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Clinical Research

Simple Renal Cysts Are Associated With 24-Month Prognosis of Patients With Type B Aortic Dissection and Hypertension

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ABSTRACT

Background: The association of simple renal cyst (SRC) with type B aortic dissection (BAD) has recently been established. However, no studies have examined adverse events after BAD hospitalization among patients with SRC. In this study, we assessed the prognostic value of SRC in BAD patients with hypertension after thoracic endovascular aortic repair (TEVAR).

Methods: We studied all BAD patients with hypertension who were admitted for TEVAR (n = 238; age 56.1 ± 9.8 years, 84.0% male). Aortic-related adverse events (ARAE) were evaluated as outcomes at 3 months and 24 months after TEVAR.

Results: Among the total number of patients, 104 (43.7%) had at least 1 SRC. Patients with SRC were significantly older than those without (59.6 ± 8.8 vs 53.3 ± 9.7; *P* < 0.001). Patients with SRC were also more likely to suffer from peripheral arterial disease (55.8% vs 40.3%; *P* = 0.018) and cerebrovascular accidents (47.1% vs 29.9%;

RÉSUMÉ

Contexte : L'existence d'un lien entre la présence d'un kyste rénal simple (KRS) et celle d'une dissection aortique de type B (DAB) a récemment été établie. Toutefois, aucune étude ne s'était penchée sur les événements indésirables survenant après une hospitalisation visant à traiter une DAB chez les patients présentant un KRS. Nous avons évalué la valeur pronostique de la présence d'un KRS chez les patients présentant une DAB et de l'hypertension après une réparation endovasculaire de l'aorte thoracique (TEVAR).

Méthodologie : Nous avons examiné les cas de tous les patients présentant une DAB et de l'hypertension qui ont été admis pour une TEVAR (n = 238; âge : 56,1 ± 9,8 ans, 84,0 % de sexe masculin). Les événements indésirables liés à l'aorte (EIA) ont été évalués 3 mois et 24 mois après la TEVAR.

Résultats : Parmi tous les patients, 104 (43,7 %) avaient au moins 1 KRS. Les patients ayant un KRS étaient significativement

Type B aortic dissection (BAD) is a cardiovascular disease with high mortality and complication rates worldwide. Currently, the survival rate of patients with BAD has increased owing to thoracic endovascular aortic repair (TEVAR). Moreover, TEVAR is the best treatment option for BAD, with long-term mortality and complication rates that are lower than those of other therapeutic methods.[1-4] Nevertheless, adverse events, such as recurrence of aortic dissection, aortic rupture, malperfusion, paraplegia, and endoleaks, could still emerge,[4] and these events are considered to be challenges in the management of BAD after TEVAR. Therefore, identification of influencing factors is needed to significantly reduce the mortality rate and the occurrence of adverse events in patients with BAD after TEVAR.

An association between simple renal cysts (SRCs) and aortic dissection has been reported in the current literature.[5,6] Kim et al. demonstrated that the prevalence rate of SRCs was significantly higher in patients with BAD (42.2%) than in healthy subjects (22.0%).[5] Many years later, a higher prevalence rate of SRCs was again observed in patients with BAD (47.3%) than in the general population (15.3%).[6] These results elucidated a possible common mechanism of connective tissue degeneration underlying the development of BAD and SRCs. However, there is a lack of evidence regarding whether the presence of an SRC affects the long-term follow-up prognosis of patients with BAD.

Several authors have reported that SRCs are related to hypertension,[7,8] and aspiration or removal of large cysts reduces blood pressure.[9,10] In addition, hypertension is the most important risk factor for BAD. Accordingly, hypertension should also be taken into consideration in studies involving SRCs and BAD.

Taken together, limited information is known regarding the potential role of SRCs in BAD. Therefore, in the present study, we examined whether the presence of SRCs could

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$P = 0.006$) than those without. Median follow-up for the 238 patients was 18.5 (range 6.4-24.0) months. Cumulative ARAE-free rates were 94.5 ± 1.5% at the 3-month follow-up and 81.5 ± 2.8% at the 24-month follow-up. Independent predictors of 3-month ARAE were found to be insertion of ≥ 2 stents (hazard ratio [HR] 3.977, 95% confidence interval [CI] 1.224-12.920; $P = 0.022$). For 24-month follow-up, SRC (HR 1.962, 95% CI 1.023-3.764; $P = 0.043$) was evaluated as the only independent predictive factor. SRC (HR 8.841, 95% CI 1.726-45.294; $P = 0.009$) was also evaluated as an independent predictive factor for 24-month ARAEs in the chronic group, but not in the acute or the subacute group.

