JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

HYDRONEPHROSIS AND ITS CLINICAL IMPACT ON RENAL FUNCTION: EVIDENCE FROM RECENT LITERATURE

Maulafi Alhamdi S*
Master's Student in Public Health, Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Aceh, Indonesia*

Keywords:
Acute kidney injury,
Chronic kidney disease,
Hydronephrosis,
Obstructive uropathy,
Renal recovery.

Received: 7 February 2026
Revised: 2 March 2026
Accepted: 2 March 2026
Available online: 01 November 2026

Corresponding Author:
E-mail: Maulafialhamdis@gmail.com

ABSTRACT

Background: Hydronephrosis is frequently identified on imaging and is commonly regarded as a marker of urinary tract obstruction. Although recent studies have clarified several mechanisms of obstructive uropathy, the relationship between hydronephrosis severity and renal functional impairment remains inconsistent. Increasing evidence indicates that radiological dilatation does not reliably predict the extent of renal injury, particularly in acute kidney injury (AKI) and in the presence of delayed decompression, infection, or malignancy. **Objective:** This narrative integrative review aims to synthesize recent evidence on the clinical impact of hydronephrosis on renal function, focusing on pathophysiological mechanisms, diagnostic challenges, acute and long-term renal outcomes, and determinants of renal recovery after obstruction relief. **Methods:** This study was conducted as a narrative integrative review of peer-reviewed literature published between 2021 and 2026. Relevant articles were identified from major biomedical databases and selected based on thematic relevance to obstructive uropathy, hydronephrosis, AKI, acute kidney disease (AKD), and chronic kidney disease (CKD). The review provides a conceptual synthesis of experimental studies, observational cohorts, systematic reviews, and consensus statements. No formal systematic review methodology, PRISMA framework, or quality appraisal tool was applied. **Results:** The evidence demonstrates that obstruction induces hemodynamic disturbance, tubular epithelial injury, inflammation, microvascular dysfunction, and fibrotic remodeling, processes that may persist after decompression and limit renal recovery. Hydronephrosis severity correlates poorly with renal dysfunction, and clinically significant obstructive AKI may occur without marked dilatation. Renal recovery is heterogeneous and influenced by obstruction duration, AKI severity, baseline renal reserve, infection, malignancy-related obstruction, and timing of intervention. Obstructive AKI may progress to AKD and increase long-term CKD risk. **Conclusion:** Hydronephrosis should be interpreted as a context-dependent indicator of renal vulnerability rather than a purely anatomical finding. Integrated clinical and functional assessment, together with structured follow-up, is essential to optimize renal recovery and mitigate chronic kidney disease progression.

Copyright ©2026 by Authors. Published by Faculty of Medicine, Universitas Diponegoro Semarang Indonesia. This is an open access article under the CC-BY-NC-SA (https://creativecommons.org/licenses/by-nc-sa/4.0/).

INTRODUCTION

Hydronephrosis is defined as dilatation of the renal pelvis and calyces resulting from impaired urinary drainage and is most commonly associated with urinary tract obstruction.[1] Although frequently identified through imaging, hydronephrosis represents more than a structural abnormality, as it may reflect clinically significant obstructive uropathy with direct consequences for renal function.[2] Within the broader context of kidney disease, obstructive uropathy is a well-recognized cause of post-renal acute kidney injury (AKI) and is considered one of the potentially reversible etiologies of renal dysfunction when timely intervention is achieved.[2] However,

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

accumulating evidence indicates that delayed or prolonged obstruction can lead to persistent renal injury, incomplete recovery, and progression to chronic kidney disease (CKD), underscoring the clinical importance of understanding the functional implications of hydronephrosis rather than relying solely on its anatomical description.[3]

The rationale for reviewing the literature on hydronephrosis and renal function is driven by growing recognition that the relationship between radiological findings and renal functional impairment is complex and frequently underestimated. While hydronephrosis is commonly used as a surrogate marker for urinary tract obstruction, several studies have demonstrated that the severity of pelvicalyceal dilatation does not consistently correlate with the degree of renal dysfunction or the potential for recovery after relief of obstruction. Experimental and translational studies have shown that obstruction initiates a cascade of pathophysiological changes, including increased intratubular pressure, altered renal hemodynamics, inflammatory activation, and progressive tubulointerstitial fibrosis, mechanisms that are central to irreversible nephron loss when obstruction is sustained.[4, 5] Clinically, AKI secondary to obstructive uropathy has been associated with adverse long-term renal outcomes and increased mortality, particularly in patients with severe AKI or malignancy-related obstruction, reinforcing the concept that obstruction-related renal injury is not always benign or transient.[6]

Despite the growing body of literature addressing obstructive uropathy and acute kidney injury, recent reviews have largely focused on either mechanistic pathways of fibrosis or clinical management algorithms in isolation. Few syntheses have explicitly examined the discordance between radiological hydronephrosis severity and functional renal impairment, nor have they systematically integrated acute injury, intermediate acute kidney disease (AKD), and long-term chronic kidney disease (CKD) trajectories within a unified conceptual framework. Consequently, hydronephrosis continues to be interpreted predominantly as an anatomical descriptor rather than a dynamic indicator of renal vulnerability.

This review addresses that conceptual gap by reframing hydronephrosis as a context-dependent marker of renal susceptibility and recovery potential. By integrating recent mechanistic evidence, diagnostic limitations, and longitudinal outcome data, this manuscript aims to provide a clinically oriented synthesis that links structural findings to functional prognosis across the AKI–AKD–CKD continuum.

METHODS

This study was conducted as a narrative integrative review aimed at synthesizing recent evidence regarding the clinical impact of hydronephrosis on renal function. The review focused on literature published between January 2021 and February 2026 to ensure inclusion of contemporary mechanistic and clinical data.

A structured but non-systematic search strategy was performed using major biomedical databases, including PubMed, Scopus, and Web of Science. Combinations of keywords and Medical Subject Headings (MeSH) terms were used, including “hydronephrosis,” “obstructive uropathy,” “acute kidney injury,” “acute kidney disease,” “chronic kidney disease,” “renal fibrosis,” “urinary obstruction,” and “renal recovery.” Boolean operators (AND/OR) were applied to refine search combinations.

Articles were selected based on relevance to the following thematic domains: (1) pathophysiology of obstructive renal injury, (2) diagnostic challenges and imaging–function discordance, (3) hydronephrosis-associated acute kidney injury, (4) progression from AKI to AKD and CKD, and (5) determinants of renal recovery after obstruction relief. Eligible sources included experimental studies, translational research, observational cohort studies, systematic reviews, meta-analyses, and consensus statements. Case reports were included selectively when they illustrated emerging diagnostic phenomena such as non-dilated obstructive nephropathy.

This review did not follow a formal systematic review protocol (e.g., PRISMA), and no structured quality appraisal tool or quantitative meta-analysis was performed. Instead, evidence was synthesized using a thematic and conceptual integration approach to provide a clinically oriented framework linking structural findings of hydronephrosis with functional renal outcomes.

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

Pathophysiology of Obstructive Renal Injury

Obstructive renal injury is initiated when urinary outflow is impaired, producing upstream increases in hydrostatic pressure within the renal pelvis and tubular system. This pressure-loading state rapidly perturbs filtration dynamics and renal perfusion, leading to an early fall in glomerular filtration rate (GFR) that reflects both reduced net filtration pressure and evolving intrarenal hemodynamic dysregulation. Contemporary syntheses emphasise that obstruction is not a purely mechanical problem; rather, it triggers a coordinated neurohumoral and paracrine response involving vasoregulatory pathways including RAAS and endothelin-related signalling that drives a biphasic renal blood flow pattern. Over time, these hemodynamic shifts contribute to regional hypoxia and create a milieu that favours progressive parenchymal injury.[4]

At the tubular level, pressure overload and hypoxia converge to induce epithelial stress responses that are increasingly understood as central determinants of the obstruction phenotype. Persistent mechanical strain and impaired microcirculatory oxygenation promote tubular epithelial injury characterised by loss of polarity, transport dysfunction, and activation of regulated cell death and stress programmes. Importantly, these epithelial changes are not merely downstream consequences; they actively shape the interstitial microenvironment through secretion of cytokines, chemokines, and profibrotic mediators that amplify inflammatory recruitment and stromal activation. Recent integrative reviews of kidney fibrosis mechanisms place tubular epithelial maladaptive repair as a pivotal step linking an initial insult (such as obstruction) to sustained interstitial remodelling and functional decline.[7]

