

13 - 331 Urinary Tract Obstruction

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Calcium Phosphate Calcium phosphate stones share risk factors with calcium oxalate stones, including higher concentrations of urine calcium and lower concentrations of urine citrate, but additional factors deserve attention. Higher urine phosphate levels and higher urine pH (typically ≥ 6.5) are associated with an increased likelihood of calcium phosphate stone formation. Calcium phosphate stones are more common in patients with distal renal tubular acidosis and primary hyperparathyroidism. There are no randomized trials on which to base preventive recommendations for calcium phosphate stone formers, so the interventions are focused on modification of the recognized risk factors. Thiazide diuretics (with sodium restriction) may be used to reduce urine calcium, as described above for calcium oxalate stones. In patients with low urine citrate levels, alkali supplements (e.g., potassium citrate or bicarbonate) may be used to increase urine citrate. However, the urine pH of these patients should be monitored initially because supplemental alkali can raise urine pH, thereby potentially increasing the risk of stone formation. Because these patients tend to have a urinary acidification defect, reducing the urine pH is not an option. Reduction of dietary phosphate may be beneficial by reducing urine phosphate excretion.

Uric Acid The two main risk factors for uric acid stones are persistently low urine pH and higher uric acid excretion. Urine pH is the predominant influence on uric acid solubility; therefore, the mainstay of prevention of uric acid stone formation entails increasing urine pH. Alkalinizing the urine can be achieved by increasing the intake of foods rich in alkali (e.g., fruits and vegetables) and reducing the intake of foods that produce acid (e.g., animal flesh). If necessary, supplementation with bicarbonate or citrate salts (preferably potassium-based) can be used to reach the recommended pH goal of 6.5 throughout the day and night. Urine uric acid excretion is determined by uric acid generation. Uric acid is the end product of purine metabolism; thus, reduced consumption of purine-containing foods can lower urine uric acid excretion. It is noteworthy that the serum uric acid level is dependent on the fractional excretion of uric acid and, therefore, does not provide information on urine uric acid excretion. For example, an individual with high uric acid generation and concurrent high fractional excretion of uric acid will have high urine uric acid excretion with a normal (or even low) serum uric acid level. If alkalinization of the urine alone is not successful and if dietary modifications do not reduce urine uric acid sufficiently, then the addition of a xanthine oxidase inhibitor, such as allopurinol or febuxostat, can reduce urine uric acid excretion by 40–50%.

Cystine Cystine excretion is not easily modified. Long-term dietary cystine restriction is not feasible and is unlikely to be successful; thus, the focus for cystine stone prevention is on

increasing cystine solubility. This goal may be achieved by treatment with medication that covalently binds to cystine (tiopronin or penicillamine) and a medication that raises urine pH. Tiopronin is the preferred choice owing to its better adverse event profile. The preferred alkalinizing agent to achieve a urine pH of 7.5 is potassium citrate or bicarbonate as sodium salts may increase cystine excretion. As with all stone types, and especially in patients with cystinuria, maintaining a high urine volume is an essential component of the preventive regimen. Struvite stones, also known as infection stones or triplephosphate stones, form only when the upper urinary tract is infected with urease-producing bacteria such as *Proteus mirabilis*, *Klebsiella pneumoniae*, or *Providencia* species. Urease produced by these bacteria hydrolyzes urea and may elevate the urine pH to a supraphysiologic level (>8.0). Struvite stones may grow quickly and fill the renal pelvis (staghorn calculi). Struvite stones require complete removal by a urologist. New stone formation can be avoided by the prevention of UTIs. In patients with recurrent upper UTIs (e.g., some individuals with surgically altered urinary drainage or spinal cord injury), the urease inhibitor

acetohydroxamic acid can be considered; however, this agent should be used with caution because of potential side effects.

