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CASE REPORT

WILEY

Extreme diet without calcium may lead to hyperoxaluria and kidney stone recurrence—A case study

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Abstract

Background: The aim of the study was to present a case study of a 56-year-old woman with hyperoxaluria induced by calcium-free diet that resulted in kidney stone recurrence.

Methods: A 24-hour urine collection and serum tests for kidney stone risk factors identification were performed. The monitoring of urine risk factors was done by untimed urine samples and 24-hour urine collections. Polarized light microscopy was performed for kidney stone analysis.

Results: The results of urine collection showed hyperoxaluria of 0.551 mmoL per 24 hours. After adding calcium-containing products to the diet the oxaluria decreased to reference range value of 0.352 mmoL/24 hours and all untimed oxalate to creatinine ratios returned to reference ranges. Polarized light microscopy revealed 100% calcium oxalate kidney stone composition (It was 50% Weddellite and 50% Whewellite).

Conclusions: The case study shows the importance of calcium intake in the prevention of calcium oxalate kidney stone recurrence. Particularly, unsuitable diet without calcium can induce kidney stone recurrence. Knowledge of diet habits is important for interpretation of kidney stone risk factors and their inhibitors excreted in urine.

KEYWORDS

calcium oxalate, hyperoxaluria, diet, risk factors, urolithiasis

1 | INTRODUCTION

Diet plays a key role in kidney stone development. Maintaining a normal BMI, drinking an adequate amount of fluid, eating a diet high in fruits, vegetables, and low-fat dairy products with an adequate intake of calcium, and avoiding frequently drinking sugar-sweetened beverages were associated with a clinically meaningful lower risk of incident kidney stones during follow-up.[1] Salt intake is a very important predictor of urinary calcium excretion. A 100 mmoL higher daily sodium intake predicted a 1.04 mmoL higher daily urinary calcium excretion.[2] Dietary calcium should not be restricted, since calcium combines with oxalate in intestine and only free oxalate can be absorbed. Conversely, free fatty acids in intestine bind calcium in patients with diseases associated with fat malabsorption, which increases amount of free oxalate that can be absorbed and excreted in urine. It is estimated that people on a typical western diet absorb about 10% of ingested oxalate.[3]

The aim of this study was to present a case study of a patient who developed calcium oxalate kidney stones after avoiding calcium in diet for a time period of 1 year.

2 | CASE REPORT

A female aged 56 years suffered from kidney stones since 2012. She had a left-side renal colic in June 2012. The 5-mm stone in left ureter was broken down by extracorporeal shock wave lithotripsy. She was without stones after this procedure. The kidney stone analysis by polarized light microscopy revealed 100% calcium oxalate composition (It was 50% Weddellite and 50% Whewellite).

Further medical history included gallstones, hypertension, and high plasma cholesterol. She was taking losartan 50 mg every 24 hours, metoprolol 200 mg daily, and rosuvastatin 20 mg daily. Her body mass index was 27.3 kg/m^2.

In September 2015, the patient had again a left-side renal colic. Low-dose radiation computer tomography showed 3 mm stones in both upper and lower left renal calyx. She also had a 4-mm stone in left ureter that probably caused the renal colic.

In November 2015, the patient was referred to kidney stone outpatient metabolic clinic. The laboratory blood tests and 24-hour urine collection was performed to find the risk factors for nephrolithiasis. The 24-hour urine sample was collected in unused plastic container with 30 mL of toluene.[4] The completeness of urine collection was checked by calculation of creatinine excretion per body kilogram.

Results of blood tests and 24-hour urine collection are presented in Tables 1 and 2. The main result is the presence of hyperoxaluria. Lower urine volume (the target for kidney stone formers is at least 2.0 L/24 hours) and borderline hypocitraturia are further risk factors of kidney stones development.

The discussion with the patient on her high oxalate content in urine revealed that she did not eat calcium-containing products from December 2014 to December 2015.

At the Metabolic outpatient clinic that is a part of the Department of Clinical Biochemistry and Pharmacology, the patient was advised to eat milk products and drink more fluids, mainly water. After this discussion, she has started to eat balanced diet containing 1000-1200 mg of calcium daily. The patient was prescribed potassium citrate but she discontinued this treatment for stomach discomfort. The long-term monitoring urinary test results of risk factors are included in Table 2. All six urine oxalate monitoring results were within reference ranges. In July 2016, after an endoscopic removal of one of the kidney stones, the stone in lower left calyx remained in its previous position. Polarized light microscopy again revealed 100% calcium oxalate kidney stone composition (It was again 50% Weddellite and 50% Whewellite).

