Urolithiasis
(calcium base renal stone)

Dr. Elham Kabiri naeini

Assistant professor of Isfahan University of Medical Sciences

- Introduction
- Classification
- Epidemiology
- Clinical Features
- Complication
- Treatment
- Conclusion

Introduction

Urolithiasis (nephrolithiasis, kidney stone disease) is the abnormal formation and retention of solid phase inorganic and organic concretions in the lumen of the urinary tract.

Kidney stone is not a diagnosis per se but the common manifestation of a variety of underlying causative and pathophysiologic factors.

Nephrolithiasis, or kidney stone disease, is a common, painful, and costly condition.

EPIDEMIOLOGY

- The prevalence of kidney stones has increased steadily over 4 decades.
- The increase was greater in females than males and higher in the aging population.
- African-Americans had a lower risk of urolithiasis compared with whites and Mexican Americans.

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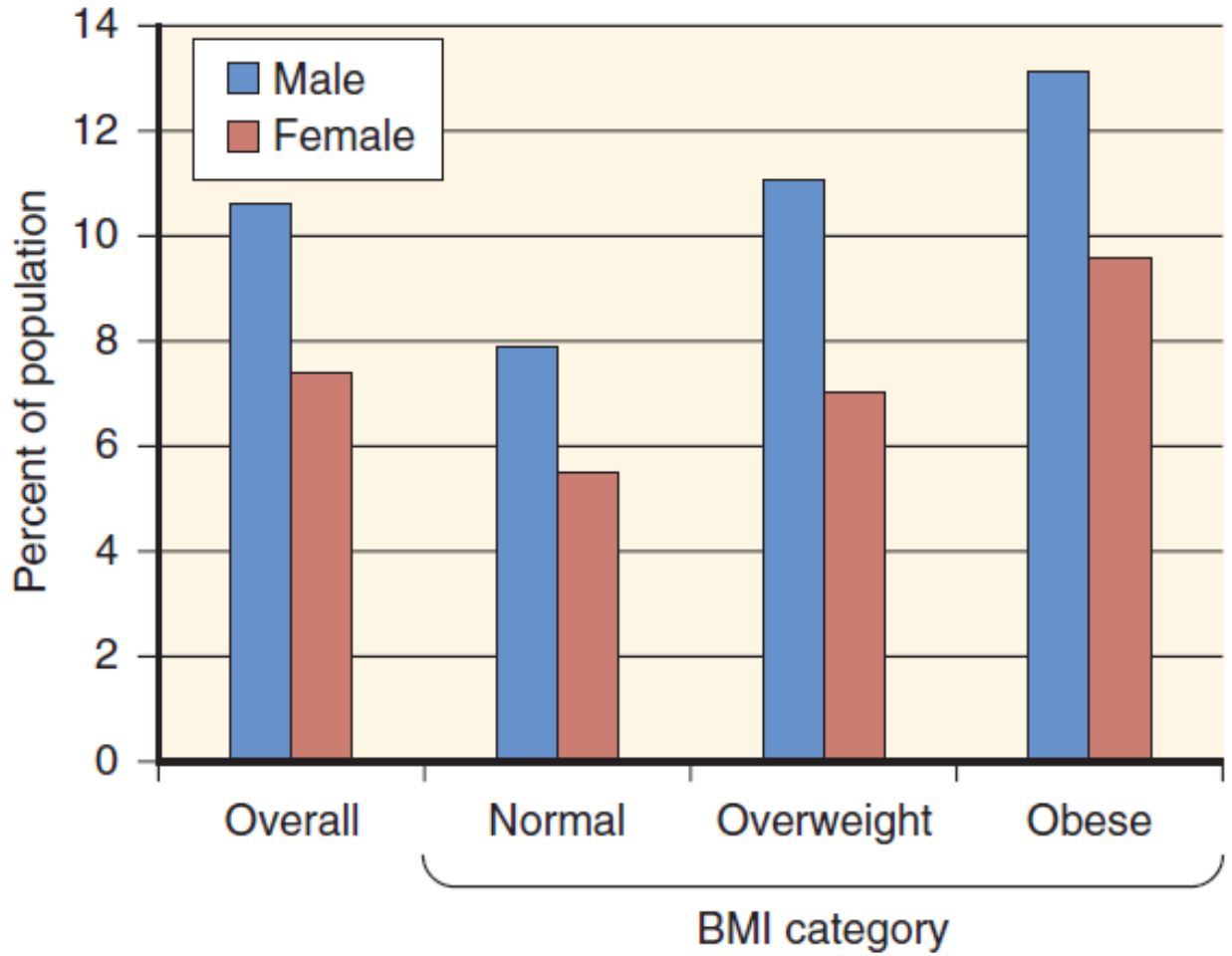
14
12
10
8
6
4
2
0
Percent of population
Male
Female
Overall
Normal
Overweight
Obese
BMI category

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- The association between features of the metabolic syndrome, such as obesity and diabetes, and prevalence of kidney stone disease is consistent with a prospective study showing that the risk of incident kidney stone disease increases with obesity and weight gain.

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- Both age and (BMI) increased with time in both uric acid and calcium stone formers
- uric acid stone formers were consistently older and had a higher BMI and lower urinary pH than calcium stone formers.
- Urinary pH, phosphorus ,oxalate, and sodium increased with time in calcium stone formers, but remained unchanged in uric acid stone formers.

Types of kidney stones

- Calcium oxalate stones are most common (~75%)
- calcium phosphate (~15%)
- Uric acid (~8%)
- struvite (~1%)
- cystine (<1%) stones

ASSOCIATED MEDICAL CONDITIONS

- gastrointestinal malabsorption (e.g., Crohn’s disease, gastric bypass surgery)
- primary hyperparathyroidism, obesity, type 2 diabetes mellitus, and distal renal tubular acidosis
- hypertension, gout, cardiovascular disease
- cholelithiasis, reduced bone mineral density, and chronic kidney disease

PATHOGENESIS

- A clinically useful concept is supersaturation (the point at which the concentration product exceeds the solubility product).

RISK FACTORS

- Dietary Risk Factors

- Dietary factors that are associated with an increased risk of nephrolithiasis include:

Animal protein, oxalate, sodium, sucrose, and fructose.

- Dietary factors associated with a lower risk include:

Calcium, potassium, and phytate, magnesium.

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Calcium intake

Higher dietary calcium intake is related to a lower risk of stone formation.

The reduction in risk associated may be due to a reduction in intestinal absorption of dietary oxalate that results in lower urine oxalate.

Low calcium intake is contraindicated as it increases the risk of stone formation and may contribute to lower bone density in stone formers.

Despite similar bioavailability, supplemental calcium main increase the risk of stone formation.

Risk factors

- Dietary oxalate is only a weak risk factor for stone formation, urinary oxalate is a strong risk factor for calcium oxalate stone formation, and efforts to avoid high oxalate intake should thus be beneficial.

