

Evaluation of Multimodal MRI Diffusion and Multiphase CT Parameters for Estimating Glomerular Filtration Rate in Patients with Acute Renal Obstruction

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Objective: This exploratory study aims to develop a multimodal model predicting estimated glomerular filtration rate (eGFR) by integrating functional Magnetic Resonance Imaging (MRI) diffusion metrics and multiphase Computed Tomography (CT) hemodynamic indices in acute urinary obstruction.

Methods: Thirty-five patients with acute urinary obstruction underwent multiphase CT and Diffusion-Weighted Imaging (DWI). We measured DWI-based indices—including Apparent Diffusion Coefficient (ADC), Slow Diffusion Coefficient (SDC), and normalized ratios—alongside CT-derived metrics, specifically Difference of Renal Attenuation (DRA), Corticomedullary Differentiation (CMD), and Difference in Corticomedullary Differentiation (DCMD). These indices were compared between obstructed and contralateral (normal) kidneys to assess diagnostic value. Predictive models for eGFR were developed using various index combinations, selecting the optimal model based on the highest Adjusted R-squared (R^2). Model accuracy was validated using Mean Absolute Error (MAE) and Root Mean Square Error (RMSE), with age and sex as fixed covariates ($p < 0.05$).

Results: Obstructed kidneys exhibited significantly lower native CT density (32.5 ± 4.2 vs 34.3 ± 2.6 HU, $p = 0.011$) and reduced CMD ($p < 0.001$) than normal kidneys. Although ADC and ADC ratios showed no significant differences ($p > 0.05$), the SDC and SDC ratios (rSDC) were significantly higher in obstructed kidneys (SDC: 0.14 ± 0.03 vs 0.13 ± 0.03 ; rSDC_Muscle: 7.05 ± 2.69 vs 6.18 ± 2.34 ; rSDC_Spleen: 1.70 vs 1.47 ; all $p < 0.05$). Combining diffusion metrics with multiphase CT attenuation enhanced eGFR prediction. The best model, integrating ADC, CMD, and Mean_rADC_Muscle, achieved an adjusted $R^2 = 0.433$ ($p = 0.0005$). The similar MAE (0.552) and RMSE (0.695) values demonstrate consistent eGFR estimation with minimal variability.

Conclusion: This exploratory investigation highlights the potential of a multimodal framework that integrates CT hemodynamic indices with MRI microstructural metrics for noninvasive eGFR estimation in acute urinary obstruction. This quantitative methodology demonstrates promising capability in identifying localized renal impairment, serving to complement established laboratory diagnostics.

Keywords: acute urinary obstruction, eGFR prediction, multimodal imaging, diffusion-weighted imaging, multiphase CT, slow diffusion coefficient

Introduction

Acute urinary tract obstruction is a common urological condition affecting both pediatric and adult populations. It typically presents with sudden-onset renal colic due to partial or complete blockage of urinary flow within the collecting

system. Obstruction remains a significant cause of renal failure, accounting for approximately 10% of cases.^{1,2} In acute obstruction, the transition from increased intrarenal pressure to irreversible parenchymal damage is driven by micro-circulatory compromise. Histological changes primarily involve the renal interstitium, characterized by tubular dilation, progressive interstitial fibrosis, and secondary parenchymal loss due to cell death. The resulting urinary stasis raises pressure throughout the upper collecting system, which is transmitted backward to the Bowman's capsule. When intratubular pressure approaches glomerular filtration pressure, the estimated glomerular filtration rate (eGFR) begins to decline gradually, especially after complete ureteral blockage.^{1,3,4} While current imaging methods are effective for diagnosing obstructive causes, they are limited in measuring their functional impact.⁵ This reliance on qualitative assessment leaves clinicians without a reliable, non-invasive metric for real-time parenchymal viability. Serum creatinine and eGFR are insensitive markers for acute unilateral injury because the contralateral kidney compensates for functional loss, masking localized parenchymal damage. Furthermore, creatinine exhibits a 2-to-4-day kinetic lag relative to the actual decline in GFR and is confounded by non-renal factors, delaying the diagnosis of acute renal failure by up to a week.^{6,7} Quantitative multimodal imaging provides a robust solution to these diagnostic limitations. However, current research usually evaluates computed tomography (CT) and magnetic resonance imaging (MRI) parameters separately,^{8,9} and rarely examines the interaction between CT-derived hemodynamics—specifically the quantitative markers of delayed and reduced nephrograms—and MRI-based microstructural changes. By combining these metrics, we can better understand the complex relationship between macrovascular perfusion and intrinsic tissue health during acute obstruction. Specifically, the Slow Diffusion Coefficient (SDC) offers a more detailed assessment of tissue-level diffusion by distinguishing true molecular motion from acute perfusion noise. This enables a more precise measurement of parenchymal damage, regardless of hemodynamic fluctuations.^{10,11}

We aimed to evaluate the predictive value of combining CT hemodynamic metrics with MRI diffusion parameters (ADC and SDC) for noninvasive eGFR estimation in acute obstructive uropathy. Given its exploratory nature, this study sought to determine whether this integrated quantitative approach could effectively stratify patient risk and optimize the timing of surgical intervention.

Materials and Method

Patient Selection

The study employed a single-center, cross-sectional descriptive design conducted from January 2025 to December 2025 and included patients aged 18 years or older presenting with unilateral acute urinary tract obstruction caused by urolithiasis. Diagnosis was confirmed by definitive CT and MRI findings. The following exclusion criteria were implemented to reduce confounding effects on renal function and imaging parameters: (1) clinical or radiological evidence of acute pyelonephritis or systemic infection related to the obstructing calculus; (2) history of renal surgery or structural interventions; (3) pre-existing chronic systemic diseases or renal conditions known to influence baseline kidney function; (4) co-existing renal tumors or significant space-occupying lesions; (5) bilateral obstruction or anuria at presentation. Baseline serum creatinine levels were measured upon admission, approximately 1–5 hours prior to the CT examination. The estimated glomerular filtration rate (eGFR) was subsequently calculated using the 2021 Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.

Image Acquisition Protocols

All patients underwent multiphasic CT and MRI—including diffusion-weighted imaging (DWI)—to evaluate suspected acute urinary tract obstruction immediately upon admission. MRI was performed after CT, with the inter-modality interval typically not exceeding 4 hours.

Multiphase CT examinations were performed on a 64-detector CT scanner (Revolution Maxima, GE Healthcare). Non-contrast CT images were obtained first to detect urinary calculi and assess baseline attenuation. Subsequently, intravenous iodinated contrast medium (1.5–2.0 mL/kg of body weight) was injected at 3–4 mL/sec via a power injector. Contrast-enhanced phases were obtained as follows: corticomedullary phase at 30–40 seconds after injection; nephrographic phase at 80–100 seconds after injection; and excretory phase at 5 minutes after injection. Scanning parameters

included a tube voltage of 120 kVp and a tube current of 100 mA with automatic exposure control. Helical scanning was performed with a rotation time of 0.5 seconds, a pitch of 1.375:1, and a table speed of 55 mm per rotation, yielding a coverage speed of 110 mm/sec. Detector coverage was 40 mm, with a helical slice thickness of 5.0 mm. Images were reconstructed with an AR60 algorithm, using a slice thickness of 1.25 mm and a reconstruction interval of 1.0 mm. The display field of view (FOV) was 36 cm, with a matrix size of 512×512. Standard soft-tissue window settings were used, with a window width/level (W/L) of 400/40. Images were reconstructed in axial planes, and coronal and sagittal reformations were generated as needed.