Microvascular dysfunction has emerged as a reinforcing axis in obstructive nephropathy, particularly through peritubular capillary impairment and chronic hypoxia. The concept is clinically relevant because microvascular rarefaction or capillary dysfunction can persist even after relief of obstruction, thereby sustaining hypoxic signalling and limiting epithelial recovery. This hypoxia–fibrosis coupling is increasingly supported by mechanistic work connecting microvascular injury to ongoing profibrotic transcriptional programmes and extracellular matrix accumulation. In practice, this helps explain why timely decompression does not guarantee full functional recovery, especially after prolonged obstruction with established microvascular compromise.[8, 9]

Inflammation is a core driver of progression from functional impairment to structural damage in obstructive kidney injury. Following obstruction, innate immune activation particularly macrophage recruitment amplifies epithelial injury and accelerates fibroblast activation via cytokine and chemokine networks. In unilateral ureteral obstruction (UUO) models, complement-related signalling and inflammasome pathways have been implicated in promoting interstitial fibrosis, illustrating how immune effector mechanisms can directly couple obstruction to scarring. These inflammatory pathways are clinically meaningful because they provide a mechanistic rationale for why sustained obstruction can produce durable structural changes, even when the initial trigger is later relieved.[10]

A distinguishing feature of chronic obstructive nephropathy is tubulointerstitial fibrosis the final common pathway of irreversible renal functional loss. Recent literature emphasises that fibrosis reflects not only fibroblast proliferation but also phenotypic transitions and activation across multiple stromal compartments, resulting in expansion of myofibroblast populations and excessive extracellular matrix deposition. High-resolution approaches, including single-cell transcriptomics in fibrotic kidney models, have refined this concept by mapping discrete stromal and immune states associated with either adaptive repair or progressive fibrosis. In parallel, lineage-tracing and mechanistic studies continue to clarify the cellular origins of myofibroblasts in renal fibrosis, supporting a multi-source model in which resident mesenchymal populations play a central role.[11, 12]

Maladaptive repair has also been linked to cell-cycle dysregulation in tubular epithelial cells. Recent mechanistic work indicates that epithelial G2/M arrest is not merely a marker of injury severity but may actively promote fibrogenesis through sustained profibrotic signalling and impaired regenerative progression. Studies describing regulatory nodes controlling tubular cell-cycle arrest and its downstream fibrotic consequences reinforce the concept that obstruction can lock in a maladaptive epithelial state, thereby favouring chronic

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

remodelling rather than restoration of normal architecture.[13, 14]

Contemporary clinical evidence aligns with these mechanistic insights by demonstrating that obstruction-associated AKI is not uniformly benign. In adult obstructive uropathy, AKI has been associated with worse long-term kidney outcomes and reduced survival, supporting the biological plausibility that the hemodynamic–hypoxic–inflammatory–fibrotic cascade can translate into durable clinical harm, particularly when obstruction is severe, prolonged, or malignancy-related. Moreover, diagnostic pitfalls such as non-dilated obstructive nephropathy underscore that clinically significant obstructive renal injury can occur even in the absence of evident hydronephrosis, potentially prolonging exposure to injurious pathways before decompression is pursued (Figure 1).[15, 16]

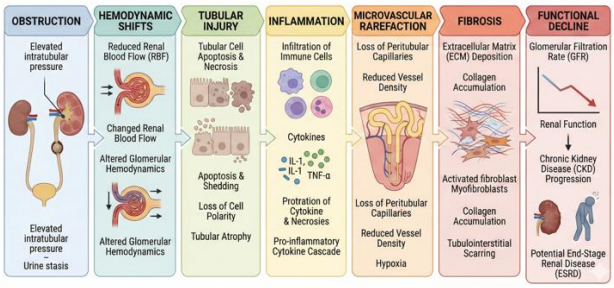


Figure 1 Pathophysiology of Obstructive Renal Injury.

Hydronephrosis and Acute Kidney Injury

Hydronephrosis becomes clinically consequential when it reflects functionally significant urinary tract obstruction capable of precipitating post-renal acute kidney injury (AKI), either through bilateral obstruction, obstruction in a solitary kidney, or unilateral obstruction complicated by superimposed infection, sepsis, or severe hemodynamic instability. Recent literature frames obstructive uropathy as a time-sensitive cause of AKI in which delayed recognition allows the transition from predominantly functional decline to structural injury and incomplete recovery, reinforcing the need to interpret hydronephrosis in the acute setting as a potential marker of ongoing nephron stress rather than a purely anatomical finding.[17, 18]

From an AKI pathophysiology standpoint, urinary outflow obstruction produces a low-flow AKI phenotype in which filtration drops rapidly due to increased tubular pressure and adaptive-to-maladaptive changes in intrarenal perfusion, while ongoing obstruction sustains renal congestion, hypoxia, and inflammatory amplification that can prolong or deepen AKI even before obvious parenchymal thinning appears. Contemporary AKI frameworks explicitly position urinary tract obstruction alongside other low-flow states as a distinct and reversible driver of AKI when timely decompression is achieved, but emphasize that reversibility is highly contingent on obstruction severity, duration, and concurrent infection.[19, 20]

Hydronephrosis-associated AKI spans a spectrum from asymptomatic creatinine rise detected incidentally on imaging to obstructive anuria with severe electrolyte derangements and urgent need for urinary diversion. The risk of AKI is particularly salient when hydronephrosis coexists with systemic infection or pyelonephritis, where obstruction may accelerate intrarenal pressure–mediated dysfunction while also impairing drainage of infected urine, increasing the likelihood of sepsis-associated kidney injury layered upon a post-renal component. A large observational analysis in hospitalized UTI patients found that the presence of urolithiasis with hydronephrosis was associated with 2-3 times higher risk of AKI than urolithiasis without hydronephrosis, supporting the clinical intuition that obstruction severity (as reflected by hydronephrosis) modifies AKI risk in infectious contexts.[21]

In patients presenting with AKI and pelvicalyceal dilatation, the diagnostic task is to decide whether the imaging abnormality represents active obstruction that is driving potentially reversible renal dysfunction and therefore warrants urgent drainage, rather than assuming that any hydronephrosis is clinically actionable.[22]

Because the yield of any imaging test is contingent on the pre-test probability generated by the bedside phenotype—urine output trajectory, pain pattern or lower urinary tract symptoms, fever/systemic inflammatory features, and the tempo of creatinine and potassium rise—recent guidance has increasingly treated radiologic findings as one strand of evidence that must be interpreted within a dynamic clinical frame.[22] This integrated approach is not merely philosophical, obstructive AKI can present with little or no collecting-system dilatation (including the recognised entity of non-dilated

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

obstructive nephropathy), particularly early in the course or when extrinsic compression and reduced urine production limit visible distension, so a negative or mild ultrasound cannot reliably exclude functionally important obstruction when suspicion is high.[16]

Management principles follow directly from the pathobiology, once obstructive AKI is confirmed (or strongly suspected in a high-risk presentation), the priority is prompt relief of obstruction by the most appropriate route (e.g., bladder catheterization for outlet obstruction, ureteral stent or percutaneous nephrostomy for upper-tract obstruction), while simultaneously correcting life-threatening derangements (hyperkalemia, severe acidosis, uremic complications) and treating infection when present. Recent nephrology–urology syntheses underline that time-to-decompression is a major determinant of renal recovery, and that concomitant infection is a key modifier that should lower the threshold for urgent drainage.[3]

After decompression, clinicians must anticipate the biphasic recovery pattern typical of post-renal AKI that is an early diuretic phase (post-obstructive diuresis, POD) may be physiologic, reflecting solute and volume excretion, but can become pathologic with sustained high urine output and clinically significant electrolyte disturbances. Contemporary literature in both adult and pediatric settings reiterates standard diagnostic thresholds (e.g., ≥200 mL/hour for two consecutive hours or >3 L/24 hours after relief of obstruction) and highlights clinically relevant complications including dehydration, hypovolemia, hypokalemia, and dysnatremias—particularly hazardous when monitoring is inadequate. Recent European Urology Open Science work further emphasizes that post-obstruction polyuria is common and that clinically significant complications such as hematuria, dehydration, and electrolyte abnormalities are not rare in real-world practice, strengthening the argument for protocolized post-decompression monitoring in hydronephrosis-associated AKI.[23, 24]