- Higher intake of animal protein may lead to increased excretion of calcium and uric acid

- Vitamin C supplements are associated with an increased risk of calcium oxalate stone formation in men

FLUIDS AND BEVERAGES

- The risk of stone formation increases as urine volume decreases.
- When the urine output is <1 L/d, the risk of stone formation more than doubles.
- Fluid intake is the main determinant of urine volume, and the importance of fluid intake in preventing stone formation

Urinary Risk Factors

- URINE VOLUME
- URINE CALCIUM
- URINE OXALATE
- URINE CITRATE
- URINE URIC ACID
- URINE pH

CLINICAL PRESENTATION AND DIFFERENTIAL DIAGNOSIS

Renal colic and painless gross hematuria are two common presentations for individuals with an acute stone event

Other diagnoses may be confused with acute renal colic:

- acute cholecystitis
- Appendicitis
- diverticulitis
- UTI

DIAGNOSIS AND INTERVENTION

- The diagnosis is often made on the basis of the history, physical examination, and urinalysis.

- The diagnosis is confirmed by an appropriate imaging preferably helical computed tomography (CT), which is highly sensitive, allows visualization of uric acid stones (traditionally considered “radiolucent”)

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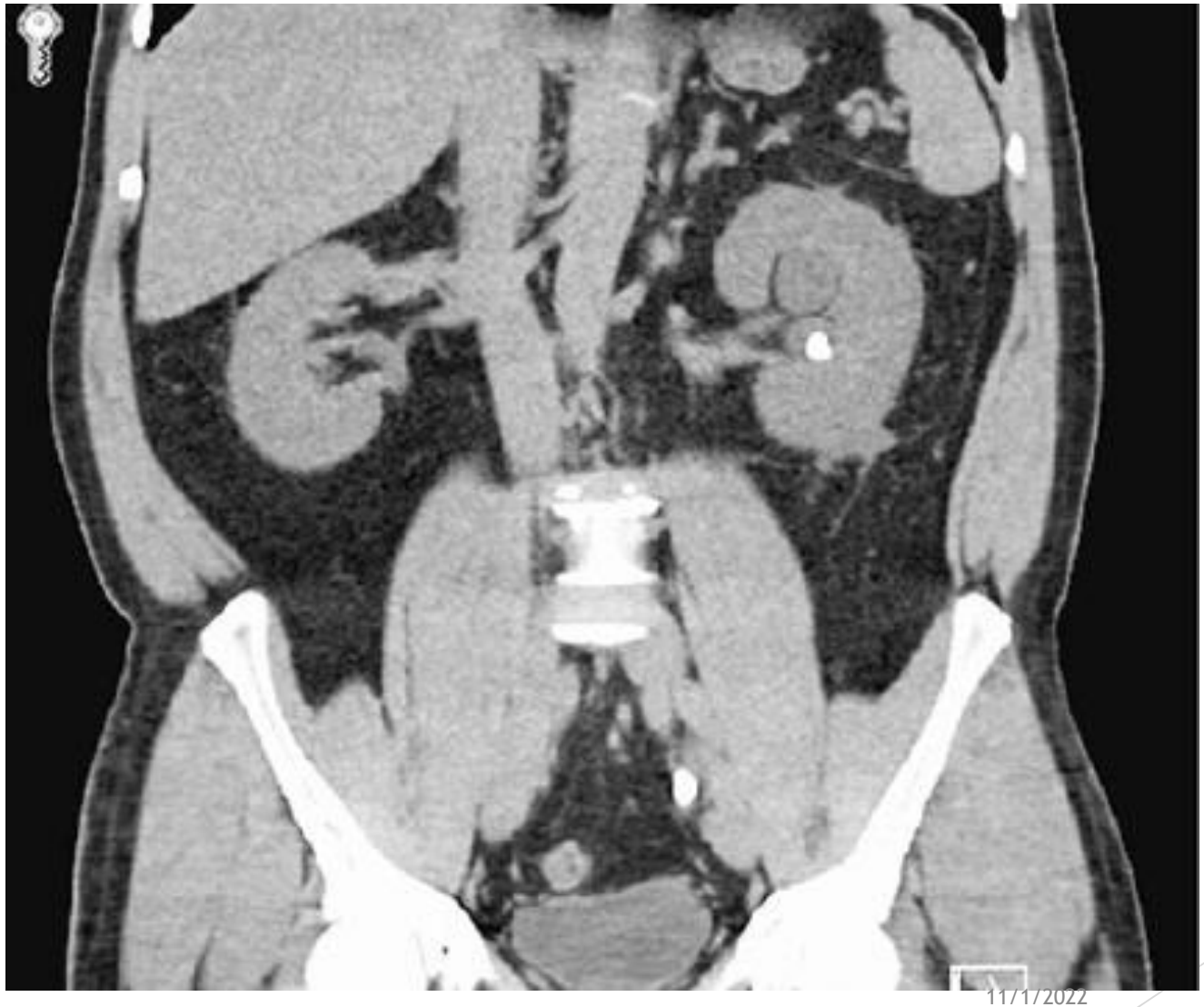

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Urologic intervention should be postponed unless there is:

- evidence of UTI
- a low probability of spontaneous stone passage (e.g., a stone measuring ≥6 mm or an anatomic abnormality)
- intractable pain

EVALUATION FOR STONE PREVENTION

It is appropriate to proceed with an evaluation even after the first stone if the patient is interested because recurrences are common.

The physical examination should assess weight, blood pressure, costovertebral angle tenderness, and lower-extremity edema as well as signs of other systemic conditions

LABORATORY EVALUATION

- creatinine, calcium, and uric acid.
- electrolytes
- The PTH level
- 25-Hydroxy vitamin D
- Urinalysis
- VBG
- 24-h urine collections:

(total volume, calcium, oxalate, citrate, uric acid, sodium, phosphorus, Creatinine,cystin,urate

Random 24-Hour Urinary Profile	Expected Values (per day)	Interpretation
Total volume	≥2.5 L	Indicative of daily fluid intake (minus insensible losses); diminishes with low fluid intake, sweating, and diarrhea
pH	5.9–6.2	<5.5—increases risk of uric acid precipitation; commonly found in idiopathic uric acid stone patients, subjects with intestinal disease and diarrhea, and in those with intestinal bypass surgery >6.7— increases risk of calcium phosphate precipitation; commonly found in patients with dRTA, primary hyperparathyroidism, alkali, and carbonic anhydrase treatment >7.0–7.5— indicates urinary tract infection from urease-producing bacteria.
Creatinine	15–25 mg/kg body weight (0.13–0.22 mmol/kg body weight)	Assessment of completeness of collection: 15–20 mg/kg body weight (0.13–0.15 mmol/kg body weight) in females, 20–25 mg/kg body weight (0.15–0.22 mmol/kg body weight) in males; valid only in steady state of constant serum creatinine concentration with time
Sodium	100 mEq (100 mmol)	Reflects dietary sodium intake (minus extrarenal loss); much lower than dietary intake in diarrhea and with excessive sweating; high sodium intake is major cause of hypercalciuria
Potassium	40–60 mEq (100 mmol)	Reflects dietary potassium intake (minus extrarenal loss); much lower than dietary intake in diarrhea states; gauge of dietary alkali intake because most dietary potassium accompanied by organic anions
Calcium	≤250–300 mg (≤6.24–7.49 mmol)	A higher value expected in males; in states of zero balance, urinary calcium excretion is net gut absorption minus net bone deposition; secondary causes should be ruled out before making the diagnosis of idiopathic hypercalciuria
Magnesium	30–120 mg (1.23–4.94 mmol)	Low urinary magnesium detected with low magnesium intake, intestinal malabsorption (small bowel disease), and following bariatric surgery; low magnesium may increase risk of calcium stones.
Oxalate	≤45 mg (≤0.51 mmol)	Commonly encountered with intestinal disease with fat malabsorption, such as inflammatory bowel disease and following bariatric surgery; values >100 mg/day

