Milk-Alkali syndrome, an udder failure of acid control

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Introduction:

Milk-Alkali syndrome is characterized by a triad of lab findings: elevated calcium levels, metabolic alkalosis, and acute kidney injury. This is found in the setting of substantial intake of calcium and an absorbable alkali. It now accounts for more than 10% of hypercalcemia cases and is the third most common cause of hypercalcemia in hospitalized patients, after hyperparathyroidism and malignancy. In Milk-Alkali syndrome, the hypercalcemia is caused due to the exogenous calcium supplementation. The metabolic alkalosis is due to increased alkali intake (such as carbonate) with concurrent hypercalcemia-mediated hypovolemia and decreased GFR. The alkalosis then leads to increased calcium reabsorption from the nephron's distal tubule, further creating a vicious cycle of calcium reabsorption and further kidney injury.

Case:

A 37 y/o F with PMHx Sjogren Syndrome, Dandy-Walker Syndrome s/p VP shunt, and bipolar disorder presented for a gradual worsening and persistent weakness in her distal BUE and BLE for the 3 weeks prior to presentation. She also noted numbness and paresthesias in her distal extremities, brain fog, and memory deficits. The pt had baseline sensory dysfunction due to her Sjogren's, however she noticed new onset proximal progression of the deficits with associated new lower extremity weakness. She visited her PCP and on routine labs was found to have several lab abnormalities, including severe hypokalemia to 2.6, severe hypercalcemia to 15.9, metabolic alkalosis to 41, and an acute kidney injury with Cr to 2.22 with a baseline of 1.07. She not on immunosuppressives for her Sjogren's and was only being treated for her peripheral neuropathy. She also took valproic acid for her bipolar disorder management. Pt was started on aggressive fluid and electrolyte replacement with appropriate response and noted improvement in kidney and neurological function. With further interview, pt revealed that she had been taking 4 tablets of extra-strength TUMS 3x daily for 2 months due to her severe acid reflux symptoms. She had not previously reported this problem to her PCP and had not tried proton-pump inhibition or H2 blocker therapy. She was counseled to stop calcium supplementation, started PPI therapy, and arranged follow up with Gastroenterology for her severe GERD symptoms and her PCP for further monitoring.

Discussion:

There has been a drop off in incidence in milk-alkali syndrome due to the prevalence of PPI therapy for GERD symptoms. In a study of patients that were hospitalized for emergent hypercalcemia, only 12% of the patients admitted over a 4-year period had milk-alkali syndrome as the underlying etiology[2]. Another study that evaluated hospitalized patients from 1998-2003 with hypercalcemia as their primary diagnosis, found similar results with 8.8% in those without ESRD, and 9 out of 25 patients with severe hypercalcemia (>14) were found to have milk-alkali syndrome[3]. This case shows the importance of keeping this syndrome on the differential while obtaining the history, because given her past medical history, there were other indications for possible tubular dysfunction such as a distal RTA secondary to her Sjogren's or a nephrogenic diabetes insipidus from her valproic acid use.

References

[1]https://www.ncbi.nlm.nih.gov/books/NBK557500/#:~:text=The%20milk%2Dalkali%20syndrome%20is,of%20calcium%20and%20absorbable%20alkali.

[2] https://read.qxmd.com/read/7891547/milk-alkali-syndrome-associated-with-calcium-carbonate-consumption-report-of-7-patients-with-parathyroid-hormone-levels-and-an-estimate-of-prevalence-among-patients-hospitalized-with-hypercalcemia?redirected=slug

[3] https://read.qxmd.com/read/16268810/milk-alkali-syndrome-is-a-major-cause-of-hypercalcaemia-among-non-end-stage-renal-disease-non-esrd-inpatients?redirected=slug