■ ■LONG-TERM FOLLOW-UP In general, the preventive regimens described above do not cure the underlying pathophysiologic process. Thus, these recommendations typically need to be followed for the patient's lifetime, and it is essential to tailor recommendations in a way that is acceptable to the patient. Because the memory of the acute stone event fades and patients often return to old habits (e.g., insufficient fluid intake), long-term followup, including repeat 24-h urine collections typically annually, is important to ensure that the preventive regimen has been implemented and has resulted in the desired reduction in the risk of new stone formation. Follow-up imaging should be planned thoughtfully. Many patients with recurrent episodes of renal colic that lead to emergency room visits often undergo repeat CT studies. While CT does provide the best information, the radiation dose is higher than that with plain abdominal radiography (KUB). Small stones may be missed by KUB, and ultrasound has a limited ability to determine the size and number of stones. Minimizing radiation exposure should be a goal of the long-term follow-up plan and must be balanced against the gain in diagnostic information. CHAPTER 331 ■ ■FURTHER READING Coe FL: The physiology of kidney stone prevention: A narrative for patients and physicians.

Available at [https://bpb-us-w2.wpmucdn.com/](https://bpb-us-w2.wpmucdn.com/voices.uchicago.edu/dist/c/3637/files/2022/10/KIDNEY-STONES_Pathogenesis-and-Prevention.pdf)

[voices.uchicago.edu/dist/c/3637/files/2022/10/KIDNEY-STONES_Pathogenesis-and-Prevention.pdf](https://bpb-us-w2.wpmucdn.com/voices.uchicago.edu/dist/c/3637/files/2022/10/KIDNEY-STONES_Pathogenesis-and-Prevention.pdf).

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Urinary Tract Obstruction Obstruction to the flow of urine, with attendant stasis and elevation in urinary tract pressure, impairs renal and urinary conduit functions and is a common cause of acute and chronic kidney disease (obstructive nephropathy). Early recognition and prompt treatment of urinary tract obstruction (UTO) can prevent or reverse devastating effects on kidney structure and function, and decrease susceptibility to hypertension, infection, and stone formation. Chronic

obstruction may lead to permanent loss of renal mass (renal atrophy) and excretory capability. Because obstructive disease may be secondary to serious underlying inflammatory, vascular, or malignant disease, familiarity with clinical findings, appropriate diagnostic testing, and the therapeutic approach is of great importance to the clinician. Recent developments have advanced our knowledge of fetal and genetic causes of lower urinary tract obstruction (LUTO) and chronic anomalies of the kidneys and urinary tracts (CAKUT), the rubric for a cluster of developmental uropathies associated with cysts and obstruction. In the last few years, studies in mice, zebrafish, and pluripotent stem cells (kidney organoids) have identified possible genetic mutations implicated in cases of CAKUT producing low nephron number, including duplications and deletions (copy number variants) and familial candidate genes (HNF1 β , PAX2, SIX1, GATA3, and

many others). Given that there is no nephrogenesis in humans after birth, obstructive disease manifesting in early or middle adulthood may in fact have origins in a decreased number of nephrons during fetal development. Fetal LUTO, if untreated, often results in pulmonary hypoplasia and death in infancy; kidney failure may be a more long-term outcome. Since amniotic fluid is produced by fetal ingestion of urine, LUTO should be suspected when the mother develops oligohydramnios or anhydramnios. Improvements in second-trimester diagnosis and treatment with vesicoamniotic shunting have improved neonatal survival.

One of the most significant concerns in renal medicine is progression to a chronically fibrotic failing kidney. To date, there has been no demonstration of reversal of scarring, but many investigations are underway to find antifibrosis drugs, including those that address fibrosis in genetic diseases that lead to obstructive nephropathy. Many mechanisms contribute to this progression, including inflammatory responses, hemodynamic consequences of the declining number of healthy nephrons, and compression of normal nephrons by cysts in genetic diseases such as autosomal dominant polycystic kidney disease (ADPKD), where a gene mutation in polycystin causes proliferation of cystic masses. In ADPKD, primary cilia of tubule epithelial cells have been implicated in the development of cysts, resulting in inflammatory processes and cell death. Polycystic disease is one of several such ciliopathies that ultimately lead to fibrosis.