MRI examinations were performed on a Siemens Magnetom Amira 1.5T system with a 13-channel phased-array body coil. The MRI protocol included axial T1-weighted imaging, axial and coronal T2-weighted imaging, and coronal DWI. DWI was performed with a single-shot echo-planar imaging sequence, with parameters: TR of 7900 ms, TE of 67 ms, a 20% distance factor, and phase-encoding direction set to anterior–posterior. The FOV was 380 mm in the read direction with a 100% phase FOV. Images were acquired with a slice thickness of 4.0 mm, a voxel size of 1.4×1.4 × 4.0 mm, a base resolution of 134, and a phase resolution of 100%. Parallel imaging used a PAT factor of 2. Diffusion gradients were applied at three b-values (50, 400, and 800 s/mm²). Fat suppression was achieved with a spectral attenuated inversion recovery (SPAIR) technique in strong mode. The total acquisition time was 4 minutes and 5 seconds.

CT Image Analysis and DDN Quantification

The delayed and diminished nephrogram (DDN) was quantitatively evaluated by measuring renal parenchymal attenuation in Hounsfield Units (HU) across three phases: non-contrast, corticomedullary, and nephrographic.⁸ For each kidney, standardized 2–4 mm radius regions of interest (ROIs) were placed at the superior, middle (at the renal hilum), and inferior poles. In the non-contrast phase, ROIs were placed within the overall parenchyma. In the contrast-enhanced phases, ROIs were strictly localized to the renal cortex (corticomedullary phase) and medulla (nephrographic phase) (Figure 1).

Difference of Renal Attenuation (DRA)

The DRA was used to quantify the reduced nephrogram by comparing the enhancement of the obstructed kidney with that of the healthy control kidney.⁸ The specific formulas were as follows:

$$\text{Cortical DRA} = \text{Attenuation}_{\text{obstructed_cortex}} - \text{Attenuation}_{\text{normal_cortex}}$$

$$\text{Medullary DRA} = \text{Attenuation}_{\text{obstructed_medulla}} - \text{Attenuation}_{\text{normal_medulla}}$$

$$\text{Total DRA} = \text{Cortical DRA} + \text{Medullary DRA}$$

$$\text{Normalized Total DRA} = \frac{\text{Medullary DRA}}{\text{Attenuation}_{\text{normal_medulla}}} + \frac{\text{Cortical DRA}}{\text{Attenuation}_{\text{obstructed_cortex}}}$$

A DRA value < 0 indicates a diminished nephrogram in the obstructed kidney.

Corticomedullary Differentiation (CMD)

The CMD measures the attenuation gradient between the cortex and medulla within the same kidney, reflecting the kinetics of contrast medium transit.⁸ It was calculated as follows:

$$\text{CMD} = \text{Attenuation}_{\text{kidney_cortex}} - \text{Attenuation}_{\text{kidney_medulla}}$$

An elevated CMD value indicates delayed contrast movement through the renal parenchyma.

Difference in Corticomedullary Differentiation (DCMD)

To quantify the delayed nephrogram, the DCMD was calculated by comparing CMD values between the two kidneys:⁸

$$\text{DCMD} = \text{CMD}_{\text{obstructed}} - \text{CMD}_{\text{normal}}$$

Higher DCMD values indicate more pronounced delays in parenchymal enhancement in the obstructed kidney.

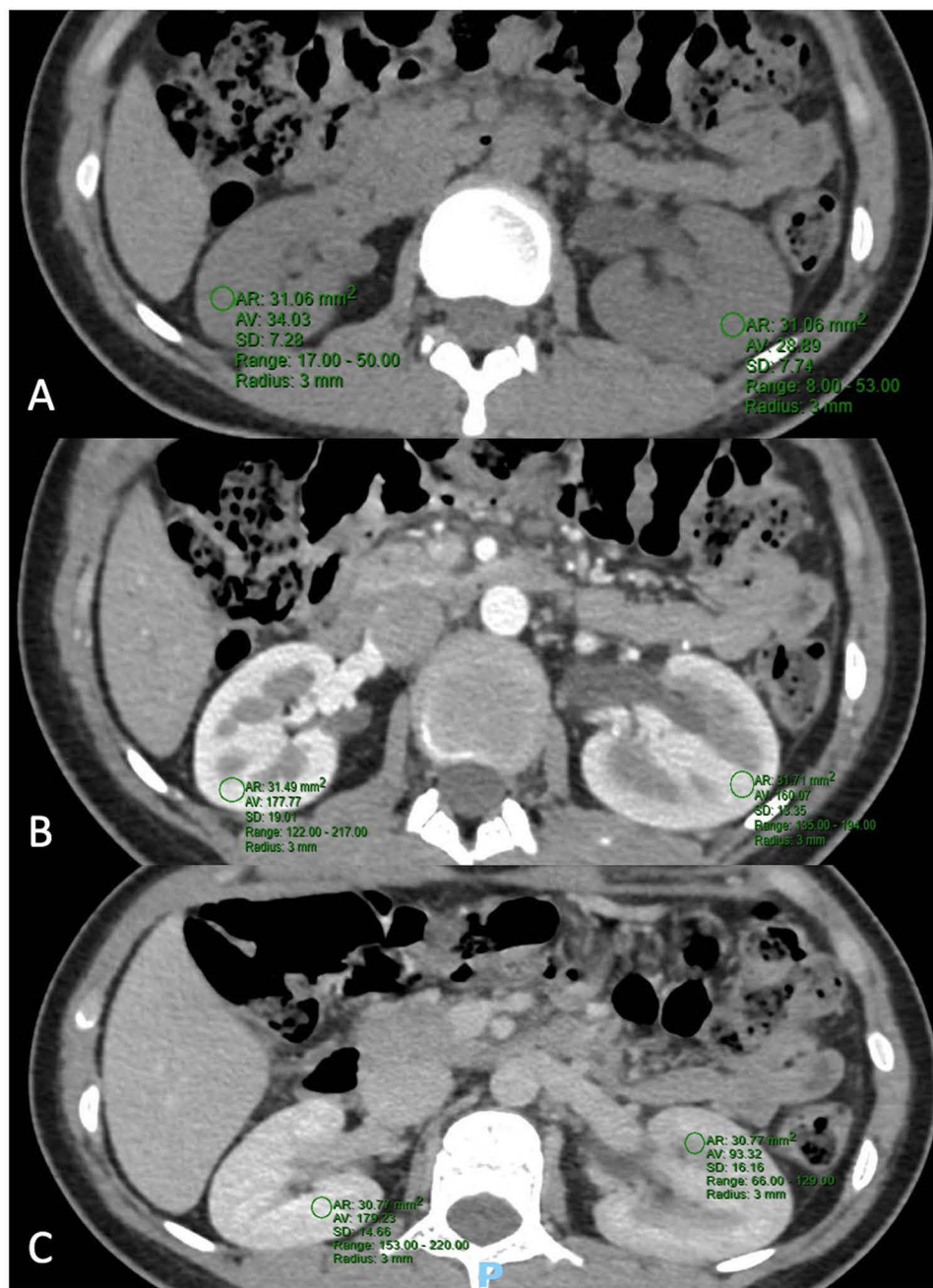


Figure 1 Quantitative assessment of renal attenuation using standardized regions of interest (ROIs) across non-contrast (A), corticomedullary (B), and nephrographic (C) CT phases.

