

Chalk Pica Presenting as Hypercalcemia-Induced Pancreatitis

by Bhaavya Pinnala, Sindhu Naresh, Saleha Asghar

Acute pancreatitis is most commonly associated with gallstone disease, chronic alcohol use or hypertriglyceridemia while metabolic abnormalities such as hypercalcemia are far less frequent.¹ Hypercalcemia accounts for only about 1% of acute pancreatitis cases, making it a cause that is not typically encountered in routine clinical practice.¹ Among the causes of hypercalcemia-induced pancreatitis (HIP), primary hyperparathyroidism is the most common and accounts for approximately 90% of all hypercalcemia cases.³

Other causes of hypercalcemia include malignancy, particularly lung cancer, breast cancer, and multiple myeloma, granulomatous diseases (tuberculosis and sarcoidosis), vitamin A or D toxicity, hypothyroidism and medications like thiazide diuretics, lithium, and antacids.^{2,3}

An under-recognized cause of hypercalcemia is chalk ingestion, which often occurs in the setting of pica. Chalk is primarily composed of calcium carbonate and can become a substantial calcium source for individuals who consume it regularly.⁵ This behavior can raise serum calcium to harmful levels, leading to metabolic disturbances and in this case, hypercalcemia-induced pancreatitis.

Case Presentation

A 52-year-old African American female with past medical conditions of hypothyroidism and newly diagnosed insulin-dependent type 2 diabetes mellitus, presented to the emergency department after a witnessed fall and several days of worsening

fatigue and confusion. According to her family, she had been complaining of abdominal pain over the past two days. Additionally, the patient had been experiencing increased urinary frequency.

Her exam was notable for pale appearance and disorientation. The remainder of the physical examination was notable for diffuse abdominal tenderness with guarding. Her vital signs included a blood pressure of 90/60 mmHg, heart rate of 120 beats-per-minute, afebrile and oxygen saturation of 97% on room air.

Initial laboratory evaluation revealed serum calcium was markedly elevated at 25.4 mg/dL with an ionized calcium of 2.48 mmol/L. Additional findings included an albumin of 3.9 g/dL and an elevated lactate of 3.2 mmol/L. Her lipase was markedly elevated at 1,739U/L. Her liver enzymes, lipid panel and ethanol level were in normal range.

Computed tomography (CT) imaging of the abdomen and pelvis with contrast demonstrated acute interstitial pancreatitis without gallstones or biliary dilation [Imaging 1 & 2]. Additionally, large heterogeneous pelvic mass, suggestive of a uterine leiomyoma, was noted.

She was admitted to the intensive care unit for management of hypercalcemic crisis. The patient received empiric antibiotics, intravenous fluids and underwent two sessions of emergent hemodialysis. Patient's hypercalcemia improved with hemodialysis and intravenous fluid, without the need for bisphosphonates, or calcitonin. Her calcium levels normalized from 25.4 to 9.4 mg/dL, accompanied by a gradual recovery in mental status. Serum calcium levels normalized within approximately 48 hours of initiating treatment. The patient's pancreatitis gradually improved as she was able to tolerate oral intake.

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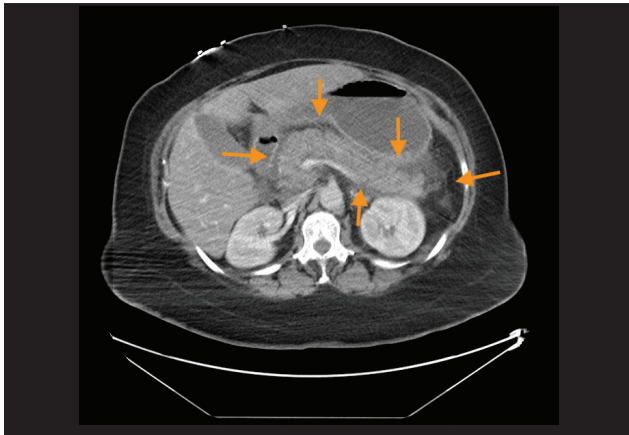


Image 1. The arrows show diffuse edema throughout the pancreas especially around the pancreatic head. Increased attenuation is seen surrounding the pancreatic head and body.

