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Imaging features of renal malperfusion in aortic dissection

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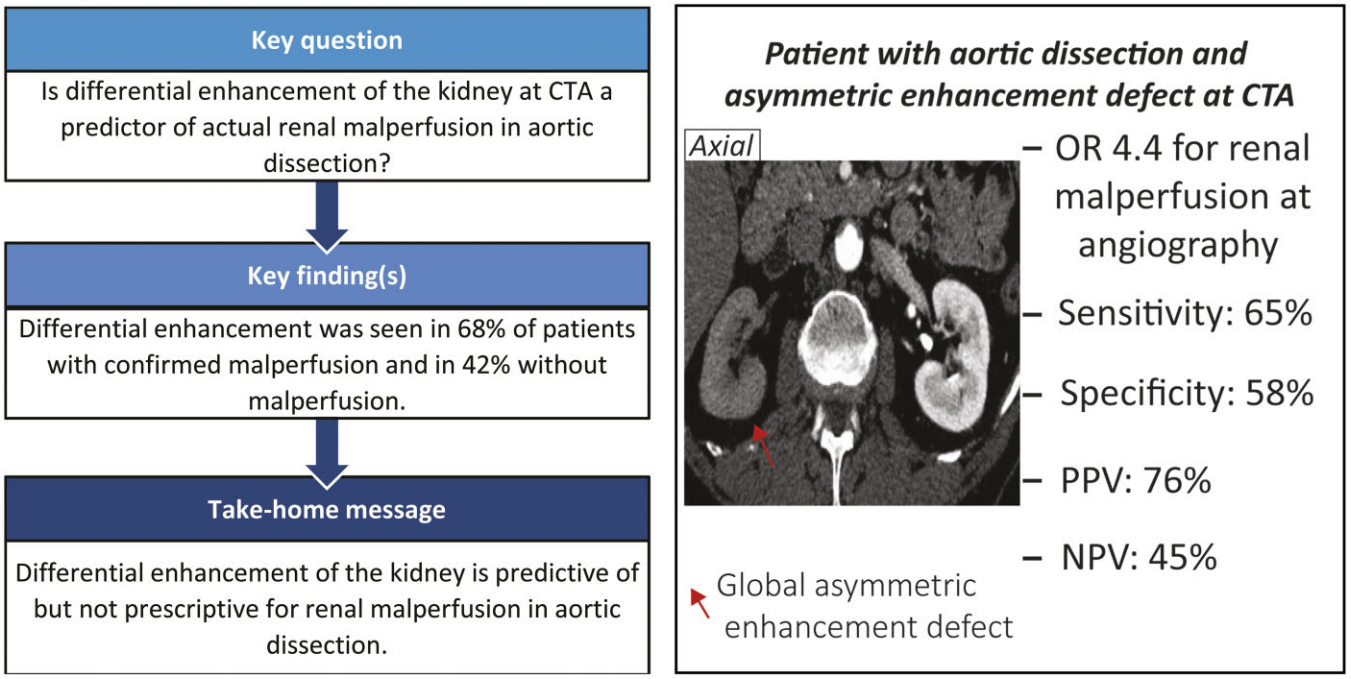

Abstract

OBJECTIVES: Malperfusion syndrome accompanying aortic dissection is an independent predictor of death with in-hospital mortality rates >60%. Asymmetrically decreased renal enhancement on computed tomography angiography is often considered evidence of renal malperfusion. We investigated the associations between renal enhancement, baseline laboratory values and the diagnosis of renal malperfusion, as defined by invasive manometry, among patients with aortic dissection.

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METHODS: In this retrospective cohort study, we included all patients who were referred to our institution with acute dissection and suspected visceral malperfusion between 2010 and 2020. We determined asymmetric renal enhancement by visual assessment and quantitative density measurements of the renal cortex. We collected invasive renal artery pressures during invasive angiography at the aortic root and in the renal arteries. Logistic regression was performed to evaluate independent predictors of renal malperfusion.

RESULTS: Among the 161 patients analysed, the majority of patients were male (78%) and had type A dissection (52%). Invasive angiography confirmed suspected renal malperfusion in 83% of patients. Global asymmetric renal enhancement was seen in 42% of patients who did not have renal malperfusion during invasive angiography. Asymmetrically decreased renal enhancement was 65% sensitive and 58% specific for renal malperfusion. Both global [odds ratio (OR) 4.43; 1.20–16.41, $P = 0.03$] and focal (OR 11.23; 1.12–112.90, $P = 0.04$) enhancement defects were independent predictors for renal malperfusion.

CONCLUSIONS: In patients with aortic dissection, we found that differential enhancement of the kidney as seen on the computed tomography angiography is predictive, but not prescriptive for renal malperfusion. While detection of renal malperfusion is aided by computed tomography angiography, its diagnosis requires close monitoring and often invasive assessment.

