Urinary Tract Obstruction

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Obstruction of the urinary tract is a condition that physicians in all disciplines frequently encounter. Regardless of the cause, obstruction of urinary outflow leads to renal impairment that is initially reversible if obstruction is alleviated. The tubules are primarily affected; however, with time, progression results in parenchymal atrophy with glomerular sparing until late in the clinical course. Long-standing obstruction affects the kidney grossly as well as microscopically, resulting in dilatation of the pelvis and calices and thinning of the cortex. Whether the obstruction is unilateral or bilateral, acute or chronic, it produces changes in the anatomy and physiology of the kidney involved.

Therapeutic efforts are often aimed solely at relieving the cause of obstruction. Therefore, the sequelae of obstruction and its management may be overlooked. Often a significant salt-and-water diuresis, known as post-obstructive diuresis (POD), results and the physician's thought process must shift from relieving and perhaps treating the obstructing lesion to treating the physiologic derangements associated with the POD.

Nephrologists, urologists, and internists should have a working knowledge of the renal response to obstruction and its alleviation. This chapter encompasses diagnoses, pathophysiology, and treatment of obstruction and management of POD.

Incidence

Impairment of normal urine flow by functional or structural changes in the urinary tract is a common occurrence in all ages. Pyelocaliectasis has been found on autopsy of 3.5% of adults with a 1 : 1 male to female ratio. Additionally, 2% of children display hydronephrosis at autopsy, mostly because of congenital anomalies. After age 60, obstruction occurs more often in males, resulting from benign prostatic hypertrophy and prostatic cancer, whereas middle-aged women develop obstructive uropathy as a result of pelvic cancer or retroperitoneal fibrosis. Currently in the United States, the incidence of patient visits attributed to obstructive uropathy is about 380 out of 100,000 patient visits.

Causes of Partial or Complete Urinary Obstruction

Obstruction of the urinary tract can occur at any level and has many causes [1, 2]. Furthermore, obstruction can be complete, implying obstruction of all functioning nephrons, or partial. In the former scenario, obstruction is

Table 1. Causes of Urinary Tract Obstruction

Kidney
- Urolithiasis – pelvis
- Ureteropelvic junction obstruction
- Papillary necrosis
- Tumor (malignant or benign)
- Intratubular obstruction (crystalline nephropathy)

Ureter
- Tumor (papilloma, transitional cell carcinoma)
- Urolithiasis
- Congenital obstructive megaureter
- Eagle Barrett syndrome (prune belly)
- Ureterocele, orthotopic or ectopic
- Ectopic ureter
- Retrocaval ureter
- Retroperitoneal fibrosis (idiopathic, drugs, irradiation)
- Inflammatory bowel disease
- Metastatic tumor, retroperitoneal adenopathy
- Infection (tuberculosis, *Schistosoma haematobium*, fungus ball)
- Ureteral valve
- Ureteral polyp
- Pelvic lipomatosis
- Lymphocele

Bladder
- Neurogenic bladder (spinal cord defect, trauma, diabetes, multiple sclerosis, Parkinson's disease,
- strokes)
- Bladder neck contracture
- Transitional cell carcinoma
- Hemorrhagic cystitis
- Blood clots
- Infection (pyocystis, schistosomiasis)
- Detrusor, sphincter dyssynergia
- Bladder stones

Urethra
- Stricture
- Detrusor-sphincter dyssynergia
- Trauma/obliteration
- Meatal stenosis
- Posterior and anterior urethral valves
- Prostatic hypertrophy or cancer
- Calculus
- Polyp or urethral carcinoma
- Phymosis, paraphymosis
- Diverticulum

Table 1. Causes of Urinary Tract Obstruction (Part 2)

Extrinsic compression
- Cervical tumor
- Uterine tumor or pregnancy
- Endometrial tumor
- Endometriosis
- Uterine prolapse
- Vaginal distension
- Aneurysms
- Aberrant crossing vessels
- Abscess
- Gartner's duct cyst
- Crohn's disease
- Diverticulitis

bilateral or involves an anatomically or functionally solitary kidney. The causes of complete or partial obstruction are listed in Table 1.

Intratubular obstruction is the result of crystalline nephropathy and cannot be demonstrated radiographically or sonographically. In many cases, such as in tumor lysis syndrome, sulfa drug administration, or ethylene glycol poisoning, there will be crystals in the urinary sediment (uric acid, sulfa, oxalate, etc.) that yield a timely diagnosis. Because the crystal deposits are the result of filtration from the blood and crystallization in the lumen of all nephrons of the kidney, the obstruction is, by definition, bilateral and complete.

Obstruction of the renal calix or pelvis may be unilateral or bilateral, complete or partial. Unilateral obstruction may result in ipsilateral loin pain, microhematuria, gross hematuria, or it may be asymptomatic. However, there will be little or no change in blood urea nitrogen (BUN), creatinine, or electrolytes if the unobstructed kidney is functioning normally. Causes of obstruction in the pelvis or calices include renal stones of any type, including staghorn calculi, renal papilla sloughed during papillary necrosis, and benign and malignant tumors.