Conclusions: **SRC could predict 24-month ARAE in BAD patients with hypertension after TEVAR, especially in the chronic group.**

plus âgés que les autres (59,6 ± 8,8 vs 53,3 ± 9,7; $p < 0,001$). Ils étaient aussi plus susceptibles d'être atteints d'une artériopathie périphérique (55,8 % vs 40,3 %; $p = 0,018$) et d'avoir subi un accident vasculaire cérébral (47,1 % vs 29,9 %; $p = 0,006$). La période de suivi médiane pour les 238 patients s'établissait à 18,5 mois (min.-max. : 6,4-24,0 mois). La proportion cumulative de patients sans EIA s'établissait à 94,5 ± 1,5 % à l'évaluation de suivi à 3 mois et à 81,5 ± 2,8 % à l'évaluation de suivi à 24 mois. Le facteur de prédiction indépendant d'EIA à 3 mois était l'implantation de ≥ 2 endoprothèses (rapport des risques instantanés [RRI] de 3,977; intervalle de confiance [IC] à 95 % : de 1,224 à 12,920; $p = 0,022$). Au cours de la période de suivi de 24 mois, la présence de KRS (RRI de 1,962; IC à 95 % : de 1,023 à 3,764; $p = 0,043$) était le seul facteur de prédiction indépendant. La présence de KRS (RRI de 8,841; IC à 95 % : de 1,726 à 45,294; $p = 0,009$) était aussi un facteur de prédiction indépendant d'EIA à 24 mois chez les patients dont la maladie était chronique, mais pas chez les patients dont la maladie était aiguë ou subaiguë.

Conclusions : **La présence de KRS est un facteur de prédiction d'EIA à 24 mois chez les patients présentant une DAB et de l'hypertension après une TEVAR, en particulier chez les patients dont la maladie est chronique.**

determine the prognosis of patients with BAD and hypertension after TEVAR.

Patients and Methods

Study population

A retrospective study was conducted on prospectively collected data of patients with BAD and hypertension who underwent TEVAR from January 2011 to April 2017 at Wuhan Asia Heart Hospital (Wuhan, China). The major exclusion criteria were medical history of aortic dissection, Marfan syndrome or other connective tissue diseases, bicuspid aortic valve, iatrogenic or traumatic dissection, syphilis or other inflammatory diseases of the aorta, chronic renal failure, chronic heart failure, chronic respiratory failure, pregnancy, and cancer. Patients with a history of acute myocardial infarction, cerebrovascular accident (CVA), or major surgery 30 days before or after study enrollment also were excluded.[11,12] In addition to the patients with the above-mentioned conditions, which would markedly affect the prognosis, patients with a predisposing factor for SRC formation, such as polycystic kidney disease, hydronephrosis, and polycystic liver disease, were excluded from the study as well.[6,9]

The study was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. All procedures were approved by the Ethics Committee of Wuhan Asia Heart Hospital. Written informed consent was obtained from every participant.

Treatment

Standard criteria were used to acquire demographic data, medical history, clinical characteristics, and examination variables. Hypertension was defined as a self-reported diagnosis of hypertension, treatment with antihypertensive medications, or having a clinical record of a blood pressure ≥ 140/90 mm Hg.[13] All subjects were classified into an acute group (< 14 days), a subacute group (15-90 days), and a chronic group (> 90 days), based on the time interval from the symptom onset date to the procedure date.[12] Body mass index (BMI) was computed as the weight (kg) divided by the square of the height (m^2). Overweight was defined as a BMI between 25 kg/m^2 and 30 kg/m^2 and obesity as a BMI of ≥ 30 kg/m^2.[14]

