Too Much of a Good Thing ...

Nancy Fuller, MD
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Objectives:

- Rouse a few brain cells to remember Milk-Alkali Syndrome
- Create a few new brain connections to consider the latest presentation of this syndrome

Or: Everything Old is New Again!
AB: 60 yo woman

Presents to the clinic for follow up of recently discovered hypertension following urgent care visit for URI symptoms

Blood pressure 180/90 at UC

No previous personal history of hypertension although both parents had hypertension

Had been taking Claritin-D regularly for her URI symptoms

PMHx: osteopenia, otherwise healthy
• Works as a custodian, a physically demanding job
• ROS: No chest pain, SOB, DOE; headache recently with URI
• Meds: Alendronate, calcium, cod liver oil, occasional Excedrin, MVI, acetaminophen, fluticasone
• PE: BP 160/102 P 88, BMI 23,
• rest of exam unremarkable
• A/P: hypertension, check chem 7, start HCTZ 12.5 mg; she was told not to start it until I called with the results of the chem 7.
• Call from lab: Ca level 14.2
• Rest of lytes: Na 136, K 4.9, chl 100, gluc 97, BUN 50, Cr 3.28, e-GFR 14
• Next stop: UW Hospital
• Extensive workup for cancer, hyperparathyroidism: all negative
• More history: she eats a diet high in dairy (lots of milk, cheese, cottage cheese) and takes 2 Citracal per day, plus an additional 1200 calcium supplement per day, plus taking a lot of Tums for dyspepsia
• Milk-Alkali Syndrome: hypercalcemia + metabolic alkalosis + renal insufficiency
• Originally described with the ingestion of large amounts of milk and sodium bicarb for the treatment of PUD (Sippy powder: 600mg magnesium carbonate and 600mg sodium bicarbonate alternating with 600 mg bismuth subcarbonate and 1200-1800 mg sodium bicarbonate with milk or cream HOURLY ( circa 1915)
• Acute: nausea, vomiting, weakness, mental changes, severe metabolic alkalosis, acute renal insufficiency. Withdrawal of milk and alkali: rapid normalization

• Chronic: after long history of high milk/alkali intake. Polyuria, polydipsia, muscle aches, pruritus, metastatic calcifications like nephrocalcinosis. Many patients continue to have CRF after treatment

• Subacute: after intermittent use of milk alkali; symptoms of both acute and chronic hypercalcemia

• This syndrome faded after the introduction of H2 blockers, then PPIs
The road to hell is paved with good intentions...
• “Calcium alkali Syndrome” rather than Milk-alkali Syndrome?

• Caused by ingestion of large amounts of calcium either as an antacid, or more commonly, for treatment/prevention of osteoporosis

• Now the 3rd most common cause of hypercalcemia after hyperparathyroidism and malignant neoplasms (approximately 10% of hospitalized patients with hypercalcemia)

• Among a subset of patients with severe hypercalcemia (>14) this is more common than malignancy

• Only half of patients have alkalosis
• Pathogenesis:
  1. Excessive calcium intake: typically 4-5 gms or more
  2. Preexisting renal insufficiency
  3. Medications that interfere with calcium excretion like thiazide diuretics
  4. Hypercalcemia worsens renal function by afferent arteriole constriction and reduced GFR.
  5. Hypercalcemia has a diuretic effect, leading to intravascular depletion
  6. Reduced GFR results in even more limited excretion of calcium:

=VICIOUS CYCLE!
• Diagnosis requires a history of excessive calcium intake, and findings of hypercalcemia and variable degrees of renal impairment, and the absence of hyperparathyroidism or malignancy
• Treatment: withdrawal of calcium, hydration; after hydration, consider furosemide to enhance calciuresis
• Hypercalcemia usually resolves after a few days but may take up to 6 months
• Renal failure usually improves but may have persistent reduced GFR
• AB: Hospitalized for 3 days
• Treated with hydration, blood pressure control (amlodipine)
• Labs at time of discharge: Ca 10.6, Cr 2.25
• Most recent labs: Ca 9.3, Cr 0.95, eGFR 65