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ORIGINAL ARTICLE

# The Relationship Between Renal Artery Involvement in Stanford B-Type Aortic Dissection and the Short-Term Prognosis: A Single-Centre Retrospective Cohort Study

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**Background** Renal artery involvement has not received sufficient attention despite the fact that aortic computed tomography angiography (CTA) examinations of Stanford B-type aortic dissection patients usually show renal artery involvement [3]. To study the influence of renal artery involvement on aortic dissection, we performed a retrospective study on acute Stanford B-type aortic dissection patients with or without renal artery involvement to investigate its effect on the prognosis of aortic dissection.

**Methods** A total of 221 patients with acute Stanford-B type aortic dissection between January 2012 and January 2014 were enrolled. The patients were divided into a renal artery involvement group and a non-renal artery involvement group based on aortic computed tomography angiography (CTA) results. The clinical data of the two groups were compared. Univariate analyses and multiple logistic regression analyses were performed to determine risk factors related to in-hospital mortality. The effect of renal artery involvement on the prognosis of Stanford B-type aortic dissection patients was analysed.

**Results** Among the 221 patients with acute Stanford type-B aortic dissection, 100 patients (45.2%) exhibited renal artery involvement. The percentage of patients with a past history of hypertension in the renal artery involvement group was significantly higher than that in the non-renal artery involvement group (84.0% vs. 74.8%, $p = 0.025$). The estimated glomerular filtration rate (eGFR), creatinine level, and urea nitrogen level at admission were not significantly different between the renal artery involvement group and the non-renal artery involvement group. The in-hospital mortality rate in the renal artery involvement group was higher than that in the non-renal artery involvement group; the difference in the percentage of in-hospital mortality between these two groups was statistically significant (12.0% vs. 4.1%, $p < 0.05$). The results of multiple logistic regression analysis showed that renal artery involvement was a risk factor for in-hospital mortality in acute Stanford B-type aortic dissection patients (odds ratio (OR) = 3.536 (1.127 ~ 11.095)). In the renal artery involvement group, the in-hospital mortality rate in the conservative treatment group was significantly higher than that in the interventional treatment group (30.8% vs. 5.4%, $p = 0.001$).

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**Conclusions** Although renal artery involvement was not associated with short-term renal function damage, it was a risk factor for in-hospital mortality after acute Stanford B-type aortic dissection.

**Keywords** Aortic dissection • Renal artery • In-Hospital mortality • Hypertension

# Introduction

Aortic dissection is a very dangerous aortic disease commonly observed in clinical settings. It is also a cardiovascular emergency with one of the highest mortality rates. Determining possible risk factors at the early stage, providing prognostic information, and conducting timely intervention may effectively reduce the risk of death due to aortic dissection. Previous studies [1,2] have suggested that visceral ischaemia, especially involvement of the superior mesenteric artery and celiac artery, is an important factor that affects mortality in aortic dissection patients. Renal artery involvement has not received sufficient attention despite the fact that aortic computed tomography angiography (CTA) examinations of Stanford B-type aortic dissection patients usually show renal artery involvement [3]. To study the influence of renal artery involvement on aortic dissection, we performed a retrospective study on acute Stanford B-type aortic dissection patients with or without renal artery involvement to investigate its effect on the prognosis of aortic dissection.

# Methods

## Study Subjects

A total of 221 cases of aortic dissection patients admitted and treated between January 2012 and January 2014 were retrospectively analysed. The inclusion criteria consisted of (1) Stanford B-type aortic dissection confirmed by aortic CTA or aortic angiography and (2) acute aortic dissection patients admitted to the hospital for treatment. The exclusion criteria consisted of (1) patients who did not receive aortic CTA, (2) patients with Stanford A-type aortic dissection, and (3) patients with chronic Stanford B-type aortic dissection.

## Study Content

### Analysis of the General Conditions of Patients

The aortic dissection type; presence of hypertension, diabetes mellitus, or atherosclerosis; past surgical history (percutaneous coronary intervention [PCI], coronary artery bypass grafting [CABG], etc.); laboratory related indicators (white blood cell, creatinine, uric acid, and urea nitrogen levels at admission); treatment methods (surgical treatment or conservative medication treatment); and complications and mortality during hospitalisation were analysed.

### Criteria of Renal Artery Involvement [4]

The renal artery was considered to be involved if it completely emerged from the false lumen and the blood supply was independently provided by the false lumen, or if the intimal flaps of the renal artery were torn and formed true and false lumen blood supplies. Patients were divided into a renal artery involvement group and a non-renal artery involvement group based on whether the false lumen of the aortic dissection exhibited renal artery involvement according to the aortic CTA results of patients.