Keywords: Renal malperfusion • Aortic dissection • Asymmetric enhancement

ABBREVIATIONS

BUN	Blood urea nitrogen
CTA	Computed tomography angiography
FL	False lumen
HU	Hounsfield Unit
NPV	Negative predictive value
PPV	Positive predictive value
TL	True lumen

INTRODUCTION

Acute aortic dissection is the most frequent acute aortic pathology with a high morbidity and mortality [1–3]. Population-based studies suggest an incidence of approximately 3 cases per 100 000 people per year [4, 5]. Among the many potentially lethal complications of acute aortic dissection (i.e. rupture, stroke, tamponade, aortic valve insufficiency), visceral malperfusion is a dreaded complication that can be difficult to manage, occurring in ∼20–30% of patients with both type A and B aortic dissection [6–12].

Visceral malperfusion in aortic dissection is defined by insufficient blood supply to the organ due to either dynamic or static obstruction of a visceral artery, leading to end-organ dysfunction or malperfusion syndrome, characterized by cell death, tissue necrosis and organ failure. Patients with malperfusion and end-organ dysfunction have poor survival, with reported in-hospital mortality rates ranging from 4% to 90% [13, 14]. The optimal management of patients with malperfusion remains debated with regard to initial management (i.e. endovascular treatment of malperfusion versus open surgical repair of the ascending aorta/root), as well as the timing of treatment (i.e. acute or delayed) [12, 15–19].

Early visceral malperfusion at patient presentation can be assessed by physical findings (i.e. peritoneal signs, oliguria), laboratory evaluation (e.g. elevated serum lactate or creatine) and findings on computed tomography angiography (CTA) imaging (e.g. decreased organ enhancement). Unlike the liver and bowel, which may be supplied by multiple collateral pathways from the superior/inferior mesenteric and coeliac arteries, decreased renal enhancement on CTA is often considered a *sine qua non* of renal malperfusion [20, 21]. However, the link between renal enhancement abnormalities at CTA and true renal perfusion deficits may be confounded by transit time differences of intravenous contrast material between the true lumen (TL) and false lumen (FL) and by the transient nature of dynamic branch artery obstruction. The relationship between CTA findings of asymmetrical decreased renal enhancement and true reductions in renal perfusion pressure is not well supported by data. Considering that patient management and interventions can be influenced by findings on initial CTA imaging, further analysis is warranted to better define the relationship between renal enhancement at CTA and malperfusion assessed through invasive angiography.

The objective of the current study was to investigate associations between asymmetric renal enhancement on CTA, renal artery anatomy, baseline laboratory abnormalities and invasive measurements of renal artery pressure data at catheter angiography among patients with AD and suspected visceral malperfusion. Furthermore, we aimed to determine the predictive value of renal enhancement abnormalities on CTA for diagnosing renal malperfusion at invasive assessment and perform multivariable analysis to identify independent predictors of renal malperfusion at initial presentation.

PATIENTS AND METHODS

Ethical statement

This retrospective analysis was performed as part of an Institutional Review Board approved, HIPAA-compliant study (HUM00159928, approved 03-27-2019) with the waiver of informed consent at the University of Michigan, Ann Arbor, MI, USA.

Study population

We performed a retrospective cohort study of patients that were treated at our centre with a diagnosis of acute aortic dissection and clinical suspicion of visceral or lower extremity malperfusion between January 2010 and December 2020. This timeframe was chosen to ensure optimal CT imaging quality (e.g. thin slice images) and full availability of clinical data from the electronic medical record. A combination of clinic and hospital records and imaging studies was used to obtain clinical and short-term information. Clinical suspicion of malperfusion was based on the treating physicians judgement at the time of presentation but generally included imaging characteristics, clinical features and