Beyond the binary presence or absence of hydronephrosis, emerging data suggest that pattern recognition within imaging phenotypes may refine AKI risk stratification. Bilateral symmetrical dilation, asymmetric upper tract distension with cortical thinning, and rapidly progressive pelvicalyceal expansion represent distinct hemodynamic contexts with different implications for intrarenal pressure transmission and compensatory capacity.[25] These radiologic nuances may provide incremental prognostic information beyond traditional grading systems, particularly when interpreted alongside urine output trajectory and dynamic creatinine trends.[22]

Another underappreciated dimension of hydronephrosis-associated AKI is the phenomenon of biochemical–radiologic discordance. In some patients, creatinine rise is disproportionate to the degree of visible dilation, reflecting either early obstruction before anatomical remodeling or significant parenchymal vulnerability due to limited nephron reserve. Conversely, marked dilation with stable renal indices may represent compensated obstruction. This discordance reinforces the concept that obstruction severity cannot be inferred from imaging alone and that renal functional reserve critically shapes the clinical phenotype.[3, 26]

The masking effect of contralateral compensation in unilateral obstruction further complicates interpretation. Systemic creatinine may underestimate injury when the non-obstructed kidney hyperfilters, delaying recognition of clinically meaningful damage in the affected kidney. This compensatory phenomenon becomes particularly relevant in individuals with reduced baseline renal mass or pre-existing chronic kidney disease, in whom reserve capacity is limited and minor obstruction may precipitate overt AKI.[27-29]

Recent translational research also raises interest in subclinical tubular stress markers that precede overt creatinine rise. While not yet incorporated into routine practice, biomarkers reflecting tubular injury or cell-cycle arrest have been explored in broader AKI populations and may eventually refine early detection of obstructive injury before irreversible structural damage occurs. Their potential role in hydronephrosis-associated AKI highlights the evolving intersection between structural imaging and molecular injury signaling.[30]

Health-system factors introduce another layer of variability. Delays in imaging, under-recognition of non-dilated obstruction, and misattribution of AKI to sepsis or volume depletion may prolong obstruction duration before decompression is achieved. Observational data consistently show that time-to-relief influences recovery probability, suggesting that

JURNAL KEDOKTERAN DIPONEGORO
(DIPONEGORO MEDICAL JOURNAL)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

diagnostic latency not merely biological severity contributes to outcome heterogeneity.[31, 32]

Malignant ureteric obstruction represents a distinct hydronephrosis–AKI phenotype in which obstruction often signals advanced systemic disease, and decompression decisions must balance renal recoverability against procedural burden, complication risk, and overall prognosis. Large contemporary datasets and systematic reviews in malignant upper urinary tract obstruction consistently show poor survival for many patients despite intervention, but also document that decompression can restore renal function sufficiently to enable systemic therapy in selected individuals. A large Scottish cohort study highlighted malignant ureteric obstruction as a marker of advanced malignancy with poor survival across cancer types, underscoring the need for individualized decision-making and consistent care pathways.[33, 34]

Within oncology populations, AKI is common, multifactorial, and prognostically significant; obstructive mechanisms particularly pelvic malignancies causing bilateral ureteric obstruction are among the reversible contributors that clinicians must actively identify. Contemporary cohort work in hospitalized cancer patients with severe AKI has emphasized cause attribution and prognostic determinants of mortality and renal replacement therapy, supporting structured evaluation that does not overlook post-renal drivers when hydronephrosis is present.[35]

Predicting renal recovery after hydronephrosis-associated AKI remains an active area of study, with recent clinical investigations focusing on baseline kidney function, AKI stage, duration of obstruction/anuria, infection, and post-decompression urine output patterns as pragmatic predictors. Recent BMC Nephrology work evaluating predictors of renal function recovery after treatment for severe unilateral upper urinary tract obstruction provides contemporary evidence that recovery is heterogeneous and measurable, supporting the clinical approach of early decompression plus close longitudinal reassessment rather than assuming uniform reversibility.[36]

The hydronephrosis–AKI relationship should be situated within the broader continuum from AKI to acute kidney disease (AKD) and chronic kidney disease (CKD).[37] Recent study emphasizes that a substantial subset of patients transition from AKI to AKD with persistent dysfunction beyond the acute window, which is clinically consistent with obstructive AKI cases where decompression is delayed, infection is superimposed, or baseline CKD is present.[38] This broader framing helps justify why hydronephrosis-associated AKI should trigger not only urgent decompression when indicated, but also deliberate follow-up for residual dysfunction and risk mitigation.[39]

While narrative synthesis highlights pathophysiological plausibility, quantitative cohort data provide essential perspective regarding the magnitude of AKI risk and downstream renal outcomes. These findings are summarized in Table 1.

Table 1. Key cohort findings linking hydronephrosis/obstructive uropathy with AKI and downstream outcomes

Study (year)	Population / setting	Sample size (N)	AKI incidence / definition	Effect size (AKI risk)	Long-term CKD progression	Mortality risk
Hsiao et al. (2021)[21]	Hospitalized UTI patients undergoing imaging (2006–2019)	1,113	14.8% AKI during admission	Among UTI patients with urolithiasis, ureteral stone + hydronephrosis independently predicted AKI: OR 2.299 (95% CI 1.112–4.755); additionally, in UTI patients without hydronephrosis, urolithiasis still predicted AKI: OR 2.451 (95% CI 1.369–4.389)	Not reported	In-hospital mortality 0.6% (7/1113)
Yang et al. (2021)[6]	Adults with obstructive uropathy,	1,683	AKI prevalence	AKI group had worse kidney outcomes vs non-AKI group	Progression to ESKD reported	Overall mortality 34%;

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

Study (year)	Population / setting	Sample size (N)	AKI incidence / definition	Effect size (AKI risk)	Long-term CKD progression	Mortality risk
	stratified by AKI vs non-AKI		78% (study definition)	(group-comparison design; effect sizes reported in paper)	(AKI vs non-AKI groups)	highest in malignancy-related obstruction (reported in paper)
Blackmur et al. (2024)[34]	Malignant ureteric obstruction managed with stent or nephrostomy (national cohort; Scotland)	~852 (derived from reported deaths 758 = 89%)	AKI incidence not the primary endpoint	Not primarily an AKI-risk study; evaluates outcomes after diversion	Not reported	High mortality: 758 deaths (89%), median survival ~141 days (reported in paper)

Long-Term Renal Consequences and Chronic Kidney Disease

Hydronephrosis secondary to urinary tract obstruction has traditionally been regarded as a condition with favorable renal prognosis, provided that timely decompression is achieved. However, evidence accumulated over the past several years has substantially modified this view, indicating that obstructive uropathy can initiate renal injury trajectories that extend well beyond the acute phase. Clinical analyses suggest that renal dysfunction following obstruction frequently persists despite apparent anatomical resolution, with a notable proportion of patients progressing toward chronic kidney disease (CKD). These findings highlight that hydronephrosis should not be conceptualized solely as a reversible insult, but rather as a potential trigger of sustained renal vulnerability.

The persistence of renal impairment after relief of obstruction reflects a fundamental dissociation between mechanical decompression and biological recovery. While urinary drainage may normalize intrapelvic pressure and resolve hydronephrosis on imaging, cellular and molecular injury within the renal parenchyma may continue to evolve. Recent mechanistic reviews emphasize that obstruction initiates a cascade of maladaptive repair processes, including prolonged epithelial stress responses, incomplete redifferentiation of tubular cells, and sustained activation of fibrogenic pathways. Once established, these processes may progress independently of ongoing obstruction, thereby explaining why renal function often stabilizes at a lower level than pre-obstruction baseline.[40]

Long-term observational studies provide clinical corroboration for these mechanistic insights. Cohorts of patients hospitalized with obstructive uropathy demonstrate heterogeneous recovery patterns, ranging from complete normalization of renal function to persistent impairment and accelerated decline over subsequent months or years. Importantly, incomplete recovery is not limited to patients with severe obstruction or overt anuria; delayed diagnosis, recurrent episodes of hydronephrosis, and subtle or intermittent obstruction have also been associated with adverse renal trajectories. These findings mirror broader epidemiological evidence from post-AKI populations, in which even partial or transient renal insults confer long-term risk for CKD development.[41, 42]