## Statistical Analysis

Statistical analyses of all data were performed using the statistical software SPSS 21.0. (IBM, Armonk, NY, USA). A two-tailed value of $p < 0.05$ was defined as statistically significant. Measurement data that followed a normal distribution are presented as means ± standard deviation. Intragroup comparisons were performed using a t-test. Count data are presented as frequencies and percentages, and intragroup comparisons were performed using the c2 test. For risk factor analysis, univariate analysis was first performed for screening; next, variables with statistical significance were entered into the multiple logistic regression analysis. The odds ratio (OR) and 95% confidence intervals were calculated.

# Results

## Comparison of Baseline Data

Among the 221 patients, 100 patients exhibited renal artery involvement, which accounted for 45.2% of the participants. An analysis of factors related to renal artery involvement showed that the renal artery involvement group had a higher percentage of individuals with a past hypertension history (84.0% vs. 74.8%, $p = 0.025$), past aortic dissection (7.0% vs. 0%, $p = 0.003$), and past aortic dissection repair (6.0% vs. 0%, $p = 0.007$). The other clinical data were not significantly different between these two groups (Table 1).

## Comparison of Clinical Characteristics

The percentage of patients with hypertension at admission in the renal artery involvement group was significantly higher than that in the non-renal artery involvement group (72% vs. 56.2%, $p = 0.012$). The diastolic pressure at admission in the renal artery involvement group was significantly higher than that in the non-renal artery involvement group (91.0 ± 20.2 vs. 85.7 ± 18.4, $p = 0.042$). However, indicators such as systolic pressure at admission, heart rate at admission, white blood cell count, creatinine level, estimated glomerular filtration rate (eGFR), uric acid level, and urea nitrogen level were not significantly different between these two groups ($p > 0.05$) (Table 2).

**Table 1** General Characteristics of Participants According to Renal Artery Involvement.

| | Group A non-renal artery involvement | Group B renal artery involvement | $t/\chi^2$ | P |
|---|---|---|---|---|
| Total Patients (n) | 121 | 100 | | |
| Demographics | | | | |
| Age | 51.38 ± 13.13 | 49.11 ± 11.164 | 1.368 | 0.173 |
| Female (%) | 88(72.7) | 83(83.0) | 3.301 | 0.069 |
| Cardiovascular Risk Factors (%) | | | | |
| Hypertension | 85(70.8) | 84(84.0) | 5.310 | 0.025* |
| Diabetes | 18.(15.1) | 14(14.0) | 0.030 | 0.862 |
| Smoking | 58(50.9) | 56(56.0) | 1.284 | 0.257 |
| Atherosclerosis | 28(23.1) | 21(21.0) | 0.117 | 0.732 |
| Cardiovascular History (%) | | | | |
| Marfan Syndrome | 0 | 1(1.0) | 1.228 | 0.268 |
| Prior Aortic Dissection | 0 | 7(7.0) | 8.534 | 0.003* |
| Prior Aortic Aneurysm | 0 | 2(2.0) | 2.382 | 0.123 |
| Aortic Repair | 0 | 6(6.0) | 7.280 | 0.007* |
| PCI | 3(2.5) | 5(5.0) | 0.997 | 0.318 |
| CABG | 0 | 1(1.0) | 1.216 | 0.270 |
| Valve Replacement | 2(1.7) | 2(2.0) | 0.037 | 0.847 |

Abbreviations: CABG, coronary artery bypass graft; PCI, percutaneous coronary intervention.
*Statistically significant.

## Comparison of the Development of Complications During Hospitalisation Between the Two Groups

Among the 221 patients, 17 patients (7.7%) died during hospitalisation; 12 patients died in the renal involvement group, and five patients died in the non-renal artery involvement group. The mortality during hospitalisation in the renal artery involvement group was higher than that in the non-renal artery involvement group (12.0% vs. 4.1%, $p = 0.029$). The percentages of patients with shock (9.0% vs. 0.8%, $p = 0.004$) and aortic rupture (11.0% vs. 3.3%, $p = 0.024$) in the renal artery involvement group were significantly higher than those in the non-renal artery involvement group. During hospitalisation, the incidences of dialysis, stroke, and acute myocardial infarction were not significantly different between these two groups ($p > 0.05$) (Table 3). The results of multiple logistic regression analysis showed that renal artery involvement, arteriosclerosis, and conservative treatment were risk factors for in-hospital mortality in acute Stanford B-type aortic dissection patients (Table 4).