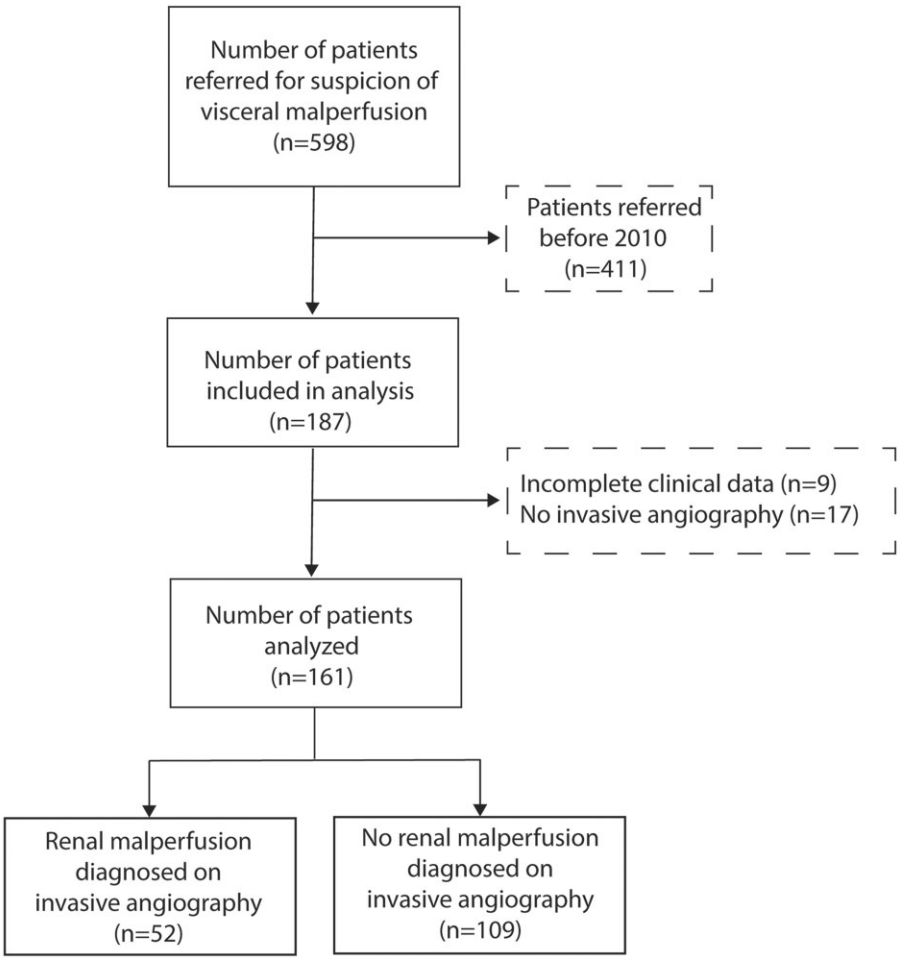

Figure 1: Patient selection flowchart.

laboratory findings (e.g. abdominal pain, bloody stool, decreased urine output, elevated lactate, liver or pancreatic enzymes, bilirubin or creatinine, absence of peripheral pulses, motor or sensory deficit of the extremity, neurological deficit) [22].

Inclusion criteria were aortic dissection (Stanford types A and B) with available CTA data, admission between 2010 and 2020, and a clinical suspicion for peripheral vascular malperfusion involving visceral or lower extremity vessels. Patients were excluded if CTA data were absent, if there were no invasive pressure measurements, or if a patient did not have clinical suspicion for visceral malperfusion (see Fig. 1 for a flowchart of the patient selection). Patients were analysed in 2 groups, either with renal malperfusion or without. Renal malperfusion was diagnosed using invasive angiography measurements of the branch vessel.

Clinical characteristics

Patient demographics, clinical history and laboratory data were collected by chart review. Demographics variables included age, sex and race. Clinical variables included history of hypertension, smoking and diabetes. Laboratory variables included serum creatinine, blood urea nitrogen (BUN), estimated glomerular filtration rate and serum lactate, and these serologic parameters were recorded at baseline (time of initial presentation).

Radiologic characteristics

Patients underwent CTA imaging either at our institution ($n = 17$) or at their referring institution ($n = 144$). A variety of anatomic variables were recorded including: renal artery supply (TL/FL/both), dissection involvement of the renal artery, presence and number of accessory renal arteries and maximal aortic diameter at the renal segment. Furthermore, renal enhancement was measured for each kidney in both a qualitative and quantitative manner. For qualitative assessments, it was noted if there was asymmetric renal enhancement by visual assessment on angiographic windows (level = 200, width = 1000), and the kidney with lower enhancement was recorded. In addition, it was noted if renal enhancement was global (i.e. involving the entire kidney) or focal (i.e. if segmental enhancement abnormalities within 1 kidney). For quantitative assessments, we measured CT Hounsfield unit (HU) attenuation values in the renal cortex using a region of interest tool at the super, mid and inferior renal poles (Fig. 2). All imaging characteristics and measurements were performed by a medical doctor with 3 years of aortic analysis experience (Pieter A.J. van Bakel) and a medical student (Matthew Henry) under the supervision of an experienced radiologist with 15 years of cardiovascular imaging experience (Nicholas S. Burris).

