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# Over-the-Counter Drug Causing Acute Pancreatitis

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## Abstract

Acute pancreatitis is caused by alcohol, gall stone disease, drugs, trauma, infections, and metabolic causes such as hypercalcemia and hyperlipidemia. Hypercalcemia-induced acute pancreatitis has been well documented but only rarely occurs due to over-the-counter calcium carbonate. In this article, we present a case of over-the-counter calcium carbonate-induced acute pancreatitis.

## Keywords

OTC, over the counter, CT, computed tomography, MAS, milk-alkali syndrome, PUD, peptic ulcer disease

## Introduction

Acute pancreatitis is defined as inflammation of the pancreas and is one of the common reasons for hospital admissions in the United States.<sup>1</sup> It has multiple causes, with alcohol and gall stone disease contributing to 80% to 90% of cases.<sup>2</sup> Other reported causes include drugs, common bile duct obstructions secondary to neoplasms and sphincter of Oddi dysfunction, and metabolic alterations such as hypercalcemia or hypertriglyceridemia. Hypercalcemia associated with primary hyperparathyroidism contributes to 3.6% cases of pancreatitis cases, while pancreatitis induced by excess consumption of over-the-counter (OTC) calcium carbonate is rare and very seldom reported in the literature.<sup>3,4</sup> In this article, we present the case of a young male with pancreatitis secondary to OTC calcium carbonate medications.

## Case Description

A 35-year-old male with no significant past medical history was admitted for epigastric pain with radiation to the back associated with nausea and vomiting. He complained of fatigue, poor appetite, and increased urination. He had a history of gastroesophageal reflux disease for which he was taking OTC medications. His vital signs were stable. On examination, he appeared slightly confused, with dry skin and generalized abdominal tenderness. Blood work: white blood cells 16400/ $\mu$ L, sodium 139 mmol/L, blood urea nitrogen 23 mg/dL, creatinine 2.6 mg/dL, aspartate aminotransferase 34 U/L, alanine aminotransferase 26 U/L, bicarbonate 34 mmol/L, alkaline phosphatase 114 U/L, calcium 15.1 mg/dL, triglyceride level 79 mg/dL, low-density lipoprotein 110

mg/dL, lipase 560 U/L, albumin 3.9 g/dL, and negative for ethanol. Computed tomography scan of abdomen/pelvis showed pancreatitis with a normal biliary tract. He was treated with pain medications and ringer lactate fluid.

Further investigation showed low parathyroid hormone (PTH) = 18 pg/mL, low vitamin D (25) = 17 ng/mL, along with normal PTH-related peptide, vitamin D (1,25), serum protein electrophoresis, urine protein electrophoresis, and free light chain. The patient admitted that he had been taking 7 to 8 tablets of 600 mg calcium carbonate per day over the past 3 weeks for acid reflux. His symptoms and calcium level gradually improved with ringer lactate, and he was discharged after 6 days. He was educated on the side effects of excessive OTC drug use.

## Discussion

Milk-alkali syndrome (MAS) is caused by increased consumption of calcium and alkali products. MAS is characterized by the triad of hypercalcemia, renal failure, and metabolic alkalosis. Increased use of OTC drugs in patients

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with osteoporosis and kidney failure can lead to a life-threatening illness. In the early days after its discovery, MAS was associated with an increased mortality rate of 4.4%.<sup>5</sup> MAS was first described by Hardt and Rivers in 1923.<sup>6</sup> They attributed this syndrome to the Sippy regimen, which was developed in 1915 for peptic ulcer disease. The Sippy regimen comprised the hourly consumption of milk, cream, and a mixture of alkaline powders. Prior to the discovery of H2 blockers and proton pump inhibitors, the Sippy regimen was commonly used in peptic ulcer disease.<sup>7</sup> In the 1970s and 1980s, MAS contributed to less than 2% of hospitalized patients with hypercalcemia, but this incidence has increased to 12% in recent years.<sup>8</sup> MAS is now considered to be the third most common cause of hypercalcemia, after hyperparathyroidism and malignant neoplasms. Some authors have suggested that this increased incidence is due to increased use of OTC drugs.<sup>9</sup>

The pathophysiology of MAS is classified into 2 phases: a generation phase and a maintenance phase. Excess calcium consumption increases serum calcium levels; this results in volume depletion from diuresis and natriuresis by activation of the calcium sensing receptor (CaSR), which stimulates renal tubular absorption of bicarbonate and decreases glomerular filtration rate. This decreased glomerular filtration rate reduces filtration of calcium, while volume depletion and metabolic alkalosis lead to increased renal absorption of calcium in order to maintain calcium homeostasis. The pathophysiology of hypercalcemia-induced pancreatitis is unclear, but high calcium levels are thought to lead to intracellular activation of proteases.<sup>10-13</sup> In addition to OTC drugs as an inciting factor for MAS, other cyclic pathophysiologic pathways that promote alkalosis, hypercalcemia, and renal failure can mimic MAS. MAS has been reported with the consumption of more than 4 to 5 g of calcium carbonate per day.<sup>9</sup> These cases present in 3 forms: acute, subacute (cope syndrome), and chronic (Burnett syndrome).<sup>14</sup> In the acute phase, patients present with symptoms of toxemia, such as nausea, vomiting, anorexia, dizziness, vertigo, and confusion; and in chronic phase, they present with muscle aches, polyuria, polydipsia, psychosis, tremor, pruritis, and abnormal calcifications. Ocular calcification, renal calcinosis, and rarely breast calcifications have been reported.<sup>15</sup> The diagnosis is clinical, and a detailed history along with a physical can aid the diagnosis of MAS. Treatment is mostly supportive, with hydration and removal of the underlying causative agent.

According to the evidence discussed above, we conclude that medications should be reconciled on every patient encounter, including OTC medications, and patients should be educated about the side effects of excessive use. Daily elemental calcium intake should not exceed 2 g per day. However, MAS has also been reported with a lower calcium intake in patients on thiazides and with preexisting renal failure, as in our patient. Therefore, it is very important that all

patients should be educated on various drug interactions in addition to information about daily calcium intake.

### Authors' Note

The work has been published previously as an abstract at the American College of Gastroenterology Annual Scientific Meeting on October 25, 2019, in San Antonio, Texas.

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### Ethics Approval

Our institution does not require ethics approval for reporting individual cases.

### Informed consent

Verbal consent was obtained from the patient(s) for their anonymized information to be published in this article.

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