PART 9 Disorders of the Kidney and Urinary Tract ■ ■ ETIOLOGY Obstruction to urine flow can result from intrinsic or extrinsic mechanical blockade as well as from functional defects not associated with fixed occlusion of the urinary drainage system. Mechanical obstruction can occur at any level of the urinary tract, from within the renal tubules or the renal calyces to the external urethral meatus (obstructive uropathy). Normal points of narrowing, such as the ureteropelvic and ureterovesical junctions, bladder neck, and urethral meatus, are common sites of obstruction. When lower UTO is above the level of the bladder, unilateral dilatation of the ureter (hydroureter) and renal pyelocalyceal system (hydronephrosis) occurs; lesions at or below the level of the bladder cause bilateral involvement. Common forms of obstruction are listed in Table 331-1. Childhood causes include congenital malformations, such as narrowing of the ureteropelvic junction (UPJ) and abnormal insertion of the ureter into the bladder, the most common cause. Vesicoureteral reflux in the absence of urinary tract infection or bladder neck obstruction often resolves with age. Reinsertion of the ureter into the bladder is indicated if reflux is severe and unlikely to improve spontaneously, if renal function deteriorates, or if urinary tract infections recur despite chronic antimicrobial therapy. Vesicoureteral reflux may cause prenatal hydronephrosis and, if severe, can lead to recurrent urinary infections, hypertension, and renal scarring in childhood. Posterior urethral valves are the most common cause of bilateral hydronephrosis in boys. In adults, UTO has

usually been attributed to acquired defects. Pelvic tumors, calculi, and urethral stricture predominate. Ligation of, or injury to, the ureter during pelvic or colonic surgery can lead to hydronephrosis, which, if unilateral, may remain undetected. Obstructive uropathy may also result from extrinsic neoplastic (carcinoma of cervix or colon) or inflammatory disorders. Lymphomas, particularly follicular, and pelvic or colonic neoplasms with retroperitoneal involvement are causes of ureteral obstruction. As many as 50% of men aged >40 years may have lower urinary tract symptoms associated with benign prostatic hypertrophy, but these symptoms may occur without bladder outlet obstruction. Regardless of the primary cause, the chronic kidney disease that results may be exacerbated by a secondary contributor like diabetes mellitus, hypertension, kidney stones, infection, and metabolic disturbances. Functional impairment of urine flow occurs when voiding is altered by abnormal pontine or sacral centers of micturition control. It may be asymptomatic or associated with lower urinary tract symptoms such as frequency, urgency, and postmicturition incontinence, nocturia, straining to void, slow stream, hesitancy, or a feeling of incomplete emptying. A history should be sought for trauma, back injury, surgery,

TABLE 331-1 Common Mechanical Causes of Urinary Tract Obstruction

URETER	BLADDER	OUTLET	URETHRA
Congenital	Ureteropelvic junction narrowing or obstruction	Ureterovesical junction narrowing or obstruction and reflux	Ureterocele
Retrocaval ureter	Bladder neck obstruction	Ureterocele	Posterior urethral valves
Anterior urethral valves	Stricture	Meatal stenosis	Phimosis
Acquired	Intrinsic Defects	Calculi	Inflammation
Infection	Trauma	Sloughed papillae	Tumor
Blood clots	Benign prostatic hyperplasia	Cancer of prostate	Cancer of bladder
Calculi	Diabetic neuropathy	Spinal cord disease	Anticholinergic drugs and α -adrenergic agonists
Stricture	Tumor	Calculi	Trauma
Phimosis	Acquired	Extrinsic Defects	Pregnant uterus
Retroperitoneal fibrosis	Aortic aneurysm	Uterine leiomyomata	Carcinoma of uterus, prostate, bladder, colon, rectum
Lymphoma	Pelvic inflammatory disease, endometriosis	Accidental surgical ligation	Carcinoma of cervix, colon
Trauma	Trauma	diabetes mellitus, neurologic or psychiatric conditions, and medications.	Causes include neurogenic bladder, often with adynamic ureter, and vesicoureteral reflux.