Quantitative MRI Diffusion Parameters and Normalized Ratios

Post-processing and image analysis were carried out performed using pMRI software (Parametric MRI, Philadelphia, PA, USA), where. Apparent Diffusion Coefficient (ADC) maps were generated using mono-exponential fitting models. All measurements were performed by a board-certified radiologist with 10 years of experience.

Circular ROIs with a radius of 5–6 mm were placed on the DWI image to exclude macro-vessels, the collecting system, and focal lesions. Renal parenchyma: ROIs were positioned at the superior, middle, and inferior poles of each kidney, consistent with the CT densitometry protocol. Reference tissues: Two ROIs were placed in the spleen and in the bilateral psoas muscles (Figure 2).

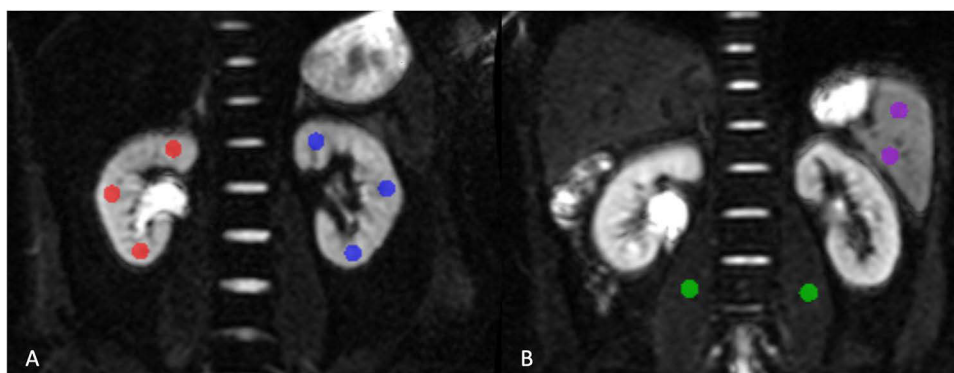


Figure 2 Spatial localization of color-coded circular regions of interest (ROIs) (5–6 mm radius) on Diffusion-Weighted Imaging (DWI). Representative images demonstrate standardized sampling of the renal parenchyma (red, **A**) and reference tissues, including the contralateral kidney (blue, **A**), the spleen (purple, **B**) and psoas muscles (green, **B**), to ensure inter-organ comparability.

Apparent Diffusion Coefficient and Normalized ADC Ratios

The final ADC value for each organ was the average across its respective ROIs. To reduce inter-individual variability, normalized ADC ratios (rADC) were calculated for both obstructed and normal kidneys as follows:

$$\text{rADC_Spleen} = \frac{\text{ADC_kidney}}{\text{ADC_spleen}}$$

$$\text{rADC_Muscle} = \frac{\text{ADC_kidney}}{\text{ADC_muscle}}$$

Slow Diffusion Coefficient (SDC) and Normalized SDC Ratios

The SDC, expressed in arbitrary units (au)/s, was calculated from signal intensities (SI) at $b = 400 \text{ s/mm}^2$ and $b = 800 \text{ s/mm}^2$ as follows:^{10,12}

$$\text{SDC} = \frac{\text{SI}_{b400} - \text{SI}_{b800}}{800 - 400}$$

To ensure accurate anatomical and functional co-registration, SDC and ADC values were extracted simultaneously from the same ROI coordinates. This metric reflects the rate of signal intensity decline, providing a specialized assessment of slow diffusion components within the renal parenchyma.

To reduce interindividual variability, normalized SDC ratios (rSDC) were calculated for both obstructed and normal kidneys relative to reference tissues.

$$\text{rSDC_Spleen} = \frac{\text{SDC_kidney}}{\text{SDC_spleen}}$$

$$\text{rSDC_Muscle} = \frac{\text{SDC_kidney}}{\text{SDC_muscle}}$$

Computation of Bilateral Renal Diffusion Parameters and Ratios

To reflect overall renal function, all DWI indices were calculated as the arithmetic mean of the values from both kidneys. This bilateral averaging was applied to the main quantitative parameters (ADC and SDC) and to their normalized ratios relative to the psoas muscle and spleen (rADC_Muscle, rADC_Spleen, rSDC_Muscle, and rSDC_Spleen). The resulting predictors, called Mean_ADC, Mean_SDC, Mean_rADC_Muscle, Mean_rADC_Spleen, Mean_rSDC_Muscle, and Mean_rSDC_Spleen, were then used as standardized inputs for the subsequent multivariate regression models.

Statistical Analysis

Statistical analyses were performed using SPSS v26.0 (IBM Corp., Armonk, NY, USA) and Python v3.12 (Python Software Foundation). Normality of the data distribution was assessed with the Shapiro–Wilk test. Continuous variables were reported as mean $\pm$ standard deviation (SD) for normally distributed data and as median [interquartile range, IQR] for non-normal data. To compare obstructed and contralateral normal kidneys, the paired *t*-test was used for parametric data, and the Wilcoxon signed-rank test was used for non-parametric data. The relationship between eGFR and quantitative imaging parameters was assessed using Pearson or Spearman correlation coefficients and visualized in a correlation heatmap.

Additionally, a systematic combinatorial modeling approach was used to predict eGFR by combining fixed clinical covariates (Age and Sex) with multiple functional imaging parameters. Imaging parameters were grouped into four main categories: ADC (ADC, rADC_Muscle, and rADC_Spleen), SDC (SDC, rSDC_Muscle, and rSDC_Spleen), CT_DRA (including Cortical, Medullary, Total DRA, CMD, and DCMD), and MRI_DWI_Mean (covering the mean values of SDC, ADC, and their respective muscle/spleen ratios from both kidneys). All continuous variables were Z-score standardized to enable direct comparison of their relative impacts. We tested all possible model combinations using Ordinary Least Squares (OLS) regression, selecting the best models based on the highest Adjusted R^2 and p-values less than 0.05. The robustness of the top models was further evaluated using multicollinearity diagnostics (Variance Inflation Factor, $VIF < 5$) and residual analysis to ensure prediction accuracy and reliability. The models' predictive performance was carefully assessed using Mean Absolute Error (MAE) and Root Mean Square Error (RMSE) to quantify the magnitude of prediction errors. For all tests, a two-tailed p-value under 0.05 was deemed statistically significant.

Results

The study enrolled 35 patients with a mean age of 50.9 ± 14.3 years; 19 (54.3%) were male and 16 (45.7%) were female. The clinical and radiological characteristics are summarized in Table 1. Regarding the obstructed side, the distribution was approximately equal between the right (51.4%) and left (48.6%) kidneys. Acute obstruction was most often caused by a single calculus (91.4%), with the proximal ureter the most common site of obstruction identified by both CT (62.9%) and MRI (68.8%). Hydronephrosis was present in all cases, with Grade II the most common presentation on CT (45.7%), while MRI showed a higher proportion of Grade III obstruction (25.7% vs 11.4% on CT). Significant secondary radiological signs of acute obstruction on CT included ureteral dilation (91.4%), ureteral wall edema (48.6%), and renal enlargement (48.6%).

