THE AMERICAN JOURNAL of MEDICINE®

Juicing Is Not All Juicy

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Juicing has become a popular health trend in the last few years. The term "juicing" or "juice cleanse" usually refers to a period of 3-10 days when a person's diet consists mainly of fruit and vegetable juices. It is widely marketed as providing health benefits, including weight loss, flushing toxins from the body, and increasing energy. Up to now there is no strong scientific evidence to support these benefits as compared with eating fruits and vegetables. However, the gospel of juicing is here to stay, mostly through social media. Juicing is clearly an understudied phenomenon in the legitimate medical world. Is it possible that juicing is harmful? Up to now, there have been no reports of juicing-induced damage, until this issue of ***The American Journal of Medicine***, which reports a case of oxalate nephropathy due to 6 weeks of a juicing fast.[1]

For this case report, we are grateful to the patient who kept detailed records of his food consumption. For over 6 weeks, he had taken a daily average of 1260 mg oxalate from beets, collard greens, kiwi, parsley, spinach, and soy products, which are considered a healthy diet and commonly consumed by people practicing juicing. Other co-factors include high vitamin C intake (2 g from supplementary vitamin C and about 0.5 g from juice), low calcium intake, and chronic kidney disease stage 3 with an estimated glomerular filtration rate of 48 mL/min. He had acute renal failure with a high serum oxalate level and required temporary hemodialysis. Fortunately, he recovered his kidney function partially, but had a loss of glomerular filtration rate by 14 mL/min due to the juicing program.[1]

Oxalate is a well-known nephrotoxin and is rich in certain fruits, vegetables, and nuts. The understanding of oxalate absorption in the gastrointestinal tract has been advanced recently after identifying 2 nonspecific oxalate transporters, SLC26a1 and SLC26a6; both are anion exchangers.[2,3] As shown in **Figure 1**, the SLC26a1 is located at the basolateral membrane, while SLC26a 6 is in the apical membrane of intestinal epithelial cells. The knock-out of either transporter leads to hyperoxalemia, hyperoxaluria, and calcium oxalate stones in mice.[2,3] Knauf et al elegantly demonstrated that oxalate is absorbed in the intestine via a passive paracellular pathway.[4] The soluble oxalate is then secreted via a transcellular pathway by entering cells via the SLC26a1 and leaving cells via SLC26a6. The 2 driving forces of this paracellular absorption of oxalate are: oxalate gradient from lumen to blood; and oxalate drag through the tight junction coupled with the water flux.[4] When high-oxalate juice is consumed, the oxalate gradient is increased. In addition, because of increased water flux, the paracellular absorption of oxalate is enhanced, more so than if the same food is eaten.

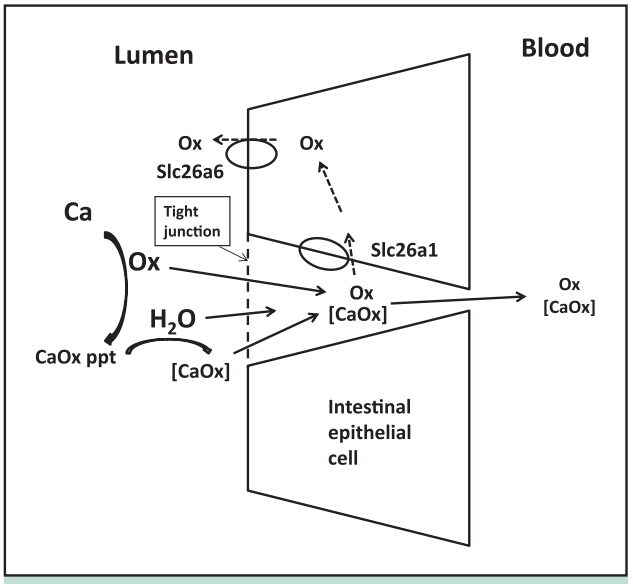

Figure 1 Intestinal oxalate absorption: Oxalate (Ox) is absorbed via a passive paracellular pathway (solid arrows) driven by lumen to blood oxalate gradient and water flux and crosses the tight junction (dashed line between 2 cells on the apical side). Soluble oxalate is secreted via transcellular pathway (broken arrows) via 2 anion exchangers: Slc26a1 at the basolateral membrane and Slc26a6 at the apical membrane. Calcium (Ca) in the lumen binds oxalate and promotes luminal calcium oxalate precipitation (ppt), thus reducing oxalate absorption. In the presence of high water content, more calcium oxalate complex remains soluble ([CaOx]) and can be absorbed via paracellular pathway, but rejected by transcellular pathway.

Funding: None.

Conflict of Interest: None.

Authorship: The author is solely responsible for the content of this manuscript.

0002-9343/$ -see front matter
http://dx.doi.org/10.1016/j.amjmed.2013.04.007

Calcium plays an important role in oxalate absorption.[5] It binds to oxalate and precipitates oxalate out, so oxalate would not be absorbed. Consuming juice with a low calcium intake would allow oxalate to be in soluble form and easily absorbed. In addition, because of high water content, more calcium oxalate complex may remain soluble and be absorbed via the paracellular pathway. Unlike the soluble oxalate, the calcium oxalate complex cannot be secreted out via oxalate transporters (**Figure 1**).[4] This is how juice enhances oxalate absorption even in the presence of calcium.

If the absorbed oxalate is beyond the capacity of the secretion system of the intestine, oxalate will enter the circulatory system and become a burden to the kidney, because oxalate is not metabolized in the body and has to be excreted by the kidney. In addition, oxalate is the end product of many chemicals, including vitamin C.[6] It appears that a high percentage of juicers are taking high-dose vitamin C, as in the case described. As a result, vitamin C is metabolized to oxalate and contributes to the pathogenesis of oxalate nephropathy.

Is this case representing the tip of the iceberg, given that millions are practicing juicing? The index patient did have chronic kidney stage 3, had been on an unusually prolonged juicing program, and unknowingly consumed an excessive amount of oxalate. Patients with chronic kidney disease are prone to oxalate nephropathy due to diminished renal capacity to excrete oxalate. For those with normal renal function and who do not consume high oxalate juice, the risk of juicing nephropathy is likely low. However, because juice consumption enhances oxalate absorption, it is prudent to calculate daily oxalate consumption. For commercial juicing products, it is critical to label the oxalate content and provide the limit of daily consumption for consumer protection. For those who have consumed a high quantity of oxalate, an evaluation including kidney function and urinalysis with microscopic examination to look for oxalate crystals is warranted. Lastly, for physicians encountering patients with acute kidney injury of unknown origin, it is appropriate to ask, "Have you been juicing lately?"

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