urine volume, pH, and levels of calcium, uric acid, citrate, oxalate, sodium, urea nitrogen, phosphate, and creatinine to assess the completeness of the collection. Preferably, two collections are performed in outpatient settings while consuming a usual diet. The findings are important for prevention of recurrence.

In the acute setting, noncontrast helical CT has replaced intravenous urography as the test of choice for the diagnosis of renal stones. CT can detect opaque and radiolucent stones with high sensitivity and specificity. Ultrasound can also detect radiolucent and radiopaque stones in kidneys but may miss ureteral stones. Ultrasound has a role in evaluating stones in pediatric and pregnant patients.

Treatment and Prevention

In most cases, small (<4 mm), nonobstructive stones can be managed conservatively because they have a good chance of spontaneous passage. With increases in stone size, the spontaneous passage rate decreases from 55% for stones less than 4 mm in diameter to 35% for stones 4 to 6 mm in diameter and 8% or less for stones greater than 6 mm in diameter.

During an acute episode of renal colic, pain management is essential, and it can be controlled with the use of NSAIDs or narcotics. Patients should be instructed to increase their fluid intake to increase their urine output to at least 2 L/day to hasten stone passage. α_1-Adrenergic receptor blockers and calcium-channel blockers can be used to facilitate stone passage. The α_1-adrenergic receptor blockers decrease ureteral smooth muscle tone and decrease the frequency and force of peristalsis, whereas calcium-channel blockers suppress smooth muscle contraction and reduce ureteral spasm.

Signs of urinary tract infection, inability to take oral fluids, or obstruction of a single functioning kidney warrants hospitalization. In acute kidney injury, anuria, or sepsis with an obstructive stone, urgent urologic consultation should be obtained. Similarly, a urology consultation should be obtained for stones greater than 10 mm in diameter due to the low probability of spontaneous clearance, for failure of conservative management, or for anatomic abnormalities that would prevent passage of the stone.

The type of surgical intervention is determined by stone size, type, and location and by the existence of infection. Shock wave lithotripsy is often recommended as the first-line therapy for non–lower pole renal calculi less than 2 cm in diameter and for lower pole renal calculi less than 1 cm in diameter. Percutaneous nephrolithotomy is suggested for larger stones.

General measures to prevent recurrent stone formation include increases in oral fluid intake to between 2 and 2.5 L/day and limitation of dietary sodium intake to less than 2 g/day and protein intake to 0.8 to 1 g/day. Dietary calcium restriction is not recommended because calcium in food binds to oxalate in the bowel and reduces urinary excretion of the highly lithogenic oxalate. However, calcium supplementation between meals should be avoided in patients with calcium stones.

Types of Renal Stones

Specific treatment modalities can be implemented when the metabolic risk factors for stone formation are identified (Table 29-9).

TABLE 29-9 TREATMENT MODALITIES FOR NEPHROLITHIASIS RISK FACTORS

URINARY ABNORMALITY	DIETARY CHANGE	MEDICATION
High calcium	Adequate dietary calcium intake Reduce animal protein intake Reduce sodium intake to <3 g/day	Thiazide
High oxalate	Avoid high oxalate foods Adequate dietary calcium intake	Consider vitamin B_6
High uric acid	Reduce purine intake	Allopurinol
Low citrate	Increase fruit and vegetable intake Reduce animal protein intake	Alkali (potassium citrate)
Low volume	Increase total fluid intake Goal urine output >2 L/day	—

TABLE 29-10 PRINCIPAL RISK FACTORS FOR CALCIUM STONE FORMATION

Low urinary volume
High urinary oxalate
High urinary calcium
Low urinary citrate
Dietary factors
  Low dietary intake of fluids, calcium, phytates, potassium
  High intake of oxalates, sodium, protein, sucrose
Medical conditions: obesity, metabolic syndrome, diabetes mellitus, primary hyperparathyroidism, gout, medullary sponge kidneys

Calcium Stones

Approximately 80% of stones are calcium stones, most of which are composed primarily of calcium oxalate, mixed oxalate, and phosphate, and less often, they are pure calcium phosphate. Calcium oxalate supersaturation is not pH dependent in the physiologic range, whereas alkaline urine promotes calcium phosphate supersaturation. The pathophysiologic mechanisms for calcium kidney stone formation are complex and are associated with several metabolic derangements (Table 29-10).

Hypercalciuria is the most common metabolic abnormality found in recurrent calcium stones formers and is detected in 30% to 60% of adults with nephrolithiasis. It is most often familial or idiopathic. One pathophysiologic mechanism for hypercalciuria is increased intestinal calcium absorption (i.e., absorptive hypercalciuria), which is the most common abnormality. Gut calcium absorption is increased in persons with idiopathic hypercalciuria, but serum calcium values remain unchanged because the absorbed calcium is promptly excreted. A second mechanism is enhanced calcium mobilization from bone (i.e., resorptive hypercalciuria), which leads to urinary loss of bone calcium. This is seen in patients with primary hyperparathyroidism, immobilization, and metastatic tumors. A third mechanism is decreased renal calcium reabsorption (i.e., renal leak), the pathogenesis of which is unclear. It is thought to result from a primary defect in renal tubular of calcium. High-sodium intake decreases proximal sodium reabsorption, and the ensuing urinary sodium excretion causes physiologic increases in calcium excretion, promoting stone formation. Consumption of large amounts of animal protein can increase the acid load, causing calcium release from bones and increased urinary calcium excretion. Acidosis results