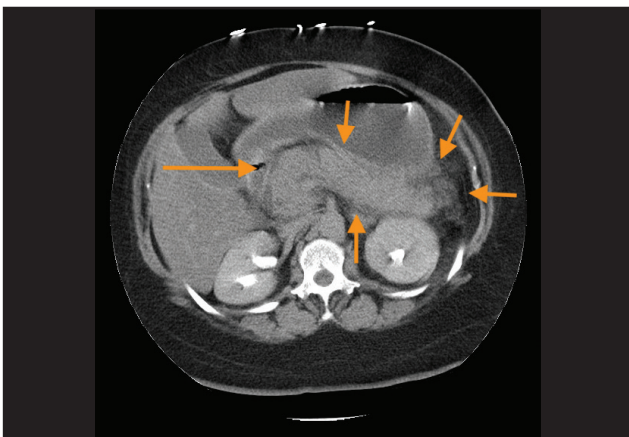


Image 2. The arrows show diffuse edema throughout the pancreas especially around the pancreatic head.

A comprehensive metabolic evaluation demonstrated a suppressed PTH level (5.9 pg/mL) while phosphate, PTHrP, 25-hydroxy vitamin D, and 1,25-dihydroxy vitamin D were within normal limits. Serum and urine electrophoresis, serum free light chains, TSH, lithium, vitamin A level, and urine calcium were all unremarkable. Hematologic studies revealed microcytic anemia with hemoglobin level of 9 g/dl, MCV of 63 fL, low iron (8 µg/dL), a TIBC of 281 µg/dL, ferritin of 37.2 ng/mL, a reticulocyte count of 1.5%, and a peripheral smear showing microcytic, hypochromic red blood cells.

After resolution of her symptoms, the patient denied the use of diuretics, lithium, antacids, vitamin

and calcium supplements. Her workup revealed no identifiable cause of hypercalcemia, raising concern about the source of her severe calcium elevation. It was only later in her hospitalization, when we specifically asked about her dietary habits, that she disclosed regularly consuming powdered chalk for almost three months. She consumed two bags of chalk (approximately four kilograms per day). She had not previously disclosed this due to embarrassment and a belief that the behavior was harmless to her health.

She received intravenous iron and was discharged on oral iron supplementation. She was evaluated by gynecology, and the uterine leiomyoma was removed. At an outpatient follow-up six weeks later, serum calcium (8.9 mg/dl) and hematologic studies returned to normal, and her chalk-ingestion behavior fully resolved.

Discussion

Hypercalcemia is an uncommon but recognized trigger of acute pancreatitis, accounting for approximately 1–2% of cases. Most HIP are attributed to primary hyperparathyroidism or malignancy-associated hypercalcemia.¹ Literature on exogenous calcium leading to pancreatitis is extremely limited, with chalk ingestion described only in isolated cases. Hypercalcemia can precipitate pancreatitis by increasing intracellular calcium levels within pancreatic acinar cells, triggering premature trypsinogen activation and leads to autodigestion and inflammation.²

HIP is infrequently encountered, but hospitalist and gastroenterology management is pivotal for acute care and recurrence prevention. Priorities include confirming pancreatitis, excluding common pancreatitis etiologies, initiating lactated ringer's-based resuscitation with adequate analgesia, and starting early enteral nutrition; contrast-enhanced CT is reserved for diagnostic uncertainty or lack of improvement after 48–72 hours, and ERCP is performed only for biliary indications. Close monitoring of electrolytes, with recognition that fat saponification can lead to secondary hypocalcemia as inflammation evolves. Preventing another episode requires addressing the underlying cause of hypercalcemia through a multidisciplinary approach, while counseling patients to avoid hidden or non-nutritive calcium sources.