The introduction of the acute kidney disease (AKD) construct has further refined understanding of the temporal evolution of renal injury after obstruction. AKD captures the intermediate phase during which renal dysfunction persists beyond the acute insult but has not yet met criteria for chronicity. International consensus documents emphasize that this phase is prognostically meaningful, as persistent renal impairment during AKD strongly predicts subsequent CKD progression and mortality. In patients recovering from hydronephrosis-associated

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

AKI, AKD is common and often under-recognized, particularly when creatinine levels partially improve yet fail to return to baseline. This transitional period represents a critical window for surveillance and intervention that is frequently overlooked in routine clinical practice.[43, 44]

Etiology-specific factors further modulate long-term renal outcomes after hydronephrosis. Malignant ureteric obstruction exemplifies a high-risk scenario in which persistent or recurrent obstruction, systemic inflammation, and exposure to nephrotoxic oncologic therapies converge to limit renal recovery. Outcome studies indicate that, although decompression may transiently improve renal parameters and permit continuation of anticancer treatment, sustained improvement in renal function is uncommon. Instead, many patients experience progressive renal decline or remain dependent on reduced renal reserve, underscoring that decompression in this context often serves a palliative or enabling role rather than reversing chronic injury.[33, 45]

Beyond etiology, host factors critically influence susceptibility to long-term renal damage.[17] Advanced age, pre-existing CKD, diabetes mellitus, and hypertension consistently emerge as modifiers that reduce renal resilience after obstruction.[15, 46, 47] In these populations, baseline nephron loss and microvascular dysfunction limit adaptive capacity, rendering the kidney less capable of compensating for obstruction-induced injury.[47-49] As a result, even short-lived hydronephrosis may precipitate disproportionate functional decline, reinforcing the need for individualized risk stratification rather than uniform assumptions of reversibility.[50, 51]

At the tissue level, increasing attention has been directed toward the role of microvascular injury and chronic hypoxia in sustaining renal dysfunction after obstruction. Recent translational studies indicate that obstruction-induced peritubular capillary rarefaction and hypoxia-responsive signaling may persist after decompression, perpetuating fibrotic remodeling and limiting regenerative responses. This vascular–interstitial axis provides a biologically plausible link between an acute obstructive event and later CKD progression, suggesting that long-term renal outcomes are shaped not only by obstruction severity but also by the kidney's capacity to restore microvascular integrity.[43, 45]

Diagnostic Challenges: Discordance Between Hydronephrosis Severity and Renal Dysfunction

Accurate diagnosis of obstructive renal injury requires recognition that urinary tract obstruction is fundamentally a functional disorder whose structural manifestations may lag behind, fluctuate, or remain subtle despite substantial impairment of renal physiology.[16] Contemporary nephrology literature increasingly emphasizes that hydronephrosis, while historically treated as a defining diagnostic marker, represents only one dimension of a multifactorial disease process and cannot be relied upon in isolation to infer either obstruction severity or renal prognosis. This evolving understanding has prompted a shift away from purely morphology-based diagnostic reasoning toward integrated functional assessment.[16,52]

Hydronephrosis reflects dilation of the renal pelvis and calyces secondary to impaired urinary drainage, yet its presence and degree are modulated by multiple variables including obstruction completeness, duration, collecting system compliance, and baseline renal architecture.[4] Several recent analyses demonstrate that severe renal dysfunction may develop in the absence of marked dilation, particularly in cases of acute complete obstruction or extrinsic compression, where rapid intrarenal pressure transmission precedes anatomic remodeling.[53, 54] Conversely, longstanding partial obstruction may produce impressive dilation with relatively preserved renal function, further complicating interpretation and underscoring the non-linear relationship between structure and function.[4]

The recognition of non-dilated obstructive nephropathy has formalized this diagnostic discordance. This entity describes post-renal acute kidney injury occurring with minimal or absent hydronephrosis on imaging and is now acknowledged as a clinically significant, though underdiagnosed, phenotype of obstructive uropathy. Contemporary reviews highlight that failure to consider this diagnosis often leads to delayed decompression, prolonged exposure to hypoxia and intratubular pressure, and avoidable nephron loss. The existence of this entity challenges entrenched diagnostic heuristics that equate obstruction with dilation and reinforces the need for heightened clinical vigilance.[55]

Ultrasonography remains the cornerstone of initial diagnostic evaluation due to its accessibility,

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

safety profile, and utility in identifying overt obstruction. However, its limitations are increasingly well characterized in recent literature.[56] Ultrasound sensitivity declines in early obstruction, in patients with low urine output, obesity, or retroperitoneal pathology, and in conditions where obstruction is complete but short-lived.[57, 58] Importantly, false reassurance from a normal ultrasound has been identified as a recurrent contributor to diagnostic delay in obstructive AKI, particularly in emergency and inpatient settings.[58]

Computed tomography provides superior spatial resolution and facilitates detection of ureteral calculi, strictures, malignancy-related compression, and retroperitoneal fibrosis, often clarifying equivocal ultrasound findings.[59, 60] Despite these advantages, CT imaging does not resolve the core diagnostic dilemma, as the extent of collecting system dilation visualized on CT correlates poorly with renal functional impairment and does not reliably predict reversibility after intervention.[59, 61] Recent outcome studies caution against using hydronephrosis grade on CT as a proxy for injury severity, emphasizing instead the integration of imaging with biochemical trends and clinical trajectory.[17, 61]

Functional assessment therefore occupies a central role in contemporary diagnostic algorithms.[52] Nuclear medicine techniques such as MAG3 or DTPA renography allow quantification of differential renal function and drainage kinetics, providing organ-specific information that cannot be inferred from serum creatinine alone.[62, 63] This distinction is particularly critical in unilateral obstruction, where contralateral compensation may normalize systemic renal indices while irreversible injury progresses silently in the obstructed kidney.[63] Multiple recent studies support the prognostic value of split renal function in guiding both urgency of decompression and expectations of recovery.[62, 64]

Adjunctive imaging modalities have been explored to refine diagnostic precision further. Doppler ultrasonography, through assessment of renal resistive indices, offers indirect insight into intrarenal vascular resistance and microvascular compromise, phenomena that often accompany advanced obstruction.[65] Elevated resistive indices have been associated with worse renal outcomes in obstructive uropathy, although variability and operator dependence limit their standalone diagnostic utility.[66] Nevertheless, when interpreted alongside structural and functional data, Doppler findings may contribute to a more nuanced assessment of injury severity.[17]

Laboratory evaluation remains indispensable in detecting and contextualizing obstruction-related renal injury.[17] Disproportionate elevations in serum creatinine, urea, or potassium relative to imaging findings should prompt reconsideration of obstruction severity and raise suspicion for functionally significant disease despite minimal dilation.[17] Recent nephrology literature emphasizes that trends in renal biomarkers, rather than absolute values alone, often provide the earliest indication of evolving obstruction and should trigger expedited diagnostic escalation when discordant with imaging.[67-69]

The diagnostic complexity of obstructive renal injury is further amplified in the presence of infection. Obstruction combined with urinary tract infection or sepsis represents a synergistic insult in which inflammatory injury compounds pressure-mediated dysfunction, often resulting in rapid deterioration of renal function with limited or delayed radiographic change. Contemporary reviews stress that in this context, diagnostic thresholds must be lowered, and equivocal imaging should not delay decompression when clinical suspicion is high, given the substantial morbidity associated with infected obstruction.[17]

Malignancy-associated obstruction constitutes a distinct diagnostic category characterized by extrinsic compression, intermittent obstruction, and asymmetric or evolving hydronephrosis.[34] In oncology populations, renal dysfunction is frequently multifactorial, and obstruction may be overlooked or underestimated when imaging findings appear modest.[70] A cohort studie has demonstrate that functional impairment in malignant ureteric obstruction often exceeds what would be predicted by hydronephrosis severity alone, reinforcing the need for systematic functional assessment in this population.[33]

Temporal factors further complicate diagnosis. Hydronephrosis may persist after relief of obstruction, reflecting residual dilation rather than ongoing functional impairment, while early obstruction may produce profound functional decline before dilation develops. This temporal mismatch highlights the limitations of static imaging and supports serial assessment incorporating urine output, biochemical

JURNAL KEDOKTERAN DIPONEGORO
(DIPONEGORO MEDICAL JOURNAL)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

recovery, and functional imaging where appropriate.[54,71]

To enhance translational applicability and reduce diagnostic ambiguity, a structured stepwise assessment model integrating clinical suspicion, imaging, functional testing, biomarker evaluation, and longitudinal monitoring is proposed (Figure 2).

