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Research paper

Renal artery involvement in acute aortic dissection: Prevalence and impact on renal atrophy in non-interventional treatment patients[☆]Chun-Chieh Wang^{a,b}, Huey-Shyan Lin^c, Yi-Luan Huang^{a,b}, Fu-Zong Wu^{a,b}, Chiung-Chen Chuo^a, Yu-Jeng Ju^d, Carol C. Wu^e, Ming-Ting Wu^{a,b,*}^a Department of Radiology, Kaohsiung Veterans General Hospital, No. 386, Ta-Chung 1st Road, Kaohsiung, 813, Taiwan^b Faculty of Medicine, School of Medicine, National Yang Ming University, Taipei, Taiwan^c Program of Health-Business Administration, School of Nursing, Fooyin University, Kaohsiung, Taiwan^d Department of Psychology, National Taiwan University, Taipei, Taiwan^e Department of Diagnostic Radiology, University of Texas MD Anderson Cancer Center, Houston, TX, USA

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ABSTRACT

Background: To evaluate the frequency of renal artery dissection (RAD) and renal hypoperfusion in aortic dissection (AD) and its effect on subsequent renal atrophy in patients who did not undergo therapeutic intervention. **Methods:** Initial CT data of 155 patients with acute AD (Stanford type A = 88, B = 67) were retrospectively analyzed. The false lumen statuses were patent (n = 94), partially thrombosed (n = 25), and completely thrombosed (n = 36) (also called as intramural hematoma (IMH)). Follow-up CT images of the surviving 122 patients (6–62.6 months, median, 28.9 months) were reviewed for analysis of sequential changes in renal volume. A regional decrease of ≥ 20 Hounsfield units in the renal cortex was defined as a renal hypo-enhancement sign (RHS). Simplified CT estimations of renal volume and estimated glomerular filtration rates (eGFR) were calculated. The generalized estimating equations (GEE) method was used to predict renal atrophy.

Results: Fifty of the 122 patients presented with 59 RAD in the current study, and a positive RHS was noted in 33.9% (20/59) of these involved kidneys. GEE analysis showed hypertension, surgical treatment for AD, presence of RAD, and positive RHS as significant risk factors for renal atrophy. Patients with RHS had the most severe form of renal atrophy. The severity of renal atrophy was mildly correlated with GFR change ($\gamma^2 = 0.044$, $p < 0.001$).

Conclusion: Renal atrophy in AD was predicted by the CT findings of RAD and RHS. The severity of renal atrophy was weakly reflected by eGFR.

1. Introduction

Malperfusion syndrome occurs in approximately 18%–50% cases of aortic dissection (AD), and can include myocardial, visceral, renal, cerebral, or limb ischemia.^{1–3} Renal malperfusion, secondary to AD, is associated with high surgical mortality and is a significant independent predictor of operative mortality.^{2,4,5} Previous studies have addressed the hemodynamic impact of renal artery dissection (RAD) morphology, which serves as a treatment guide for renal malperfusion.^{6,7} With recent advances in endovascular stenting, the minimal invasive treatment of RAD is now possible. However, the criteria for renal artery stenting in AD remains controversial.^{8,9} Therefore, an observational study on the

natural course of untreated RAD and its effects on renal function and renal volume in AD survivors could potentially be used to guide renal artery intervention.

Multidetector computed tomography (MDCT) provides high-resolution morphological details in AD,¹⁰ thereby yielding favorable visualization of the relationship between the intimal flap and the branch arteries. In the present study, we used MDCT to determine the frequency of renal hypoperfusion in AD and to evaluate its effect on subsequent renal atrophy in patients who did not undergo therapeutic intervention.

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Institutional Review Board approval was obtained. Written informed consent was waived by the Institutional Review Board.

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Abbreviations and acronyms

AD	Aortic dissection
eGFR	Estimated glomerular filtration rate
EVAR	Endovascular aneurysm repair
GEE	Generalized estimating equation

H	Hounsfield unit
IMH	Intramural hematoma, also named "complete thrombosis of false lumen"
MDCT	Multi-detector computed tomography
RAD	Renal artery dissection
RHS	Renal hypoenhancement sign

2. Methods**2.1. Patients**

We retrospectively analyzed the MDCT data of 177 consecutive patients diagnosed with acute AD (including those with patent, partial thrombosed, and completely thrombosed false lumens), presenting within 14 days of symptom onset, from August 2009 to November 2014. Clinical information including surgical history (endovascular aneurysm repair [EVAR]), history of hypertension and diabetes mellitus, creatinine level (mg/dL), and clinical outcomes were obtained from their medical records. Estimated glomerular filtration rate (eGFR) (mL/min/1.73 m²) came from estimation by the Modification of Diet in Renal Disease formula.¹¹ The current study was approved by the institutional review board. The requirement to obtain informed consent from the patient was waived, and all authors had full control of the data and information in this study.