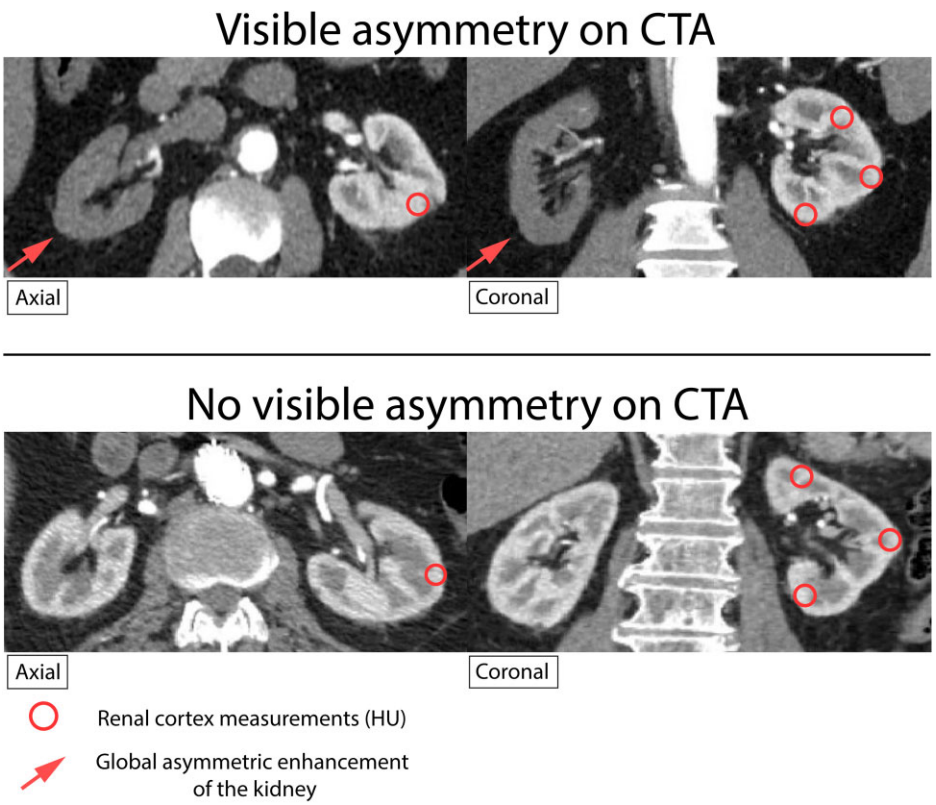

Figure 2: Assessment of renal enhancement on computed tomography angiography. CTA: computed tomography angiography; HU: Hounsfield units.

Table 1: Baseline patient demographics and characteristics

Variable	Total	Renal malperfusion ($n = 52$)	No renal malperfusion ($n = 109$)	*P*-Value
Age, years, mean ± SD	57.2 ± 13.3	54.6 ± 10.9	58.9 ± 13.3	0.14
Male, *n* (%)	126 (78.2)	36 (69.2)	90 (82.6)	0.06
Type A, *n* (%)[a]	84 (52.2)	30 (58.8)	54 (49.5)	0.27
Type B, *n* (%)[a]	76 (47.2)	21 (41.2)	55 (50.5)	0.27
White, *n* (%)	115 (71.4)	36 (69.2)	81 (74.3)	0.31
Hypertension, *n* (%)	124 (77.0)	38 (73.1)	86 (78.8)	0.48
Smoking, *n* (%)	69 (42.9)	25 (55.6)	44 (45.4)	0.26
Diabetes, *n* (%)	7 (4.3)	2 (3.9)	5 (3.1)	0.84

[a]One patient had a traumatic aortic dissection and therefore was not classified as a type A or type B dissection.
SD: standard deviation.

Invasive angiography and pressure measurements

All patients included in this analysis were evaluated with invasive angiography performed by an expert interventional radiologist (David M. Williams) at our institution using a standardized protocol. The indication for invasive angiography was suspicion of visceral or lower extremity malperfusion based on clinical characteristics and imaging characteristics. All invasive measurements were performed prior to any surgical or endovascular intervention. Angiographic evaluation for malperfusion was performed using intravascular ultrasound. Intravascular ultrasound examination was performed along the length of the aorta from the ascending aorta to the external iliac arteries to determine the anatomy of the dissection flap in relation to the branch vessels. Due to the modest prevalence of concurrent suspected and unsuspected malperfusion in different territories, pressure measurements were performed across superior mesenteric, bilateral renal and bilateral external iliac arteries in all patients regardless of suspected vascular territory, comparing blood pressure at the aortic root with simultaneous pressures in the branch arteries. In instances of renal artery dissection with a re-entry tear (thus kidney perfusion through both lumens), we excluded these pressure measurements from the analysis. In literature significant gradients for malperfusion are defined between 10 and 20 mmHg [23–25]. In our institution, we define visceral malperfusion as an aorto-branch artery gradient of >15 mmHg by manometry as previously described [16, 22, 26].

Table 2: Diagnostic variables

	Parameter	Total	Renal malperfusion (*N* = 52)	No renal malperfusion (*N* = 109)	*P*-Value
Renal artery pressures	SBP kidney supplied by true lumen, median (IQR)	91 (70–104)	84 (52–103)	92 (80–104)	**0.02**
	DBP kidney supplied by true lumen, median (IQR)	49 (42–61)	49 (35–55)	50 (42–56)	0.61
	SBP kidney supplied by false lumen, mean ± SD	89 ± 22	88 ± 25	89 ± 20	0.84
	DBP kidney supplied by false lumen, mean ± SD	49 ± 8	49 ± 9	49 ± 7	0.85
CTA parameters	Renal artery dissected, *n* (%)	39 (24)	16 (31)	23 (21)	0.12
	Global enhancement defect, *n* (%)	81 (50)	35 (67)	46 (42)	***<0.01***
	Focal enhancement defect, *n* (%)	12 (7.5)	8 (15)	4 (4)	***<0.01***
	True lumen renal cortex enhancement (HU), median (IQR)	157.0 (110.9–196.2)	113.8 (70.1–167.2)	161.7 (121.4–201.0)	***<0.01***
	False lumen renal cortex enhancement (HU), median (IQR)	144.3 (97.5–179.1)	148.5 (125.5–179.3)	142.2 (86.5–180.0)	0.35
Laboratory values	eGFR, median (IQR)	56.5± 26.8	50.5 (39.3–59.3)	56.0 (41.8–78.0)	0.08
	Creatinine (mg/dl), median (IQR)	1.47± 1.17	1.4 (1.3–1.9)	1.4 (1.0–1.9)	0.17
	Blood urea nitrogen (mmol/l), median (IQR)	23.0± 11.9	21.0 (17.8–27.3)	22 0.0 (18.0–28.3)	0.40
	Lactate (mmol/l), median (IQR)	1.6 (1.0–2.8)	1.5(1.0–2.9)	1.5 (1.1–2.5)	0.72

Pressures are in mmHg.