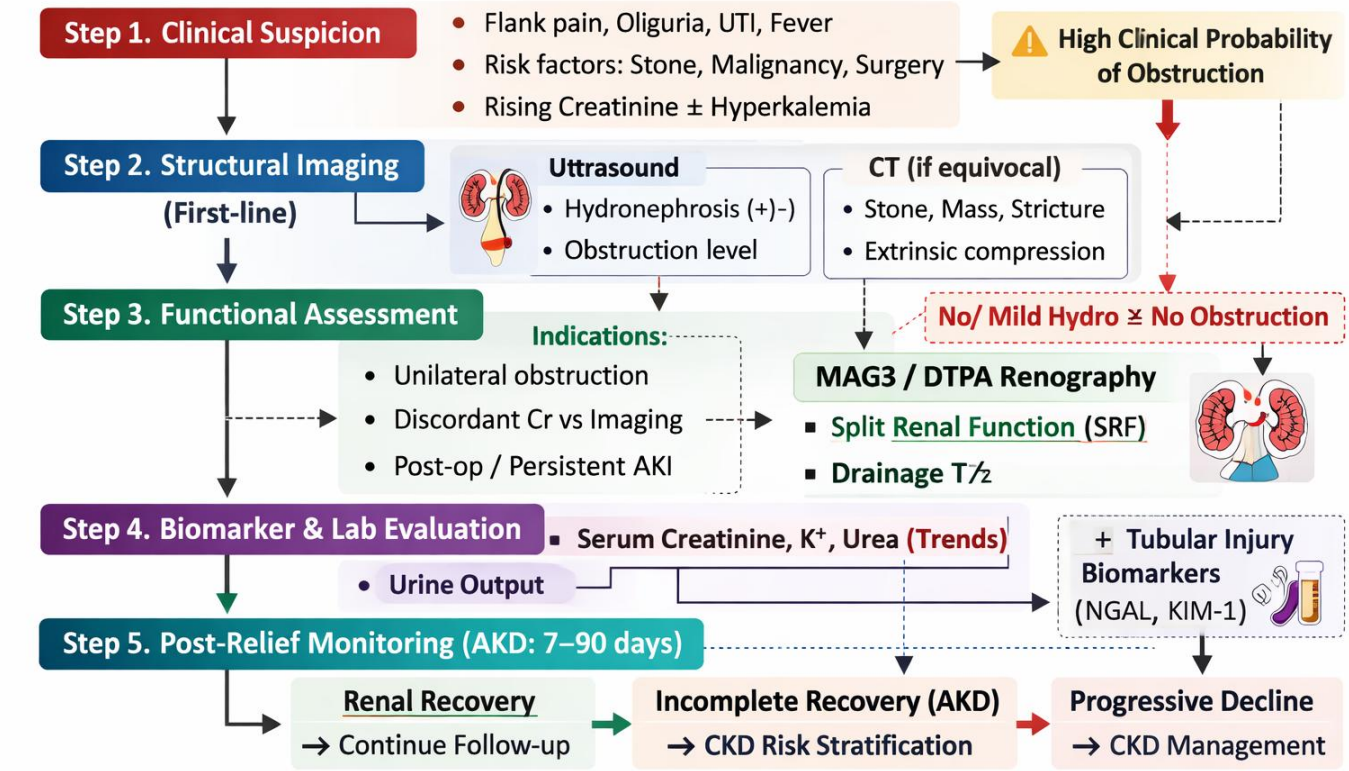


Figure 2. Proposed stepwise clinical framework for evaluation of hydronephrosis-associated renal dysfunction.

Determinants of Renal Function Recovery After Obstruction Relief

Renal recovery following relief of urinary tract obstruction is best understood as a probabilistic rather than deterministic outcome, shaped by the biological depth of injury at the time of intervention rather than by decompression alone. While restoration of urinary flow remains the sine qua non of management, contemporary evidence increasingly challenges the assumption that decompression equates to recovery. Clinical trajectories after obstruction reveal marked heterogeneity, reflecting the extent to which injury has already transitioned from functional derangement to structural remodeling. This reframing aligns obstructive uropathy with broader paradigms of kidney injury in which reversibility is constrained by maladaptive repair rather than by persistence of the inciting insult.[3, 72, 73]

Among obstruction-related variables, duration of obstruction exerts the most consistent and biologically plausible influence on renal recovery. Experimental models and clinical cohorts converge on the observation that short-lived obstruction predominantly induces hemodynamic and tubular functional changes, whereas prolonged obstruction facilitates interstitial inflammation, fibroblast activation, and irreversible nephron loss.[71] Importantly, duration is rarely measured directly in clinical practice; instead, it is inferred retrospectively from symptom burden or biochemical severity, a limitation that likely contributes to delayed intervention.[3]

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

The completeness and laterality of obstruction further modulate recovery in ways that complicate reliance on serum creatinine as a global marker of renal outcome.[74] Bilateral obstruction or obstruction in a solitary kidney predictably produces more severe acute kidney injury and portends a lower probability of complete recovery.[75] In unilateral obstruction, however, compensatory hyperfiltration by the contralateral kidney may normalize systemic creatinine despite permanent functional loss in the affected kidney.[76] Several observational studies underscore this dissociation, cautioning against equating biochemical recovery with true nephron preservation and emphasizing the importance of kidney-specific functional assessment in prognostication.[77]

Severity of acute kidney injury at presentation represents another determinant that integrates both temporal and biological dimensions of injury. Higher AKI stage, prolonged oliguria or anuria, and severe metabolic derangements are consistently associated with inferior recovery, reflecting cumulative tubular and microvascular damage. These findings resonate with the broader AKI literature, in which both magnitude and persistence of injury independently predict long-term renal outcomes. In obstructive uropathy, AKI severity therefore functions not merely as a marker of acute illness but as a surrogate for the extent of irreversible parenchymal injury already incurred.[78]

Patient-specific factors exert a parallel influence by defining the kidney's reparative capacity. Baseline renal function emerges as one of the strongest predictors of recovery, with pre-existing chronic kidney disease markedly constraining post-obstructive improvement. Advanced age, diabetes mellitus, hypertension, and systemic vascular disease further diminish renal resilience by limiting nephron reserve and impairing microvascular adaptability. In such contexts, even transient obstruction may precipitate a disproportionate and sustained decline in function, reinforcing the inadequacy of uniform prognostic assumptions across heterogeneous patient populations.[79]

Superimposed infection represents a critical modifier that accelerates injury and compromises recovery. Obstructive uropathy complicated by urinary tract infection or sepsis exposes the kidney to synergistic insults such as pressure-mediated tubular injury coupled with inflammatory and ischemic damage. Contemporary reviews emphasize that infection lowers the threshold for irreversible injury and amplifies fibrotic signaling, thereby diminishing the restorative potential of decompression. These data provide a mechanistic rationale for aggressive early drainage in infected obstruction and caution against delays attributable to diagnostic uncertainty.[80]

Management-related factors, particularly the timing and durability of decompression, further shape recovery trajectories.[34] Multiple recent studies demonstrate that earlier relief of obstruction is associated with greater functional improvement, whereas delayed intervention permits consolidation of maladaptive repair pathways.[31, 81] The specific method of decompression ureteral stenting versus percutaneous nephrostomy appears less consequential than the adequacy and persistence of drainage, provided obstruction is effectively relieved.[82] Conversely, recurrent obstruction due to stent failure or catheter dysfunction has been linked to poorer outcomes, underscoring the need for vigilant post-procedural surveillance.[83]

The post-decompression phase itself offers prognostic insight. Post-obstructive diuresis, while often interpreted as evidence of tubular recovery, may also unmask impaired concentrating ability and residual tubular dysfunction.[84] Excessive or prolonged diuresis has been associated with delayed stabilization of renal function and increased risk of electrolyte disturbances, particularly in older or comorbid patients.[23] However, absence of diuresis in the setting of severe pre-decompression AKI may signal advanced tubular injury and limited regenerative capacity, highlighting the value of early post-relief physiology in outcome prediction.[85]

At a mechanistic level, growing translational evidence implicates maladaptive repair as the principal barrier to full recovery after obstruction.[86] Persistent epithelial cell cycle arrest, unresolved

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

inflammation, microvascular rarefaction, and sustained fibrotic signaling constrain regenerative responses even after the inciting obstruction is removed.[87] These processes mirror those described in other forms of progressive kidney disease and provide a unifying biological explanation for why recovery plateaus despite timely decompression.[88] Obstructive nephropathy thus emerges not as an isolated entity but as a model of injury-induced chronic kidney disease.[86]

The concept of acute kidney disease (AKD) has sharpened clinical understanding of recovery after obstruction relief.[86] Persistent renal dysfunction extending weeks beyond decompression identifies a high-risk state for chronic kidney disease progression and mortality.[78, 89] Recent consensus statements emphasize that failure to recover baseline renal function within this window warrants intensified follow-up and secondary prevention strategies.[38] In hydronephrosis-associated obstruction, systematic recognition of AKD offers a practical framework for transitioning care from acute management to long-term renal preservation.[78]

Table 2 Clinical Determinants of Renal Function Recovery After Relief of Urinary Tract Obstruction.