Ureteric obstruction can be unilateral or bilateral, partial or complete. Ureteral colic and flank pain result from acute obstruction and may be accompanied by nausea, vomiting, scrotal pain and micturitional urgency; however, chronic indolent obstruction may be asymptomatic.

Malignant tumors such as transitional cell carcinoma, stones, and retroperitoneal lymphadenopathy constitute the more common causes of ureteral obstruction. Ureteric strictures may be caused by multiple stone passage, instrumentation or a previous operation, pelvic irradiation, infections such as renal tuberculosis or *Schistosoma haematobium* infection, and inflammatory bowel disease. Additionally, in cases of inflammatory bowel disease, radiation, and penetrating trauma, a urinary fistula can develop from the ureter or bladder with resorption of urine by the peritoneal membrane and resultant elevation in BUN, creatinine, chloride, and other electrolytes. This urinary resorption may mimic obstruction, although no obstruction exists per se.

Extrinsic causes of ureteral obstruction are best classified by the system of origin. Most extrinsic lesions obstructing the ureters originate in the reproductive system, including pregnancy and cervical, endometrial, and ovarian cancer as well as uterine prolapse and endometriosis. Vascular abnormalities such as aortic or iliac aneurysms or aberrant vessels and retroperitoneal lymphadenopathy from malignancy cause extrinsic obstruction. As previously stated, inflammatory bowel disease, particularly Crohn's disease, can cause strictures and fistulas of the ureters and bladder, respectively. Finally, retroperitoneal fibrosis results in encasement of the ureters, inhibiting peristalsis and causing significant partial obstruction. Patients usually present with vague back pain and an elevated erythrocyte sedimentation rate (ESR), BUN, and creatinine. There may also be a history of methyldopa, methysergide, or beta blocker drug use or retroperitoneal malignancy requiring chemotherapy or irradiation. Unlike other causes of obstruction, hydronephrosis may not be apparent radiographically or sonographically because of fibrous encasement of the upper urinary tract.

Pathophysiology of Obstruction

Obstruction of the urinary tract generally causes dilatation of all portions of the urinary tract proximal to the level of obstruction ascending to the renal parenchyma. The initial response is one of muscular hypertrophy of the proximal ureter and renal pelvis followed by production of collagen and elastic tissue. The latter connective tissue impairs myogenic impulse transmission, thereby disturbing peristalsis [3, 4]. Hydronephrosis causes tubular dilatation and tubule cell atrophy, appearing within 7 days of obstruction in the distal tubule and within 14 days in the proximal tubule. By day 28, approximately 50% of the medulla is lost, and there is obvious cortical atrophy because of the associated loss of proximal tubules. Glomerular changes occur only after 28 days of obstruction. Although there is an eventual reduction of blood flow in hydronephrosis, it appears to be the result of impaired venous drainage and not only to an alteration in arterial flow [5, 6, 7].

When complete obstruction occurs, the urine in the obstructed kidney is not static; rather, there is a turnover of urine in hydronephrosis as most urine extravasates via the calyceal fornix. Urine can exit the renal pelvis by extravasation, pyelolymphatic back-

flow, and pyelovenous backflow while glomerular filtrate replenishes the urine and maintains the hydronephrosis. With lower pressure, the urine exits into the lymphatics; with higher pressure, the renal venous system resorbs the urine [8, 9]. In chronic obstruction of 6 to 34 days' duration, the quantity of urine escaping the renal pelvis ranges from 0.04 to 0.16 mL/min. Glomerular filtration in complete obstruction is 1.2 mL/min after 2 weeks and 0.4 mL/min after 5 weeks [10].

Renal Compensation for Obstruction

When a single kidney fails, regardless of the etiology, there is adaptation within the remaining kidney to restore total renal function toward normalcy. Such adaptation occurs both by hypertrophy and hyperplasia [11]. As a result of unilateral obstruction, there is an ipsilateral and contralateral increase in renal mass in the first week. Thereafter, there is progressive hypertrophy in the nonobstructed kidney while the obstructed kidney slowly atrophies [12, 13]. During this compensatory hypertrophy, the glomeruli increase in size but not in number. Perhaps this is the result of single nephron hyperfiltration.

Upon alleviation of obstruction, the obstructed kidney regains some function, and although compensatory hypertrophy of the contralateral kidney persists, total renal function does not recover to normal by 4 months. The degree of recovery of renal function after relief of obstruction varies with the duration of obstruction and the severity of pyelolymphatic or pyelovenous backflow of urine [14]. Upon releasing total ureteral obstruction of 4 weeks' duration, the glomerular filtration rate (GFR) returns to 35% of normal by 5 months; however, there is no recovery of function after 6 weeks of total ureteral obstruction.