CTA: computed tomography angiography; DBP: diastolic blood pressure; eGFR: estimated glomerular filtration rate; HU: Hounsfield units; IQR: interquartile range; SBP: systolic blood pressure; SD: standard devaiation. Bold = *p*-value <0.05.

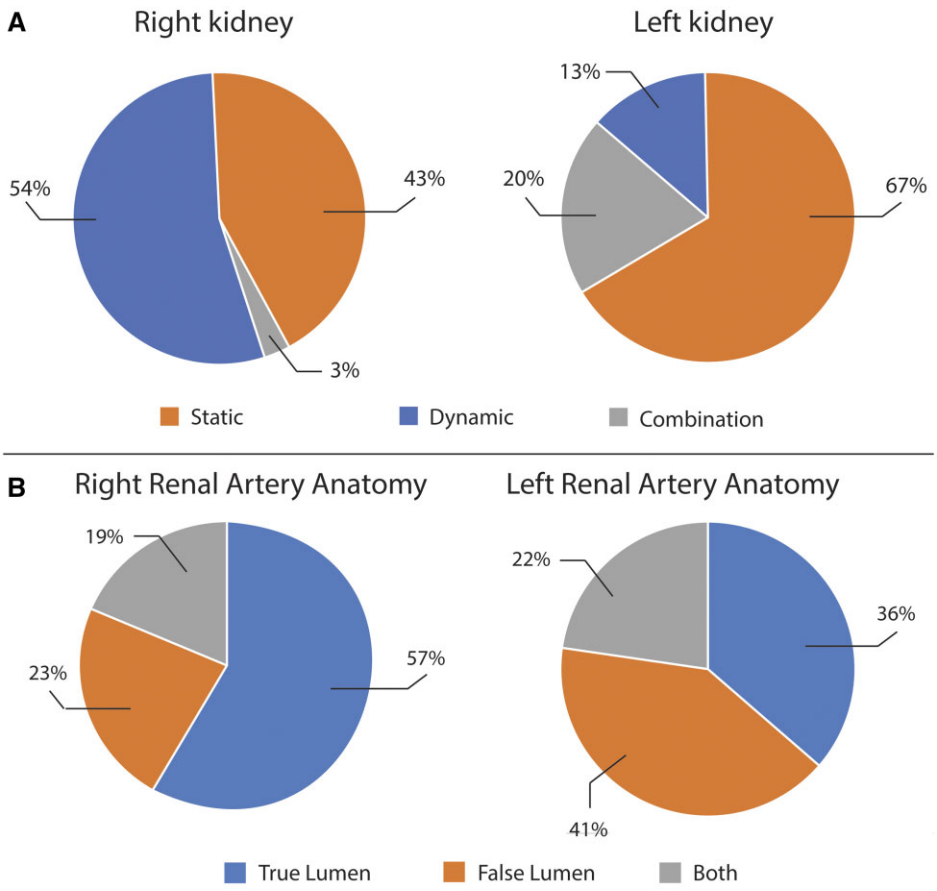

Figure 3: (**A**) Occlusion type by kidney. (**B**) Luminal supply of renal arteries.

Statistical analysis

Categorical variables were analysed using univariate analysis, which included use of Chi-square tests or Fisher's exact test as appropriate. Continuous variables are expressed as mean (SD) or median with interquartile range depending on their normality. Kolmogorov–Smirnov test was used to determine if the variable had a normal distribution. Continuous variables were analysed using Student's *T*-test or Mann–Whitney *U*-test as applicable. The sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) of asymmetric renal enhancement were calculated by using a 2 × 2 contingency table. To identify independent predictors for renal malperfusion, a multivariable logistic regression was performed in which independent variables were selected for the model based on a priori background knowledge. All variables that were included had a biologically plausible and independent relationship with the dependent variable of invasive renal malperfusion. A *P*-value of <0.05 was considered statistically significant. Data analysis was performed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA).

RESULTS

Patient characteristics

After the application of the inclusion and exclusion criteria, 161 patients were deemed appropriate for analysis. A total of 26 patients were excluded from analysis due to incomplete clinical data ($n = 9$) or the absence of invasive angiography measurements ($n = 17$) (Fig. 1). The mean age of the study cohort was 57.2 ± 13.3 years and the majority of patients were male (78.2%, 126/161). Over 75% of patients had a history of hypertension at baseline (77.0%, 124/161) and 42.9% (69/161) had a history of smoking. Type A dissection was diagnosed in 52.2% of cases (84/161) and type B dissection in 47.2% (76/161). There were no statistically significant differences in patient demographics and patient-history between patients with renal malperfusion and patients without renal malperfusion. Patient demographics are found in Table 1.

Radiologic and laboratory parameters at baseline

CTA measurements showed a good agreement between the 2 raters for the quantitative assessment of renal cortical attenuation [intraclass correlation coefficient: 0.88, 95% confidence interval (CI) 0.74–0.95] (Supplementary Material, Table S1) [27]. Qualitative assessment of asymmetric renal enhancement showed an excellent interobserver agreement with a Cohen's kappa of 0.85. Radiologic and laboratory parameters are found in Table 2. The median time between CTA imaging and invasive angiography was 11 h (IQR 7.0–32.5), with no differences between patients with renal malperfusion and those without renal malperfusion (9.0 h, IQR 6.0–35.0 and 11.0 h, IQR 7.0–30.5, $P = 0.30$, respectively). There was no difference in median time between the onset of symptoms related to aortic dissection and the invasive angiography between patients with renal malperfusion and those without renal malperfusion (16 h, IQR 10.0–55.5 and 16 h, IQR 10.3–40.5, $P = 0.86$, respectively).

