

Obstructive uropathy – acute and chronic medical management

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Abstract

Obstructive uropathy is an important cause of acute and chronic kidney disease. Decompression of the urinary tract is an essential aspect of treatment. The cause and aetiology of obstruction typically determine the surgical approach. Acute relief of obstruction is frequently complicated by fluid and electrolyte imbalance. Standard therapeutic interventions for acute or chronic renal failure also apply for cases of obstructive uropathy. This narrative review summarises the early and long-term medical management of obstructive uropathy.

Key Words: Obstructive uropathy; Nephrology; Urology; Post-obstructive; Diuresis

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Core Tip: Obstructive uropathy is a prevalent cause of acute and chronic kidney disease. Urinary tract decompression is the single most beneficial aspect of management; renal impairment is frequently reversible and the long-term renal prognosis is generally excellent. Subsequent medical care is an important but underappreciated supplement to surgical treatment. Acute relief of obstruction is often complicated by disorders of sodium and potassium balance and post-obstructive diuresis. Longer-term management principles are similar to those for other forms of chronic kidney disease.

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INTRODUCTION

Obstructive uropathy is an important and potentially reversible cause of acute and chronic kidney disease. It may be broadly defined as a functional or structural impediment to urinary flow with resulting renal injury. Some sources label obstructive ‘uropathy’ as the condition causing a blockage to the flow of urine and obstructive ‘nephropathy’ as the ensuing renal parenchymal disease. With an incidence of 1.7 per 1000 people[1], obstructive uropathy accounts for approximately 10% of all cases of both acute and chronic kidney disease[2,3], including 5% of the chronic dialysis population[4]. Post-renal causes of kidney disease are particularly common in paediatric and geriatric groups.

Obstructive uropathy can be classified as acute or chronic, unilateral or bilateral, partial or complete, and intrinsic or extrinsic. There is an array of causes (Table 1) affecting both the upper and lower urinary tract; obstruction may occur anywhere from the renal calyces to the urethral meatus. The single most common cause of obstructive uropathy is benign prostatic hyperplasia, probably followed by neurogenic bladder[2]. In women the most frequent aetiology is a pelvic mass, while ureteric calculi are the major contributor in middle-aged adults and patients with a solitary kidney[2,5].

Prompt urinary system decompression is vital, which may be achieved through a variety of methods depending on the cause and site of blockage. Urologists are primarily responsible for relieving obstruction and for treatment of the underlying lesion. However, collaboration with nephrologists may be requested because sudden decompression is frequently complicated by abnormal fluid and electrolyte balance and long-term renal insufficiency. Although longstanding experience and high-quality data support a spectrum of urologic interventions, limited published evidence exists to guide the medical management of obstructive kidney diseases in the acute and chronic context. Obstructive uropathy is less represented in the literature than other causes of medical kidney disease. This evidence-based narrative review outlines the acute and post-decompression medical management of obstructive uropathy.

SEARCH METHODS

A structured search of the PubMed database was undertaken from inception to September 2022, using a range of applicable search terms in various combinations, such as “obstructive uropathy”, “post-obstructive diuresis”, and “bladder decompression”. Results were screened for relevance. A broad selection of articles were obtained, including clinical trials, commentaries, and case series. Additional papers were retrieved by manually searching guidelines or article reference lists. Articles were limited to the English language. Results of this literature search were synthesised to generate this narrative review.

PATHOPHYSIOLOGY OF URINARY TRACT OBSTRUCTION AND DECOMPRESSION

Obstructive uropathy pathogenesis begins with a post-renal obstructing lesion that impedes urinary flow. A sequence of physiologic and pathophysiologic events develop thereafter, the severity of which is related to the degree of obstruction. Continuous urine production is initially maintained through normal glomerular filtration and urinary tract peristalsis, resulting in increased pressure proximal to the blockage. The high-pressure system is perpetuated by compensatory smooth muscle stretching and hypertrophy (shown macroscopically by hydroureteronephrosis). Raised upper tract pressure reduces net hydraulic pressure thereby lowering the glomerular filtration rate (GFR), and reduces renal perfusion which subsequently causes ischaemic injury. These processes beget inflammation, tubular atrophy, and interstitial fibrosis within 2 h of total obstruction[3,4]. Urinary acidification and concentration mechanisms become impaired (‘hyposthenuria’), manifesting as blood electrolyte derangements and polyuria, which are relatively unpredictable and determined by affected sections of renal cortex and medulla[6].