All patients underwent contrast-enhanced computed tomography (CT) at admission. On contrast-enhanced CT, BAD was defined as disruption of the medial layer and the existence of an intimal flap located in the descending aorta, resulting in separation of the aortic wall layers and formation of true and false lumens of the aorta, with or without communication.[12] CT images were used to analyze the presence of SRCs in the study population. All imaging data were retrieved, re-read, and reanalyzed by an experienced radiology attending physician as part of standard clinical care without knowledge of this study. The presence of SRCs was defined according to the Bosniak classification system.[7] All SRCs in our study belonged to category I.[7] An SRC was defined as a thin-walled, low-attenuation, usually round- or oval-shaped mass, having a regular contour with a clear interface within the renal parenchyma, without evidence of enhancement, calcification, or septa on CT.[7] Patients with BAD were divided into a group with SRCs and a group without SRCs.

All patients were treated with optimal antihypertensive medications and TEVAR.[12] Intravenous calcium channel blockers, β-blockers, or a combination of both was administered to reduce the systolic blood pressure to < 120 mm Hg as initial therapy in the emergency department and intensive care unit. Standardized procedures were performed throughout TEVAR under general anaesthesia. To identify the site of the entry tear, digital subtraction angiography was performed before stent deployment. Subsequently, a stent was delivered and deployed to cover the entry tear, and completion angiography was performed to confirm the satisfactory deployment of the stent.[15] Oral antihypertensive medications, including calcium channel blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, β-blockers, and diuretics, either alone or in combination, were prescribed instead of intravenous medications after TEVAR. These medications were administered by the treating clinicians according to the related guidelines.[13] On discharge from the hospital, the clinicians advised the patients appropriately regarding their condition.

This study had an observational design, and there was no interference in the clinical management of the patients. Demographic data, medical history, clinical presentation, electrocardiography results, echocardiography results, CT results, treatment methods, medications, and follow-up outcomes were recorded on a standardized form. All data were recorded by 2 cardiologists and reviewed by a third cardiologist for consistency and validity.

Follow-up

Follow-ups were performed by 3 clinical cardiologists who were neither aware of the study nor involved in the clinical management of the patients with BAD. Data regarding the symptoms, medications, contrast-enhanced CT findings, and other related examinations were collected during follow-up. Standardized electronic follow-up forms were completed. Follow-up was performed 1, 3, 12, and 24 months after completion of TEVAR. Each patient was advised to go back to the hospital for a review and to undergo related examinations according to the follow-up intervals. For missed visits, telephone conversations were conducted with the patients, their designated relative or contact, or their physician. If patients could not be contacted via telephone on 3 attempts, questionnaires were sent to the address on file at the hospital. If there was still no response, they were considered to be lost to follow-up. To ensure the authenticity and accuracy of follow-ups, 10% of the follow-up results were randomly selected, and the corresponding patients were followed again by 2 cardiologists.

The primary outcomes of our study were 3-month and 24-month aortic-related adverse events (ARAEs), which included aortic-related mortality, new aortic dissection, aortic rupture, requirement for surgery or repeated TEVAR after discharge, malperfusion (mainly mesenteric or lower-extremity ischemia), paraplegia, major stroke, and endoleaks.[11,16]

Statistical analysis

One-sample Kolmogorov-Smirnov test was used to evaluate the distribution of all continuous variables. Data are presented as mean ± SD for continuous variables of normal distribution and percentage for categoric variables. The independent-samples *t* test and chi-square test were used to compare the differences between the normal continuous variables and categoric variables, respectively. The relationships of SRCs and 3-month and 24-month ARAEs were assessed by means of univariate and multiple Cox proportional hazard analyses, with the forward stepwise method. Hazard ratios (HRs) were presented with 95% confidence intervals (CIs), and a 2-tailed $P < 0.05$ was considered to be statistically significant. All data were analyzed with the use of the SPSS software (SPSS 19.0 for Windows, Chicago, IL).

Results

Demographics

A total of 256 patients with hypertension and BAD after TEVAR were enrolled in the present study. Among them, 1 patient who presented with another aortic lesion besides BAD, 1 with a history of aortic dissection, 1 with iatrogenic or traumatic dissection, 4 with cancer, 3 with chronic renal failure, 2 who had CVA within 30 days of study enrollment, 4 with hydronephrosis, and 2 without follow-up records were excluded. The 238 remaining patients (200 men and 38 women, overall mean age 56.1 ± 9.8 years) were included in the present analysis. The flow chart is shown in Figure 1.