The prevalence of renal artery dissection was 24.2% (39/161) of patients, with no significant difference between patients with renal malperfusion and without renal malperfusion [30.7% (16/52) vs 21.1% (23/109), $P = 0.12$]. The TL provided exclusive arterial supply to the right kidney in 57.2% of the cases and to the left kidney in 35.9% of the cases (Fig. 3). Dynamic obstruction occurred in 54.3% (19/35) of obstruction in the right kidney, as opposed to 13.3% (4/30) of obstructions in the left kidney (Fig. 3).

Global enhancement asymmetry was found in half the patients (50.3%, 81/161) but was found more often in patients with renal malperfusion than those without renal malperfusion [67.3% (35/52) vs 42.2% (46/109), $P < 0.01$]. Focal enhancement defects were found in 7.5% of patients (12/161), with a higher prevalence in patients with renal malperfusion than without renal malperfusion [15.4% (8/52) vs 3.6% (4/109), $P < 0.01$].

Patients with renal malperfusion had lower renal cortex enhancement when supplied by the TL than those without renal malperfusion [113.8 HU (IQR 70.1–167.2) vs 161.7 HU (IQR 121.4–201.0), $P < 0.01$]. No differences were observed in renal cortex enhancement when the kidney was supplied by the FL [148.5 HU (IQR 125.5–179.3) vs 142.2 HU (IQR 86.5–180.0), $P = 0.35$]. Asymmetric renal enhancement (visual assessment) at CTA had a sensitivity of 65% and a specificity of 58% for renal malperfusion, and the PPV of asymmetric renal enhancement was 76% and the NPV 45% for renal malperfusion, when assuming the prevalence of 67% for renal malperfusion among patients suspected clinically of visceral malperfusion (Table 3) [22].

The median baseline estimated glomerular filtration rate was similar between patients with renal malperfusion versus patients with no renal malperfusion [50.5 (IQR 39.3–59.3) vs 56.0 (IQR 41.8–78.0), $P = 0.08$]. Baseline creatinine levels were not significantly different between the 2 groups [1.4 mmol/l (IQR 1.3–1.9) vs 1.4 mmol/l (IQR 1.0–1.9), $P = 0.17$]. BUN was not different for patients with renal malperfusion as opposed to no renal malperfusion [21.0 mmol/l (IQR 17.8–27.3) vs 22.0 mmol/l (IQR 18.0–28.3), $P = 0.40$]. There was no difference between the lactate levels of the renal malperfusion group and the no renal malperfusion group [1.5 mmol/l (IQR 1.0–2.9) vs 1.5 mmol/l (1.1–2.5), $P = 0.72$] (Table 2).

Table 3: 2 × 2 contingency table for analysis of sensitivity and specificity of renal enhancement with renal malperfusion

	Renal Malperfusion	No renal Malperfusion	
Asymmetric enhancement	48 (TP)	76 (FP)	$N = 124$
Symmetric enhancement	26 (FN)	103 (TN)	$N = 129$
	$N = 74$	$N = 179$	
Sensitivity: 65%, specificity: 58			
Positive predictive value[a]: 76%			
Negative predictive value[a]: 45%			

[a]Assumes renal malperfusion prevalence of 67% in aortic dissection patients [22].
TP: true positive; FP: false positive; FN: false negative; TN: true negative.

Invasive angiography measurements

The median systolic blood pressure in the renal artery that was supplied by the TL was lower in patients with renal malperfusion than those without renal malperfusion [84 mmHg (IQR 52–103) vs 92 mmHg (IQR 80–104), $P = 0.02$]. The median diastolic blood pressure in the renal artery that was supplied by the TL was not

Table 4: Results of multivariate regression analysis for predictors of renal malperfusion

Variables	Odds ratio	95% confidence interval	*P*-Value
Age	0.95	0.91–0.99	**0.04**
Sex (female)	3.44	0.86–13.86	0.08
Hypertension	1.02	0.19–5.56	0.98
Blood urea nitrogen at presentation	0.98	0.93–1.05	0.61
eGFR at presentation	0.98	0.95–1.01	0.21
Lactate at presentation	0.87	0.63–1.20	0.39
Dissected renal artery	2.41	0.52–11.23	0.26
True lumen supply	0.95	0.51–1.75	0.86
Global enhancement defect	4.43	1.20–16.41	**0.03**
Focal enhancement defect	11.23	1.12–112.90	**0.04**

eGFR: estimated glomerular filtration rate. Bold = *p*-value <0.05.

different between the 2 groups [49 mmHg (IQR 35–55) vs 50 mmHg (IQR 42–56), $P = 0.61$]. Systolic and diastolic pressure in the renal artery that was supplied by the FL was not different between patients with renal malperfusion and those without renal malperfusion [88 (25) vs 89 (20) mmHg, $P = 0.84$ and 49 (9) vs 49 (7) mmHg, $P = 0.85$, respectively]. Invasive measurements are found in Table 2.