Reflux in children may result in severe unilateral or bilateral hydronephrosis and hydronephrosis. Overflow urinary incontinence combined with sudden-onset fecal incontinence, severe lower back pain, and saddle anesthesia, requires emergency evaluation for possible cauda equina syndrome. Urinary retention may be the consequence of α -adrenergic and anticholinergic agents, as well as opiates. Hydronephrosis in pregnancy is due to relaxational effects of progesterone on smooth muscle of the renal pelvis, as well as ureteral compression by the enlarged uterus, more often on the right side. Diagnostic tools to identify anatomic obstruction include urinary flow measurements and a postvoid residual measurement. Bladder volume may be readily assessed by bedside ultrasound. Cystourethroscopy and urodynamic studies may be reserved for the symptomatic patient to assess the filling phase (cystometry), pressure-volume relationship of the bladder, bladder compliance, and capacity. Pressure-flow analysis evaluates bladder contractility and bladder outlet resistance during voiding. Bladder obstruction is characterized by high pressures in women, whereas in men, a diagnosis of bladder outlet obstruction is based on flow rate and voiding pressures. A voiding cystourethrogram may be useful in evaluating incomplete emptying and bladder neck and urethral pathology.

■ ■ **CLINICAL FEATURES AND PATHOPHYSIOLOGY** The pathophysiology and clinical features of UTO are summarized in Table 331-2. Flank pain, the symptom that most commonly leads to medical attention, is due to distention of the collecting system or renal capsule. Pain severity is influenced more by the rate at which distention develops than by the degree of distention. Acute supraventricular

TABLE 331-2 Pathophysiology of Bilateral Ureteral Obstruction

HEMODYNAMIC EFFECTS	TUBULE EFFECTS	CLINICAL FEATURES
Acute Pain (capsule distention)	Azotemia, oliguria, or anuria	↑ Renal blood flow
↓ GFR	↓ Medullary blood flow	↑ Vasodilator prostaglandins, nitric oxide
↑ Ureteral and tubule pressures	↑ Reabsorption of Na ⁺ , urea, water	Chronic Azotemia
Hypertension	AVP-insensitive polyuria	Natriuresis
Hyperkalemic, hyperchloremic acidosis	↓ Renal blood flow	↓ GFR
↑ Vasoconstrictor prostaglandins	↑ Renin-angiotensin production	↓ Medullary osmolarity
↓ Concentrating ability	Structural damage; parenchymal atrophy	↓ Transport functions for Na ⁺ , K ⁺ , H ⁺
Release of Obstruction	Postobstructive diuresis	Potential for volume depletion and electrolyte imbalance due to losses of Na ⁺ , K ⁺ , PO ₄
Slow ↑ in GFR (variable)	↓ Tubule pressure	↑ Solute load per nephron (urea, NaCl)
Natriuretic factors present	2 ⁻ , Mg ²⁺ , and water	Abbreviations: AVP, arginine vasopressin; GFR, glomerular filtration rate.