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A CASE REPORT

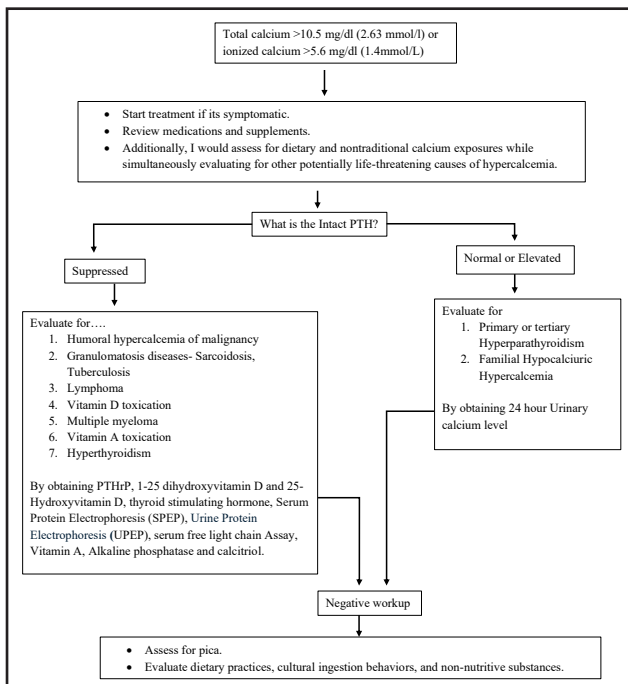


Image 3. Diagnostic Algorithm for Hypercalcemia with Inclusion of Dietary and Non-Nutritive Calcium Sources

Pica is often underreported and maybe normalized by patients who have engaged in the behavior for years. Although geophagia is a recognized form of pica in the DSM-5, psychiatric consultation was not obtained during this hospitalization. An additional consideration, in Central Georgia, kaolin (a form of chalk) is sometimes consumed as part of a cultural practice, particularly among African American women.⁶ However, both the patient and her family denied any cultural or personal use of kaolin or similar substances.

The current diagnostic algorithm for hypercalcemia begins by confirming elevated calcium levels and reviewing medications and supplements, including thiazide diuretics, lithium, vitamin D, vitamin A, calcium-containing antacids, and other over-the-counter products. Once hypercalcemia is established, intact PTH is measured to distinguish PTH-mediated from non-PTH-mediated causes. Elevated or inappropriately normal PTH levels suggest primary hyperparathyroidism or familial hypocalciuric hypercalcemia, while low PTH levels prompt evaluation for malignancy, granulomatous disease, vitamin D intoxication, and monoclonal gammopathies through testing such as PTHrP,

1,25-dihydroxyvitamin D, 25-hydroxyvitamin D, SPEP, UPEP, and serum free light chains. Although this algorithm captures the major etiologic categories, it does not include assessment of dietary or non-traditional calcium exposures.

Our case demonstrates that ingestion of calcium-rich substances, including chalk or culturally rooted forms of kaolin, can lead to significant hypercalcemia and may remain unrecognized without targeted questioning. Adding a step that evaluates dietary practices, cultural ingestion behaviors, and non-nutritive substances could improve detection of these overlooked causes and enhance diagnostic accuracy.

Pica is managed through a combination of education, nutritional counseling, and behavioral strategies implemented by an interdisciplinary team that may include physicians, nursing staff, psychologists, social workers, dietitians, and family members. Patients and families are taught to focus on understanding the behavior and adopt safer alternative coping skills.⁷

CONCLUSION

This case illustrates the importance of considering nontraditional and dietary sources of calcium in the evaluation of severe hypercalcemia and hypercalcemia-induced pancreatitis. Uncommon etiologies such as chalk ingestion can be easily overlooked without targeted questioning, particularly when the clinical picture remains unexplained after routine evaluation. Adding this step to clinical algorithms may facilitate earlier recognition of atypical causes, prevent delayed diagnosis, and improve patient outcomes. ■

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