Hydrostatic Pressures in the Ureter, Pelvis, and Renal Tubules During Obstruction

Normally the pressure in the renal pelvis is between 6 and 7 mm Hg, exceeding intraperitoneal pressure and that within the bladder and ureter. The pressure in the normal proximal tubule is 14 mm Hg, and the pressure from glomerular filtration (glomerular capillary pressure [60 mm Hg], less capillary oncotic pressure [25 to 30 mm Hg], less the hydrostatic pressure in Bowman's space [15 mm Hg]) is between 15 and 20 mm Hg [15].

During urinary tract obstruction, renal pelvis and ureteral pressures rise acutely only to decline to 50% of the peak value within 24 hours. Over the next 2 months, the intraureteric pressure steadily decreases to a nadir of 15 mm Hg [14, 16]. This higher ureteral pressure, sometimes measured at 50 – 70 mm Hg during acute obstruction, results from filtration pressure and active muscle contractions in the renal pelvis and ureter [15, 17]. Administration of mannitol or volume expansion with saline can increase ureteral pressures to 100 mm Hg [18, 19]. Proximal tubular pressures may acutely rise to 40 mm Hg; however, within 24 hours the proximal tubular pressure is below normal because of afferent renal arteriolar vasoconstriction [20, 21].

Glomerular Filtration, Renal Blood Flow, and Tubular Function in Acute Obstruction

Glomerular filtration and renal blood flow (RBF) are affected as a consequence of afferent renal arteriolar constriction. As proximal tubular pressure increases, the GFR falls because of afferent renal arteriolar vasoconstriction beginning within 5 hours of the onset of

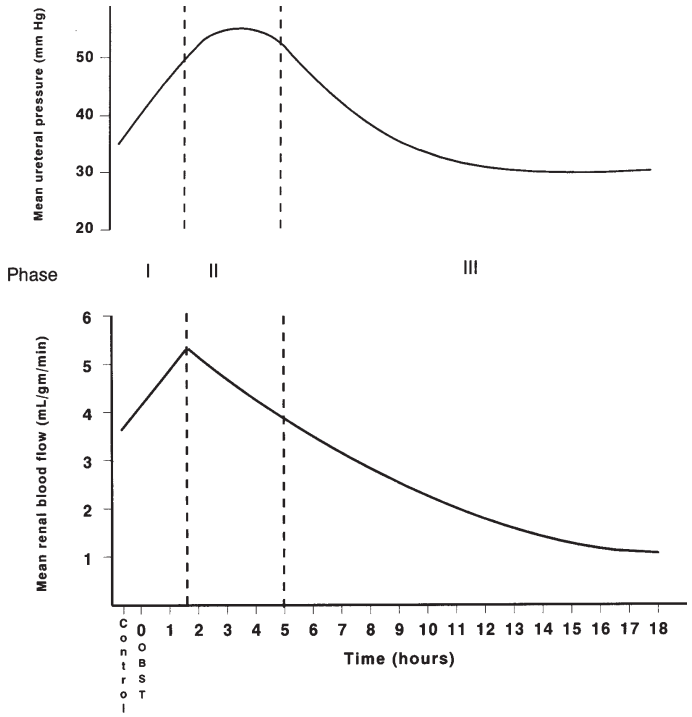

Figure 1. The triphasic response of ureteral pressure and renal blood flow in response to unilateral obstruction. Adapted from [Vaughan ED Jr, Bruce RG, Waid TH, Lucas BA et al 1997 Understanding postobstructive diuresis. Contemp Urol 9: 53-66] with permission.

obstruction. Additionally, as the proximal tubular pressure rises, there is a declining pressure gradient between the glomerulus and the proximal tubule. GFR is 52% of normal 4 hours after the onset of complete obstruction but only 2% of normal after 48 hours [22]. Additionally, during the first 24 hours of obstruction, RBF and ureteral pressure in the ipsilateral kidney display a triphasic relationship (Figure 1) [16, 23]. In the first 90 minutes, there is an increase in RBF and ureteral pressure consistent with preglomerular vasodilatation, a phenomenon that is prostaglandin mediated [24]. The increase in RBF appears to be confined to the cortex with the majority being distributed to the inner cortex [25].

The second phase occurs from 90 minutes to 5 hours after acute obstruction and is the result of preglomerular arteriolar resistance. The mean RBF begins to decline; however, the mean ureteral pressure continues to rise and then plateau. The third phase then ensues from 5 – 18 hours, marked by declines in both ureteral pressure and RBF. The presumed mechanism would appear to be persistent preglomerular vasoconstriction, but the exact mechanism remains unknown [26].

Tubular function is altered in partial acute ureteral obstruction because of a slower rate of tubular fluid flow. As a result, urine volume decreases, osmolality increases, and urinary sodium concentration may be reduced [27]. After complete acute ureteral obstruction, there is a further decline in GFR, and tubular function becomes impaired, which results in a temporary concentrating defect after the release of acute ureteral obstruction [28].

