


# A Rocky Discontinuation of Diet Mountain Dew

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## Abstract

A 62-year-old man with a past medical history of recently diagnosed type II diabetes mellitus presented for multiple episodes of nephrolithiasis after stopping Diet Mountain Dew ingestion. Stone analysis confirmed calcium oxalate stones. It was theorized that the high citrate in Diet Mountain Dew was protective against his newly recurrent nephrolithiasis. For lifestyle preference, the patient chose lemonade-flavored Crystal Light—known to be high in citrate—instead of potassium citrate 30–40 mEq supplementation. To date, the patient's nephrolithiasis has not recurred. Potassium citrate is a preventive strategy against calcium oxalate stones in patients with suspected or confirmed hypocitraturia. Citrate binds calcium, therefore, preventing the interaction between calcium and oxalate. Alternative supplementation strategies, such as citrus-flavored sodas (eg, Diet Mountain Dew), powdered drinks (eg, Crystal Light), and natural juices (eg, lemon juice), may be plausible alternatives to potassium citrate. Patient lifestyle and the risks and benefits to a particular supplemental choice must be considered for every patient.

## Keywords

calcium oxalate, kidney stone, nephrolithiasis, citrate, soft drink

## Case

A 62-year-old man with a past medical history of atrial fibrillation and recently diagnosed with type II diabetes mellitus (DM) presented to our institution with acute left-sided flank pain. He was found to have acute kidney injury due to nephrolithiasis. Four months prior to admission, the patient received a new diagnosis of DM. After this diagnosis, he eliminated all Diet Mountain Dew (DMD) consumption from his diet (average of four 12-ounce cans or about 1.4 L of DMD per day) and replaced it with water hydration. Two months later, the patient presented to his primary care provider's office after acute onset of severe right flank pain. Computed tomography (CT) stone protocol showed a 6-mm calculus at the right ureterovesicular junction with associated right hydronephrosis and a 5-mm nonobstructing left upper pole renal calculus. The stone passed after starting increased oral hydration, tamsulosin 0.4 mg daily, and oxycodone for pain control. Nephrolithiasis analysis was ordered, but the patient did not return his passed stone to the lab.

Two weeks prior to admission, the patient developed acute left-sided flank pain. The CT stone protocol showed a nonobstructing 5-mm stone in the proximal left ureter and a 4-mm stone in the right ureter with minimal hydronephrosis.

Outpatient stone treatment was again initiated with increased oral hydration, tamsulosin, and oxycodone. Unfortunately, the stone was not passed, and the patient continued to experience flank pain. On the day of admission, he was seen in his cardiologist's office for a routine follow-up of atrial fibrillation controlled with dofetilide. He was found to have an elevated Cr 2.99 g/dL (baseline ~ 1.3 g/dL) and an increase in QTc from 412 to 560 ms with u-waves. Dofetilide and apixaban were stopped, and he was directed to the emergency department for admission.

On admission, the patient was afebrile with blood pressure 142/85 mm Hg, pulse 69 bpm, respiratory rate 16, and O<sub>2</sub> sat 98% on room air. On examination, he was alert, oriented, and in no acute distress. He had no costovertebral or abdominal tenderness. Repeat CT stone protocol showed an obstructing left mid-ureteral 7-mm calculus with mild

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**Figure 1.** Computed tomography stone showed a 7-mm left mid-ureteral calculus (yellow arrow left figure) with mild upstream hydronephrosis (yellow arrow right figure).

upstream hydronephrosis (Figure 1) and 2 adjacent nonobstructing 2-mm distal right ureteral calculi. Urology was consulted, and bilateral ureteral stents were placed. Biochemical urine studies showed normal urine oxalate at 26 mg/day (reference 16-49 mg/day) and low urine calcium 0.05 g/volume (reference 0.1-0.3 g/volume). A 24-hour urine citrate was not available at our institution, and a send-out test was not sent during his admission. After stent placement, the patient's pain and creatinine improved to 1.9 g/dL and QTc improved to 411 ms. He was discharged home and approximately 2 weeks later underwent outpatient lithotripsy. Stone analysis showed calcium oxalate stones.

With this information and on review of his dietary changes, it was theorized that the high potassium citrate content of DMD had previously prevented stone formation. The patient was prescribed potassium citrate supplementation. However, he did his own research and found that low-calorie lemonade-flavored Crystal Light was high in citrate. Out of lifestyle preference, he began drinking crystal light (averaged 1.9 L per day, which was found to contain about 220 mEq of citrate)<sup>1</sup> instead of taking citrate supplements. Following this, he no longer had recurrent nephrolithiasis and, thus, declined additional testing. Twelve months later, the patient's nephrolithiasis has not recurred. His creatinine had slightly improved to 1.8, but never returned back to his original baseline of 1.1.

