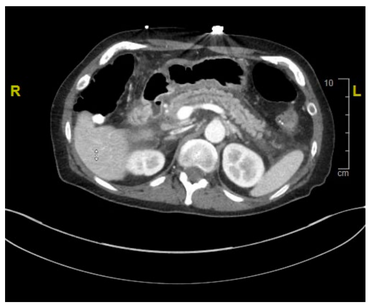
Day 5: She felt significantly better and was discharged with patient education regarding both alcohol and antacid use.

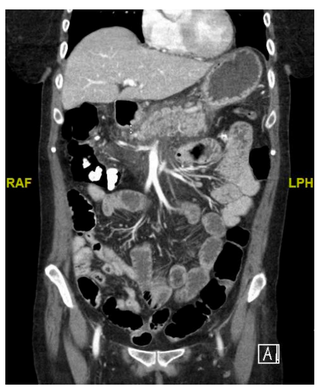
Initial Labs



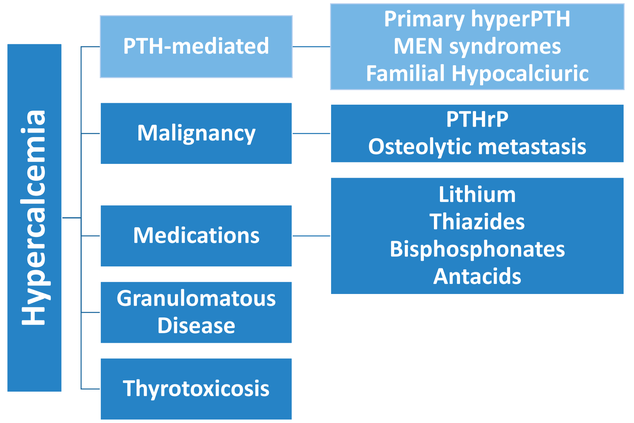
Ca: 16.9
Mg: 1.6
Ph: 1.1
PTH: 3.856
1,25 Dihydroxy Vit D: <5
Lipase: 1,482
Cocaine: Positive
Cannabinoids: Positive

Imaging





Differential Diagnosis



Discussion

- Antacid-associated hypercalcemia can result in transient hypocalcemia due to a rebound overcorrection of PTH.[1]
- Hypercalcemia reduces GFR, resulting in AKI. Furthermore, alkalosis enhances calcium reabsorption aggravating hypercalcemia.
- This case highlights the value of a comprehensive history that ascertains all medications without stopping prematurely at the most common etiologies of disease.
- Early diagnosis also becomes important for appropriate treatment as bisphosphonates, a mainstay of traditional hypercalcemia therapy, is contraindicated in MAS.[4]

References

[1] Beall DP, Henslee HB, Webb HR, Scofield RH. Milk-alkali syndrome: a historical review and description of the modern version of the syndrome. Am J Med Sci. May 2006;331(5):233-42.
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[3] Lafferty FW. Differential diagnosis of hypercalcemia. J Bone Miner Res. 1991;6 Suppl 2:S51-S61.
[4] Brandwein SL, Sigman KM. Case report: milk-alkali syndrome and pancreatitis. Am J Med Sci. 1994;308(3):173-176.