The effects of partial and complete chronic obstruction have also been studied. In chronic partial obstruction, there is a reduction in RBF, GFR, urinary concentrating ability, so-

dium reabsorption, and urinary acidification (hydrogen ion excretion) [27, 29]. Indeed, impairment of urinary acidification is a result of impairment in all aspects of tubular hydrogen ion handling, including ammonia excretion, titratable acidity, and bicarbonate reabsorption [30]. Complete chronic obstruction displays a decline in ureteral pressures after 24 hours, and the decline continues over 6 – 8 weeks. Proximal tubular pressures may normalize or become 30% lower than normal [26, 31]. Because of afferent arteriolar vasoconstriction, RBF progressively declines to 70% of normal at 24 hours and 50%, 30%, and 12% at 3, 6, and 50 days, respectively [16, 23]. The most significant reductions in RBF are seen in the outer renal cortex and the inner medulla [25, 32].

As stated, the rate of glomerular filtration declines progressively in chronic complete obstruction, with fluid exiting the collecting system by pyelolymphatic, pyelovenous, and pyelotubular backflow. The GFR is 0.4 mL/min at 5 weeks of obstruction; however, this is enough to replace the exiting tubular fluid [10]. One week after release of complete obstruction of 2 weeks' duration, the GFR is restored to 15% of normal with the maximum attainable recovery being 46% of normal. No recovery of GFR is ever noted after 6 weeks of chronic obstruction [33].

Complete urinary obstruction impairs all tubular function except urinary dilution, and upon release of obstruction, urinary concentrating ability and sodium conservation are severely impaired. Although urinary concentrating ability can be recovered after release of 2 weeks of complete obstruction, it remains permanently impaired after 4 weeks of obstruction [14]. Other tubular functions are likewise impaired, including glucose transport, potassium excretion, sodium resorption, and urinary acidification. Inability to concentrate urine remains the primary defect [14, 34].

Obstruction and Hypertension

Hypertension may be associated with either unilateral or bilateral obstruction. Acute unilateral obstruction is associated with renin elaboration and renin-dependent hypertension [14, 23]. Chronic unilateral obstruction may be associated with hypertension. However, renin is rarely elevated in bilateral obstruction, and patients usually have volume-dependent hypertension in this setting.

Clinical Presentation and Diagnosis of Obstruction

The clinical manifestations of urinary tract obstruction vary, depending on the location, duration, and degree of obstruction. Patients with complete bilateral obstruction or with an obstructed solitary kidney may present with acute oligoanuric renal failure, whereas partial obstruction of both kidneys or a solitary kidney may result in chronic azotemia with polyuria or urine output alternating from oliguria to polyuria. Pain is more likely to be associated with acute obstruction; however, obstruction may be totally asymptomatic and occur without laboratory findings or clinical manifestations [35].

As stated previously, hypertension may occur as a consequence of obstruction and may be volume or renin dependent. Polycythemia because of erythropoietin secretion has been described; however, in severely azotemic patients a normochromic, normocytic anemia is more commonly seen. Physical examination may uncover a palpable flank mass or bladder, and rectal or pelvic examination may reveal an enlarged prostate gland or gynecologic pathology. In thin patients, bladder masses may

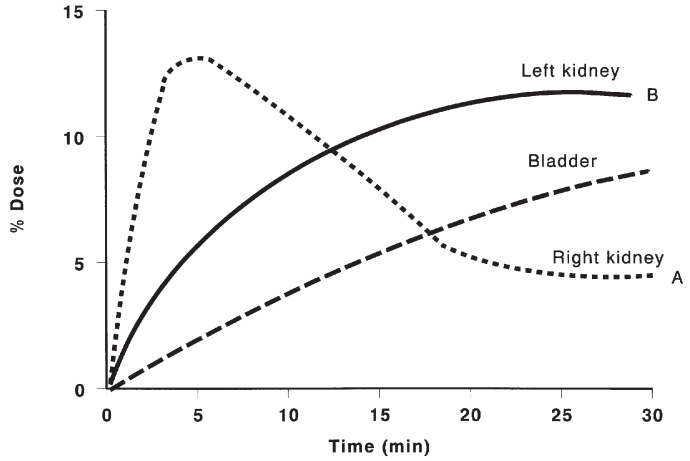

Figure 2. Renogram showing unobstructed pattern in the right kidney (A) and an obstructed pattern in the left kidney (B). Appearance of radionuclide in the bladder is seen in the bladder curve.

be palpated bimanually. Finally, assessment of volume status is very important in determining how to best manage the patient. Complete obstruction usually results in volume expansion, which in severe cases can produce congestive heart failure (CHF) and pulmonary edema. Volume depletion can occur in unilateral obstruction in which the contralateral kidney excretes salt and water in a compensatory fashion.