## Discussion

Calcium oxalate stones are the most common type of kidney stones (70%-80%), with the highest prevalence in white, elderly men. Modifiable risk factors include low dietary intake of calcium, potassium, and fluids and high intake of

oxalate-rich foods, non-dairy animal protein, and vitamin C supplements, among others. Urine biochemical abnormalities may include hypercalciuria, hyperoxaluria, hypocitraturia, or high urine uric acid.<sup>2</sup> Hypocitraturia has been classified as less than 320 mg excreted per day.<sup>3</sup> In our case, the patient developed recurrent kidney stones following cessation of about 1.4 L/day of DMD. Different citations and biochemical techniques report varying ranges of citrate in DMD. One study using ion chromatography references citrate content at 6.8 mEq citrate per liter,<sup>4</sup> while another using nuclear magnetic resonance spectroscopy references it at 26.98 mEq citrate per liter.<sup>1</sup>

For patients with calcium oxalate stones and suspected or confirmed hypocitraturia, supplementation with potassium citrate 30 to 40 mEq/day is a preventative strategy.<sup>5</sup> Supplementation in this range is associated with an approximately 2-times increase in urinary citrate.<sup>6</sup> Citrate inhibits stone formation by forming a soluble complex with urinary calcium, thus reducing the amount of calcium available for oxalate binding. There is also evidence to suggest citrate directly dissolves calcium oxalate stones.<sup>7</sup> However, potassium citrate supplements are not always preferred or tolerated by patients. Side effects may include abdominal distress, diarrhea, and nausea.<sup>8</sup> Furthermore, some patients desire alternative nonpharmacological supplementation strategies based on lifestyle and compliance.

Diet-citrus soft drinks—such as DMD (Table 1)—have variable citrate levels reported.<sup>1,4</sup> A small cross-over study (n = 9) investigated 1 L diet Sunkist orange soda (citrate 8.4 mEq/L) versus water consumption in healthy adults with normocitraturia. It found an increase in urinary citrate by 60 mg/day; however, this was not statistically significant ( $P = 0.34$ ).<sup>9</sup> Powdered mix drinks may provide another option for

**Table 1.** Possible Alternatives to Potassium Citrate Supplementation.

	Citrate composition	Approximate commercial cost	Approximate cost per 30 mEq/day	Special considerations
Potassium citrate tablets (10 mEq each)	Potassium citrate	60 tabs at \$20-42 <sup>a</sup>	\$1.00-2.10	Concentrated doses can cause gastrointestinal upset, including abdominal distress, nausea, and diarrhea
Diet Mountain Dew (6.8 <sup>4</sup> to 27 <sup>1</sup> mEq/L)	Citric acid, potassium citrate, sodium citrate	1 L at \$1.00	\$1.11 <sup>3</sup> -4.40 <sup>4</sup>	Consumption of water and other non-sweetened beverages is generally encouraged over diet drinks. Studies looking at the effects of artificial sweeteners on health are mixed. Artificial sweeteners reduce the risk of dental caries. However, there are mixed findings (negative, neutral, and positive) regarding the impact on weight, glycemic control, and cardiovascular disease <sup>12</sup>
Orange juice (145 mEq/L) <sup>1</sup>	Citric acid	1 L at \$2.50	\$0.64	Consumption of high sugar beverages risks weight gain, diabetes, and metabolic syndrome
Freshly squeezed lemon juice (147 mEq/L) <sup>1</sup>	Citric acid	8 oz of juice at \$2.00-\$2.70 for 5 lemons	\$1.63-2.20	A lower calorie option than many other juices. However, high acidity and citrate concentration risk gastrointestinal upset
Crystal Light (117 mEq/L) <sup>1</sup>	Citric acid, potassium citrate	30 packets (where 1 packet makes a 21 oz drink) at \$17	\$0.23	Contains artificial colors and sweeteners. The impact on human health remains controversial (as briefly discussed above).

<sup>a</sup>Dollar range estimate is taken from the top 10 quotes from the GoodRx application.

patients. As seen in our case, lemonade-flavored Crystal Light is a low-calorie option with 117 mEq/L citrate (or 55 mEq in a standard 16 oz bottle).<sup>1</sup> Other nonpharmacologic products found to be high in citrate include lemon juice and orange juice. A randomized cross-over study evaluating commercial orange juice versus lemonade (ie, Minute Maid) found orange juice to have a greater alkalizing and citraturia effect than its lemonade counterpart.<sup>10</sup> However, excess calories and weight gain must be carefully weighed with fruit juices—as an 8 oz serving of orange juice can exceed 110 calories. Freshly squeezed lemon juice has been shown to increase urine citrate 2-fold and is relatively low-calorie (approximately 60 calories per 8 oz) compared with other juices.<sup>11</sup> A reasonable strategy is to add 4 ounces of lemon juice concentrate with 2 liters of water, which can increase both urine citrate and urine volume.<sup>11</sup>

There are a couple of limitations to our case. First, we could not quantify the volume of intake and urine output when our patient transitioned off DMD to water and then Crystal Light. The amount of total fluid intake could influence stone formation. We were also unable to test for urine citrate levels, so—despite our clinical suspicion—it is unknown if our patient had baseline hypocitraturia or if urinary citrate levels increased following Crystal Light supplementation.

In conclusion, we present a fascinating case on the importance of dietary history and how Crystal Light—and other high-citrate products—may act as nonpharmacologic potassium citrate supplement strategies to prevent calcium oxalate kidney stones. In addition to cost, risks, and benefits, patient lifestyle should be weighed in choosing potassium citrate replacements.

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### Ethics Approval

Our institution does not require ethical approval for reporting individual cases or case series.

### Informed Consent

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

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