		should be ruled out before making the diagnosis of idiopathic hypercalciuria
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Oxalate	≤45 mg (≤0.51 mmol)	Commonly encountered with intestinal disease with fat malabsorption, such as inflammatory bowel disease and following bariatric surgery; values >100 mg/day (1.14 mmol/day) suggest primary hyperoxaluria (PH); the diagnosis of PH I and PH II is further established with high urinary glycolate and L-glycerate levels.
Phosphorus	≤1100 mg (35.5 mmol)	Indicative of dietary organic and inorganic phosphorus intake and absorption; a higher excretion may increase the risk of calcium phosphate stone formation.
Uric acid	600–800 mg (3.57–4.76 mmol)	Hyperuricosuria is encountered with overproduction of endogenous uric acid or overindulgence of purine-rich foods such as red meat, poultry, and fish; mainly a risk factor for calcium oxalate stones when UpH is >5.5 but is a risk factor for uric acid stone when UpH < 5.5.
Sulfate	≤20 mmol	Sulfate is a marker of dietary acid intake (oxidation of sulfur-containing amino acids).
Citrate	≥320 mg (≥1.67 mmol)	Inhibitor of calcium stone formation; hypocitraturia is commonly encountered in metabolic acidosis, dRTA, chronic diarrhea, excessive protein ingestion, strenuous physical exercise, hypokalemia, intracellular acidosis, with carbonic anhydrase inhibitor drugs (e.g., acetazolamide, topiramate, zonisamide), but rarely with ACE inhibitors
Ammonium	30–40 mEq (30–40 mmol)	Ammonium is a major carrier of H^+ in the urine; its excretion corresponds with urinary sulfate (acid load); a higher ammonium-to-sulfate ratio indicates GI alkali loss.
Chloride	100 mEq (100 mmol)	Chloride varies with sodium intake.
Cystine	<30–60 mg (<0.12–0.25 mmol)	Cystine has a limited urinary solubility, at 250 mg/L.

CALCIUM STONES

- PREVALENCE OF CALCIUM STONES

A study from France has shown an increased prevalence of calcium phosphate stone in men versus women from 1980 to 2004, from 9.4% to 26.6% and the increase was associated with a gradual rise in BMI during this period.

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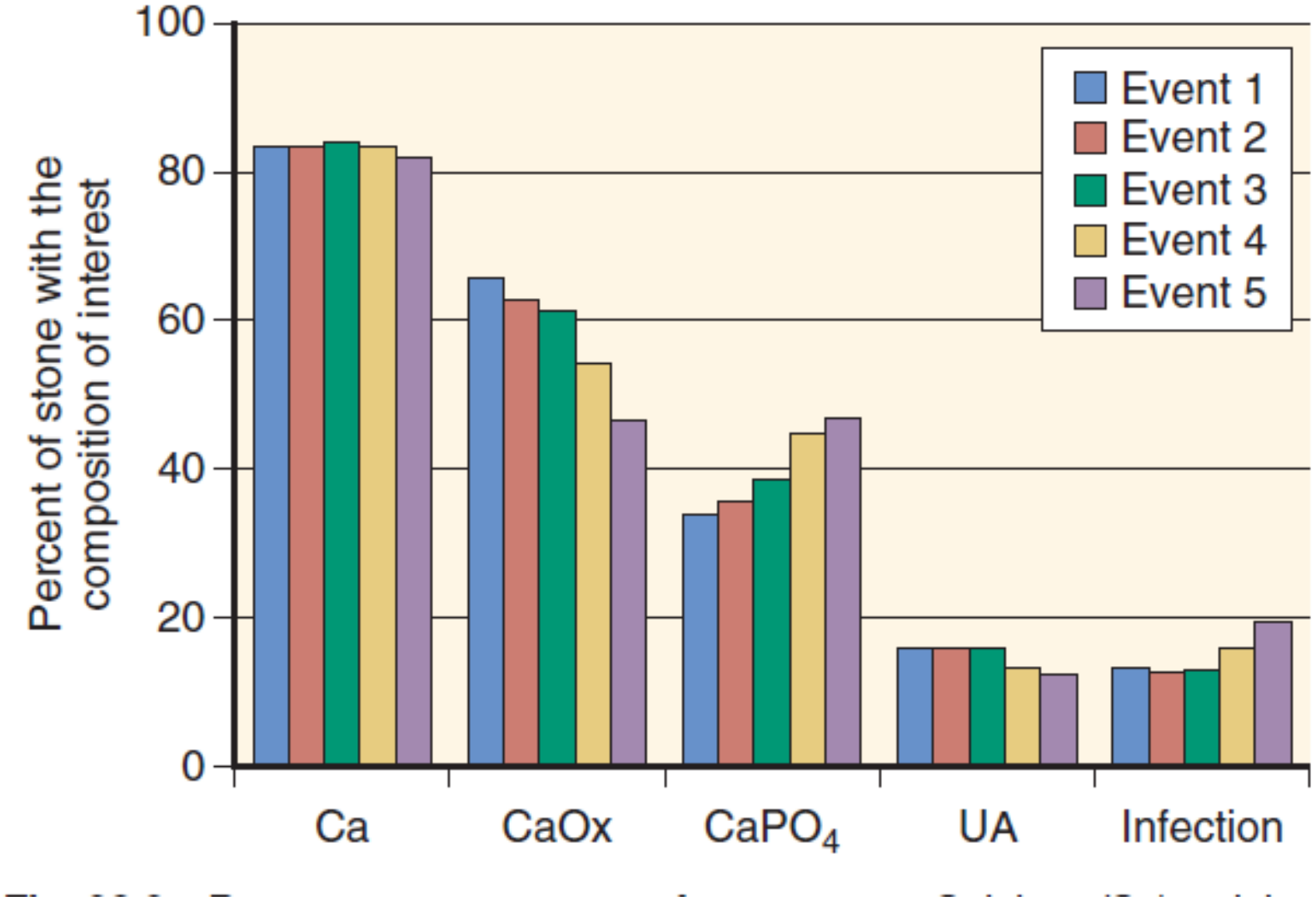

Percent of stone with the composition of interest
100
80
60
40
20
0
Event 1
Event 2
Event 3
Event 4
Event 5
Ca
CaOx
$CaPO_4$
UA
Infection

CALCIUM INTAKE

- The role of dietary calcium as a risk factor was assessed showing that low dietary calcium intake is associated with a higher risk of kidney stones in women and young men.