Multivariable model

A multivariable model identified global enhancement defect (OR 4.43, 95% CI 1.20–16.41, $P = 0.03$) and focal enhancement defect (OR 11.23, 95% CI 1.12–112.90, $P = 0.04$) as predictive for renal malperfusion. Age was protective for renal malperfusion (OR 0.95, 95% CI: 0.91–0.99, $P = 0.04$) (Table 4).

DISCUSSION

The present study is unique in that we were able to define renal malperfusion using invasive catheterization in a relatively large cohort of patients ($n = 161$) suspected of having visceral malperfusion based on clinical and radiologic findings at presentation. Furthermore, we were able to examine differences in baseline clinical and imaging characteristics between patients with and without renal malperfusion and investigate baseline characteristics that independently predicted renal malperfusion. Key findings from this study include: (i) visible asymmetry of the kidneys, both global and focal, was independently predictive of renal malperfusion when controlling for covariates such as renal artery dissection and baseline GFR and serum lactate, (ii) 20% of patients who had symmetric renal enhancement were still found to have renal malperfusion confirmed by invasive angiography, (iii) over half of patients (61%) with asymmetric enhancement did not have renal malperfusion confirmed by invasive angiography, (iv) differential renal enhancement at CTA had a sensitivity of 65%, a specificity of 58%, a PPV of 76% and an NPV of 43% among patients suspected of having renal malperfusion.

In this study, over 80% of patients with confirmed renal malperfusion at the time of angiography showed either focal or global lower enhancement of the kidney on CTA. Conversely, over half of patients (53%) with either a focal or global decreased enhancement did not have renal malperfusion at the time of angiography. This lack of strong concordance between imaging characteristics and renal malperfusion at invasive assessment has been described previously. For example, Barnes *et al.* [26] found that invasive angiography confirmed 67% of suspected renal malperfusion. However, their study (1996–2004) occurred in an era of significantly older CT technology and not surprisingly imaging characteristics were only reported as reason for suspected malperfusion in 8% of patients [23, 26]. With the emergence of CT scanners capable of fast, high-resolution, whole-aorta imaging, the value of renal imaging characteristics has been increasingly investigated, yet the relationship between renal CT enhancement and changes in renal perfusion pressure has not been well-defined in prior studies. One explanation for why visual asymmetric enhancement of the kidneys is often not confirmed at invasive angiography could be simply related to differences in transit time of the contrast bolus to the FL-perfused kidney [26, 28, 29]. A representative case demonstrating transient differences in luminal and renal enhancement related to contrast bolus timing is shown in Fig. 4. Another explanation could be found in the phenomenon of dynamic branch artery obstruction, which is a time-variable process that may either develop or resolve between the time of CT imaging and invasive assessment. Furthermore, dynamic obstruction may be underappreciated by CTA, given that the dynamic nature of the dissection flap is not well appreciated on standard, static CTA images. Lastly, CTA is often conducted near the time of presentation when patients are more likely to be hypertensive, whereas invasive measurements are typically performed after some delay (mean of 11 h later in our study) once the patient's blood pressure has been pharmacologically controlled and dynamic obstruction alleviated. Our observation that over 40% of patients with asymmetric renal enhancement on CT were not found to have renal malperfusion at invasive assessment may reflect the ability of prompt and aggressive antihypertensive medical management, as practiced at many centres, to alleviate dynamic obstruction. Interestingly, ~1 in 5 patients with symmetric renal enhancement were found to have renal malperfusion at invasive angiography. This observation highlights the importance of close monitoring of patients with aortic dissections and emphasizes the need for a low threshold for angiographic evaluation for any clinical evidence of developing malperfusion. Renal duplex ultrasound has been investigated and showed promising results in patients with aortic dissection and suspicion of renal malperfusion, due to the dynamic nature of the imaging [30]. Patients with renal malperfusion have a modest concurrence of malperfusion in other