Laboratory evaluation may or may not be helpful. The diagnosis can be aided by the presence of azotemia, a normal anion gap (8 – 12 mmol/L), hyperchloremic metabolic acidosis with normal potassium, or hyperkalemia. The urinalysis will usually have a pH of > 5.5 on a fresh specimen. The urinary sodium will be ≥ 40 mmol/L and the fractional excretion of filtered sodium (FENa) will be > 1. First voided morning urine will reveal the patient's lack of concentrating ability with a low specific gravity (1.002 to 1.010) and osmolality ≤ 400 mOsm/kg. If renal failure is far advanced, the patient may develop a uremic metabolic acidosis in which the anion gap will be elevated. Urinary sediment can range from bland to active including erythrocytes (due to tumor, BPH, clots, or stones), infection; leukocytes (due to infection, or stones), or crystals (due to stones, infection, or crystalline nephropathy) [35]. Finally, hypernatremia may occur if patients are partially obstructed and sustain severe water losses because of tubular insensitivity to antidiuretic hormone (ADH), i.e. nephrogenic diabetes insipidus.

Ultrasonography remains the most useful test in diagnosing urinary tract obstruction. It is noninvasive, relatively inexpensive, and both sensitive and specific. Sonography will rarely yield a false-positive result because of anatomic variations of the pyelocaliceal system that may be misinterpreted as hydronephrosis. Sonography may provide false-negative results in patients who are both obstructed and volume depleted or in patients obstructed because retroperitoneal fibrosis has encased the entire collecting system. In the latter case, retrograde pyelography with drainage films, placement of ureteral catheters or stents, or placement of percutaneous nephrostomies and antegrade nephrostograms may diagnose and treat the obstruction.

Radioisotope renography can be useful in diagnosing urinary tract obstruction and differentiating between mechanical (anatomic) obstruction and functional (aperistaltic) obstruction (Figure 2). Isotope scanning is unique in the investigation of obstruction be-

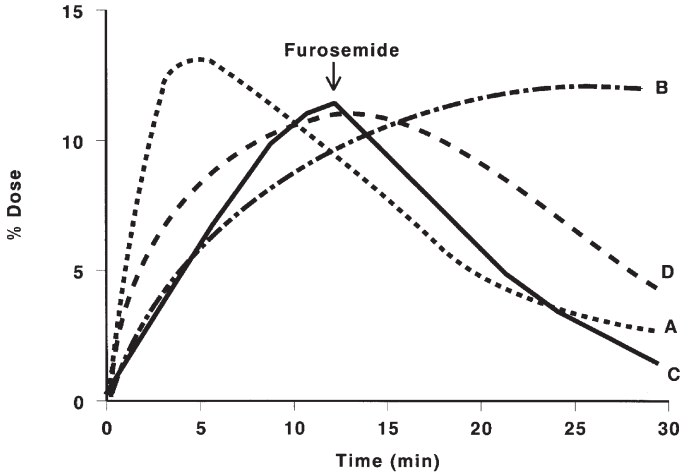

Figure 3. The diuretic renogram. Results are followed on a time activity curve with a normal curve excluding obstruction (A). A rising curve unaffected by diuretic administration indicates obstruction (B). Response to diuretics suggests a dilated collecting system, which is not obstructed (C). A partial diuretic response indicates subtotal obstruction (D).

cause it offers simultaneous quantification of renal function and dynamic analysis of urine flow rates. Urinary tract dilitation without a demonstrable anatomic lesion may occur in ureteric reimplantation, pyeloplasty, ureterolithotomy, pyelolithotomy, primary megaureter or vesicoureteral reflux. If a standard renogram is abnormal with no or sluggish elimination of radionuclide tracer at 10 – 30 min, furosemide is given intravenously (IV) while the study continues. One of 4 responses may occur (Figure 3):

- The renogram is normal, excluding obstruction (A).
- The renogram curve remains obstructive, confirming anatomic obstruction (B).
- The obstructive curve is converted to a nonobstructive curve with rapid and complete elimination of the tracer (C).
- The obstructive curve displays a partial response to diuresis, indicating subtotal or partial obstruction (D).

It must be noted that in patients with abnormal voiding or in patients with free vesicoureteral reflux, the bladder must be drained with a catheter or the test result may suggest mechanical obstruction, when, in fact, it is not present. A repeat scan with a Foley catheter in place is then warranted.

Finally, perfusion-pressure flow studies can be obtained to rule out obstruction. Also known as the Whitaker test, the procedure measures the perfusion pressure of a solution passing antegrade through a percutaneous nephrostomy tube at 10 mL/min. A pressure rise of ≥ 22 cm water indicates obstruction, whereas a rise of < 15 cm of water excludes obstruction. Values ranging between 15 and 22 cm water are said to be equivocal [36, 37]. The major disadvantage of the test is its invasiveness. The results of numerous studies comparing perfusion pressure flow with the less invasive diuretic isotope renography have been variable, with correlations ranging from 53 – 86% [37, 38]. Additionally, studies comparing diuretic renography with renal pelvic morphology appear to correlate well (r = 0.88) [39].

Summary of Physiologic Conditions During and After Release of Obstruction

Changes in renal physiology depend on whether ureteral obstruction is partial or complete. Complete obstruction often results in the uremic state because of retention of waste products normally excreted. Anatomically the tubules look normal in complete obstruction, whereas in partial obstruction, the tubules of the nonobstructed kidney are collapsed and the nephrons are poorly perfused. Pressures are elevated in the proximal and distal tubules in complete obstruction but are lower than normal in the unobstructed units in partial obstruction. Afferent arterial pressure is elevated in complete obstruction and diminished in the unobstructed units in partial obstruction.