- On the contrary, calcium supplementation was associated with a higher risk of kidney stones solely in older women.

VITAMIN D INTAKE

- Vitamin D deficiency is highly prevalent in kidney stone formers, of which 20% are hypercalciuric.

- Thus, vitamin D repletion should not be avoided in the kidney stone population, but urinary calcium excretion should be assessed during the repletion period.

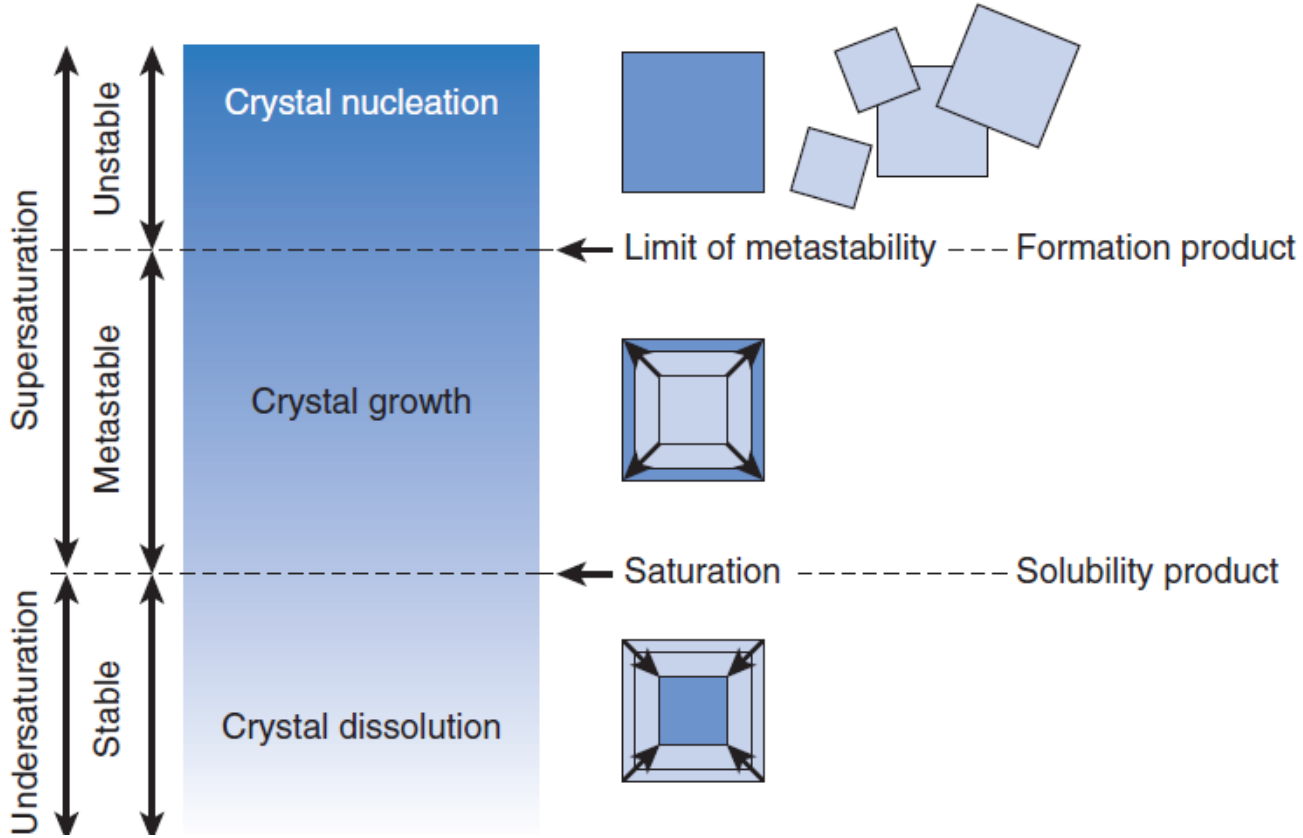
OXALATE INTAKE

- The heterogeneity of cause among the stone formers, and the magnitude of the effect of dietary oxalate.

PROTEIN CONSUMPTION

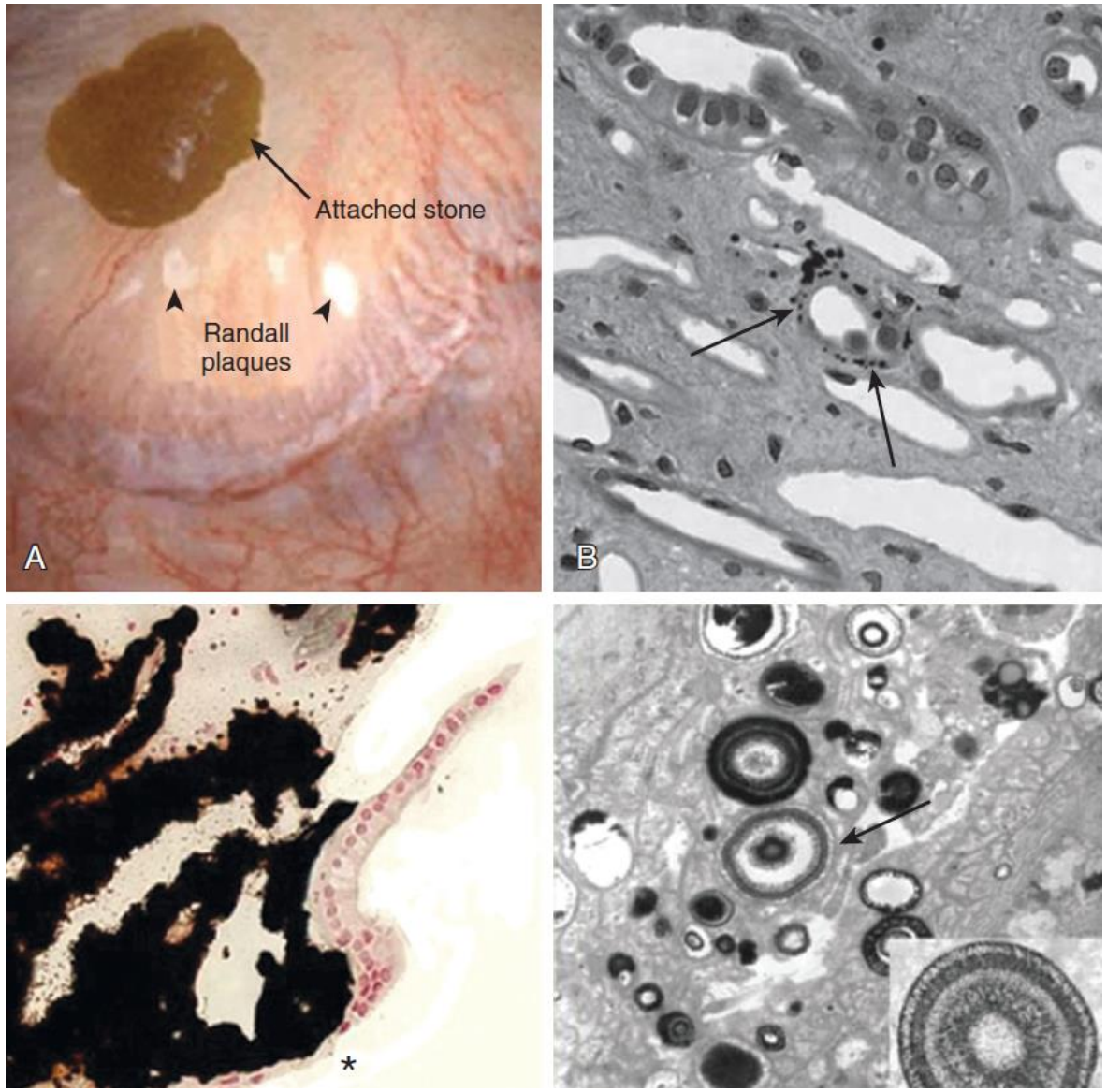
- Epidemiologic studies have demonstrated a positive relationship between animal protein consumption and kidney stone formation in men, but not as much in women.

