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A Case Report of Hypercalcemia and Mucosal Injury after Calcium Chloride Intoxification

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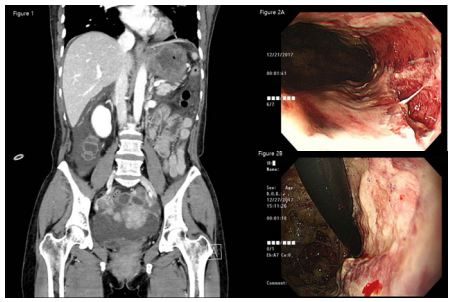
Calcium chloride is a substance that is used for medical purposes as it is known to be relatively less harmful. However, it is an alkali solution that can cause severe damage to skin and mucous membrane. In general, taking calcium chloride does not cause severe electrolyte abnormalities when the skin barrier is intact, but it can lead to dangerous hypercalcemia if accompanied by skin injury. Herein, we reported a case of gastrointestinal mucosal damage and subsequent severe hypercalcemia after ingestion of calcium chloride. A 53-year-old woman who did not have any underlying disease took about half a cup of calcium chloride, which was used to shovel the snow, for the purpose of committing a suicide. She vomited 5 to 6 times after taking it and spitted out blood 3 to 4 times accompanied with nausea and abdominal pain. The patient's consciousness was not deteriorated. In the laboratory findings, severe hypercalcemia of 18.0 mg/dL was observed. Bisphosphonate and calcitonin were administered with massive normal saline hydration. Calcium, magnesium, electrolytes, and electrocardiography were monitored, and as the patient complained of persistent abdominal pain, nausea, and vomiting, peripheral nutrition was maintained at the fasting state. On esophagogastroduodenoscopy, multiple gastric ulcers were found, and the endoscopy was performed once a week to check if the patient could start a meal. After the patient's recovery was confirmed at week 2, she began eating and was discharged without complications. It is interesting that although calcium chloride is known to be a relatively less harmful substance, it caused mucosal injury in the stomach and then was absorbed into the body and caused hypercalcemia. Fortunately, the patient recovered quickly without facing any crisis due to hypercalcemia or other complications, which was probably because of the early visit to the hospital and proper management after taking calcium chloride. Figure 1) Computed Tomography taken after admission: There is a finding with which ulcer perforation can be suspected Figure 2A)EGD performed 2 days after admission 2B)EGD performed 9 days after admission



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SLC12A3gene(a single heterozygous c.964+1G > A in intron7)mutation strongly suggests Gitelman Syndrome

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GITELMAN SYNDROME INTRODUCTION: A widely used system classifies Bartter syndrome on the basis of the underlying genetics, as follows Type 1 Mutations in SLC12A1 Type 2 Mutations in the ROMK gene Type 3 Mutations of the chloride voltage-gated channel Kb gene (CLCNKB); classic Bartter syndrome Type 4 Mutations in BSND Type 5 Gitelman syndrome: Results from mutations SLC12A3, the sodium-chloride cotransporter **BACKGROUND:** Understanding Bartter syndrome and Gitelman syndrome as a spectrum of disease rather than distinct disorders. Aim to review the clinical diagnostic features of two renal tubular disorders may be broadly classified as renal tubulopathies, salt-losing tubulopathies or channelopathies. Gitelman syndrome causes metabolic abnormalities resembling treatment with high dosage of thiazide diuretics while Bartter syndrome resembles treatment with high dosage of loop diuretics. **DATA SOURCES:** Selected clinical case references concerning these syndromes were analyzed, together with PubMed search of the literature. CASE REPORT A 23 year old male a trainee soldier was referred to our hospital by military hospital for further diagnostic evaluation of episodic exacerbation of muscle weakness, muscle aches, fatigue, hematuria, proteinuria, hypokalemia, heavy meals might also be accompanied by abdominal pain, vomiting since early childhood. He was otherwise healthy. In FH, grandfather, father &older sister had kidney problems with shared similar clinical features. On examination, V/S, EKG, CXR, TFT, Rapid ACTH, US&CT of abdomen did not reveal any abnormality. Serum potassium 2.88 mEq/L, serum sodium135.4 mEq/L, serum chloride99.9 mEq/L, serum bicarbonate32 mMol/L, serum magnesium1.05 mg/dL, 24hrs.urine Mg172 mg/d,24hrs.urine TCa15.7 mg/d, serum PH7.50, urine TTKG4.25 **CONCLUSIONS:** The severe, steady state hypokalemia in Bartter syndrome and Gitelman syndrome may abruptly become life-threatening under certain aggravating conditions. Clinicians need to be cognizant of such renal tubular disorders, and peromptly treat at-risk patients. **DISCUSSION:** The signs and symptoms of Gitelman syndrome vary widely, even among affected members of the same family