Among the 238 patients, 104 patients (43.7%) had at least 1 SRC. Their detailed baseline clinical characteristics were evaluated. The clinical characteristics were compared between the patients with BAD with and without SRCs (Table 1). The mean age of the patients with SRCs was significantly higher than that of the patients without SRCs (59.6 ± 8.8 years vs 53.3 ± 9.7 years; $P < 0.001$). The patients with SRCs were also more likely to experience peripheral arterial disease (55.8% vs 40.3%; $P = 0.018$) and CVA (47.1% vs 29.9%; $P = 0.006$) than those without. No differences were observed between the patients with and without SRCs regarding sex (81.7% vs 85.8% male; $P = 0.393$), prevalence rates of diabetes mellitus (7.7% vs 12.7%; $P = 0.213$), coronary artery disease (18.3% vs 20.9%; $P = 0.614$), dyslipidemia (63.5% vs 74.6%; $P = 0.063$), or other characteristics. Detailed information is presented in Table 1.

Survival analysis

The median follow-up in all 238 patients was 18.5 (range 6.4-24.3) months. The cumulative ARAE-free rates were

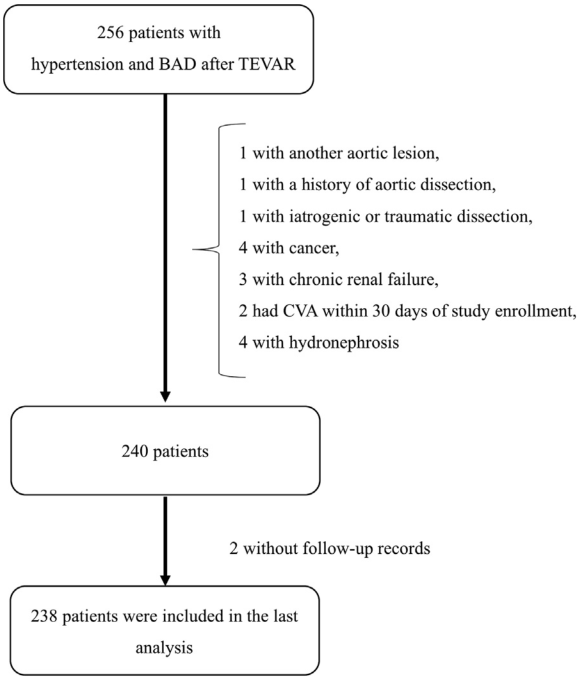

Figure 1. Flow chart of included patients. BAD, type B aortic dissection; CVA, cerebrovascular accident; TEVAR, thoracic endovascular aortic repair.

Table 1. Baseline characteristics of patients with type B aortic dissection and hypertension after TEVAR

Characteristic	Overall (n = 238)	Patients with renal cysts (n = 104)	Patients without renal cysts (n = 134)	*P* value
Male	200 (84.0)	85 (81.7)	115 (85.8)	0.393
Age (y), mean ± SD	56.1 ± 9.8	59.6 ± 8.8	53.3 ± 9.7	< 0.001
BMI				0.359
Normal	128 (53.8)	53 (51.0)	75 (56.0)	
Overweight	97 (40.7)	47 (45.2)	50 (37.3)	
Obese	13 (5.5)	4 (3.8)	9 (6.7)	
Tobacco abuse	155 (65.1)	67 (64.4)	88 (65.7)	0.841
Alcohol abuse	52 (21.8)	20 (19.2)	32 (23.9)	0.389
DM	25 (10.5)	8 (7.7)	17 (12.7)	0.213
PAD	112 (47.1)	58 (55.8)	54 (40.3)	0.018
CVA	89 (37.4)	49 (47.1)	40 (29.9)	0.006
CAD	47 (19.7)	19 (18.3)	28 (20.9)	0.614
Dyslipidemia	166 (69.7)	66 (63.5)	100 (74.6)	0.063
Stage of BAD				0.633
Acute	150 (63.0)	64 (61.5)	86 (64.2)	
Subacute	44 (18.5)	18 (17.3)	26 (19.4)	
Chronic	44 (18.5)	22 (21.2)	22 (16.4)	
Operative procedure				0.735
TEVAR	172 (72.3)	74 (71.2)	98 (73.1)	
Hybrid operation	66 (27.7)	30 (28.8)	36 (26.9)	
Number of stents				0.880
1	212 (89.1)	93 (89.4)	119 (88.8)	
≥ 2	26 (10.9)	11 (10.6)	15 (11.2)	
Medications at discharge				
ACEI/ARB	219 (92.0)	93 (89.4)	126 (94.0)	0.193
CCB	214 (89.9)	96 (92.3)	118 (88.1)	0.280
β-Blocker	229 (96.2)	99 (95.2)	130 (97.0)	0.465
Diuretic	66 (27.7)	24 (23.1)	42 (31.3)	0.158