## Comparison of Treatment Programs Within the Renal Artery Involvement Group

A statistical analysis of the conditions of patients with regard to in-hospital mortality showed that the percentage of patients who received conservative treatment (drug treatment only) in the mortality group was significantly higher than that in the survival group (58.8% vs. 20.6%, $p < 0.001$); the difference was statistically significant. In addition, conservative treatment was a risk factor for in-hospital mortality in acute Stanford B-type aortic dissection patients. In the renal artery involvement group, a comparison between patients who received conservative treatment and patients who received non-conservative treatment showed that the in-hospital mortality rate in the conservative treatment group was significantly higher than that in the non-conservative treatment group (30.8% vs. 5.4%, $p = 0.001$), and this difference was also significant between the two groups. These results indicated that, for Stanford B-type aortic dissection patients with renal artery involvement, the risk of in-hospital mortality in the conservative treatment group was higher than that in the non-conservative group.

# Discussion

This study showed that patients with past hypertension had a higher probability of exhibiting renal artery involvement during the development of aortic dissection. The onset of aortic dissection is an acute process; however, dissection results from vascular intimal damage due to the combined action of a variety of factors. Therefore, blood flow enters the

**Table 2 Haemodynamics, Laboratory Data and Therapies According to Renal Artery Involvement.**

| | Group A non-renal artery involvement | Group B renal artery involvement | t/$\chi^2$ | P |
|---|---|---|---|---|
| Haemodynamics on Admission | | | | |
| SBP (mmHg) | 145.455 ± 29.0849 | 151.100 ± 27.9767 | −1.461 | 0.145 |
| DBP (mmHg) | 85.711 ± 18.3655 | 91.020 ± 20.1905 | 2.045 | 0.042* |
| Hypertension (%) | 68(56.2) | 72(72.0) | 6.429 | 0.012* |
| Heart Rate (bpm) | 82.264 ± 14.8811 | 84.380 ± 16.3454 | 1.006 | 0.316 |
| Laboratory Data on Admission | | | | |
| WBC(×109) | 10.4486 ± 4.0625 | 11.6408 ± 5.4636 | 1.851 | 0.066 |
| Urea (umol/L) | 6.2698 ± 4.0387 | 5.8728 ± 2.4695 | −0.859 | 0.391 |
| Cr (umol/L) | 107.3634 ± 134.94538 | 95.7476 ± 58.24365 | −0.801 | 0.424 |
| eGFR (ml/min·1.73m2) | 90.8764 ± 40.1054 | 87.4468 ± 34.0432 | 0.677 | 0.499 |
| Uric Acid(umol/L) | 310.99 ± 105 | 334.32 ± 106.8148 | 1.621 | 0.107 |
| Total Cholesterol (umol/L) | 2.6996 ± 2.06709 | 2.7079 ± 1.63842 | 0.300 | 0.976 |
| Triglycerides (umol/L) | 3.0304 ± 1.48870 | 2.7919 ± 1.48360 | −1.116 | 0.266 |
| HDL (umol/L) | 0.9997 ± 0.70129 | 0.8963 ± 0.27179 | −1.310 | 0.192 |
| LDL (umol/L) | 4.2606 ± 19.89294 | 2.2380 ± 0.66561 | −0.958 | 0.339 |
| Therapy (%) | | | | |
| Only Medications | 26(21.5) | 26(26.0) | 0.808 | 0.668 |
| Interventional Therapy | 90(74.4) | 70(70.0) | | |
| Surgery | 5(4.1) | 4(4.0) | | |

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; WBC, white blood cells; eGFR, estimated glomerular filtration rate; HDL, high density lipoprotein; LDL, low density lipoprotein.
*Statistically significant.

area between the vascular intima and the adventitia to form a dissection. When patients have experienced long-term high blood pressure levels, the impact on the arterial wall is larger [5]; when rupture occurs during acute aortic dissection, the pressure on the blood vessels is larger, and thus, the tear in the false lumen is larger, which can easily cause renal artery involvement. Therefore, we found that aortic dissection patients with past hypertension had a higher probability of developing a false lumen of the involved renal artery during dissection onset.

In this study, renal artery involvement was not associated with long-term renal function damage in patients with Stanford B-type aortic dissection. Renal artery involvement is common in clinical studies of Stanford B-type dissection patients. A previous study [6] showed that renal injury in aortic dissection patients after hospital admission was due to renal ischaemia induced by renal artery involvement. However, our study showed that the urea nitrogen and creatinine levels, eGFR, and in-hospital dialysis at admission were not significantly different between patients with or without renal

**Table 3 Early Outcomes and In-Hospital Mortality According to eGFR.**

| | Group A non-renal artery involvement | Group B renal artery involvement | t/$\chi^2$ | P |
|---|---|---|---|---|
| Cardiogenic Shock(%) | 1(0.8) | 9(9.0) | 8.342 | 0.004* |
| Acute Myocardium Infarction(%) | 3(2.4) | 1(1.0) | 0.674 | 0.412 |
| Dialysis(%) | 3(2.5) | 3(3.0) | 0.056 | 0.813 |
| Cerebmvascular Accident(%) | 1(0.8) | 3(3.0) | 1.455 | 0.228 |
| Aortic Rupture (%) | 4(3.3) | 11(11.0) | 5.123 | 0.024* |
| In-Hospital Mortality (%) | 5(4.1) | 12(12.0) | 4.773 | 0.029* |

Abbreviation: eGFR, estimated glomerular filtration rate.
*Statistically significant.