obstruction, as from a stone lodged in a ureter (Chap. 330), is associated with excruciating, sometimes intermittent, pain, known as renal colic. This pain often radiates to the lower abdomen, testes, or labia. By contrast, more insidious causes of obstruction, such as chronic narrowing of the UPJ, may produce little or no pain and yet result in total destruction of the affected kidney. Flank pain that occurs only with micturition is pathognomonic of vesicoureteral reflux. Obstruction of urine flow results in an increase in hydrostatic pressures proximal to the site of obstruction. It is this buildup of pressure that leads to the accompanying pain, the distention of the collecting system in the kidney, and elevated intratubular pressures that initiate tubular dysfunction. In the first days of obstruction, the dilatation of the poorly compliant collecting system may be minimal. As the increased hydrostatic pressure is expressed in the urinary space of the glomeruli, further filtration decreases or stops completely. Azotemia develops when overall excretory function is impaired, often in the setting of bladder outlet obstruction, bilateral renal pelvic or ureteric obstruction, or unilateral disease in a patient with a solitary functioning kidney. Complete bilateral obstruction should be suspected when acute renal failure is accompanied by anuria. Any patient with renal failure otherwise unexplained, or with a history of nephrolithiasis, hematuria, diabetes mellitus, prostatic enlargement, pelvic surgery, trauma, or tumor should be evaluated for UTO. In the acute setting, partial, bilateral obstruction may mimic pre renal azotemia with a high blood urea nitrogen-to-creatinine ratio, concentrated urine, and sodium retention. Renal vascular resistance may be increased. However, with more prolonged obstruction, symptoms of polyuria and nocturia commonly accompany partial UTO and result from loss of medullary hypertonicity with diminished renal concentrating ability. Failure to produce urine free of salt (natriuresis) is due to downregulation of salt reabsorption in the proximal tubule and of transport proteins including the Na⁺, K⁺ adenosine triphosphatase (ATPase), Na:K:2Cl cotransporter (NKCC2) in the thick ascending limb, and the epithelial Na⁺ channel (ENaC) in collecting duct cells. In addition to direct effects on renal transport mechanisms, increased prostaglandin E₂ (PGE₂) (due to induction of cyclooxygenase-2 [COX-2]), angiotensin II (with its downregulation of Na⁺ transporters), and atrial or B-type natriuretic peptides (ANP or BNP) due to volume expansion in the azotemic patient contribute to decreased salt reabsorption along the nephron. Nitric oxide synthases (NOS)

in ureteral smooth muscle and urothelial tissues have been found to oppose the high ureteral pressure in unilateral obstruction.

Dysregulation of aquaporin-2 water channels in the collecting duct contributes to the polyuria. The defect usually does not improve with administration of vasopressin and is, therefore, a form of acquired nephrogenic diabetes insipidus. Wide fluctuations in urine output in a patient with

azotemia should always raise the possibility of intermittent or partial UTO. If fluid intake is inadequate, severe dehydration and hypernatremia may develop. However, as with other causes of poor renal function, excesses of salt and water intake may result in edema and hyponatremia. Partial bilateral UTO often results in acquired distal renal tubular acidosis, hyperkalemia, and renal salt wasting. The H^+ -ATPase, situated on the apical membrane of the α -intercalated cells of the collecting duct, is critical for distal H^+ secretion. The trafficking of intracellular H^+ pumps from the cytoplasm to the cell membrane is disrupted in UTO. The decreased function of the ENaC, in the apical membrane of neighboring collecting duct principal cells, contributes to decreased Na^+ reabsorption (salt wasting) and, therefore, decreased K^+ secretion via K^+ channels. Ammonium (NH_4^+) excretion important to the elimination of H^+ is impaired. These defects in tubule function are often accompanied by renal tubulointerstitial damage. Azotemia with hyperkalemia and metabolic acidosis should prompt consideration of UTO.

CHAPTER 331 The renal interstitium becomes edematous and infiltrated with mononuclear inflammatory cells early in UTO. Later, interstitial fibrosis and atrophy of the papillae and medulla occur and precede these processes in the cortex. The increase in angiotensin II noted in UTO contributes to the inflammatory response and fibroblast accumulation through mechanisms involving profibrotic cytokines. With time, this process leads to chronic kidney damage.

Urinary Tract Obstruction UTO must always be considered in patients with urinary tract infections or urolithiasis. Urinary stasis encourages the growth of organisms. Urea-splitting bacteria are associated with magnesium ammonium phosphate (struvite) calculi that may take on a staghorn appearance. Hypertension is frequent in acute and subacute unilateral obstruction and is usually a consequence of increased release of renin by the involved kidney. Chronic kidney disease from bilateral UTO, often associated with extracellular volume expansion, may result in significant hypertension. Erythrocytosis, an infrequent complication of obstructive uropathy, is secondary to increased erythropoietin production.