Study (Author, Year)	Design / Population	Definition of Recovery	Determinants Evaluated	Key Findings	Impact on Recovery
Low et al., 2021[63]	Prospective cohort; chronic unilateral ureteric obstruction	Improvement in split renal function after intervention	Baseline differential renal function, obstruction duration	Baseline renal function independently predicted post-intervention improvement	Lower baseline SRF → reduced likelihood of recovery
Wijaya et al., 2022[90]	Observational cohort; obstructive uropathy	Serum creatinine normalization post-treatment	AKI severity, infection, obstruction duration	Higher AKI stage and infection associated with incomplete recovery	Severe AKI and infection ↓ recovery probability
Medina et al., 2024[91]	Retrospective cohort; malignant ureteral obstruction	Renal function stabilization after stenting/PCN	Malignancy type, bilateral obstruction, baseline CKD	Recovery limited in advanced malignancy despite decompression	Malignancy-related obstruction ↓ sustained recovery
Rigonalli et al., 2025[24]	Observational clinical study	Post-obstructive diuresis and renal stabilization	Post-decompression urine output pattern	Absence or abnormal POD associated with delayed stabilization	POD pattern reflects tubular injury severity

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

CONCLUSION

Hydronephrosis should not be viewed as a purely anatomical finding or assumed to resolve without consequence after decompression. Current evidence shows that urinary tract obstruction may initiate structural and microvascular injury that persists beyond mechanical relief, leading to incomplete renal recovery in a substantial proportion of patients.

In clinical practice, the severity of pelvicalyceal dilation alone should not determine management decisions. Evaluation requires integration of clinical presentation, urine output, biochemical trends, and, when appropriate, functional assessment of individual kidney performance. Early decompression remains essential, particularly in patients with infection, bilateral obstruction, or limited baseline renal reserve.

Relief of obstruction should also be followed by structured reassessment of renal function within the weeks after intervention. Persistent dysfunction during this period identifies patients at risk for chronic kidney disease and warrants ongoing monitoring and kidney-protective management.

CONFLICTS OF INTEREST

The author declares no conflict of interest.

FUNDING

No specific funding was provided for this article

AUTHOR CONTRIBUTIONS

Conceptualization, MAS.; methodology, MAS.; investigation, MAS.; data curation, MAS.; writing—original draft preparation, MAS.; writing—review and editing, MAS.; visualization, MAS.; supervision, MAS.; project administration, MAS.

ACKNOWLEDGMENTS

The author would like to acknowledge Zainoel Abidin General Hospital, Banda Aceh, Aceh, Indonesia, for providing an academic environment that supported the preparation of this manuscript.

REFERENCES

1. Thotakura R, Anjum F. Hydronephrosis and Hydroureter. StatPearls. Treasure Island (FL): StatPearls Publishing
Copyright © 2025, StatPearls Publishing LLC.; 2025.
2. Bingaman S, Leslie SW, Hinson MR. Obstructive Uropathy. StatPearls. Treasure Island (FL): StatPearls Publishing
Copyright © 2025, StatPearls Publishing LLC.; 2025.
3. Gembillo G, Spadaro G, Santoro D. Link between obstructive uropathy and acute kidney injury. World J Nephrol. 2025; 14:99120.
4. Nørregaard R, Mutsaers HAM, Frøkiær J, Kwon TH. Obstructive nephropathy and molecular pathophysiology of renal interstitial fibrosis. Physiol Rev. 2023; 103:2827-72.
5. Nan QY, Piao SG, Jin JZ, Chung BH, Yang CW, Li C. Pathogenesis and management of renal fibrosis induced by unilateral ureteral obstruction. Kidney Res Clin Pract. 2024; 43:586-99.
6. Yang J, Sun BG, Min HJ, Son YB, Kim TB, Lee J, et al. Impact of acute kidney injury on long-term adverse outcomes in obstructive uropathy. Sci Rep. 2021; 11:23639.
7. Yamashita N, Kramann R. Mechanisms of kidney fibrosis and routes towards therapy. Trends in Endocrinology & Metabolism. 2024; 35:31-48.
8. Lyu G, Liao H, Li R. Ferroptosis and renal fibrosis: mechanistic insights and emerging therapeutic targets. Ren Fail. 2025; 47:2498629.
9. Long Z, Luo Y, Yu M, Wang X, Zeng L, Yang K. Targeting ferroptosis: a new therapeutic opportunity for kidney diseases. Frontiers in Immunology. 2024; Volume 15 - 2024.
10. You D, Weng M, Wu X, Nie K, Cui J, Chen Y, et al. C3aR contributes to unilateral ureteral obstruction-induced renal interstitial fibrosis via the activation of the NLRP3 inflammasome. Life Sciences. 2022; 308:120905.
11. Rudman-Melnick V, Adam M, Stowers K, Potter A, Ma Q, Chokshi SM, et al. Single-cell sequencing dissects the transcriptional identity of activated fibroblasts and identifies novel persistent distal tubular injury patterns in kidney fibrosis. Res Sq. 2023.
12. He S, Xia T, Guo Z, Song X, Wang H, He H, et al. Tracing the origin of myofibroblasts in kidney fibrosis. Nature Communications. 2025; 17:653.
13. Zhang Y, Yang Y, Yang F, Liu X, Zhan P, Wu J, et al. HDAC9-mediated epithelial cell cycle