The quantitative assessment of DDN parameters revealed significant deviations in renal enhancement patterns (Table 2). All Density Reduction Areas (DRA) were negative, confirming a systematic decrease in contrast opacification within the obstructed kidneys. This reduction was more pronounced in the medullary region (-44.9 ± 44.9) than in the cortical region (-16.5 ± 18.5). Consequently, the obstructed kidneys had a significantly higher CMD (44.3 ± 35.0) than the normal contralateral side (14.9 ± 35.0), yielding a substantial positive DCMD of 29.6 ± 32.0 .

Further comparative analysis revealed significant differences in several quantitative imaging parameters between obstructed and normal contralateral kidneys (Table 3). The analysis indicated significantly lower native CT density (32.5 ± 4.2 vs 34.3 ± 2.6 HU, $p=0.011$) and substantially higher CMD ($p<0.001$) in obstructed kidneys. Similarly, SDC and its ratios (rSDC_Muscle, rSDC_Spleen) were significantly increased ($p<0.05$). In contrast, no significant differences were observed in ADC values or their ratios ($p>0.05$).

Correlation analysis between renal function and quantitative imaging parameters showed that ADC and CMD had the strongest associations ($R = 0.319$ and $R = -0.214$, respectively). However, none of the observed correlations were statistically significant ($p > 0.05$) (Figure 3). Similarly, other parameters, including DRA variants and rSDC ratios, exhibited only weak or negligible correlations with eGFR ($|R| < 0.1$).

Among the 20 most robust predictive models identified (Figure 4), the top 5 were selected for detailed analysis based on their adjusted R^2 values. Multivariable regression analysis identified these 5 models for renal function, with R^2 values exceeding 0.4 (Table 4). The best-performing model (Model 1) integrated Age, Sex, ADC, CMD, and

Table 1 Imaging Characteristics of Obstructed Kidneys on CT and MRI

Characteristics	CT	MRI
	n (%)	n (%)
Affected Side		
Right	18 (51.4)	18 (51.4)
Left	17 (48.6)	17 (48.6)
Obstruction Site		
Renal Pelvis	1 (2.9)	1 (2.9)
Proximal Ureter (1/3 upper)	22 (62.9)	24 (68.8)
Mid Ureter (1/3 middle)	5 (14.3)	3 (8.6)
Mid Ureter (1/3 middle)	7 (20.0)	7 (20.0)
Hydronephrosis		
Grade I	14 (40)	12 (34.3)
Grade II	16 (45.7)	13 (37.1)
Grade III	4 (11.4)	9 (25.7)
Grade IV	1 (2.9)	1 (2.9)
Size of the affected kidney		–
Normal	18 (51.4)	–
Enlarged	17 (48.6)	–
Perinephric fat stranding		–
None	23 (65.7)	–
Grade I	7 (20.0)	–
Grade II	3 (8.6)	–
Grade III	2 (5.7)	–
Perinephric fluid collection	7 (20)	–
Gerota's fascia thickening	8 (22.9)	–
Ureteral wall edema	17 (48.6)	–
Ureteral dilation	32 (91.4)	–
Number of stones		–
Single stone	32 (91.4)	–
Two stone	3 (8.6)	–
Total	35 (100)	35 (100)

Table 2 Performance Characteristics of Delayed and Diminished Nephrograms (DDN) (n=35)

	Mean ± SD	Median [IQR]	Shapiro–Wilk test
Cortical DRA	−16.5 ± 18.5	−13.3 [18.8]	p<0.05
Medullary DRA	−44.9 ± 44.9	−35.9 [77.7]	p<0.05
Total DRA	−61.4 ± 60.1	−38.5 [93.9]	p<0.05
Normalized Total DRA	−0.36 ± 0.32	−0.26 [0.54]	p<0.05
CMD obstructed kidney	44.3 ± 35.0	45.9 [41.9]	p>0.05
CMD normal kidney	14.9 ± 35.0	3.9 [52.0]	p<0.05
DCMD	29.6 ± 32.0	23.5 [55.4]	p>0.05

Abbreviations: IQR, Interquartile Range; SD, Standard Deviation; DRA, Difference of Renal Attenuation; CMD, Corticomedullary Differentiation; DCMD, Difference in Corticomedullary Differentiation.

Table 3 Comparison of Quantitative Imaging Parameters Between Obstructed and Contralateral Healthy Kidneys (n=35)

Parameters	Obstructed Kidney	Normal Kidney	t-value	Z-Score	p-value
Native CT density (HU)	32.5 ± 4.2	34.3 ± 2.6	-2.68	-	0.011*
CMD	46.1 [43.7]	3.0 [54.2]	-	-4.30	< 0.001*
ADC (10 ⁻⁶ mm ² /s)	1989.0 [179]	2011.0 [188]	-	-1.29	0.196
rADC_muscle	1.58 [0.21]	1.62 [0.27]	-	-1.41	0.159
rADC_spleen	2.51 [0.44]	2.57 [0.49]	-	-1.39	0.164
SDC (au)/s	0.14 ± 0.03	0.13 ± 0.03	3.76	-	0.001*
rSDC_muscle	7.05 ± 2.69	6.18 ± 2.34	3.72	-	0.001*
rSDC_spleen	1.70 [2.01]	1.47 [1.9]	-	-2.95	0.003*

Notes: * Indicate significance $p < 0.05$. Continuous data were presented as Mean ± SD for normally distributed variables or as Median [IQR] for non-normally distributed variables.

Abbreviations: CMD, Corticomedullary Differentiation; ADC, Apparent Diffusion Coefficient; rADC, ratio Apparent Diffusion Coefficient; SDC, Slow Diffusion Coefficient.

Mean_rADC_Muscle ($R^2 = 0.433$, $p = 0.0005$). Across all models, MAE and RMSE values remained consistent at 0.547–0.619 and 0.694–0.742, respectively, and all associations were statistically significant ($p < 0.01$).

Analysis of the top five models ($R^2 > 0.40$) confirmed consistent predictive trends for eGFR (Figure 5). ADC and rADC_Muscle emerged as the primary positive predictors (orange bars), while Age and CMD consistently showed the strongest negative impact (blue bars). Across all top-five models, the scatter plots demonstrated stable predictive consistency.