HISTOPATHOLOGY



RANDALL'S PLAQUE

- Randall concluded that the attached stones were growing from the interstitial calcium plaque, rather than directly from the normal epithelium.


Attached stone
Randall plaques
A
B
*

CALCIUM PHOSPHATE STONES

persistently high pH is seen in CaP stone formers with distal renal tubular acidosis (dRTA) from congenital or acquired causes.

The abundance of CaP crystals is correlated with:

- Higher urinary CaP supersaturation
- High urine pH
- A lesser extent, by hypocitraturia and hypercalciuria.

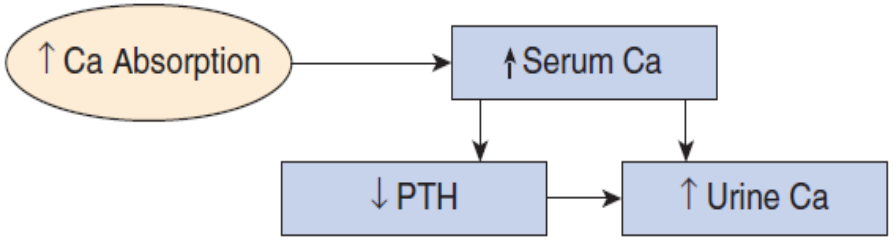
CALCIUM STONES

Risk factors for CaOx stone formation include:

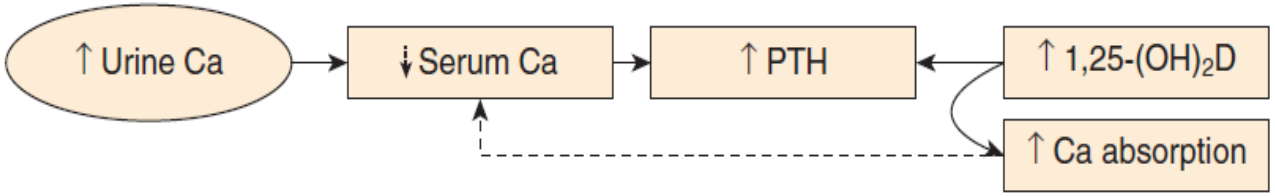
- hypercalciuria
- hyperuricosuria
- hypocitraturia
- hyperoxaluria
- Altered urinary pH

- CaP stones share some common mechanisms :in contrast to CaOx, unduly alkaline urine is characteristic among CaP stone formers but not in CaOx stone formers.