Unremitting obstruction will eventually lead to scarring and atony and culminate in end-stage renal failure (ESRF), over days to months. In experimental models, some renal function persists for at least 2 wk after ureteric ligation but not longer than approximately 4 wk[7,8]. The onset of complete obstruction does not induce an abrupt cessation of kidney function because once a pressure threshold is met, backward urine leakage into the interstitium occurs (‘pyelointerstitial backflow’) with subsequent lymphatic drainage[9]. This safety mechanism also explains why spontaneous rupture of the obstructed collecting system is very rare.

Relief of urinary tract obstruction results in reversal of the processes above. Convalescence may be brisk owing to functional hypertrophy of unaffected nephrons, but incomplete renal recovery is common.

Table 1 Causes of urinary tract obstruction by anatomical level

Level of urinary tract obstruction	Causes
Upper tract (kidney, ureter)	Calculi
	Tumour
	Upper tract bleeding (<i>e.g.</i> , bleeding renal cancers, traumatic)
	Anatomic abnormality (<i>e.g.</i> , pelvi-ureteric junction obstruction)
	Papillae sloughing (<i>e.g.</i> , pyelonephritis, tuberculosis)
	Ureteric stricture
	Ureterocoele
Lower tract (bladder, prostate, urethra)	Calculi
	Tumour
	Neurogenic bladder
	Benign prostatic hyperplasia
	Prostatitis
	Cystocoele
	Bleeding (<i>i.e.</i> , clot retention)
	Urethral stricture
	Posterior urethral valves
Extrinsic	Retroperitoneal fibrosis
	Aneurysm
	Pregnancy
	Faecal impaction
	Pelvic organ prolapse
	Phimosis

SURGICAL ISSUES

The site and aetiology of urinary tract obstruction determine the surgical approach. For example, an upper tract process may necessitate ureteric stenting or placement of a nephrostomy tube to achieve adequate decompression of the blocked collecting system, while lower tract pathology (which is typically prostatic in nature for men) may benefit from pharmacotherapy, bladder catheterization, or bladder outlet surgery depending on the acuity and degree of obstruction. There is a myriad of surgical options and techniques employed in the management of urinary tract obstruction, for which a full discussion is beyond the scope of this article.

Definitive urologic surgery should be delayed if possible until after the patient is medically stable and urgent decompression has been accomplished. Resuscitation and emergency drainage are particularly important in cases of obstruction-related severe renal failure or in the setting of an 'infected obstructed' kidney. Temporising interventions, such as percutaneous nephrostomy or bladder catheter insertion, almost always precede a secondary corrective procedure performed at a later date. Medical issues requiring optimisation are often encountered at this initial presentation rather than the time of elective surgery.

Rapid bladder drainage may result in decompression haematuria, or haematuria *ex vacuo*. Relief of a chronically distended bladder, which is associated with a friable bladder wall and capillary damage, leads to macroscopic bleeding in approximately 10% of cases[10]. Decompression haematuria is almost always transient and of little clinical importance. Irrigation is sometimes required. A small randomised control trial demonstrated no benefit in gradual bladder drainage compared to rapid drainage with respect to the risk of macrohaematuria[11].

It should be recognised that urgent decompression is not indicated for all patients with urinary tract obstruction. Those with chronic symptoms, for example men seen in the outpatient setting with lower urinary tract symptoms and bladder outflow obstruction, in the absence of complications are generally suitable for a trial of medical therapy which can be complemented by elective surgery where needed. Complications that should trigger immediate bladder catheterisation include hydronephrosis, chronic urinary retention (usually defined as post-void residual bladder volumes of greater than around 300

mL), and renal impairment (obstructive uropathy).

FLUID MANAGEMENT

Clinical background and genesis

Sudden reversal of obstruction is followed by a polyuric phase in approximately two-thirds of patients with obstructive uropathy[12]. Polyuria is typically a physiologic response in which the kidney aims to restore euvolaemia and normal plasma concentrations, conceptually similar to the polyuric phase of recovering acute tubular necrosis. This osmotic diuresis of retained fluid, urea, and other nitrogenous solutes usually resolves within 24 h.