Values are presented as n (%) unless otherwise specified.

ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BAD, type B aortic dissection; BMI, body mass index; CAD, coronary artery disease; CCB, calcium channel blocker; CVA, cerebrovascular accident; DM, diabetes mellitus; PAD, peripheral arterial disease; TEVAR, thoracic endovascular aortic repair.

94.5 ± 1.5% at the 3-month follow-up and 81.5 ± 2.8% at the 24-month follow-up.

In the univariate Cox proportional regression analysis, insertion of ≥ 2 stents (HR 3.977, 95% CI 1.224-12.920; $P = 0.022$) was a significant factor for 3-month ARAEs. Meanwhile, the presence of SRCs (HR 1.115, 95% CI 0.375-3.318; $P = 0.845$), age (HR 1.010, 95% CI 0.955-1.069; $P = 0.728$), peripheral arterial disease (HR 1.316, 95% CI 0.442-3.916; $P = 0.622$), and CVA (HR 1.440, 95% CI 0.484-4.286; $P = 0.512$) were not significant factors for 3-month ARAEs. At the 24-month follow-up, the presence of SRCs (HR 1.962, 95% CI 1.023-3.764; $P = 0.043$) was found to be a risk factor of ARAEs in the univariate Cox proportional regression analysis. Age (HR 1.004, 95% CI 0.972-1.038; $P = 0.797$), peripheral arterial disease (HR 1.161, 95% CI 0.609-2.213; $P = 0.649$), and CVA (HR 1.417, 95% CI 0.739-2.717; $P = 0.294$) were not significant factors for 24-month ARAEs in the univariate Cox proportional regression analysis.

Table 2. Multivariate Cox proportional regression analysis of risk factors for aortic-related adverse events for all of the study patients

Event	HR	95% CI	*P* value
3-month aortic-related adverse events			
Number of stents			
1	Reference		
≥ 2	3.977	1.224-12.920	0.022
24-month aortic-related adverse events			
Simple renal cysts	1.962	1.023-3.764	0.043

CI, confidence interval; HR hazard ratio.

In the multivariate Cox proportional regression analysis (Table 2), insertion of ≥ 2 stents (HR 3.977, 95% CI 1.224-12.920; $P = 0.022$) was a significant predictor of 3-month ARAEs, and the presence of SRCs was not. At the 24-month follow-up, the presence of SRCs (HR 1.962, 95% CI 1.023-3.764; $P = 0.043$) was found to be the only significant risk factor for ARAEs. The probability of a 24-month ARAE-free status based on the presence of SRCs in the Kaplan-Meier analysis showed significant differences, as shown in Figure 2.

In the acute group, the prevalence of SRCs was 42.7% and the median follow-up in all 150 patients was 20.7 (range 7.0-24.3) months. The cumulative ARAE-free rates were 95.3 ± 1.7% at the 3-month follow-up and 84.9 ± 3.3% at the 24-month follow-up. In the multivariate Cox proportional regression analysis (Table 3), insertion of ≥ 2 stents was a significant predictor of 3-month ARAEs (HR 12.236, 95% CI 2.356-63.544; $P = 0.003$) and 24-month ARAEs (HR 9.613, 95% CI 2.730-33.854; $P < 0.0001$). The presence of SRCs was not found to be a significant risk factor for ARAEs.