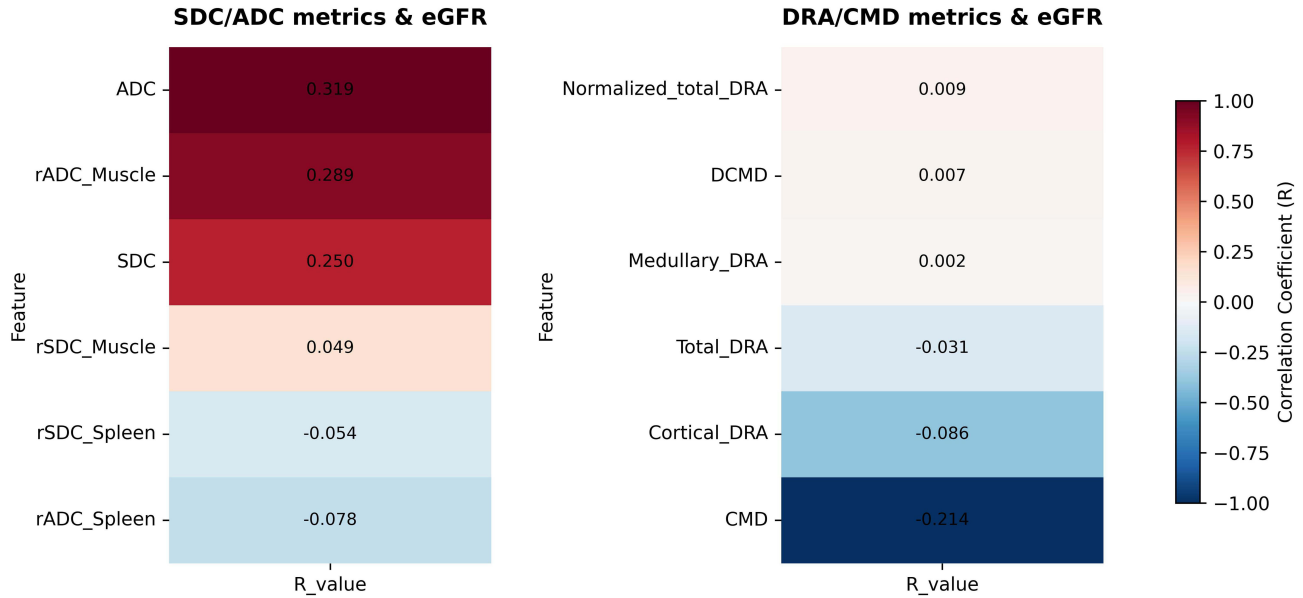


Figure 3 Heatmap profiles of correlations between eGFR and quantitative imaging parameters. The dual-panel heatmaps display Pearson correlation coefficients (R) between eGFR and SDC/ADC metrics (left) or DRA/CMD metrics (right). Features are listed on the y-axis against their R-values on the x-axis. Color intensity encodes the correlation strength and direction (blue: negative; red: positive), with exact R-values annotated within each cell. Although ADC ($R=0.319$) and CMD ($R=-0.214$) exhibited the most prominent trends, no correlations reached statistical significance across all evaluated indices ($p > 0.05$).

Abbreviations: eGFR, estimated glomerular filtration rate; Adj. R^2 , adjusted R-squared; ADC, Apparent Diffusion Coefficient; CMD, Corticomedullary Differentiation; SDC, Slow Diffusion Coefficient; rADC, ratio Apparent Diffusion Coefficient; rSDC, ratio Slow Diffusion Coefficient; DRA, Difference of Renal Attenuation; DCMD, Difference in Corticomedullary Differentiation.

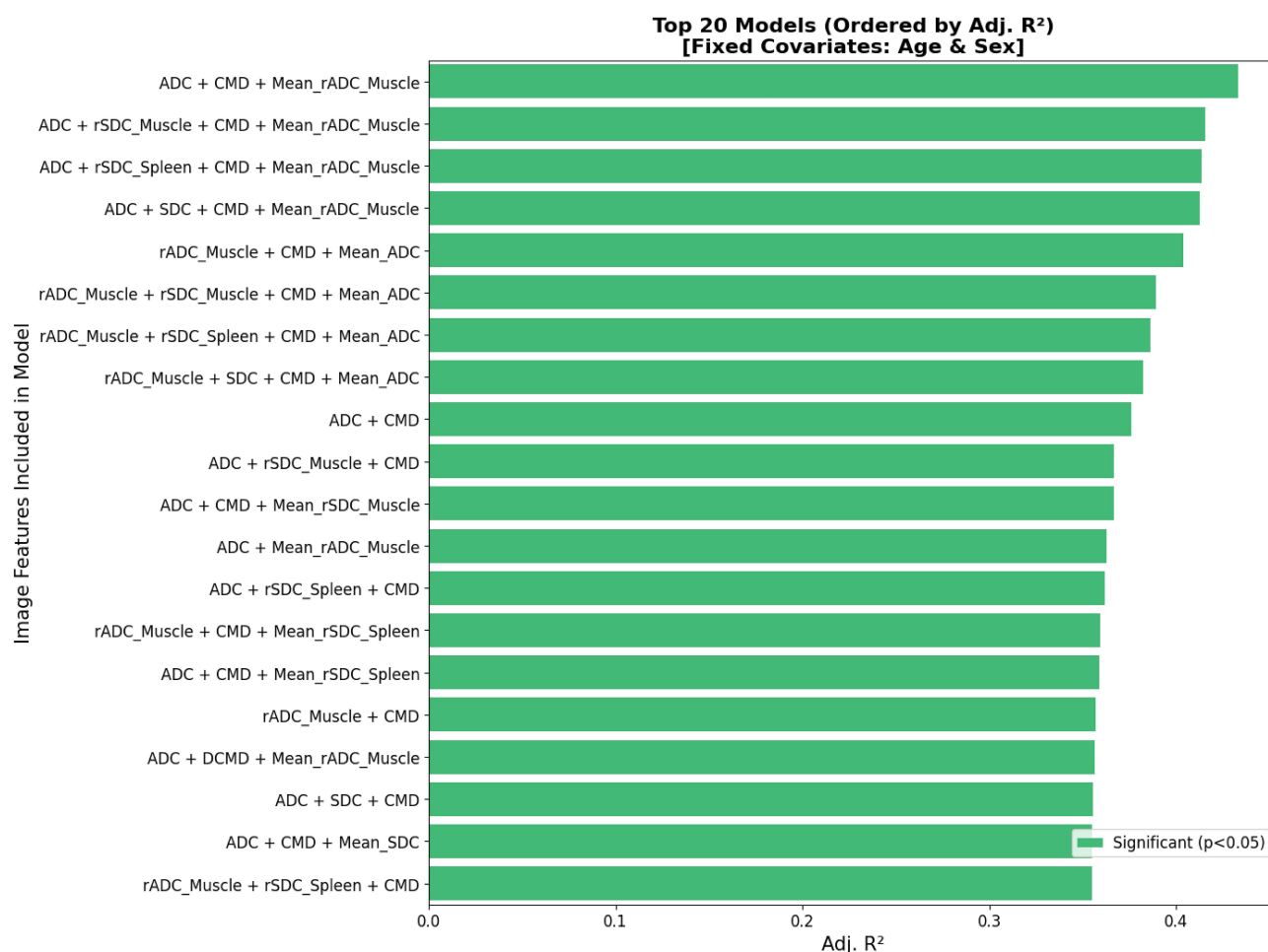


Figure 4 Comparative performance of the top 20 predictive models for eGFR. The horizontal bar chart ranks the top 20 age- and sex-adjusted regression models by Adjusted R-squared (Adj. R²). The y-axis lists the specific imaging feature combinations against the model fit on the x-axis. The green bars demonstrate that integrating hemodynamic (CMD) and microstructural (ADC, rADC, SDC) indices consistently yields Adj. R² > 0.35, with all configurations achieving statistical significance (p < 0.05). The top-performing architecture achieves the highest variance explanation by optimally combining cortical enhancement kinetics with water diffusion metrics.