Prolonged or marked polyuria may be pathologic and extend beyond the reestablishment of homeostasis. The working term for this clinical situation is post-obstructive diuresis (POD). POD is defined as urine output greater than 200 mL/h for at least 2 consecutive hours or a urine output exceeding 3 L in 24 h[12,13]. POD seldom lasts longer than 48 h. Mechanisms of POD include impaired urinary concentrating ability due to aquaporin downregulation and loss of medullary tonicity, and dysfunction of tubular transporters because of cell apoptosis. POD affects approximately 2% of patients with complete unilateral obstruction and up to 50% of patients with bilateral obstruction[14,15]. Risk factors include complete or chronic obstruction. Risk is also proportionate to the creatinine elevation and residual bladder volume at the time of presentation; a bladder volume greater than 1500 mL prior to decompression is often associated with POD[13,16].

Treatment

Mild polyuria typically resolves without any intervention within a few hours or days and oral hydration as an outpatient therapy is generally sufficient. Patients are generally asymptomatic apart from occasional postural hypotension. Patients should be instructed to drink to thirst and avoid dehydration with a minimal daily fluid intake of approximately 2 L or 25 mL/kg. Self-monitoring of urine colour can be a simple guide allowing patients to assess their hydration status.

POD, a significant diuresis beyond 2 d, or any episodes of hypotension warrant hospitalisation for observation and fluid replacement because of the risk of electrolyte abnormalities or hypovolaemia, which may precipitate cardiovascular collapse. Regular monitoring of vital signs, an accurate fluid balance chart, and daily weights are essential. Postural blood pressure checks are also useful. Hourly urine output measurement is indicated until patient condition settles. The urine drainage catheter should be allowed to drain freely without intermittent clamping as this allows accurate assessment of urine output; historical concerns surrounding rapid decompression are unfounded, as free drainage has not been reliably shown to be associated with more complications than gradual decompression[17,18].

There is no consensus on the best fluid replacement strategy in POD and treatment must be individualised. Controlled trials to inform practice are absent. Fluid prescribing can be complex in post-obstruction patients with ongoing losses, haemodynamic instability, and electrolyte abnormalities. POD is a unique scenario where replacement fluid is administered to patients who are volume-overloaded, in anticipation of steady ongoing losses. A useful strategy is to mentally separate the patient with POD into a volume-overloaded phase and a volume-depleted phase. In the initial volume-overload phase, a preferred method is to administer intravenous fluids at 50% the rate of the preceding hour's urine output. This allows for controlled weight loss and downward titration of intravenous fluid replacement without driving endless diuresis. Crystalloid solutions are recommended rather than colloid; the type of crystalloid depends on the subject's biochemical parameters. Enteral solutions may be appropriate in reliable patients who can manage comfortably. Should an ensuing hypovolaemic phase occur, standard resuscitation principles apply; therapy should account for deficits, maintenance requirements, and ongoing losses. Most patients do not reach a hypovolaemic phase as they successfully achieve homeostasis and diuresis abates with decremented fluid replacement.

ELECTROLYTE DERANGEMENTS

Clinical background and genesis

Obstructive uropathy may be complicated by a number of electrolyte abnormalities, both before and after acute relief of obstruction, particularly in the context of high-grade chronic obstruction[19]. Laboratory findings vary depending on the degree of corticomedullary damage, GFR, and volume status. Early post-renal obstruction generally produces a state of tubular solute wasting, notably of sodium, potassium, bicarbonate, magnesium, calcium, and phosphate. As renal function declines, these abnormalities may be accompanied by gradual retention of potassium, hydrogen, chloride, and ammonium. Obstructive uropathy is a relatively common cause of proximal and distal renal tubular acidosis. Findings may be indistinguishable from other causes of acute or chronic kidney injury.

Sodium and potassium disorders are the most serious and frequent considerations in practice. Although the majority of cases are mild, life-threatening or refractory presentations are sometimes encountered. An overview of the management of sodium and potassium disorders in the setting of urinary tract obstruction is presented below. Other acid-base and electrolyte problems are clinically insignificant without ESRF and will not be discussed in this review.