In the subacute group, the prevalence of SRCs was 40.9% and the median follow-up in all 44 patients was 17.3 (range 6.5-24.3) months. The cumulative ARAE-free rates were 95.4 ± 3.2% at the 3-month follow-up and 80.6 ± 6.8% at the 24-month follow-up. In the multivariate Cox proportional regression analysis, no factor was found to be a significant predictor of 3-month or 24-month ARAEs.

In the chronic group, the prevalence of SRCs was 50.0% and the median follow-up in all 44 patients was 16.3 (range 4.3-24.3) months. The cumulative ARAE-free rates were 90.9 ± 4.3% at the 3-month follow-up and 70.8 ± 7.7% at the 24-month follow-up. In the multivariate Cox proportional regression analysis (Table 4), no factor was found to be significant predictor of 3-month ARAEs. The presence of SRCs (HR 8.841, 95% CI 1.726-45.294; $P = 0.009$) and female sex

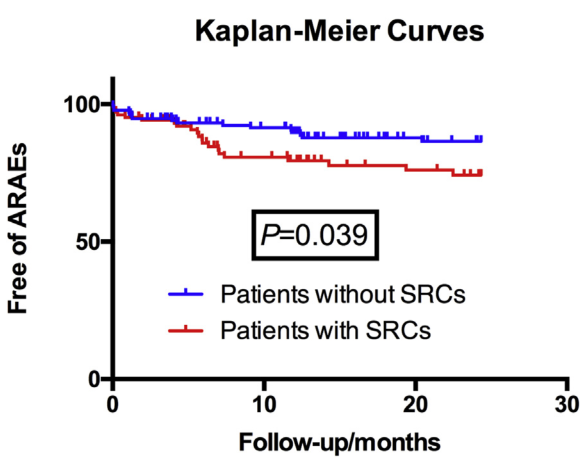

Figure 2. Kaplan-Meier analysis of freedom from the development of aortic-related adverse events (ARAEs) based on the presence of simple renal cysts (SRCs).

Table 3. Multivariate Cox proportional regression analysis of risk factors for aortic-related adverse events for acute BAD patients

Event	HR	95% CI	*P* value
3-month aortic-related adverse events			
Number of stents			
1	Reference		
≥ 2	12.236	2.356-63.544	0.003
24-month aortic-related adverse events			
Number of stents			
1	Reference		
≥ 2	9.613	2.730-33.854	< 0.0001

BAD, type B aortic dissection; CI, confidence interval; HR hazard ratio.

(HR 5.187, 95% CI 1.120-24.025; $P = 0.035$) were found to be significant risk factors for 24-month ARAEs.

Discussion

Our findings suggest that the presence of SRCs was independently associated with a high risk of 24-month ARAEs in patients with hypertension hospitalized with BAD after TEVAR, but the presence of SRCs was not found to be associated with 3-month ARAEs, even after adjusting for other risk factors. This phenomenon was more pronounced in the chronic group. In addition, the mean age of the patients with SRCs was significantly higher than those without an SRC. The patients who had SRCs were also more likely to experience peripheral arterial disease and CVA than those who had no SRCs.

In the general population, the prevalence rate of SRCs has been reported to be 5%-41%, depending on the imaging method, ethnic background, and other influencing factors.[17-20] In the present study, the prevalence rate of SRCs was 43.7% in the patients with hypertension hospitalized with BAD after TEVAR, which was higher than that reported in the general population and was consistent with previous reports on patients with BAD (42.2%-47.3%).[5,6] The increase in the prevalence rate of SRCs among the older population and in men has been correlated with the natural history of SRCs,[7] and that was also consistent with our study results, where the mean age of our patients who had SRCs was significantly higher than those who had no SRCs and male sex predominance (84.0%) was observed. The male-to-female sex ratio was 4.5, which is higher than that in the general population (2.0).[6] The higher male-to-female sex ratio in patients with SRCs who developed BAD than in subjects with SRCs in the general population is probably the result of the higher frequency of BAD in men than in women.