All patients with baseline CTs were analyzed. The following exclusion criteria were employed: initial renal atrophy due to end-stage renal disease or previous renal insult; history of nephrectomy; and a history of polycystic kidney disease. As a result, 22 patients (12.4%) were excluded, and the remaining 155 patients were enrolled in this study. AD was classified as Stanford type A (n = 88) or B (n = 67), and the status of the false lumen was classified as patent (n = 94), partially thrombosed (n = 25), and completely thrombosed (also called as intramural hematoma [IMH]; n = 36).¹²⁻¹⁴ The in-hospital mortality rate was 21.3% (n = 33, Type A/B = 22/11, EVAR = 20). The surviving 122 patients were followed up using contrast-enhanced MDCT during the subacute stage when required, followed by subsequent annual evaluations (6–62.6 months, median, 28.9 months, mean, 35 ± 23.9 months); the patients were included for analysis of renal atrophy. The

patient population is as summarized in the flow chart in Fig. 1.

2.2. Image acquisition

All initial and follow-up CT examinations were performed on a 64-detector row scanner (Aquilion 64, Toshiba Medical Systems, Tokyo, Japan). Each examination included non-enhanced and contrast-enhanced images. Nonionic contrast medium (350 mg/mL Iopromide; Bayer, Berlin, Germany) was administered with a bolus injection (rate 3–4 mL/s; volume, 80–100 mL) using a power injector (Stellant D; Medrad, Indianola, PA). Scanning was initiated using a bolus-tracking technique (threshold, 150 HU in the ascending aorta). The scanning parameters were as follows: 100–120 kVp, 140–200 mAs (effective), and reconstruction at a section thickness of 2 mm with an increment of 1.5 mm for both unenhanced and contrast-enhanced images. The scanning ranged from the thoracic inlet to the diaphragm for the unenhanced scans and from the lower neck to the pubic symphysis for the contrast-enhanced scans. Electrocardiographic gating was used for follow-up CT in patients with Stanford type A AD.

2.3. Image evaluation

All CT images were independently evaluated by 2 radiologists (C.C.W. and Y.L.H., with 6 and 9 years of experience in thoracic radiology, respectively) for category analysis, such as dissection type and renal cortical enhancement. Interpretation disagreements were resolved by consensus. All quantitative measurements, such as renal size, were performed by 1 radiologist (C.C.W.) using visual inspection.

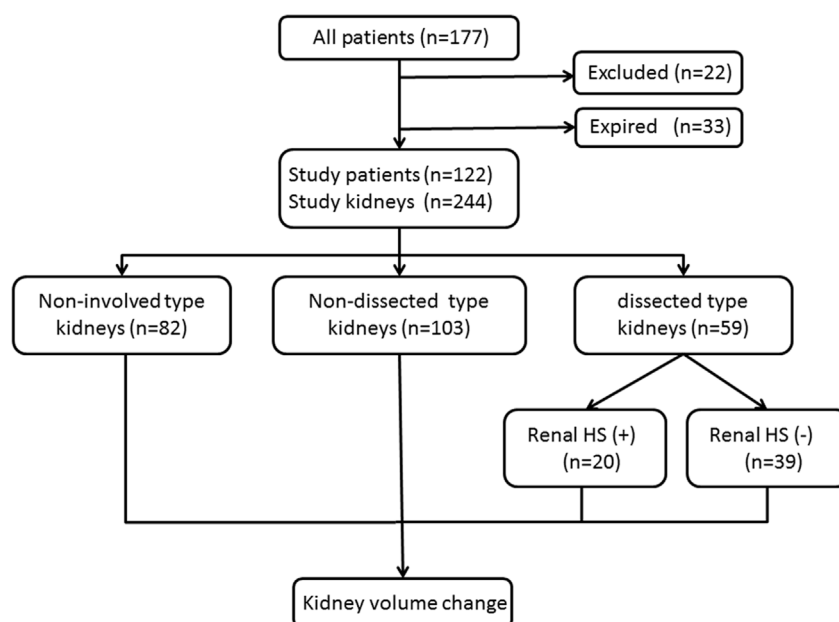


Fig. 1. A branch diagram of all patients with aortic dissection.

Branch diagram summarizing the conditions of the 155 patients with aortic dissection in this study. RHS: renal hypoenhancement sign.

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