Abbreviations: eGFR, estimated glomerular filtration rate; Adj. R², adjusted R-squared; ADC, Apparent Diffusion Coefficient; CMD, Corticomedullary Differentiation; SDC, Slow Diffusion Coefficient; rADC, ratio Apparent Diffusion Coefficient; rSDC, ratio Slow Diffusion Coefficient.

Discussion

This study shows that quantitative imaging biomarkers offer a more accurate assessment of acute urinary obstruction than qualitative methods. By measuring changes in microcirculation and water diffusion, these indicators provide a measurable framework for predicting eGFR impairment during the acute phase.

Table 4 Top 5 Predictive Models for Renal Function Assessment in Acute Urinary Obstruction

	Features	R ²	MAE	RMSE	p_value
1	Age + Sex + ADC + CMD + Mean_rADC_Muscle	0.433	0.552	0.695	0.0005*
2	Age + Sex + ADC + rSDC_Muscle + CMD + Mean_rADC_Muscle	0.415	0.550	0.694	0.0013*
3	Age + Sex + ADC + rSDC_Spleen + CMD + Mean_rADC_Muscle	0.413	0.547	0.695	0.0014*
4	Age + Sex + ADC + SDC + CMD + Mean_rADC_Muscle	0.413	0.551	0.695	0.0014*
5	Age + Sex + rADC_Muscle + CMD + Mean_ADC	0.404	0.570	0.713	0.001*

Note: * Indicate significance p < 0.05.

Abbreviations: MAE, Mean Absolute Error; RMSE, Root Mean Square Error; CMD, Corticomedullary Differentiation; ADC, Apparent Diffusion Coefficient; rADC, ratio Apparent Diffusion Coefficient; SDC, Slow Diffusion Coefficient.

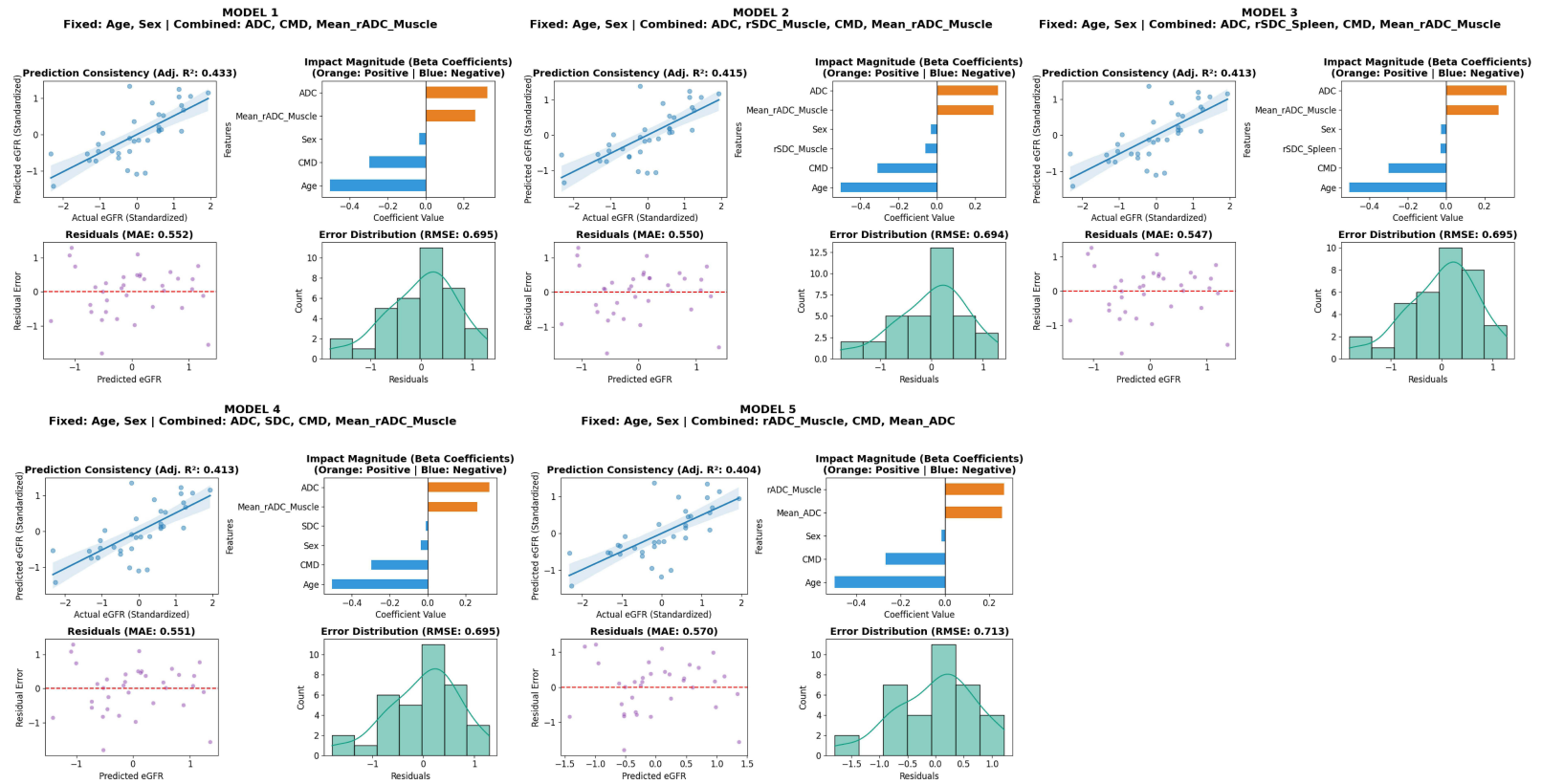


Figure 5 Comprehensive regression analysis and feature importance profiles for the top five eGFR predictive models. The multi-panel matrix displays the performance, error profiles, and feature weights for the top five age- and sex-adjusted models (Models 1–5). Each model panel contains a quadrant subplot grid: (Top-Left) Prediction Consistency: scatter plots of standardized actual versus predicted eGFR with a linear fit (solid blue line), 95% confidence interval (shaded area), and Adjusted R²; (Top-Right) Impact Magnitude: horizontal bar charts of standardized beta coefficients ranking feature importance (Orange: positive association; blue: negative association); (Bottom-Left) Residuals: scatter plots of errors against predictions with a dashed red zero-baseline and MAE; and (Bottom-Right) Error Distribution: histograms overlaid with kernel density estimate (KDE) curves and RMSE.

Abbreviations: eGFR, estimated glomerular filtration rate; ADC, Apparent Diffusion Coefficient; CMD, Corticomedullary Differentiation; SDC, Slow Diffusion Coefficient; rADC, ratio Apparent Diffusion Coefficient; rSDC, ratio Slow Diffusion Coefficient; MAE, Mean Absolute Error; RMSE, Root Mean Square Error.