The finding that the presence of SRCs was independently associated with 24-month ARAEs in patients with hypertension hospitalized with BAD after TEVAR was of great importance. Based on existing information and the literature, there are 2 possible explanations. First, BAD and SRCs may share a common genetic defect or pathophysiologic mechanism.[5,6] BAD is associated with cystic medial degeneration. Dysregulation of matrix metalloproteinase (MMP) production and activity contributes to extracellular matrix degradation and medial layer degeneration, probably resulting in weakness of the aortic wall and the occurrence of BAD. In addition, MMPs could potentially play a part in the development of SRCs.[5,6,21] Obermuller et al. reported that MMP-14 was synthesized and secreted by the epithelia lining the cysts and the renal tubules of a rat model of autosomal-dominant polycystic kidney disease.[22] Interestingly, a significant reduction in the cyst number and kidney weight was found when a tissue inhibitor of MMPs was used for treatment. Furthermore, MMP-2 and MMP-9 were found to be abundant in benign cystic fluids of humans in a study by Harada et al. in 2002.[23] These suggest that MMP activity may be the common pathophysiologic mechanism between BAD and SRCs. Second, hypertension may be the major link between SRCs and BAD. In recent years, it has been elucidated that high blood pressure could be decreased with the treatment of SRCs in patients with hypertension. In addition, it is well known that hypertension is regarded as an important risk factor for BAD.[12] Therefore, it is reasonable to conclude that SRCs may affect the prognosis of BAD through its association with hypertension. In addition to hypertension, SRCs are associated with advanced age, peripheral arterial disease, and CVA, all of which worsen prognosis of BAD patients. Although multivariate analysis did not show that these factors affected the prognosis of BAD, we cannot completely exclude the impact of these factors. In future studies, we need to include more patients and analyze the impact of these factors in detail.

Table 4. Multivariate Cox proportional regression analysis of risk factors for aortic-related adverse events for chronic BAD patients

Event	HR	95% CI	*P* value
3-month aortic-related adverse events			
None			
24-month aortic-related adverse events			
Simple renal cysts	8.841	1.726-45.294	0.009
Female	5.187	1.120-24.025	0.035

BAD, type B aortic dissection; CI, confidence interval; HR hazard ratio.

In contrast, we did not find the presence of SRCs to be associated with 3-month ARAEs even after adjusting for other risk factors. In patients with hypertension and BAD after TEVAR, insertion of ≥ 2 stents, indicating a larger extent of aortic injury, probably predisposes them to ARAEs at the 3-month follow-up more potently than the structural weakness associated with SRCs. Another explanation was that the rate of control of hypertension decreased as the time of follow-up increased.[24] At the 3-month follow-up, the blood pressure of all patients was effectively controlled. Therefore, when the shear stress and strain levels on the aorta were low, the structural weakness associated with SRCs was not responsible for the prevalence rate of 3-month ARAEs.

Another interesting finding of this study was that the prevalence rate of peripheral arterial disease and CVA was higher in the patients who had SRCs than in those who had no SRCs. The associations of SRCs with peripheral arterial disease and CVA may support the hypothesis that extracellular matrix remodeling causes structural weakness of middle and large vessel walls.[25] However, no study has yet investigated the relationships of SRCs with peripheral arterial disease and CVA. The relationships and internal pathogenesis of SRCs with peripheral arterial disease and CVA need to be examined exhaustively in the future.

The present findings have an important clinical implication. The belief, which was prevalent for many years, that an SRC is a benign finding is challenged by the findings of this report. Our results underscore the importance of the presence of SRCs in patients with BAD. Patients with BAD who develop SRCs need not only stricter control of other correctable risk factors, but also more prudent follow-ups.

Limitations

There were several limitations in the present study. First, the number and size of cysts were not considered. Second, we did not include patients with BAD who were normotensive, mainly because of the small sample size. Third, no follow-up regarding SRCs was performed, and the relationship between SRC progression and BAD prognosis was not analyzed. Finally, this study was a clinical observational study, and the molecular and pathophysiologic mechanisms that might explain the link between BAD and SRCs were not evaluated. Further studies are needed to address these issues fully.

Conclusion

Our data indicate that the presence of SRCs has an important prognostic value on 24-month ARAEs in patients with hypertension and BAD after TEVAR, especially in the chronic group. Additional research is needed to better understand the prognostic value of SRCs. It is necessary to study the mechanisms of the role that SRCs play in the development and prognosis of BAD to provide new evidence and information on the pathogenesis of BAD.

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Disclosures

The authors have no conflicts of interest to disclose.

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