■ ■ DIAGNOSIS A history of difficulty in voiding, pain, infection, or change in urinary volume is common. Evidence for distention of the kidney or urinary bladder can often be obtained by palpation and percussion of the abdomen. A careful rectal and genital examination may reveal enlargement or nodularity of the prostate, abnormal rectal sphincter tone, or a rectal or pelvic mass. Urinalysis may reveal hematuria, pyuria, and bacteriuria. The urine sediment is often normal, even when obstruction leads to marked azotemia and extensive structural damage. An abdominal scout film, although insensitive, may detect nephrocalcinosis or a radiopaque stone. As indicated in Fig. 331-1, if UTO is suspected, a bladder catheter should be inserted. Abdominal ultrasonography should be performed to evaluate renal and bladder size, as well as pyelocalyceal contour. Ultrasonography is ~90% specific and sensitive for detection of hydronephrosis. False-positive results are associated with diuresis, renal cysts, or the presence of an extrarenal pelvis, a normal congenital variant. Congenital UPJ obstruction may be mistaken for renal cystic disease. Hydronephrosis may be absent on ultrasound when obstruction is <48 h in duration or associated with volume contraction, staghorn calculi, retroperitoneal fibrosis, or infiltrative renal disease. Duplex Doppler ultrasonography may detect an increased resistive index in urinary obstruction. The radiodensity of renal stones is measured in Hounsfield units (HUs). Calcium oxalate and calcium phosphate stones may be distinguished from uric acid calculi by their HU radiodensities on noncontrast computed tomography (CT) scan, the test of choice for acute flank pain.

Unexplained renal failure Insert bladder catheter No diuresis: do ultrasound Diuresis Obstruction below bladder neck Hydronephrosis Do CT scan to identify site and etiology of obstruction Urologic evaluation Positive or negative but still high suspicion

PART 9 Disorders of the Kidney and Urinary

Tract Retrograde urography and ureteral stent considered Antegrade urography and percutaneous nephrostomy considered

FIGURE 331-1 Diagnostic approach for urinary tract obstruction in unexplained renal failure. CT, computed tomography. Recent advances in technology have led to alternatives and have replaced the once standard intravenous urogram in the further evaluation of UTO. The high-resolution multidetector row CT scan, in particular, has the advantages of visualizing the retroperitoneum, as well as identifying both intrinsic and extrinsic sites of obstruction. Noncontrast CT scans improve visualization of the urinary tract in the patient with renal impairment and are safer for patients at risk for contrast nephropathy. Magnetic resonance urography is not at this time superior to the CT scan, and certain gadolinium agents carry a risk of systemic sclerosis in patients with renal insufficiency. Recently, promising alternatives to gadolinium have emerged, including iron-based contrast and inhalation of xenon gas, although thus far, gadolinium produces the clearest images and most-established safety. CT scanning may define the site of obstruction, identify and characterize kidney stones, and demonstrate dilatation of the calyces, renal pelvis, and ureter above the obstruction. The ureter may be tortuous in chronic obstruction. Though radionuclide scans give less anatomic detail than CT scans, they are able to give differential renal function. In the case of asymmetric renal function, the clinician may decide on a preferable kidney to decompress in the case of bilateral obstruction. Furosemide is sometimes given to increase detection with imaging and to distinguish functional from anatomic obstruction. The increase in urinary flow may bring out the pain of an acute obstructive process. To facilitate visualization of a suspected lesion in a ureter or renal pelvis, retrograde or antegrade urography should be attempted. These procedures do not carry risk of contrast-induced acute kidney injury in patients with renal insufficiency. The retrograde approach involves catheterization of the involved ureter under cystoscopic control, whereas the antegrade technique necessitates percutaneous placement of a catheter into the renal pelvis. Although the antegrade approach may provide immediate decompression of a unilateral obstructing lesion, many urologists initially attempt the retrograde approach unless the catheterization is unsuccessful. Voiding cystourethrography is of value in the diagnosis of vesico ureteral reflux and bladder neck and urethral obstructions. Postvoiding films reveal residual urine. Endoscopic visualization by the urologist often permits precise identification of lesions involving the urethra, prostate, bladder, and ureteral orifices.