The main contribution of this study is the integration of CT-derived hemodynamic indices (DRA, CMD, and DCMD) with MRI-based diffusion metrics (ADC and SDC). A key feature of this multimodal approach is the use of SDC. By isolating slow-moving water components through high-b-value signal decay,¹⁰ SDC serves as a sensitive marker for early microstructural changes that often come before overall functional impairment. Conventional assessments of acute unilateral kidney injury are often limited by the insensitivity of serum creatinine and eGFR. Because these systemic markers reflect global renal function, contralateral compensation often conceals acute parenchymal damage—a phenomenon further exacerbated by a 2-to-4-day kinetic lag and confounding non-renal factors.^{6,7} Our approach addresses this diagnostic gap by opportunistically extracting perfusion and diffusion metrics from standard-of-care CT and MRI scans to quantify localized microstructural and hemodynamic impairment. The findings suggest that combining these clinico-radiological markers provides a more nuanced profile of renal status than isolated parameters, helping bridge the gap between structural imaging and functional laboratory metrics. Clinically, localized perfusion and diffusion deficits can serve as early indicators to optimize the timing of surgical decompression, potentially preventing irreversible parenchymal loss. Furthermore, these imaging phenotypes may aid in patient risk stratification, distinguishing patients likely to achieve spontaneous recovery from those requiring intensive post-discharge monitoring for chronic progression. While these implications are significant, the exploratory nature and modest sample size of this study necessitate larger prospective, multicenter trials to validate standardized thresholds for clinical decision-making.

Quantitative analysis of DDN using DRA and CMD provides an objective assessment of hemodynamic shifts in acute obstruction; these indices are pathognomonic markers of acute urinary tract obstruction. These indices (DRA and DCMD) exhibit exceptionally high specificity, although their sensitivity remains modest, ranging from 33% to 52%.⁸ Specifically, a medullary DRA threshold of < -15 HU and a DCMD threshold of > 20 HU both yielded 90% specificity for predicting obstructive status.⁸ Another study established an optimal threshold of 15 HU for cortical attenuation differences via ROC analysis, providing balanced diagnostic performance with 85.2% sensitivity and 85.1% specificity.¹³ Our results indicated a significant reduction in native renal attenuation within the obstructed kidney, consistent with interstitial edema and increased parenchymal water content.¹⁴ Using a threshold of < 5 HU for inter-renal attenuation differences, we observed 100% specificity and PPV in detecting ureteral lithiasis. The associated sensitivity, NPV, and overall accuracy were 61%, 69%, and 79%, respectively.¹⁵ Negative DRA values, coupled with elevated CMD, confirm a disrupted corticomedullary enhancement gradient. These findings align strongly with the study by Marshall C. Strother et al,⁸ further validating the clinical reliability of these quantitative thresholds in the acute setting.

Physiologically, these changes are driven by an acute rise in intrarenal pressure transmitted to Bowman's space, which reduces net glomerular filtration pressure and triggers pre-glomerular vasoconstriction.^{1,16} To minimize the impact of systemic confounders such as cardiac output and contrast injection kinetics, we used normalized DRA. This approach improves diagnostic reliability compared with absolute Hounsfield Unit (HU) measurements, which are often affected by inter-patient variability and injection rate differences that can bias results.

Our findings align with the literature, which has shown that decreased parenchymal uptake on CT correlates with reduced renal blood flow due to pressure.^{16,17}

Our study observed a reduction in ADC within the obstructed renal parenchyma compared with the contralateral healthy kidney. Normal renal ADC values generally fall within the $1.8\text{--}2.2 \times 10^{-3} \text{ mm}^2/\text{s}$ range, although these metrics can be influenced by technical parameters and b-value settings.^{9,18} Pathophysiologically, this decline is driven by the mechanical buildup of intrarenal pressure, which is transmitted to the interstitial space, compressing the microvasculature and causing localized ischemia. Since ADC values reflect both pure water diffusion and capillary perfusion (intravoxel incoherent motion), this hemodynamic impairment typically results in a decrease in the measured ADC.^{19–21} However, in our cohort, this reduction did not reach statistical significance ($p > 0.05$). This lack of significance may be due to sample heterogeneity, particularly the varying durations of acute obstruction among patients, which could lead to different stages of parenchymal response.

To better define these microstructural changes, we introduced the SDC. Unlike ADC, which is affected by perfusion effects, SDC is generated from high b-values ($b=400$ and $b=800 \text{ s/mm}^2$), effectively isolating true diffusion within the cellular microenvironment. While ADC reflects a mix of perfusion and diffusion, SDC specifically detects subtle microstructural changes and is less influenced by T2 relaxation or quick hemodynamic shifts. To our knowledge, the

use of SDC has not been previously studied in acute urinary tract obstruction. Although it is well established for characterizing liver disease, parotid gland lesions, and brain tumors, its role in renal obstructive pathology remains to be clarified.^{10–12}

While a decline in SDC typically indicates persistent parenchymal damage, our results demonstrated a slight but statistically significant increase in acutely obstructed kidneys. This pattern suggests that during the acute phase, the renal architecture remains largely intact with minimal fibrotic accumulation.^{2,3} We hypothesize that this elevation in SDC reflects acute interstitial edema and extracellular volume expansion, driven mechanically by tubular dilatation and an early inflammatory response that expand fluid-filled spaces and reduce water diffusion barriers.^{3,4,6} Consequently, SDC may offer higher specificity than ADC for capturing these early microstructural changes. However, these physiological mechanisms remain speculative in the acute setting and require validation in future longitudinal studies.

Our univariate correlation analysis revealed that while ADC ($R = 0.319$) and CMD ($R = -0.214$) exhibited the strongest individual trends with eGFR, none of these single-parameter associations reached statistical significance ($p > 0.05$, Figure 3). Similarly, individual DRA variants and rSDC ratios demonstrated negligible linear correlations. Nevertheless, the underlying biological trends of these metrics align closely with renal pathophysiology. Among all parameters, a decline in water diffusivity (ADC) within the renal parenchyma reflects the reduction in global filtration capacity. Conversely, the negative correlation of CMD indicates that a greater loss of corticomedullary differentiation on CT corresponds to lower eGFR levels. These results explain why ADC and CMD were consistently identified as the most important predictors in our top models. In contrast, indices related to DRA (attenuation loss) showed very little linear relationship with eGFR. This suggests that while DRA is highly useful for detecting acute obstruction, it may be less effective for measuring the degree of functional impairment on its own. By combining the microstructural information from ADC with the hemodynamic data from CMD, our combined models achieved good predictive accuracy, effectively connecting localized tissue changes with overall renal function.

While SDC showed a weak linear correlation with eGFR, its inclusion in the top-performing models highlights its role as a crucial modulating variable. It detects subtle microstructural changes such as early interstitial edema that ADC may miss due to capillary noise. Therefore, SDC does not simply duplicate ADC data; it provides complementary insights into parenchymal viability, ensuring the models stay reliable even during the fluctuating stages of acute obstruction.

This discrepancy between non-significant univariate correlations and the moderate multivariable performance ($R^2 > 0.40$, $p < 0.001$) reflects the multidimensional nature of acute urinary obstruction. Single physiological metrics appear insufficient to predict global functions like eGFR, which are often confounded by contralateral renal compensation and systemic kinetic lags. However, combining microstructural MRI metrics with hemodynamic CT indices and clinical demographics may offer a complementary effect. This multimodal approach tentatively addresses shared aspects of renal pathophysiology, potentially reducing the underestimation of injury associated with isolated parameters and providing a more balanced reflection of acute renal functional status.