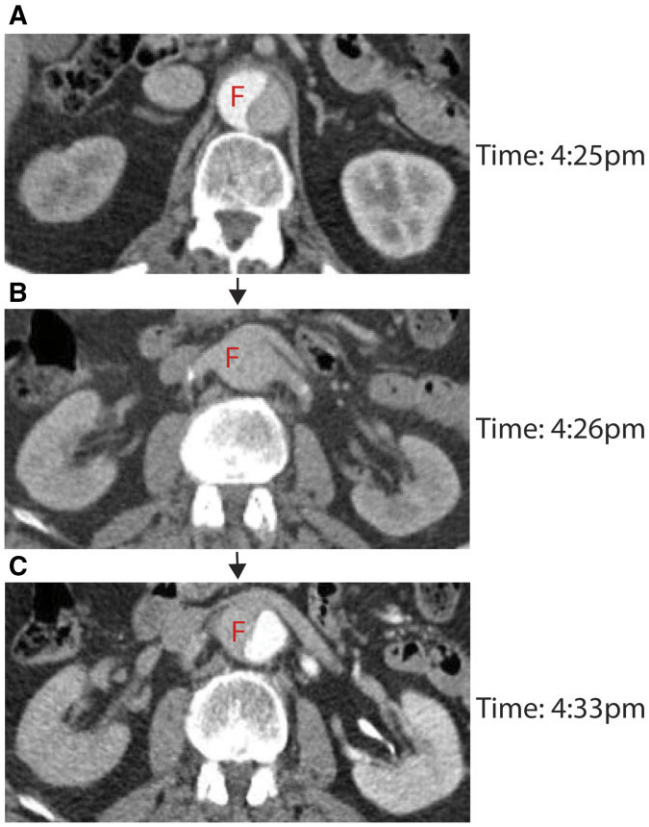

Figure 4: Representative example of dynamic changes in luminal enhancement due to contrast transit time in acute aortic dissection. (**A**) On initial angiographic images (time = 4:25 pm) the false lumen shows higher enhancement than the true lumen. (**B**) Approximately 1 min later (4:26 pm) enhancement of both lumens equilibrates to a low level. (**C**) Subsequently, the patient received another bolus of intravenous contrast and on repeat scan 7 min later (4:33 pm) the luminal enhancement pattern is reversed with the true lumen demonstrating higher enhancement. The patient underwent repair of his type A dissection, and on CT 9 years later, there was no renal atrophy. F: false lumen.

territories, and this should be kept in mind in judging the role and conclusiveness of renal duplex.

Interestingly, the rate of renal artery dissection was not significantly higher in patients with renal malperfusion, and renal artery dissection was not a significant predictor of malperfusion in our multivariable logistic regression. This observation may in part be explained by limited statistical power given the relatively low incidence of renal artery dissection (24%) in our population. Lastly, we found increasing age to have a mild protective effect on the risk of renal malperfusion. While the mechanism of this effect is not entirely clear, its postulated that a decreased flap mobility related to atherosclerosis and intimal calcification may lessen the risk of dynamic branch artery obstruction.

Limitations

Limitations of this study include the retrospective nature, with inability to capture all potential variables of interest that may have influence on renal malperfusion (e.g. timing and type of pharmacologic interventions, dynamic changes in blood pressure) from the electronic medical records. Also, visual assessment and quantitative assessment of the kidneys was performed by 2 independent observers, which could lead to interobserver variability. In addition, not all CTA images in our analysis were acquired at our centre, and thus we did not have control over the specific CT acquisition parameters, including those related to the timing of contrast bolus administration. This is due to the fact that our hospital is a referral centre for aortic dissection patients and thus many patients are transferred to our hospital with diagnostic CTAs already performed at an outside facility. Finally, there is an unavoidable delay between the time of the diagnostic CT acquisition and the time of invasive angiography, averaging 11 h in our study, during which time changes in the patients' blood pressure, pharmacologic management and overall clinical status could affect the renal perfusion pressures. Similarly, measurements of creatinine and BUN are often made from blood drawn at admission, 60–90 min from symptom onset, and may not reflect ongoing renal artery compromise. We recognize these as limitations, however, we also believe that these delays reflect the clinical reality of making treatment decisions based on clinical assessment and initial diagnostic imaging studies, especially when that study is performed at an outside institution. Finally, we recognize there are other CT findings that contribute to the impression of arterial compromise, such as complete TL collapse in the visceral segment of the abdominal aorta; while beyond the scope of this study, we plan to investigate such anatomic characteristics in future work.

CONCLUSION

This unique database with patients with acute aortic dissection and suspicion for renal malperfusion, combined with findings from CTA imaging and selective renal manometry and arteriography, allows for better understanding of the associations between imaging features and the presence of renal malperfusion in patients with aortic dissection. Differential enhancement of the kidney, seen commonly on CTA, is predictive but not prescriptive of renal malperfusion. Asymmetric renal enhancement at CTA can appropriately raise the suspicion of renal malperfusion; however, given the substantial discordance we observed between the imaging appearance of the kidneys and invasive renal artery pressure measurements, our results emphasize that accurate diagnosis and prompt management of renal malperfusion requires close patient monitoring and often invasive assessment, which can be combined with endovascular treatment of the malperfusion.

SUPPLEMENTARY MATERIAL

Supplementary material is available at *EJCTS* online.

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The data underlying this article will be shared on reasonable request to the corresponding author.

Author contributions

Pieter A.J. van Bakel: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Writing—original draft; Writing—review & editing. **Matthew Henry:** Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Validation; Visualization; Writing—original draft; Writing—review & editing. **Karen M. Kim:** Conceptualization; Supervision; Writing—review & editing. Bo Yang: Conceptualization; Supervision; Writing—review & editing. **Joost A. van Herwaarden:** Conceptualization; Supervision; Writing—review & editing. **C. Alberto Figueroa:** Conceptualization; Supervision; Writing—review & editing. **Himanshu J. Patel:** Conceptualization; Supervision; Writing—review & editing. **David M. Williams:** Conceptualization; Data curation; Investigation; Project administration; Resources; Supervision; Writing—review & editing. **Nicholas S. Burris:** Conceptualization; Data curation; Formal analysis; Funding acquisition; Investigation; Methodology; Project administration; Resources; Supervision; Validation; Writing—original draft; Writing—review & editing.

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