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

arrest in G2/M contributes to kidney fibrosis in male mice. Nat Commun. 2023; 14:3007.
14. Wang P, Huang Z, Peng Y, Li H, Lin T, Zhao Y, et al. Circular RNA circBNC2 inhibits epithelial cell G2-M arrest to prevent fibrotic maladaptive repair. Nature Communications. 2022; 13:6502.
15. Yang J, Sun BG, Min H-J, Son Y-B, Kim TB, Lee J, et al. Impact of acute kidney injury on long-term adverse outcomes in obstructive uropathy. Scientific Reports. 2021; 11:23639.
16. Feliciangeli V, Noce A, Montalto G, Germani S, Miano R, Asimakopoulos AD. Non-dilated obstructive nephropathy. Clinical Kidney Journal. 2024; 17.
17. Pérez-Aizpurua X, Cabello Benavente R, Bueno Serrano G, Alcázar Peral JM, Gómez-Jordana Mañas B, Tufet IJJ, et al. Obstructive uropathy: Overview of the pathogenesis, etiology and management of a prevalent cause of acute kidney injury. World J Nephrol. 2024; 13:93322.
18. Yaxley J, Yaxley W. Obstructive uropathy – acute and chronic medical management. World Journal of Nephrology. 2023; 12:1-9.
19. Molitoris BA. Low-Flow Acute Kidney Injury: The Pathophysiology of Prerenal Azotemia, Abdominal Compartment Syndrome, and Obstructive Uropathy. Clinical Journal of the American Society of Nephrology. 2022; 17:1039-49.
20. Xu X, Zeng T, Chen S, Tian N, Zhang C, Chen Y, et al. Acute kidney injury: pathogenesis and therapeutic interventions. Molecular Biomedicine. 2025; 6:61.
21. Hsiao C-Y, Chen T-H, Lee Y-C, Wang M-C. Ureteral stone with hydronephrosis and urolithiasis alone are risk factors for acute kidney injury in patients with urinary tract infection. Scientific Reports. 2021; 11:23333.
22. Whitworth P, Courtney KG, Oto A, Allen BC, Akin O, Barker SJ, et al. ACR Appropriateness Criteria® Hydronephrosis on Prior Imaging-Unknown Cause. Journal of the American College of Radiology. 2024; 21:S144-S67.
23. Finger E, Moses A. As The River Flows: A Case Report of Post Obstructive Diuresis. BMC Nephrology. 2025; 26:341.
24. Rigonalli U, Sigg S, Moos SV, Baumeister P, Mattei A, Fankhauser CD, et al. Prevalence and Clinical Implications of Post-obstruction Hyperdiuresis Among Patients with Urinary Retention: A Mini Review. European Urology Open Science. 2025; 73:68-70.
25. Tummalapalli SL, Zech JR, Cho HJ, Goetz C. Risk stratification for hydronephrosis in the evaluation of acute kidney injury: a cross-sectional analysis. BMJ Open. 2021; 11:e046761.
26. Tummalapalli SL, Zech J, Cho H, Goetz C. Risk stratification for hydronephrosis in the evaluation of acute kidney injury: a cross-sectional analysis. BMJ Open. 2021; 11:e046761.
27. Weissinger M, Seyfried KC, Ursprung S, Castaneda-Vega S, Seith F, von Beschwitz S, et al. Non-invasive estimation of split renal function from routine (68)Ga-SSR-PET/CT scans. Front Med (Lausanne). 2023; 10:1169451.
28. Elzaki A. The Utility Of Radioisotope Renography In Assessment Of Renal Function: A Single-Center Experience. International Journal of Pure Medical Research. 2022; 7.
29. Noel S, Parikh CR. Kidney functional reserve helps early detection of subclinical chronic kidney disease. American Journal of Physiology-Renal Physiology. 2023; 325:F885-F7.
30. Lu Z, Ni W, Wu Y, Zhai B, Zhao Q, Zheng T, et al. Application of biomarkers in the diagnosis of kidney disease. Frontiers in Medicine. 2025; Volume 12 - 2025.
31. Benea A, Porav-Hodade D, Turaiche M, Rosca O, Lighezan D-F, Rachieru C, et al. Time to Decompression in Obstructive Urosepsis from Ureteral Calculi: Thresholds, Initial Diversion, and Early Biomarkers: A Systematic Review. Journal of Clinical Medicine. 2025; 14:8546.
32. Pérez-Aizpurua X, Benavente RC, Serrano GB, Peral JMA, Mañas BG-J, i Jaumot JT, et al. Obstructive uropathy: Overview of the pathogenesis, etiology and management of a prevalent cause of acute kidney injury. World journal of nephrology. 2024; 13:93322.
33. Buckby A, David R, Kahokehr A. Renal Decompression for Malignant Ureteric Obstruction: A Tertiary Hospital Cohort Analysis. Société Internationale d'Urologie Journal. 2025; 6:62.

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

34. Blackmur J, Group SMUOS. Management of malignant ureteric obstruction with ureteric stenting or percutaneous nephrostomy. BJS. 2024; 111.
35. Calças Marques R, Reis M, Pimenta G, Sala I, Chuva T, Coelho I, et al. Severe Acute Kidney Injury in Hospitalized Cancer Patients: Epidemiology and Predictive Model of Renal Replacement Therapy and In-Hospital Mortality. Cancers (Basel). 2024; 16.
36. Li J, Pang H, Li W. Analysis of efficacy factors in unilateral obstructive severe renal insufficiency: a single-centre retrospective study. BMC Nephrology. 2025; 26:69.
37. Ostermann M, Lumlertgul N, Jeong R, See E, Joannidis M, James M. Acute kidney injury. The Lancet. 2025; 405:241-56.
38. Gameiro J, Gouveia B, Fonseca JA, Lopes JA. The burden of acute kidney disease: an epidemiological review and importance of follow-up care. Clin Kidney J. 2025; 18:sfaf169.
39. Gameiro J, Marques F, Lopes JA. Long-term consequences of acute kidney injury: a narrative review. Clinical Kidney Journal. 2020; 14:789-804.
40. Ding Y, Gao L, Chen Y, Qiao Y, Yang B. Molecular mechanisms and therapeutic advances of peritubular capillary neogenesis in acute kidney injury. Front Mol Biosci. 2025; 12:1643838.
41. Profile of End Stage Chronic Kidney Disease due to Obstructive Uropathy Patients Undergoing Hemodialysis. Brawijaya Journal of Urology. 2024; 5:8-12.
42. Veltkamp DMJ, Porras CP, Gant CM, Groenestege WMT, Kok MB, Verhaar MC, et al. Long-term risks of adverse kidney outcomes after acute kidney injury: a systematic review and meta-analysis. Nephrol Dial Transplant. 2025; 40:2143-58.
43. Lameire NH, Levin A, Kellum JA, Cheung M, Jadoul M, Winkelmayer WC, et al. Harmonizing acute and chronic kidney disease definition and classification: report of a Kidney Disease: Improving Global Outcomes (KDIGO) Consensus Conference. Kidney International. 2021; 100:516-26.
44. Koh ES, Chung S. Recent update on acute kidney injury-to-chronic kidney disease transition. Yonsei Medical Journal. 2024; 65:247.
45. Lin H, Pan J, Zhang J, He Y, Ge Y, Niu D, et al. Intermedin protects peritubular capillaries by inhibiting eNOS uncoupling through AMPK/GTPCH-I/BH4 pathway and alleviate CKD following AKI. Free Radical Biology and Medicine. 2025; 234:72-85.
46. Javier RM, Salim J, Aji BL, Pradipta BPA, Nur C, Muhammad I, et al. Risk factors for chronic kidney disease and septic shock with hypertension in adults and children. Frontiers in Nephrology. 2025; Volume 5 - 2025.
47. Ameer OZ. Hypertension in chronic kidney disease: What lies behind the scene. Front Pharmacol. 2022; 13:949260.
48. Meariman JK, Zulli H, Perez A, Bajracharya SD, Mohandas R. Small vessel disease: Connections between the kidney and the heart. American Heart Journal Plus: Cardiology Research and Practice. 2023; 26:100257.
49. Durante A, Mazzapicchi A, Baiardo Redaelli M. Systemic and Cardiac Microvascular Dysfunction in Hypertension. International Journal of Molecular Sciences. 2024; 25:13294.
50. Buckenmayer A, Siebler N, Haas CS. Pre-existing chronic kidney disease, aetiology of acute kidney injury and infection do not affect renal outcome and mortality. J Nephrol. 2024; 37:391-400.
51. Veltkamp DMJ, Porras CP, Gant CM, Groenestege WMT, Kok MB, Verhaar MC, et al. Long-term risks of adverse kidney outcomes after acute kidney injury: a systematic review and meta-analysis. Nephrology Dialysis Transplantation. 2025; 40:2143-58.
52. Volkan-Salanci B, Erbaş B. Diuretic Renal Scintigraphy in Adults: Practical Aspects and Reporting. Seminars in Nuclear Medicine. 2022; 52:445-52.
53. Ulusoy O, Aydın E, Ateş O, Hakgüder G, Özer E, Olguner M, et al. Clues for the early loss of renal function in congenital hydronephrosis: Analysis of renal pelvis collagen ratio, diuresis renography and upper urinary tract morphology. Journal of Pediatric Urology. 2023; 19:197.e1-.e7.