**Table 4 Multivariate Regression Model for Prediction of In-Hospital Mortality.**

| Predictive Factors | β | Wald$\chi^2$ | OR (95%CI) | P |
|---|---|---|---|---|
| Renal Arteries Involved | 1.263 | 4.686 | 3.536(1.127 ~ 11.095) | 0.030 |
| Only Medications | 1.643 | 9.212 | 5.172(1.790 ~ 14.948) | 0.002 |
| Atherosclerosis | 1.296 | 5.503 | 3.655(1.238 ~ 10.794) | 0.019 |

artery involvement. Therefore, this study showed that renal artery involvement was not a relevant factor that affected renal function at hospital admission. It is possible that, because aortic dissection exhibits acute disease onset and severe symptoms, patients usually seek treatment at the early stage of disease onset. Although the blood supply of the kidney is provided by the false lumen or double lumen, the kidney is still in the compensatory stage and does not suffer renal injury. To determine whether renal function is damaged, dynamic changes in renal function should be closely monitored in patients.

We found that renal artery involvement is a risk factor for in-hospital mortality in acute Stanford B-type aortic dissection patients. This study showed that the in-hospital mortality rate in the renal artery involvement group was significantly higher than that in the non-renal artery involvement group. In addition, renal artery involvement was a risk factor for in-hospital mortality in dissection patients. Renal artery involvement can easily cause death during aortic dissection, and the possible mechanisms are described below. During the onset of aortic dissection, renal artery involvement suggests that the tear in the false lumen is broad, which typically indicates a complex dissection. It may be combined with ischaemia of other organs, and the lesions are typically complex. Therefore, the surgical difficulty is increased, and the mortality risk is high. Renal artery involvement may cause peripheral sympathetic nervous system activity [7] in the kidney, increased blood vessel tension, and reflective increases in the heart rate and blood pressure. However, the blood pressure and heart rate at admission were not significantly different between the two groups in the present study. It is possible that most patients had already received treatment in local hospitals before seeking treatment in our hospital, and drugs such as β blockers had been used to control the blood pressure and heart rate; therefore, they did not present significant increases in heart rate. However, sympathetic nerve activity and increased blood vessel tension cause an increased risk of dissection rupture in acute Stanford B-type aortic dissection patients during hospitalisation, thus increasing mortality.

Acute Stanford B-type aortic dissection patients with renal artery involvement should receive surgical intervention as soon as possible because the effect of conservative treatment is poor. The current treatment programs have contradictions for these patients. A previous study [8] showed that surgery or intervention by endovascular exclusion for aortic dissection could cause the closure of the false lumen; therefore, an ischaemic condition would be present in kidneys, where the blood supply had been previously provided by the false lumen. In addition, due to the influence of factors such as intraoperative contrast agents, renal function would also suffer significant damage, which might increase in-hospital dialysis. This study showed that, within the group of patients with renal artery involvement, the percentage of patients who received conservative treatment in the mortality group was significantly higher than that in the survival group. Conservative treatment may be one of the risk factors for in-hospital mortality in aortic dissection patients. The possible mechanisms are described below. Although early stage surgical intervention in acute Stanford B-type aortic dissection patients with renal artery involvement might cause renal function damage, if the false lumen can be closed as soon as possible, then the continuous impact of blood flow on the aortic wall can be terminated to avoid further tearing of the false lumen, thus reducing the pressure on the false lumen and decreasing the risk of aortic rupture. Therefore, we recommend that acute Stanford B-type patients with renal artery involvement should receive surgical intervention as soon as possible due to the poor effect of conservative treatment.

In summary, after acute Stanford B-type aortic dissection patients are admitted to the hospital, renal artery involvement should be noted. The possibility of renal artery involvement during the development of aortic dissection is higher for patients with a past history of hypertension. Renal artery involvement can be an important factor for the prediction of in-hospital mortality in acute Stanford B-type aortic dissection patients. The effect of conservative treatment for patients with renal artery involvement is unacceptable; therefore, surgical intervention should be performed as soon as possible.

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