Hyponatraemia

Hyponatraemia as a consequence of plasma dilution and tubulopathy is a common finding. It often takes several weeks following decompression before plasma sodium concentration returns to a normal range[20]. Mild hyponatraemia between 130-135 mmol/L requires no specific treatment because it should not be associated with symptoms or clinical complications. Liberal fluid intake should be avoided and a normal diet encouraged, effectively being a fluid restriction in the face of post-decompression polyuria. However, for obvious hypervolaemic hyponatraemia, standard fluid restriction is appropriate.

Moderate or severe hyponatraemia requires strict monitoring of fluid balance and plasma sodium. The frequency of blood testing must be decided on a case-by-case basis. Severe hyponatraemia is defined as a plasma sodium level below 120 mmol/L or the presence of hyponatraemia with neurological dysfunction. Like all electrolyte disturbances, the basic treatment strategy for severe hyponatraemia must establish the body's electrolyte deficit, the desired rate of correction, and the ongoing losses. There are many methods to estimate sodium balance and suitable replacement regimens, one of which is shown in [Supplementary Table 1](#). While useful, prediction formulas are relatively inaccurate and should be interpreted within the overall clinical picture. Sodium replenishment should be adjusted according to patient progress and serial blood sampling; gradual correction is preferable. With respect to POD, although precise sodium calculations can be attempted, administration of intravenous 0.9% sodium chloride at half the urine output rate is ultimately sufficient in the majority of cases.

Transurethral resection of the prostate (TURP) is a widespread surgical treatment for patients with obstructive uropathy, and the TURP syndrome therefore warrants special mention. TURP syndrome is characterised by dilutional hyponatraemia developing early post-TURP due to absorption of hypotonic irrigation solution through open prostatic venous sinusoids. Patients can absorb more than 1 L of fluid intraoperatively in this way. This potentially life-threatening syndrome is becoming increasingly rare as recognition and preventative measures improve. Use of bipolar diathermy with isotonic irrigation solution has reduced its incidence. TURP syndrome is usually effectively managed with fluid restriction and diuretics.

Hypernatraemia

Prolonged diuresis of dilute urine may result in a hypernatraemic volume-depleted state. Such patients require net rehydration rather than tapered chasing of the urine output. The incidence of hypernatraemia in obstructive uropathy is unknown but appears relatively small. Onset is typically several days post-decompression. All episodes are serious due to the risk of cerebral oedema; hypernatraemia is probably among the strongest predictors of death in individuals with POD.

Like the management of hyponatraemia, treatment is based on an estimate of water deficit and ongoing losses and is regularly adjusted according to response. Hypotonic crystalloid such as 5% glucose should be used to reach a target plasma sodium at the upper limit of normal at 145 mmol/L ([Supplementary Table 2](#)).

Hypokalaemia

Hypokalaemia is the commonest electrolyte abnormality of obstructive uropathy. It is most often encountered immediately after relief of obstruction due to urinary wasting and is self-limiting; approximately 30% of episodes of POD are associated with hypokalaemia[21]. Potassium supplementation can be difficult to predict and usually requires repeated assessment and titration. As a general rule, each 1 mmol/L fall in plasma potassium concentration equates to a body deficiency of around 200 mmol. Oral potassium replacement is generally adequate and is guided by biochemistry results, ensuring a serum level greater than at least 2.5 mmol/L but ideally above 3.5 mmol/L. Insoluble and effervescent potassium tablets each contain approximately 8 mmol and 14 mmol of potassium, respectively. Severe hypokalaemia with plasma potassium below 2.5 mmol/L deserves cardiac monitoring and intravenous replacement. The maximum daily dose by any route must be less than 400 mmol to avoid malignant arrhythmias. Simultaneous magnesium supplementation is necessary for concurrent hypomagnesaemia.

Hyperkalaemia

Post-renal obstruction complicated by severe renal impairment or focal injury of the distal tubules may give rise to hyperkalaemia. Hyperkalaemia is mostly a sign of established renal failure from a chronic process but can also occasionally evolve after prolonged post-decompression diuresis in subjects with normal kidney function.

Potassium-lowering therapies are not usually necessary since potassium homeostasis rapidly resets following collecting system decompression. Although hyperkalaemia treatment has not been widely studied in the specific context of uropathies, it is a well-known medical emergency and typical pharmacologic regimens apply. Readers should refer to standard reference texts. Retrospective data demonstrates that haemodialysis is utilised in fewer than 15% of acute hospitalisations for obstructive uropathy[22]. Patients requiring dialysis from the time of index presentation almost always imply undetected chronic urinary tract obstruction.