Her kidney stone disease was stable according to sonography until November 2019. The reference ranges for urine excretion of calcium,

TABLE 1 Serum test results on diet without calcium on 25.11. 2015

Serum test	Result	Unit	Reference range
Glucose	5.6	mmol/L	3.9-5.5
Sodium	140	mmol/L	136-144
Potassium	4.6	mmol/L	3.8-5.1
Chloride	105	mmol/L	95-107
Inorganic phosphate	1.16	mmol/L	0.7-1.5
Magnesium	0.97	mmol/L	0.80-0.94
Calcium corrected for 40 g of albumin	2.36	mmol/L	2.10-2.55
HCO_3^-	27.2	mmol/L	22.0-28.0
Urea	4.3	mmol/L	3.0-8.0
Creatinine	72	μmol/L	49-90
eGFR[a] (CKD-EPI[b] equation) from serum creatinine	81	mL/min/1.73 m^2	90-150
Total bilirubin	12	μmol/L	<20
Alanine aminotransferase	38.4	U/L 37°C	<43.8
Aspartate aminotransferase	22.2	U/L 37°C	<40.2
Gamma-glutamyl transferase	24	U/L 37°C	<106.2
Alkaline phosphatase	61.4	U/L 37°C	<150
Total protein	70.7	g/L	64.0-79.0
Albumin	43.9	g/L	36.0-45.0
Parathyroid hormone intact (1-84)	4.3	pmol/L	1.6-6.0

[a]Estimated glomerular filtration rate.

[b]Chronic Kidney Disease Epidemiology Collaboration.

TABLE 2 Results of 24 urine collections and untimed urine samples on diet without and with calcium

Test	Results on the diet without calcium 16.12.2015	Results on the balanced diet 1.8.2016	Results on the balanced diet 22.8.2017	Unit	Reference range
pH (pH meter)	6.01	6.29	5.59	pH units	
Urine volume/24 h	1.900		2.220	L	1.5-3.0
Osmolality	296	537		mmol/kg	
Sodium/24 h	70		92	mmol/24 h	41-227
Potassium/24 h	68		84	mmol/24 h	17-77
Chloride/24 h	91		128	mmol/24 h	40-224
Calcium/24 h	3.8		5.28	mmol/24 h	2.0-5.0
Creatinine/kg/24 h	136		132	μmol/kg/24 h	105-155
Calcium/creatinine ratio	0.36	0.37	0.51	mol/mol	<0.60
Inorganic phosphate/24 h	21		29	mmol/24 h	13-35
Inorganic phosphate/creatinine ratio	2.0	2.31	2.79	mol/mol	0-2.8
Uric acid/24 h	3.6		3.3	mmol/24 h	2.0-4.0
Uric acid/creatinine ratio	0.35	0.37	0.32	mol/mol	<0.30
Magnesium/24 h	4.1		5.5	mmol/24 h	3-5
Magnesium/creatinine ratio	0.39	0.25	0.53	mol/mol	0.2-0.5
Oxalate/24 h	0.551		0.352	mmol/24 h	0-0.5
Oxalate/creatinine ratio	0.053	0.023	0.034	mol/mol	<0.04
Citrate/24 h	2.3		2.0	mmol/24 h	2.5-5.0
Citrate/creatinine ratio	0.22	0.24	0.19	mol/mol	>0.15

magnesium, inorganic phosphate, citrate, and oxalate are adopted from the Urolithiasis Guidelines by the European Association of Urology.[5]

The patient signed an informed consent regarding the publication of her case study. The local hospital Ethic committee approved the publication.

3 | DISCUSSION

We present a case study of a patient on diet without calcium-induced hyperoxaluria and calcium oxalate nephrolithiasis.

Sakhaee reported the main causes of hyperoxaluria can be divided into three categories: increased consumption from oxalate-rich foods, increased intestinal oxalate absorption, and increased hepatic oxalate synthesis as a result of an inborn error in metabolism of oxalate production. His preclinical study suggests that hyperoxaluria in obesity depends on a complex network of inflammatory responses.[6] Increased intestinal absorption is the likely mechanism of hyperoxaluria in this case study and overweight could contribute to hyperoxaluria.

Li et al found that patients with gallstones have increased the risk of development of kidney stones.[7] Our patient has also gallstones. This diagnosis may disturb digestion and lead to increased amount of free fatty acids in intestine. Free fatty acids can bind calcium, which results in increased absorption of free oxalate and urinary oxalate excretion.

In patients with calcium oxalate stones the simplest approach for stone formers is to avoid oxalate-rich foods and to increase the consumption of calcium with each meal.[8] The patient was counseled with the same recommendations.

Sakhaee reported that calcium oxalate kidney stone formers should maintain intake of 1000-1200 mg of calcium, 100 mmoL/day of sodium, and 0.8 g/kg body weight of animal protein.[9] Our patient completely avoided calcium intake, which probably led to increased oxaluria and kidney stone formation.

Curhan et al found that a higher dietary calcium intake may decrease the incidence of symptomatic kidney stones.[10] After adding calcium in diet, the oxalate excretion normalized and kidney stone disease became stable for at least 5 years.

The guideline by American Urological Association states in its guideline statement 10 – Clinicians should counsel patients with calcium oxalate stones and relatively high urinary oxalate to limit intake of oxalate-rich foods and maintain normal calcium consumption.[11] We adopted this approach.

The limitation of this case study is that we did not confirm results of polarized light microscopy by infrared spectroscopy. The polarized light microscopy method in our laboratory has regularly participated in external quality assessment system (www.sekk.cz) with excellent

results. It is useful to confirm results of polarized light microscopy by other analytical methods (infrared spectroscopy or X-ray analysis). Since 2019, we have adopted this approach and we have confirmed kidney stone analysis by infrared spectroscopy.

We can conclude that diet without calcium leads to increased oxalate intestinal absorption and calcium oxalate kidney stone formation. Knowledge of diet habits is important for interpretation of kidney stone risk factors, and their inhibitors excreted in urine.

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