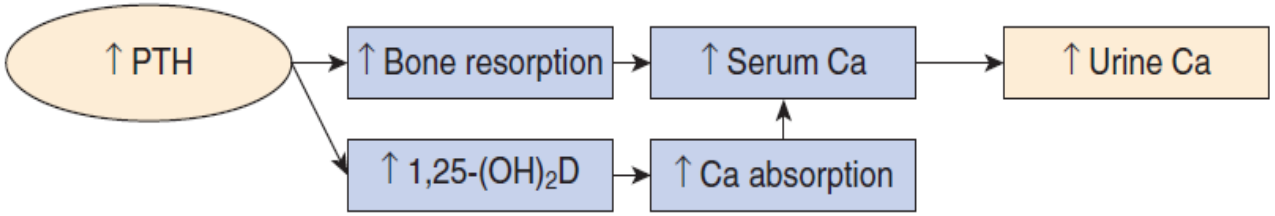
Absorptive hypercalciuria

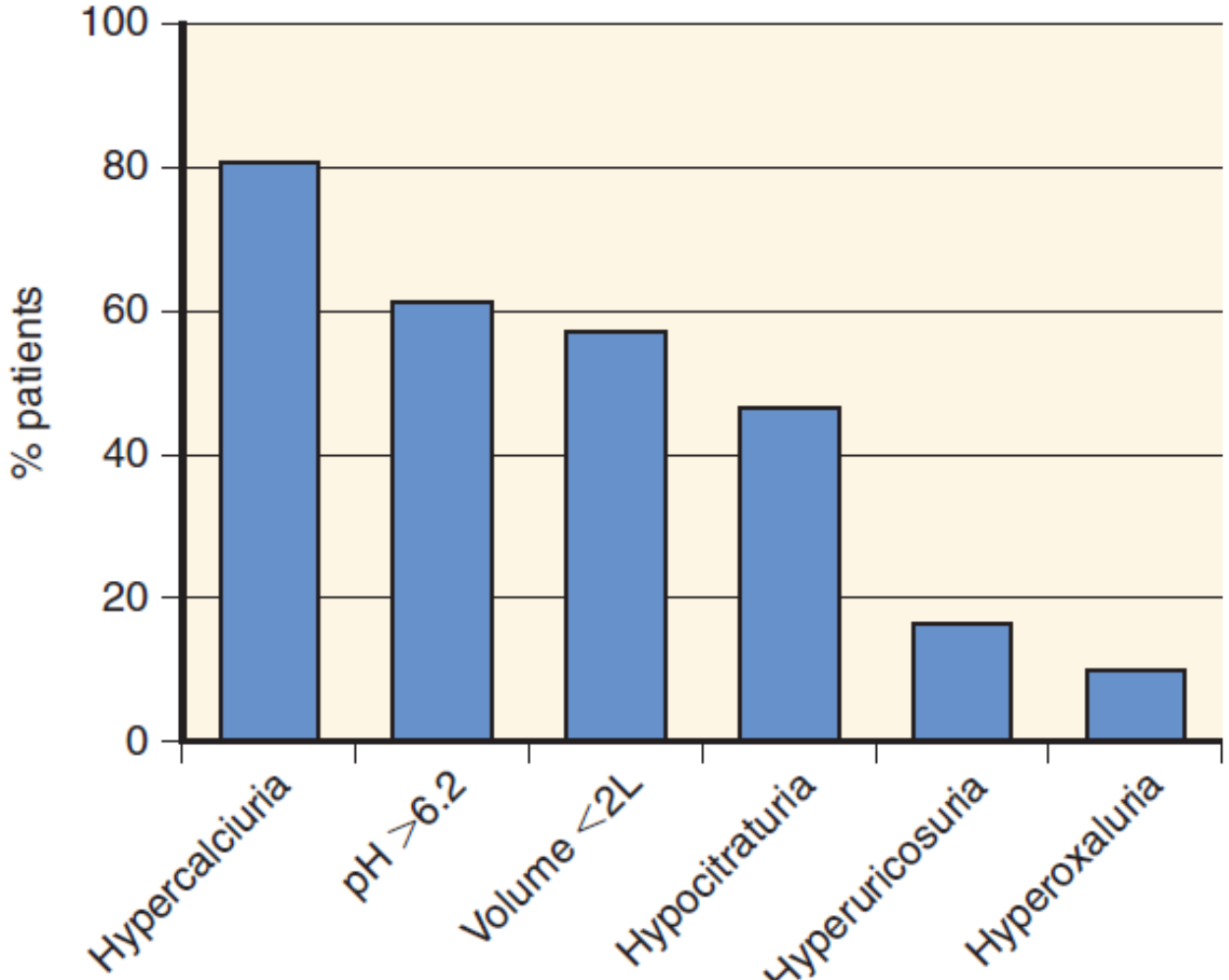


Renal hypercalciuria



Primary hyperparathyroidism




100
80
60
40
20
0
% patients
Hypercalciuria
pH >6.2
Volume <2L
Hypocitraturia
Hyperuricosuria
Hyperoxaluria

INHIBITORS

Classify inhibitors into four groups:

- multivalent metallic cations such as magnesium
- small organic anions such as citrate
- small inorganic anions such as pyrophosphate
- macromolecules such as osteopontin and Tamm-Horsfall protein

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Table 38.1 Natural Inhibitors of Stone Formation

Inhibitor	Examples
Inorganic	Magnesium
	Pyrophosphate
Small organic anion	Citrate
Macromolecules	Bikunin
	Calgranulin
	FK binding protein-12
	Glycosaminoglycans
	Nephrocalcin
	Osteopontin
	Prothrombin F1 fragment
	Lithostathine
	Matrix Gla protein
	Tamm-Horsfall protein
	Urinary trefoil factor-1

CALCIUM STONES

HYPERCALCIURIA

INTESTINAL HYPERABSORPTION OF CALCIUM

RENAL LEAK HYPERCALCIURIA

RESORPTIVE HYPERCALCIURIA

HYPERURICOSURIA

HYPOCITRATURIA

HYPEROXALURIA

Hyperoxaluria

PH typically presents during early childhood with calcium oxalate stones and nephrocalcinosis.

Enteric hyperoxaluria because of inflammatory bowel disease, jejunoileal bypass, and modern bariatric surgeries for morbid obesity are the most common cause of hyperoxaluria in the clinical practice.

(RYGB) is a weight reduction procedure theoretically combining restrictive and malabsorptive mechanisms for the treatment of obesity. Urolithiasis is is well-known complication of subjects who under RYGB.

ALTERATIONS OF URINARY PH

- Both highly acidic (≤5.5) and highly alkaline (≥6.7) urine increase the propensity for calcium kidney stone formation.

- Highly alkaline urine increases the abundance of monohydrogen phosphate which, in combination with calcium, transforms to thermodynamically unstable brushite (CaHPO42H2O) and, finally to hydroxyapatite

- The three main risk factors for the development of brushite stone are alkaline urine, hypercalciuria, and hypocitraturia

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Calcium Kidney Stones: Pathogenesis, Evaluation, and Treatment Options

Helen L. Figge, RPh, PharmD, MBA, Lean Six Sigma Certified
Senior Director, Career Services

Drug-Induced Factors:

These include:

- acetazolamide, amphotericin B
- calcium antacids, calcium supplements, glucocorticoids, loop diuretics, theophylline, and vitamin D

- Acetazolamide contributes to calcium stone formation in part by inducing a mild metabolic acidosis
- Calcium antacids and supplements increase the load of absorbed calcium and hence increase the amount of excreted calcium.
- Loop diuretics directly stimulate urinary excretion of calcium.

Patient Evaluation and Management

An unenhanced helical CT scan is generally the most sensitive method for establishing the presence of a renal stone.

- Ureteral stones smaller than 5 mm will generally pass spontaneously with adequate hydration.
- Stones greater than 10 mm will generally not pass spontaneously and will require urologic intervention.
- Stones ranging from 5 mm to 10 mm will have variable outcomes

Continue……

- Pain may be managed
- Intravenous hydration
- Alpha - adrenergic receptor blockers or calcium channel blockers are sometimes prescribed to assist with stone passage.
- Analysis of the stone composition is critical for ongoing management.
- Basic lab-oratory evaluation
- Once the acute episode has passed, certain patients should undergo a complete laboratory evaluation.

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Pathogenesis of calcium oxalate urinary stone disease: species comparison of humans, dogs, and cats

Allison L. O'Kell[a,b], **David C. Grant**[c], and **Saeed R. Khan**[b,d]

[a]Department of Small Animal Clinical Sciences, The University of Florida, 2015 SW 16th Ave, PO Box 100126 Gainesville FL 32610 USA

[b]Department of Urology, The University of Florida, 1600 SW Archer Road, Gainesville, Florida, 32610, USA

[c]Department of Small Animal Clinical Sciences, Virginia Tech, 215 Duck Pond Drive, Blacksburg VA, USA 24061

[d]Department of Pathology, Immunology, and Laboratory Medicine, The University of Florida, 1600 SW Archer Road, Gainesville, Florida, 32610, Gainesville, Florida, USA

Abstract

Idiopathic calcium oxalate nephrolithiasis is a highly recurrent disease that is increasing in prevalence. Decades of research have not identified effective methods to consistently prevent the formation of nephroliths or induce medical dissolution. Idiopathic calcium oxalate nephroliths form in association with renal papillary subepithelial calcium phosphate deposits called Randall's plaques (RPs). Rodent models are commonly used to experimentally induce calcium oxalate

Continue……

- Calcium oxalate kidney stones are more common in men
- Calcium oxalate stones are often recurrent, with a 10 year recurrence rate of 30% in first time stone formers
- The relationship of calcium oxalate kidney stones to important co-morbidities such as chronic kidney disease and metabolic syndrome is unclear. Kidney stones have been associated with a higher risk of chronic kidney disease in some studies
- Although when stone composition was evaluated, patients with calcium containing stones had better renal function than patients with non-calcium containing stones