RFB and GFR are reduced to one-third of normal in both complete and partial obstruction, in the former by increased proximal tubular pressure and in the latter by afferent arteriole vasoconstriction of the unaffected kidney. When complete obstruction is alleviated, the tubular pressures normalize; however, the GFR diminishes because of afferent arteriolar vasoconstriction. Urine flow may be increased dramatically after the release of complete obstruction, and the excretion of urea, potassium, phosphate, and magnesium is enhanced. A diuresis occurs regardless of fluid balance until the GFR can restore sodium delivery to the tubules and medullary hypertonicity can be regained. The urinary concentrating defect persists for several days beyond the salt-wasting defect, and the ability to conserve urinary sodium should herald the recovery of concentrating ability and attenuation of the diuresis.

After the release of partial obstruction, the affected kidney has a normal urine flow of dilute urine because of reduced GFR and RBF and impaired concentrating ability. The contralateral kidney maintains homeostasis and a POD is often clinically inapparent.

Mechanisms of POD

POD occurs when there is correction of complete bilateral obstruction or complete obstruction of a solitary functioning kidney. During unilateral obstruction, urinary and serum abnormalities are obscured by the unobstructed kidney and POD rarely occurs. First characterized as a syndrome of volume and electrolyte imbalance following relief of obstruction by catheterization, POD involves the production of large volumes of urine immediately after the relief of urinary obstruction [40]. This syndrome occurs when all nephrons are obstructed and patients still have reversible, albeit advanced, renal failure. Three mechanisms are postulated (Figure 4): a defect in urinary concentrating ability, impaired renal sodium reabsorption, and solute diuresis due to retained urea or iatrogenic administration of sodium-containing IV fluids [41].

Upon relief of complete urinary tract obstruction, RBF and GFR initially decrease as a result of the action of renal prostaglandins [42]. Because a defect in urinary concentration and sodium conservation exists, diuresis ensues. When the diuresis is prolonged and severe, significant loss of water, sodium, potassium, and magnesium can result in hypovolemia and electrolyte abnormalities that

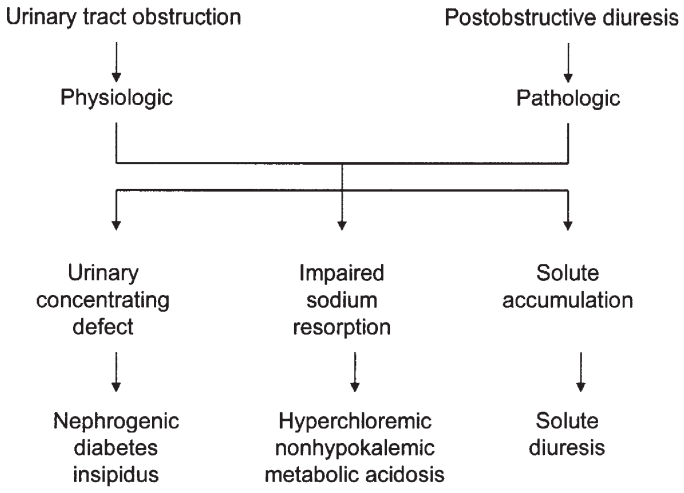

Figure 4. Pathophysiologic alterations in urinary tract obstruction and postobstructive diuresis. These mechanisms are physiologic during obstruction, continue pathologically in the postobstructive period until the hypertonicity of the renal medulla is restored. Solute accumulation and diuresis are pathologic when IV fluids are inappropriately administered. [Bruce RG, Waid TH, Lucas BA 1997 Understanding postobstructive diuresis. Contemp Urol 9:53-66] with permission.

cannot be adequately prevented or restored by oral intake of solute (diet) and water [34].

Patients with prolonged POD are insensitive to the administration of ADH or deoxycorticosterone acetate (DOCA), which explains the 2 patterns of diuresis seen in POD. The more common clinical entity is the diabetes insipidus-like nephropathy, which is usually associated with chronic obstruction and is a self-limiting concentrating defect leading to free water losses. Postobstructive sodium-losing nephropathy is rare but is a more severe and protracted diuresis that occurs in the setting of severe bilateral obstruction and reversible renal failure [43]. The question becomes how and to what degree do urinary concentrating defects and renal sodium loss contribute to pathologic water and electrolyte loss in POD.

As stated earlier, the urinary concentrating defect is the result of altered renal hemodynamics in the early phases of complete obstruction. The GFR initially decreases after obstruction is relieved, and without tubular solute, including sodium and urea, the medullary tonicity cannot be maintained. Until glomerular flow and filtration improve and provide solute to restore the medullary tonicity, hypotonic urine losses will continue. Clinically, this generally results in a water diuresis of 1 – 4 days' duration.