Limitations and Future Orientations

Our results indicate that combining imaging-derived parameters has the potential to predict eGFR in acute clinical settings. Several limitations of this study warrant consideration. First, the sample size was relatively small due to the clinical challenges of acquiring both acute CT and MRI scans sequentially, which precluded robust cross-validation or external validation. Second, the absence of post-treatment or post-decompression longitudinal follow-up restricted our capacity to assess temporal changes in renal function and structural recovery. Third, the diffusion-weighted imaging (DWI) protocol employed only three b-values, limiting the comprehensive evaluation of perfusion-related diffusion metrics that could potentially enhance predictive performance. Fourth, the exact duration from symptom onset to imaging acquisition was not systematically recorded, introducing a potential confounding variable related to the evolution of acute obstruction.⁹ To address these limitations, future research will focus on expanding the cohort size and optimizing imaging protocols. Specifically, we plan to construct and validate a joint predictive framework that integrates conventional clinical variables—including age, sex, baseline serum creatinine/eGFR, obstruction grade, and symptom duration—with our quantitative imaging biomarkers. This integrated approach will allow us to rigorously quantify the incremental predictive

value, assess model discrimination and reclassification, and evaluate the potential cost-effectiveness of this imaging framework in clinical practice.

Conclusions

In conclusion, this exploratory study demonstrates the potential of a multimodal framework combining CT hemodynamic indices with MRI microstructural metrics for noninvasive eGFR estimation in acute urinary obstruction. While this quantitative approach shows promise in capturing localized renal impairment, it is intended to complement, rather than replace, established laboratory metrics. Further large-scale validation is required to establish its clinical utility for patient risk stratification.

Data Sharing Statement

The datasets generated or analyzed during the study are available from the corresponding author on reasonable request.

Ethical Approval

This study adhered to ethical standards set by the World Medical Association's Declaration of Helsinki principles. Informed consent was obtained from the study participants prior to study commencement which was approved by University of Medicine and Pharmacy, Hue University Institutional Review Board (H2023/008).

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors have no potential conflicts of interest to disclose for this work.

References

1. Quaia E, De Paoli L, Martingano P, Cavallaro M. *Obstructive Uropathy, Pyonephrosis, and Reflux Nephropathy in Adults*; 2014. doi:10.1007/978-3-642-54047-9_14
2. Claudon M, Durand E, Grenier N, et al. Chronic urinary obstruction: evaluation of dynamic contrast-enhanced MR urography for measurement of split renal function. *Radiology*. 2014;273(3):801–812. doi:10.1148/radiol.14131819
3. Kevin PGH, Jeremy H. CHAPTER 58 - urinary tract obstruction. In: *Comprehensive Clinical Nephrology*. Elsevier; 2010:702–715.
4. K.M. K. Part VIII. Upper urinary tract obstruction and trauma. In: *Campbell-Walsh Urology*. 11th ed. Elsevier; 2016:1089–1103.
5. Ames CD, Older RA. Imaging in urinary tract obstruction. *Brazilian J Urol*. 2001;27(4):316–325.
6. Pérez-Aizpurua X, Cabello Benavente R, Bueno Serrano G, et al. Obstructive uropathy: overview of the pathogenesis, etiology and management of a prevalent cause of acute kidney injury. *World J Nephrol*. 2024;13(2):1–12. doi:10.5527/wjn.v13.i2.93322
7. Star RA. Treatment of acute renal failure. *Kidney Int*. 1998;54(6):1817–1831. doi:10.1046/j.1523-1755.1998.00210.x
8. Strother MC, Cho EY, Loecher M, et al. The delayed nephrogram: point-of-care quantitative measurement, validation as indicator of obstruction, and novel use as predictor of renal functional impairment. *Eur Urol Focus*. 2023;8(6):1809–1815. doi:10.1016/j.euf.2022.01.019
9. Düzenli K, Öztürk M, Yıldırım İO, Erdem G. The utility of diffusion-weighted imaging to assess acute renal parenchymal changes due to unilateral ureteral stone obstruction. *Urolithiasis*. 2017;45(4):401–405. doi:10.1007/s00240-016-0924-3
10. Ling GH, Wang YXJ. MRI slow diffusion coefficient (SDC) shows liver pyogenic abscess has faster diffusion than adjacent liver parenchyma. *Quant Imag Med Surg*. 2025;15(7):6547–6551. doi:10.21037/qims-2025-1010

11. Zheng WY, Shi YT, He YY, et al. A novel MRI diffusion metric 'slow diffusion coefficient' (SDC) for diagnosing isocitrate dehydrogenase (IDH) genotype in diffuse gliomas: initial promising results. *BMC Med Imag.* **2025**;25(1). doi:10.1186/s12880-025-01980-y
12. Yao DQ, King AD, Zhang R, Jiang RF, Wong LM, Wang YXJ. Added value of slow diffusion coefficient (SDC) for the separation of benign and malignant parotid gland tumors by diffusion weighted imaging: preliminary results. *BMC Med Imag.* **2025**;25(1). doi:10.1186/s12880-025-02051-y
13. Goldman SM, Faintuch S, Ajzen SA, et al. Diagnostic value of attenuation measurements of the kidney on unenhanced helical CT of obstructive ureterolithiasis. *AJR Am J Roentgenol.* **2004**;182(5):1251–1254. doi:10.2214/ajr.182.5.1821251
14. Georgiades CS, Moore CJ, Smith DP. Differences of renal parenchymal attenuation for acutely obstructed and unobstructed kidneys on unenhanced helical CT. *Am J Roentgenol.* **2001**;176(4):965–968. doi:10.2214/ajr.176.4.1760965
15. Baliyan V, Das CJ, Sharma S, Gupta AK. Diffusion-weighted imaging in urinary tract lesions. *Clin Radiol.* **2014**;69(8):773–782. doi:10.1016/j.crad.2014.01.011
16. Belyaeva K, Rudenko V, Serova N, et al. Kidney computed tomography perfusion in patients with ureteral obstruction. *Urologia J.* **2024**;91(3):486–493. doi:10.1177/03915603241244935
17. Kim DW, Yoon SK, Ha DH, Kang MJ, Lee JH, Choi S. CT-based assessment of renal function impairment in patients with acute unilateral ureteral obstruction by urinary stones. *Abdom Imag.* **2015**;40(7):2446–2452. doi:10.1007/s00261-015-0417-9
18. Pinto DS, George A, Johny J, Hoisala RV. Role of MRI in the evaluation of acute pyelonephritis in a high-risk population with renal dysfunction: a prospective study. *Emerg Radiol.* **2023**;30(3):285–295. doi:10.1007/s10140-023-02122-z
19. Tran HPD, Hoang NT, Dang CN. Benign versus malignant solid liver lesions: how can apparent diffusion coefficient value be added to the differentiation? *Investigative Magnetic Resonance Imag* **2025**;29(4):216–224.
20. Sanga Reddi BR, Bodagala VD, Lakshmi AY, Kumar NA, Kumar VS. Diffusion weighted MR imaging in the diagnosis of acute pyelonephritis and its complications: a prospective observational study. *J NTR Univ Health Sci.* **2019**;8(3):170–174. doi:10.4103/jdntruhs.jdntruhs_116_18
21. Goyal A, Sharma R, Bhalla A, Gamanagatti S, Seth A. Diffusion-weighted MRI in assessment of renal dysfunction. *Indian J Radiol Imag.* **2012**;22(3):155–159. doi:10.4103/0971-3026.107169

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