PERIOPERATIVE MEDICATION MANGEMENT

Medication adjustments are frequently necessary following urinary tract decompression. For example, dosing is influenced by kidney function which is frequently poor and fluctuating in this scenario, and nephrotoxins should be withheld. Common considerations are listed in [Table 2](#).

PHARMACOLOGIC THERAPY FOR OBSTRUCTIVE UROPATHY

It is usually appropriate for patients with obstructive uropathy to receive nephrology follow-up. There is no directly proven reno-protective medical therapy for obstructive uropathy beyond standard measures such as cardiovascular risk factor reduction and proteinuria-lowering agents. Statins have improved renal recovery in animal studies but there is no supporting data in humans[23].

Angiotensin-converting enzyme (ACE) inhibitors appear similarly as effective for long-term renal preservation as in other causes of chronic kidney disease (CKD), but may aggravate injury if introduced too early post-obstruction[3,24-26]. Hypertension is prevalent among individuals with obstructive uropathy and, like in all forms of CKD, is associated with worse outcomes. Guideline-directed antihypertensive therapy is recommended[27].

Sodium-glucose transport protein 2 (SGLT2) inhibitors demonstrated an anti-fibrotic effect when introduced in laboratory rats exposed to iatrogenic ureteric obstruction[28]. In the Dapagliflozin in Patients with Chronic Kidney Disease (DAPA-CKD) randomised control trial, the group randomised to SGLT2 inhibitor treatment, including a small number of subjects with obstructive uropathy, experienced significant renal and cardiovascular benefits[29]. SGLT2 inhibitor use in the presence of an indwelling catheter or instrumentation may increase the risk of urological infection.

Given the prevalence of bladder outlet obstruction from prostatic hyperplasia, nephrologists also require some understanding of basic pharmacologic therapies for this problem ([Table 2](#)). Other specific causes of uropathy which benefit from adjuvant medical therapy include retroperitoneal fibrosis, with immune-modulators, nephrolithiasis in the form of calcium channel blockers, and cancer through chemotherapy.

PROGNOSIS

The most important predictors of renal recovery are the completeness and duration of obstruction, and the presence or absence of coexisting infection. There is an inverse relationship between chronicity and reversibility; urinary tract obstruction for less than 1 wk is typically associated with complete recovery. The majority of patients experience complete recovery of kidney function with reversal of obstruction. Most improvement occurs in the first fortnight following decompression but ongoing renal recovery may be seen for up to 6 mo. High-grade obstructive uropathy for more than 6-8 wk is said to be irreversible. However, with good preventative care the trajectory of CKD related to obstructive uropathy tends to be benign with only 3% of patients progressing to dialysis at 10 years[22]. A significant minority of patients commenced on dialysis at the time of diagnosis gradually improve and are freed from dialysis after several months[7].

Total destruction of an infected obstructed kidney may occur within a matter of days. Without decompression the infected obstructed kidney is associated with a 40% mortality[30], compared to a mortality rate of less than 5% following successful decompression[31].

Positive prognostic features for renal recovery include POD, younger age, a normal pre-morbid GFR, and lower grades of hydronephrosis as opposed to higher[14,32]. Obstruction severity is more important for renal outcome than aetiology, though bladder outflow obstruction appears to yield a better prognosis than upper tract obstruction because vesical trabeculation and hypertrophy protect the renal parenchyma from a high-pressure system.

Although obstructive uropathy is associated with low short-term mortality, intermediate- and long-term outcomes are poor, particularly for those older than 85 years of age[5]. The median 12-mo survival for patients presenting with malignant and non-malignant obstruction is roughly 40% and 90%, respectively[22].