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

54. Xiang X, Wei X, Miao J, Sun M, Cao W, Zhao B, et al. Case Report: Acute kidney injury due to minimal dilated obstructive nephropathy in the context of gastric cancer. Frontiers in Oncology. 2025; Volume 15 - 2025.
55. Shahzad MA, Baxi PV, Rodby RA. The challenges of diagnosing nondilated obstructive uropathy: a case report. Canadian Journal of Kidney Health and Disease. 2022; 9:20543581221086683.
56. Ata F, Sharma R, Akram J, Tanous B, Arshad A, Alamin M, et al. Back to bedside: Renal point-of-care ultrasonography (POCUS) by internal medicine resident physicians for identification of hydronephrosis in patients with acute kidney injury. Qatar Med J. 2025; 2025:36.
57. Gaudreau-Simard M, Ruller S, Dann M, Woo MY, Mallick R, McInnes MDF, et al. Imaging of Medical Patients with Acute Kidney Injury: Patterns of Ultrasound Use and the Role of Point-of-care Ultrasound at a Tertiary Care Center. Journal of General Internal Medicine. 2026; 41:119-25.
58. Taus PJ, Manivannan S, Dancel R. Bedside assessment of the kidneys and bladder using point of care ultrasound. POCUS journal. 2022; 7:94.
59. Napitupulu T, Hardja Y, Susanto M, Hutahaean GD, Riantri I, Sutapa H, et al. Correlation Between the Grade of Hydronephrosis with Surgical Outcomes After Ultrasound-guided Supine Percutaneous Nephrolithotomy: A Retrospective Observational Study. Medeni Med J. 2023; 38:120-7.
60. Türk C, Petřík A, Sarica K, Seitz C, Skolarikos A, Straub M, et al. EAU guidelines on diagnosis and conservative management of urolithiasis. European urology. 2016; 69:468-74.
61. Xia J, Deng S, Hu Y, Huang Z, Chen S, Zhao C, et al. A novel diagnostic model to grade the impairment of split renal function for patients with obstructive hydronephrosis based on enhanced CT imaging. Chinese Journal of Academic Radiology. 2026.
62. Lim HJ, Choi SH. Assessment of Individual Renal Function Using (99m)Tc-MAG(3) Renography. In Vivo. 2022; 36:206-11.
63. Low ZY, Allen SE, Arumuham V, Davis LM, Allen C, Bomanji J, et al. Does relative renal function improve after intervention for chronic ureteric obstruction? Cent European J Urol. 2021; 74:64-70.
64. Ochoa-Figueroa M, Borbely K, Hasselqvist D, Askling F, Lööw T, Aljabery F, et al. 3D dynamic diuretic renal scintigraphy using a hybrid whole body CZT SPECT/CT camera protocol in the evaluation of acute ureteric obstruction caused by ureteric stone. EJNMMI Rep. 2024; 8:27.
65. Shahzad G, Raj E, Kumar K, Shaikh AH, Abbasi P, Kumar M, et al. Diagnostic Accuracy of Renal Artery Resistive Index in Diagnosing Obstructive Uropathy. Journal of Health and Rehabilitation Research. 2024; 4:1557-63.
66. Ilyas S, Waseem F, Tariq AB, Shaukat S, Mehmood A. Comparison of doppler indices between obstructed and non-obstructed kidney. Journal of Ayub Medical College Abbottabad. 2024; 36:744–8-–8.
67. Yang H, Chen Y, He J, Li Y, Feng Y. Advances in the diagnosis of early biomarkers for acute kidney injury: a literature review. BMC Nephrol. 2025; 26:115.
68. Matarneh A, Akkari A, Sardar S, Salameh O, Dauleh M, Matarneh B, et al. Beyond creatinine: diagnostic accuracy of emerging biomarkers for AKI in the ICU - a systematic review. Ren Fail. 2025; 47:2556295.
69. Kai Z, Yun K, Su Y, Li J, Liu Y. Metabolomics biomarkers and related pathways in acute renal injury: a bibliometric investigation, meta-analysis, and systematic review. Frontiers in Medicine. 2025; Volume 12 - 2025.
70. Medina AA, García IL, Tello FG, Rodríguez SÁ, Palacios MH, Alcaraz MM, et al. The challenging management of malignant ureteral obstruction: Analysis of a series of 188 cases. Current Urology. 2024; 18:34-42.
71. Khater Y, Barakat N, Shokeir A, Samy A, Karrouf G. Renal fibrosis progression following partial unilateral ureteral obstruction: mechanisms and therapeutic insights. World Journal of Urology. 2025; 43:229.
72. Vanichapol T, Gonzalez A, Delgado R, Brewer M, Clouthier KA, Menshikh AA, et al. Permanent defects in renal medullary structure and function after reversal of urinary obstruction. JCI insight. 2025; 10:e187008.

JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i6.55518
JKD (DMJ), Volume 15, Number 6, November 2026 : 575-591

73. Tang W, Wei Q. The metabolic pathway regulation in kidney injury and repair. Frontiers in Physiology. 2024; Volume 14 - 2023.
74. Toumi HR, Sallabi SM, Lubbad L, Al-Salam S, Hammad FT. The Effect of Nerolidol on Renal Dysfunction following Bilateral Ureteral Obstruction. Biomedicines. 2024; 12:2285.
75. Nishimura K, Takenouchi A, Komatsu S, Kawaguchi Y, Kudo W, Takiguchi S, et al. Effect of emergent nephrostomy on long-term total and split renal function in patients with upper urinary tract obstruction due to pelvic malignant tumors. Pediatric Surgery International. 2024; 40:234.
76. Tamara L, Brodjonegoro S, Nahdi F. Renal Function Improvement Following Bilateral Percutaneous Nephrostomy. Contagion: Scientific Periodical Journal of Public Health and Coastal Health. 2025; 7:444.
77. He K, Wan D, Li S, Yuan G, Gao M, Han Y, et al. Non-contrast-enhanced magnetic resonance urography for measuring split kidney function in pediatric patients with hydronephrosis: comparison with renal scintigraphy. Pediatr Nephrol. 2024; 39:1447-57.
78. Jensen SK, Heide-Jørgensen U, Gammelager H, Birn H, Christiansen CF. Acute Kidney Injury Duration and 20-Year Risks of CKD and Cardiovascular Disease. Kidney International Reports®. 2024; 9:817-29.
79. Koh ES, Chung S. Recent Update on Acute Kidney Injury-to-Chronic Kidney Disease Transition. Yonsei Med J. 2024; 65:247-56.
80. Zhang Z, Ding P, Yang M, Zhou X, Long K. Case Report: Iatrogenic intraperitoneal hematoma compresses the ureters leading to urogenic sepsis. Frontiers in Medicine. 2025; Volume 12 - 2025.
81. Cozma C, Georgescu D, Popescu R, Geavlete B, Geavlete P. Double-J stent versus percutaneous nephrostomy for emergency upper urinary tract decompression. J Med Life. 2023; 16:663-7.
82. Moon YJ, Jun DY, Jeong JY, Cho S, Lee JY, Jung HD. Percutaneous Nephrostomy versus Ureteral Stent for Severe Urinary Tract Infection with Obstructive Urolithiasis: A Systematic Review and Meta-Analysis. Medicina. 2024; 60:861.
83. Wang X, Ji Z, Yang P, Li J, Tian Y. Forgotten ureteral stents: a systematic review of literature. BMC Urology. 2024; 24:52.
84. Leslie SW, Sajjad H, Sharma S. Postobstructive Diuresis. StatPearls. Treasure Island (FL): StatPearls Publishing
Copyright © 2025, StatPearls Publishing LLC.; 2025.
85. Wijaya WS, Irdam GA, Rahman F. Predicting Parameters of Renal Function Recoverability after Obstructive Uropathy Treatment in Adults. Acta Med Indones. 2022; 54:500-12.
86. Yeh T-H, Tu K-C, Wang H-Y, Chen J-Y. From Acute to Chronic: Unraveling the Pathophysiological Mechanisms of the Progression from Acute Kidney Injury to Acute Kidney Disease to Chronic Kidney Disease. International Journal of Molecular Sciences. 2024; 25:1755.
87. Xu D, Zhang X, Pang J, Li Y, Peng Z. Mechanisms of Acute Kidney Injury–Chronic Kidney Disease Transition: Unraveling Maladaptive Repair and Therapeutic Opportunities. Biomolecules. 2025; 15:794.
88. Zou Y, Dai J, Li J, Liu M, Li R, Li G, et al. Role of the TGF-β/Smad signaling pathway in the transition from acute kidney injury to chronic kidney disease (Review). Int J Mol Med. 2025; 56.
89. Frydman S, Freund O, Katash HA, Rimbrot D, Banai S, Shacham Y. Persistent Renal Dysfunction After Acute Kidney Injury Among STEMI Patients Undergoing Primary Coronary Intervention: Prevalence and Predictors. Clin Cardiol. 2024; 47:e70002.
90. Wijaya WS, Irdam GA, Rahman F. Predicting Parameters of Renal Function Recoverability after Obstructive Uropathy Treatment in Adults. Acta Medica Indonesiana. 2022; 54:500-12.
91. Artiles Medina A, Laso García I, González Tello F, Álvarez Rodríguez S, Hevia Palacios M, Mata Alcaraz M, et al. The challenging management of malignant ureteral obstruction: Analysis of a series of 188 cases. Current Urology. 2024; 18:34-42.