Attending Rounds

A Woman with Recurrent Calcium Phosphate Kidney Stones

David S. Goldfarb

Summary

Kidney stones composed predominantly (50% or more) of calcium phosphate constitute up to 10% of all stones and 15%–20% of calcium stones, 80% of which are composed of calcium oxalate. Calcium phosphate is a minor component of up to 30% of calcium oxalate stones as well. The cause of calcium phosphate stones is often obscure but most often related to a high urine pH. Some patients with calcium phosphate stones may have incomplete renal tubular acidosis. Others have distal renal tubular acidosis characterized by hyperchloremic acidosis, hypocitraturia, and high urine pH. The use of carbonic anhydrase inhibitors such as acetazolamide, topiramate, and zonisamide leads to a similar picture. Treatment options to specifically prevent calcium phosphate stone recurrence have not been tested in clinical trials. Increases in urine volume and restriction of sodium intake to limit calcium excretion are important. Citrate supplementation is probably effective, although the concomitant increase in urine pH may increase calcium phosphate supersaturation and partially offset the inhibition of crystallization resulting from the increased urine citrate excretion and the alkali-associated reduction in urine calcium

Nephrology Section, New York Harbor Department of Veterans Affairs Healthcare System, New York, New York; Endourology Section, Lenox Hill Hospital, New York, New York; and Division of Nephrology, New

Therapeutic approach to calcium phosphate stone formers

Fluid therapy:

- increase fluid intake up to 3 L or more to ensure urine output 2.5 L/d
- higher fluid intake should be consistent throughout the day
- increase fluid intake with exercise
- increase fluid intake with meals
- increase fluid intake before bedtime but minimize disturbance of sleep
- most fluid intake should be water
- avoid grapefruit juice (associated with more stones)
- limit cola (may have adverse effects on urine chemistry)

David S. Goldfarb.cjasn. 2012

Dietary therapy

- prescribe diet based on 24-h urine data but educate patient about general principles
- limit sodium intake to 2 g (about 100 meq) per day
- limit oxalate intake if calcium oxalate is an important stone component
- reduce intake of high oxalate foods accompany ingested oxalate with dairy products
- moderate protein intake (e.g., 1.2 g/kg per day)
- two to three servings of dairy per day may be desirable (if calcium oxalate is a stone component)

Pharmacologic therapy

Judge efficacy based on results of 24-h urine collections

- Potassium citrate 20–30 meq two times per day
- one dose at bedtime to cover lower urine volume and higher calcium concentrations

benefit is uncertain if

- urine volume does not increase
- urine calcium does not fall
- urine citrate does not rise
- urine pH rises

Continue……

Thiazides:

- may be useful even if hypercalciuria is not present
- chlorthalidone 12.5–50 mg one time per day
- hydrochlorothiazide 25–50 mg one or two times per day
- indapamide 2.5–5.0 mg one time per day
- maintain normal serum potassium concentration with potassium citrate if hypocitraturia is present or potassium chloride if hypocitraturia is not present
- if necessary, consider amiloride 5–10 mg two times per day, spironolactone 25 mg two times per day, or eplerenone 25–50 mg one time per day
- avoid poorly soluble triamterene

PRACTICE

Strategies for preventing calcium oxalate stones

About 10% of people will experience nephrolithiasis in their lifetime, and about 70% of those will have recurrences. About 80% of stones are calcium based, and about 80% of those are calcium oxalate stones. We discuss here briefly the evidence for the prevention of calcium oxalate stones through dietary and pharmacologic measures.

Urinary risk factors

cium stones, yet its cause in most patients remains unclear. It is still often referred to as "idiopathic hypercalciuria." The efficacy of classifying hypercalciuria on the basis of cause remains controversial and of unproven value in clinical management.

Dietary modification

Fluid intake

The cornerstone of management is to increase urine volume. The effect appears to be linear, with a point of diminishing return reached at urine vol-

Calcium

Studies of dietary calcium's effect on stone recurrence rates have led to major changes in nonpharmacologic manipulation. Epidemiologic evidence shows an inverse relation between dietary calcium intake and recurrence rates. This is probably best explained by calcium's inhibition of intestinal oxalate absorption. A randomized controlled trial (RCT) assigned men with hypercalciuria to follow either a diet low in calcium (400 mg) and oxalate or a diet higher in calcium (1200 mg) with restricted intake of oxalate, protein and

Risk factor	Causes	Treatment	Level of evidence
Low urine volume	Exercise, sweating, low fluid intake, heat, bowel disease	Increase fluid intake to 2.5-3.0 L/d	RCT (Borghi et al. *J Urol* 1996;155:839-43)
Hypercalciuria	Idiopathic, vitamin D intoxication	Reduce sodium intake; begin thiazide diuretic therapy; do not reduce calcium intake except in extreme cases; increase dietary calcium intake to 1000 mg	RCT (Borghi et al. *N Engl J Med* 2002;346:77-84)
Hyperoxaluria	Dietary ingestion and endogenous metabolism; inflammatory bowel disease	Restrict oxalate intake; increase dairy intake accompanying oxalate-containing foods	No RCT (Holmes et al. *Urol Res* 2004;32:311-6)
Hypocitraturia	Renal tubular acidosis; other metabolic acidoses; chronic bowel disease; often idiopathic	Begin potassium citrate supplementation	RCT (Barcelo et al. *J Urol* 1993;150:1761-4)
Hyperuricosuria	Excessive purine ingestion as animal protein	Reduce purine ingestion; begin allopurinol therapy	RCT (Ettinger et al. *N Engl J Med* 1986;315:1386-9)
Increased sodium excretion	Excessive dietary sodium intake	Restrict sodium intake	No RCT of sodium restriction alone (Borghi et al. *N Engl J Med* 2002;346:77-84)

Box 1: Foods most commonly responsible for raising urine oxalate levels

- Rhubarb
- Dark green leafy vegetables (e.g., spinach, chard, arugula)
- Beets
- Wheat bran
- Nuts, seeds
- Soy products
- Chocolate
- Tea
- Strawberries

Source: Massey L et al. *J Am Diet Assoc* 1993;93: 901-6. See also www.ohf.org/docs/Oxalate2004.pdf and www.litholink.com/patientsDietInfo.htm

Moving Points
in Nephrology

Pathophysiology-Based Treatment of Idiopathic Calcium Kidney Stones

Fredric L. Coe, Andrew Evan,† and Elaine Worcester**

Summary

Idiopathic calcium oxalate (CaOx) stone-formers (ICSFs) differ from patients who make idiopathic calcium phosphate (CaP) stones (IPSFs). ICSFs, but not IPSFs, form their stones as overgrowths on interstitial apatite plaque; the amount of plaque covering papillary surface is positively correlated with urine calcium excretion and inversely with urine volume. The amount of plaque predicts the number of recurrent stones. The initial crystal overgrowth on plaque is CaP, although the stone is mainly composed of CaOx, meaning that lowering supersaturation (SS) for CaOx and CaP is important for CaOx stone prevention. IPSFs, unlike ICSFs, have apatite crystal deposits in inner medullary collecting ducts, which are associated with interstitial scarring. ICSFs and IPSFs have idiopathic hypercalciuria, which is due to decreased tubule calcium reabsorption, but sites of abnormal reabsorption may differ. Decreased reabsorption in proximal tubules (PTs) delivers more calcium to the thick ascending limb (TAL), where increased calcium reabsorption can load the interstitium, leading to plaque formation. The site of abnormal reabsorption in IPSFs may be the TAL, where an associated defect in bicarbonate reabsorption could produce the higher urine pH characteristic of IPSFs. Preventive treatment with fluid intake, protein and sodium restriction, and thiazide will be effective in ICSFs and IPSFs by decreasing urine calcium concentration and CaOx and CaP SS and may also decrease plaque formation by increased PT calcium reabsorption. Citrate may be detrimental for IPSFs if urine pH rises greatly, increasing CaP SS. Future trials should examine the question of appropriate treatment for IPSFs.