The natriuresis occurring in POD results from increased delivery of sodium to the distal tubule in the face of limited capacity of the distal tubule to resorb the increased sodium load [44]. This state is not unlike that produced by loop diuretics, which, when administered, block chloride and sodium resorption in the loop of Henle, increase delivery to the distal tubule, and thereby create a solute-rich urine. Additionally, disturbances in proximal sodium resorption can occur due to proximal tubular dysfunction and further add to distal tubule solute delivery. The naturesis of POD is therefore not only related to enhanced sodium excretion to relieve volume expansion, but also to a pathologic decrease in tubular sodium reabsorption [44].

Patients with POD have elevated serum levels of atrial natriuretic factor (ANF) [45]. ANF is elevated whenever there is extracellular volume expansion, whether acute or chronic, and has four effects: natriuresis, diuresis, vascular relaxation, and increasing GFR via afferent arteriole dilation and effer-

ent arteriole constriction [46]. ANF has been associated with complete urinary obstruction and to the development and control of POD [47].

Diagnosing and Treating POD

The diagnosis of POD should be considered whenever excessive diuresis occurs after obstruction is alleviated. Recognition of the patients at greatest risk for developing POD is an important first step. Complete urinary tract obstruction is usually the predisposing clinical condition. The clinical situation is often encountered in elderly males with bladder outlet obstruction from prostate disease, who develop reversible renal failure [48]. These patients have, at some time in their clinical course, high pressure chronic retention of urine and elevated serum levels of ANF, conditions which favor the development of POD. Additionally, the patient's volume status is an important indicator of the degree of diuresis. Hypervolemic patients will diurese more vigorously than those who are euvolemic or volume depleted [35].

Once the obstruction has been treated by catheterization, stenting, or percutaneous nephrostomy, treatment of POD must begin. Hourly assessment of the patient's urine flow and oral intake along with assessment of volume status is essential to prevent the patient from becoming volume depleted. Remembering that the recently unobstructed kidney requires time to recover the ability to conserve sodium and to concentrate urine, the clinician may find it useful to categorize these patients into low, moderate, and high risk of developing diuresis [33] (Figure 5).

In the low-risk and moderate-risk patient, the diuresis may not be brisk, and the thirst mechanism will compel the patient to increase oral intake and replace volume. Obviously if the patient is obtunded, then the thirst mechanism is unreliable, and oral intake will be both inadequate and unsafe. If the patient is nauseated and/or vomiting, oral intake is again unreliable, and if there is orthostatic hypotension, tachycardia, or urine volumes > 200 mL/hour, IV replacement is necessary. The high-risk patient will have evidence for volume overload, mental status changes due to uremia or other neurologic conditions, and almost always complete obstruction of both kidneys or a solitary functioning kidney. These patients should receive IV fluid replacement from the onset of POD.

When IV fluid replacement is needed, a solution with a composition similar to that of urine is desirable. A spot urine for sodium, potassium, and chloride is helpful in determining the makeup of this solution. Urine sodium and chloride are usually 70 – 80 mmol/L. Urine potassium is usually 20 – 30 mmol/L. Therefore, replacement of urine with half normal saline or 5% dextrose in half normal saline with 20 – 30 mmol K/L is ideal in this situation. Since patients requiring immediate IV therapy are often volume overloaded, replacement of each mL of urine with one half mL of IV solution will correct the volume expansion while avoiding volume depletion. If the patient is hyponatremic, then normal saline should be used initially at 1/2 mL IV /mL urine. If the patient is volume depleted on physical assessment, the patient should be given IV normal saline at least 1 IV mL urine and additional boluses of normal saline until euvolemic by physical assessment or by central venous pressure monitoring. If the patient is hyperkalemic at the start of IV replacement, monitoring of the serum potassium will be necessary until the patient is