Table 2 Common perioperative considerations and medical therapies for obstructive uropathy

Medication class	Comments
Perioperative care	
Non-steroidal anti-inflammatory drugs	<p>Should be avoided or used cautiously in any form of acute or chronic kidney disease</p> <p>There is ample experimental and clinical evidence suggesting that non-steroidal anti-inflammatory drugs (NSAIDs) may worsen kidney function in patients with renal impairment, especially during a concomitant physiologic insult, and delay renal recovery from acute kidney injury (AKI)[33-36]. NSAIDs interfere with renal auto-regulation and can directly induce <i>de novo</i> AKI through several mechanisms.</p>
Antihypertensives	<p>Hypertension is frequently seen in patients with obstructive uropathy, due to volume expansion and upregulation of renin and erythropoietin release because of focal hypoxia[37,38]</p> <p>Hypertension may reverse rapidly following acute relief of obstruction and diuresis, so antihypertensive medications should be rationalised accordingly</p> <p>Renin-angiotensin system blockade, with angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, should be avoided or prescribed carefully due to the well-recognised risks of pre-renal AKI and hyperkalaemia</p>
Antibiotics	<p>Leucocytosis and raised inflammatory markers are commonly seen in acute presentations of urinary tract obstruction as part of the stress response. A low threshold for empiric antimicrobial coverage for urinary infection is prudent</p> <p>Renal drug clearance declines roughly in proportion to the drop in GFR. Antibiotic dosing or frequency may need to be reduced, depending on the agent. Antibiotics considered 'nephrotoxic' may need to be withheld, such as vancomycin and gentamicin</p> <p>Urosepsis in the context of an obstructed collecting system is tissue-invasive. Therefore, selected antibiotics must be broad-spectrum and penetrant, and reach therapeutic levels quickly. Intravenous ampicillin or ceftriaxone are typical choices, which can be modified based on culture sensitivities</p>
Obstructive uropathy pharmacologic therapies	
Alpha-1 adrenergic receptor antagonists	<p>Common uses: Benign prostatic hyperplasia, urolithiasis (medical expulsive therapy)</p> <p>Rationale: Induce smooth muscle relaxation, thereby enlarging ureteral and urethral calibre and improving flow</p> <p>Examples: Prazosin (non-selective), tamsulosin (selective), silodosin (selective)</p>
5-alpha reductase inhibitors	<p>Common uses: Benign prostatic hyperplasia</p> <p>Rationale: Targeted antiandrogen effect, thereby reducing prostate volume and the static component of bladder outlet obstruction</p> <p>Examples: Dutasteride, finasteride</p> <p>Combination tablets with alpha-1 adrenergic receptor antagonists are also widely available</p>
Phosphodiesterase-5 inhibitors	<p>Common uses: Benign prostatic hyperplasia, erectile dysfunction</p> <p>Rationale: Exact mechanism of action in lower urinary tract symptoms is unclear but may antagonise phosphodiesterase (PDE) receptors on smooth muscle cells, thus inducing urethral relaxation and improved urine flow, or increase bladder and prostate perfusion Examples: Sildenafil, tadalafil, vardenafil</p> <p>PDE-5 inhibitors are commonly prescribed for men with erectile dysfunction and coexisting features of prostatism.</p> <p>PDE-5 inhibitors may also be used in combination with alpha-1 adrenergic receptor antagonists or 5-alpha reductase inhibitors</p>

TAKEAWAY POINTS

The therapeutic approach to obstructive uropathy is less well understood than in other forms of acute or chronic renal failure. A joint effort between urologists and physicians is necessary. Priorities include catheterisation and surgical correction of the underlying lesion.

An early post-decompression polyuric phase with electrolyte losses is common, requiring judicious monitoring and replacement. Although usually mild, post-obstructive diuresis may be a complex process that quickly transitions from volume overload to dehydration and an attentive fluid management protocol is warranted. Nephrologists should be particularly wary of evolving hypernatraemia, which must be treated urgently.

Many patients develop CKD and should receive long-term nephrology follow-up. Cardiovascular risk factor reduction is pertinent and evidence-based, including blood pressure control and use of ACE inhibitors and SGLT2 inhibitors for reno-protection. The kidney-specific prognosis is generally favourable with few patients proceeding to dialysis.

CONCLUSION

Obstructive uropathy is a potentially reversible condition. Surgical decompression of the urinary system is the key component of care, but many surrounding medical issues must also be considered. Most cases of obstructive uropathy experience complete renal recovery, but a significant minority develop chronic kidney disease or require dialysis. Patients therefore benefit from inter-speciality collaboration between urologists and nephrologists.

FOOTNOTES

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