No hydronephrosis High suspicion Low suspicion No further workup for obstruction Negative

TREATMENT Urinary Tract Obstruction UTO complicated by infection requires immediate relief of obstruction to prevent development of generalized sepsis and progressive renal damage. Sepsis necessitates prompt urologic intervention. Drainage may be achieved by nephrostomy, ureterostomy, or ureteral, urethral, or suprapubic catheterization. Prolonged antibiotic treatment may be necessary. Chronic or recurrent infections in a poorly functioning obstructed kidney may necessitate nephrectomy. When infection is not present, surgery is often delayed until acid-base, fluid, and electrolyte status is restored. Nevertheless, the site of obstruction should be ascertained as soon as feasible. Elective relief of obstruction is usually recommended in patients with urinary retention, recurrent urinary tract infections, persistent pain, or progressive loss of renal function. Benign prostatic hypertrophy may be treated medically with α -adrenergic blockers and 5 α -reductase inhibitors. Renal colic may be treated with anti-inflammatory medication as edema often contributes to an obstructing ureteral stone, and α -adrenergic blockers may also be of benefit. The clinician should be aware of the risk of intraoperative floppy iris syndrome associated with cataract surgery in patients taking α -adrenergic blockers. Use of nonsteroidal anti-inflammatory medication must take into account the potential for renal harm, and opiates in patients with decreased renal

function may be dangerous and should be used with caution. Functional obstruction secondary to neurogenic bladder may be decreased with the combination of frequent voiding and cholinergic drugs. ■ ■PROGNOSIS With relief of obstruction, the prognosis regarding return of renal function depends largely on whether irreversible renal damage has occurred. When obstruction is not relieved, the course will depend mainly on whether the obstruction is complete or incomplete and bilateral or unilateral, as well as whether or not urinary tract infection is also present. Complete obstruction with infection can lead to total destruction of the kidney within days. Partial return of glomerular filtration rate may

follow relief of complete obstruction of 1 and 2 weeks' duration, but after 8 weeks of obstruction, recovery is unlikely. In the absence of definitive evidence of irreversibility, every effort should be made to decompress the obstruction in the hope of restoring renal function at least partially. A renal radionuclide scan, performed after a prolonged period of decompression, may be used to predict the reversibility of renal dysfunction. ■ ■POSTOBSTRUCTIVE DIURESIS Relief of bilateral, but not unilateral, complete obstruction commonly results in polyuria, which may be massive. The urine is usually hypotonic and may contain large amounts of sodium chloride, potassium, phosphate, and magnesium. The natriuresis is due in part to the correction of extracellular volume expansion, the increase in natriuretic factors accumulated during the period of renal failure, and depressed salt and water reabsorption when urine flow is reestablished. The retained urea is excreted with improved glomerular filtration rate, resulting in an osmotic diuresis that increases the urine volume of electrolyte-free water. Electrolyte-free water excretion (hypotonic urine) is recognized as being present when the sum of the urinary concentrations of sodium and potassium is lower than the serum sodium concentration. Precipitating factors include suppression of antidiuretic hormone at arterial baroreceptor sites, elevation of natriuretic peptides, or nephrogenic diabetes insipidus due to obstructive tubular injury. In the majority of patients, this diuresis results in the appropriate excretion of the excesses of retained salt and water. When extracellular volume and composition return to normal, the diuresis usually abates spontaneously. Occasionally, iatrogenic expansion of extracellular volume is responsible for, or sustains, the diuresis observed in the postobstructive period. Replacement with intravenous fluids in amounts less than urinary losses usually prevents this complication. More aggressive fluid management is required in the setting of hypovolemia, hypotension, or disturbances in serum electrolyte concentrations. The loss of electrolyte-free water with urea may result in hypernatremia. Measured urinary output and serum and urine sodium, potassium, and osmolal concentrations should guide the use of

appropriate intravenous replacement. Often replacement with 0.45% saline is required because of the likelihood of appropriately matching urine electrolyte concentrations. Relief of obstruction may be followed by urinary salt and water losses severe enough to provoke profound dehydration and vascular collapse. In these patients, decreased tubule reabsorptive capacity is probably responsible for the marked diuresis. Appropriate therapy in such patients includes intravenous administration of salt-containing solutions to replace sodium and volume deficits.

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