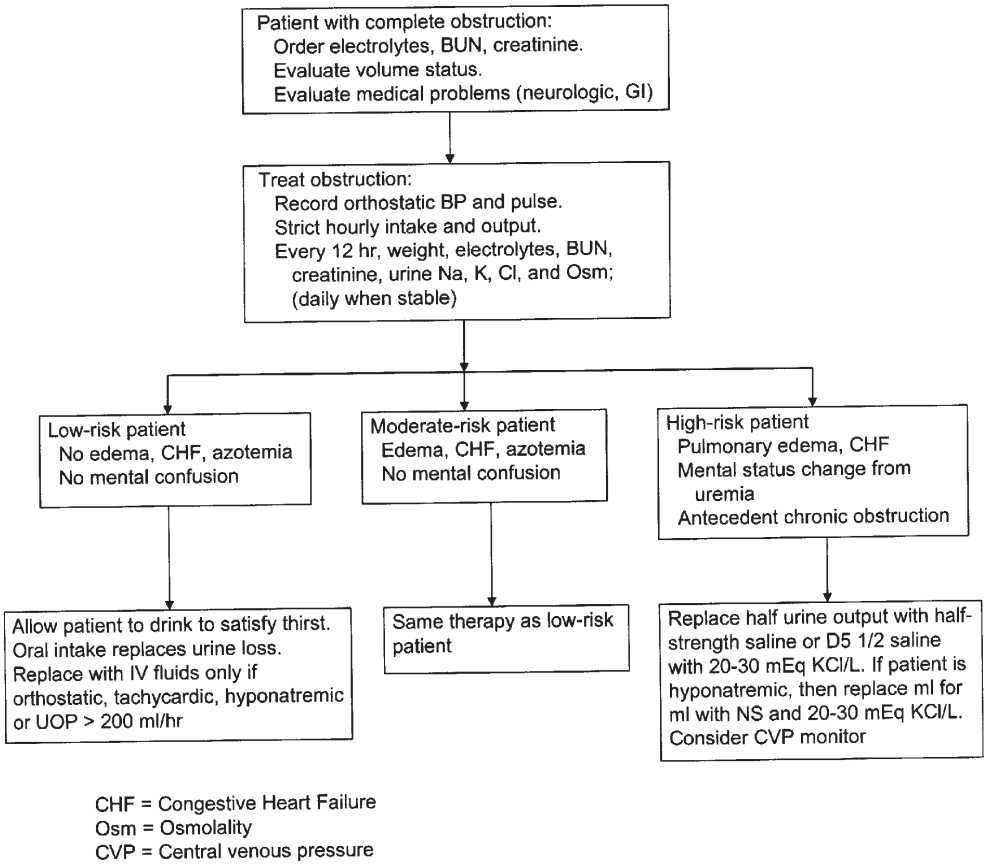

Figure 5. Algorithm for managing postobstructive diuresis. Adapted from [Vaughan ED Jr, Gillenwater JY, Bruce RG, Waid TH, Lucas BA 1997 Understanding postobstructive diuresis. Contemp Urol 9:53-66] with permission. CHF = Congestive Heart Failure, Osm = Osmolality, CVP = Central venous pressure.

normokalemic. Potassium may then be added to the IV urine replacement solution. During the period of recovery of renal function and volume replacement, serum magnesium must be monitored because magnesium wasting can occur in POD. Replacement with magnesium sulfate 0.25 to 0.5 mmol/kg in 4 divided doses daily for the next 24 – 72 hours should be adequate to replace magnesium deficits in these patients [35].

It should be noted that IV fluid replacement is recommended by some authors even in mild cases of POD and in volume overloaded patients [49]. IV fluid administration may result in increased diuresis and natriuresis; however, GFR may recover more quickly. Finally, one must understand that prolonged diuresis can be iatrogenic due to IV volume expansion. When IV fluid replacement is used in patients who are not volume depleted, the clinician should discontinue treatment every 8 – 12 hours. Discontinuation of therapy should produce a slowing of iatrogenic diuresis, whereas POD will continue unabated. One should re-

member that the obstructed kidney has transiently lost its ability to transport chloride and sodium from the lumen of the loop of Henle to the medullary interstitium. The urine concentration of sodium should be high until medullary tonicity is restored, a process requiring urea. If IV fluids are administered too vigorously, the urea concentration will be lowered and the medullary interstitium will be "washed out", delaying the recovery of concentrating ability. In this scenario, the clinician should allow the BUN to rise and follow the urinary sodium. As medullary tonicity rises and the ability to concentrate urine and conserve sodium is restored, the urinary sodium will fall, heralding the slowing of the diuresis within 24 – 48 hours [35].

Summary Points of POD

In summary, it should be remembered that POD stems from the relief of complete urinary obstruction (all functioning nephrons) and is usually, but not always, associated with significantly advanced but reversible renal failure. Urinary concentrating defects begin during the obstructive phase and are not resolved until alleviation of the obstruction and restoration of the hypertonic renal medullary gradient, via the countercurrent exchanger. Natriuresis occurs after the relief of complete obstruction because of defective sodium handling in the proximal and distal tubules. Additionally, the loss of renal medullary function (countercurrent multiplication) presents more sodium and chloride to a relatively nonresorptive distal tubule and collecting duct producing a solute-enriched urine. ANF levels are elevated in volume-expanded states, such as complete urinary tract obstruction, and facilitate a diuresis when the patient is no longer obstructed. An osmotic diuresis from retained urea also occurs.

Although volume overload may be present initially, it can progress to volume depletion when untreated diuresis occurs. In this case, IV fluid administration is needed to prevent prerenal azotemia and possibly shock. However, iatrogenic volume expansion from overzealous administration of IV fluids may prolong the diuretic phase. Advanced but generally reversible renal failure, an acquired distal nonhypokalemic renal tubular acidosis (Type IV RTA) and salt-wasting nephropathy are often present, and other significant electrolyte losses (magnesium, potassium, and chloride) can occur and require replacement. Treatment regimens must be individualized to the patient's volume status, severity of diuresis, mental status, and degree of electrolyte abnormalities. In this regard, vigilant monitoring of the patient's physical status as well as serum and urine chemistries remains the basis of optimum clinical care.

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