cell disease is proteinuria, a consequence of glomerular hyperfiltration that results from reduction in nephron mass.

The treatment of sickle cell nephropathy focuses on primary management of the hematologic disorder. Tubular dysfunction may require potassium and bicarbonate supplementation to treat hypokalemia and acidosis, and those with ESRD are treated with dialysis and renal transplantation.

Lithium

Lithium is a monovalent cation, which is freely filtered through the glomeruli. Up to 80% of filtered lithium is reabsorbed in the proximal tubule, and a small fraction is reabsorbed in the distal nephron through the epithelial sodium channel ($E_{Na}C$). Lithium causes dysregulation of the aquaporin water channel and $E_{Na}C$ expression in the cortical collecting duct. The most common manifestation of renal disease associated with lithium is CIN manifesting as a chronic, insidious decline in renal function. The course of renal disease after discontinuation of lithium is highly unpredictable, with no reliable clinical clues to identify those destined for recovery or progression.

Lithium also is associated with nephrogenic diabetes insipidus, which can occur in up to 40% of patients as early as 8 weeks after lithium initiation. Other tubular dysfunctions associated with lithium include water diuresis, natriuresis, and metabolic acidosis. Lithium-associated nephrogenic diabetes insipidus can be treated with $E_{Na}C$ blockade by amiloride.

Urinary Tract Obstruction

Urinary tract obstruction is a common cause of acute kidney injury and chronic kidney disease. When renal function is normal at baseline, unilateral or partial obstruction anywhere along the urinary tract may be asymptomatic, with no discernable change in renal function or urine output. Bilateral urinary tract obstruction, however, can lead to acute and chronic kidney injury and ESRD. It is important to address this possibility early in the clinical course of unexplained renal insufficiency or uremia.

Obstruction to urine flow causes an increase in ureteral intraluminal pressure. Over time, nephron tubules are injured, and the resulting changes in thromboxane A_2 and angiotensin levels decrease renal blood flow. Tubular damage leads to urinary concentrating defects, renal tubular acidosis, and hyperkalemia. If complete obstruction is not relieved, ischemia and nephron loss decrease the glomerular filtration rate.

The most common causes of obstructive nephropathy are shown in Table 29-4. Among elderly men, benign prostatic hypertrophy is a particular concern. Overall, the clinical presentation depends on the cause, site, and time course of obstruction. Patients with obstructive nephropathy have decreased urine output and are at risk for suprapubic pain (i.e., bladder distention from ureteral obstruction), renal colic (i.e., nephrolithiasis), urinary tract infections, fever, acute kidney injury, hypertension, and hematuria. Pain resulting from stretching of the urinary collecting system is the most common presenting symptom. Acute ureteral obstruction usually results in severe flank pain that typically radiates to the groin and is referred to as *renal colic*. Patients with complete bladder outlet obstruction have acute kidney injury and anuria. Patients with incomplete or intermittent bladder outlet obstruction have urinary hesitancy, dribbling, urgency, decreased urine stream, nocturia, and polyuria. These patients are usually pain free. Tubular injury from obstruction causes decreased urinary concentrating capability leading to polyuria.

TABLE 29-4 CAUSES OF URINARY OBSTRUCTION

CAUSE	EXAMPLES
Congenital urinary tract malformation	Meatal stenosis Ureterocele Posterior urethral valves Urethral atresia Phimosis Megaureter–prune belly syndrome
Intraluminal obstruction (urethra and bladder outlet)	Phimosis Urethral strictures Benign prostate hyperplasia Pelvic tumor Anticholinergic drugs Neurogenic bladder Tuberculosis Radiation Trauma Calculi Blood clots Papillary necrosis (sickle cell disease, diabetes mellitus)
Extrinsic compression	Pelvic tumors Prostatic hypertrophy Retroperitoneal fibrosis or tumors
Acquired anomalies	Urethral strictures Neurogenic bladder Intratubular precipitates Bladder mass or stones

The physical examination should include palpation of the kidney and bladder, as well as a rectal, pelvic, and prostate assessment. The patient may have an enlarged and palpable bladder, enlarged prostate, costovertebral tenderness, groin pain, hypertension, or gross hematuria. The mainstays of the initial evaluation include measurement of the postvoid residual volume of the bladder (>125 mL is considered significant and may indicate obstruction) and renal ultrasound to evaluate the kidneys, ureters, and bladder for distention or other abnormalities.

The initial goals of therapy are to manage volume status, electrolyte abnormalities, infection, and other complications of obstructive nephropathy and to relieve the obstruction as soon as possible to prevent further damage to renal parenchyma. If urinary obstruction is suspected, a catheter should be placed in the bladder to address possible bladder outlet obstruction. If a large postvoid residual volume (>125 mL) is detected, the urinary catheter should remain in place while the cause is ascertained. Occasionally, relief of obstruction is associated with a large postobstructive diuresis that may be sufficient in degree to cause volume depletion and hypotension.

If the obstruction is acute, complete recovery of renal function can be expected. If the anatomic site of the urinary tract obstruction is above the bladder, more sophisticated approaches to drainage (e.g., percutaneous nephrostomy tube placement) may be required to relieve obstruction.

CYSTIC KIDNEY DISEASES

Renal cysts are lined by a polarized epithelium and filled with fluid. They result from defects in the structure and the function