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*Department of Medicine, University of Chicago, Chicago, Illinois; and †Department of Anatomy and Cell Biology, Indiana University School of Medicine, Indianapolis, Indiana

Correspondence:
Dr. Elaine Worcester, University of Chicago, Section of Nephrology, 5841 South Maryland/ Mailing Code 5100, Chicago, IL 60637. Phone: 773-702-7459; Fax: 773-702-5818; E-mail: eworcest@bsd.uchicago.edu

Introduction

The common measures of reduced diet sodium and protein, increased fluids, thiazide, potassium citrate, reduced diet purine, and allopurinol have until now been viewed from the perspective of altering urine supersaturations and inhibitors of crystallization. Here, we add to this familiar theme the new work concerning how stones actually form and how the mechanisms that drive their formation actually function. The result is a new level of understanding in the use of accepted treatments that should bring to physicians and their patients a greater confidence and subtlety of management.

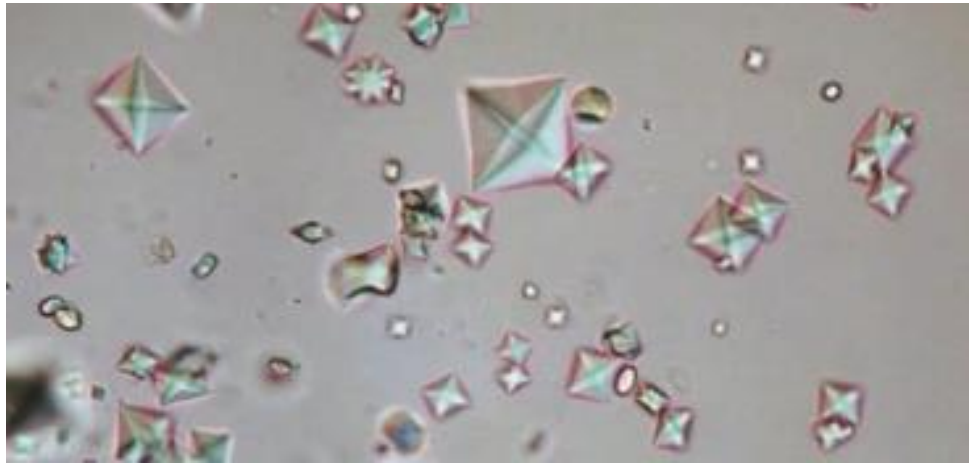
The new work has divided calcium stone-formers not only by their clinical appearances but also their renal pathology. We have always distinguished patients who form calcium stones because of systemic disease from idiopathic calcium stone-formers. However, we now know that idiopathic calcium stone-pathic." Among these, most (1) form stones for which the most abundant crystal is calcium oxalate (CaOx). The kidneys of idiopathic CaOx stone-formers (ICSFs) are normal except for papillary interstitial apatite deposits (2,3) that appear as white clouds (Figure 1A) under the urothelium during ureteroscopy (URS) or percutaneous nephrolithotomy (PERC). The kidney stones grow over these deposits, often called "white plaque," on the outside of the papilla (Figure 1B). No crystals are seen within the epithelial compartments (4). The deposits evoke no obvious inflammation, and renal papillae are completely normal in appearance except for plaque and overgrowing stones.

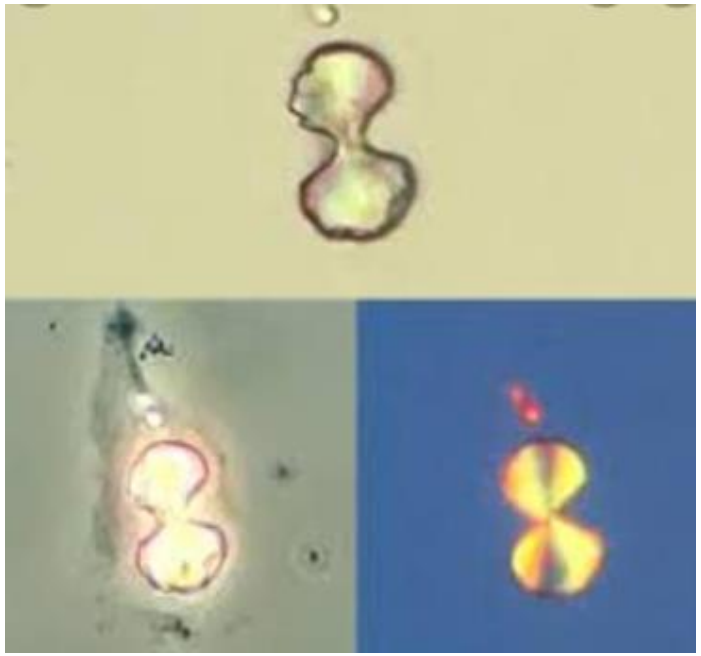
Plaque begins as collections of tiny microspherules that form in the basement membranes of the papillary thin limbs of the loops of Henle (Figure 1C) and spread from there into the interstitium and eventually beneath the urothelium and basement membranes of inner medullary collecting ducts (IMCDs) and termi-

- IPSFs, unlike ICSFs, have apatite crystal deposits in inner medullary collecting ducts, which are associated with interstitial scarring.
- ICSFs and IPSFs have idiopathic hypercalciuria, which is due to decreased tubule calcium reabsorption, but sites of abnormal reabsorption may differ.

- Decreased reabsorption in proximal tubules (PTs) delivers more calcium to the thick ascending limb (TAL), where increased calcium reabsorption can load the interstitium, leading to plaque formation.
- The site of abnormal reabsorption in IPSFs may be the TAL, where an associated defect in bicarbonate reabsorption could produce the higher urine pH characteristic of IPSFs.

Fredric L. Coe.2011 by the American Society of Nephrology*





conclusion

Stone prevention is also deemed valuable by many Patients

- Avoidance of time-consuming and humiliating emergency room visits,
- Adequate pain relief
- Exposure to repeated doses of ionizing radiation are all highly worthy goals.


Thank
you