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Research Article

Resistant Hypertension in Thoracoabdominal Aortic Dissection Involving the Renal Arteries

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Abstract

There is little longitudinal BP data in patients with thoracoabdominal aortic dissections (TAAD). In patients attending an aortic diseases clinic, thoracic aortic dissection (TAD) having false lumen extensions past the renal arteries (Group 1) were compared to TAD without renal artery involvement (Group 2). Resistant hypertension (rHTN) was present in 28.6% of Group 1, significantly ($p=0.038$) greater than the 4.8% in Group 2. Group 1 had a significantly ($p=0.04$) greater prevalence of coronary artery disease. BP's post-TAD were not significantly different between the two groups likely due to the greater number of antihypertensive drugs used in rHTN. There was a higher proportion of ARB usage in Group 1. TAAD, with the right renal artery supplied by the false lumen and the left renal artery supplied by a true lumen had the greatest prevalence (42.9%) of rHTN; significantly ($p<0.05$) greater than other types of renal artery dissections. The data suggest, rHTN is prevalent in TAAA, necessitating vigorous treatment but good BP control is possible. The nature of the renal artery involvement identifies potential rHTN.

ABBREVIATIONS

rHTN: Resistant Hypertension, CAD: Coronary Artery Disease, TAD: Thoracic Aortic Dissection, TAAD: Thoracoabdominal Aortic Dissection

INTRODUCTION

Thoracic aortic dissection (TAD) is a potentially disastrous condition with a high immediate and long term mortality [1,2]. TAD has been classified into various subtypes. The most widely used classification—the Stanford classification system [1]; considers Type A dissections as those involving the ascending aorta while Type B dissections involve the descending aorta without any involvement of the ascending aorta. Thoracic aortic aneurysms (TAA) can be more extensive and involve the abdominal aorta and are referred to as thoracoabdominal aneurysms (TAAA). Crawford proposed a TAAA classification based on the anatomic extent of the aneurysm [3]. Type I involves most of the descending thoracic aorta from the origin of the left subclavian to the suprarenal abdominal aorta. Type II is the most extensive, extending from the subclavian to the aortoiliac bifurcation. Type III involves the distal thoracic aorta to the aortoiliac bifurcation. Type IV TAAAs mainly involve the abdominal aorta below the diaphragm [3]. Thoracoabdominal dissections (TAAD) may be considered with this general classification.

Although the etiology of TAD is complex involving factors in the arterial wall that maintain aortic structure and function, hypertension is usually considered to be an important factor in producing aneurysmal expansion which predisposes to the dissection [4]. In thoracic aortic aneurysm (TAAA), hypertension exacerbates the process of aortic expansion leading to dissection [5,6]. Although the precise mechanisms responsible for the dissection remain a matter of debate, elevated systemic arterial blood pressure (BP) has been implicated in its pathogenesis [7,8]. Thoracoabdominal aortic dissection (TAAD) can involve the renal arteries and have a risk of impairment in renal blood flow producing ischemic kidney disease. Considering the role of the kidney in the genesis of hypertension, restricting renal blood flow would be expected to further elevate BP [9,19].

While HTN in chronic TAD has been studied [10-12], HTN in TAAD have not been examined in a systematic manner. The objective of this study was to assess long-term blood pressures post-TAAD, their pharmacological management, and whether dissection induced variations in renal artery involvement impact BP management in TAAD.

PATIENTS AND METHODS

Patients

The study population consisted of patients who attended the Aortic Diseases Outpatient Clinic at a University Hospital. A retrospective case review was conducted from the electronic medical records of persons attending the Clinic during a 3 year period. This study was approved by the Clinical Research Ethics

Board of the University of British Columbia (IRB Number (H16-03047). All cases were reviewed. Cases were selected if they had thoracoabdominal dissections and met the following (entry) criteria (i) thoracoabdominal aortic dissection confirmed by CT angiogram of the aorta (ii) the aortic dissection extended past the renal arteries (iii) kidneys were supplied by renal arteries. The exclusion criteria were: clinical evidence or a family history of a known genetic cause of aneurysm or dissection such as Marfan's syndrome or Ehlers Danlos.

The electronic medical records were reviewed and data extraction included the following clinical variables: age, sex, systolic BP (SBP) and diastolic BP (DBP), cardiovascular risk factors and location/site of TAD. The CT angiogram reports were examined and data was extracted as to whether the aortic false lumens extended past the renal arteries. Thoracoabdominal dissections were classified according to the Crawford classification [3]. Antihypertensive medications were recorded and classified into the following Groups: ß-blockers (BB), calcium channel blockers (CCB), angiotensin-converting enzyme inhibitors (ACEi), angiotensin receptor antagonists (ARB), diuretics (DIU) and centrally acting agents (CAA). The number as well as combination of antihypertensive agents was recorded.

A control Group was selected, matching 1:1, of individuals who had a TAD that did not involve the renal arteries. Individuals in the control Group were matched by age and sex without knowledge of their other clinical data.

Patients were considered as belonging to one of two groups according to renal artery involvement. Renal artery involvement was defined as CT imaging evidence of a dissection induced false lumen extending to or past the renal arteries (Group 1). Patients of Group 2 served as the control/comparison, defined as having TAD's without false lumen extension to the renal arteries. The reported perfusion of the kidney from the true or false lumen was recorded.

Statistical analysis

Data are presented as the mean + standard deviation for continuous variables. Univariate continuous variables were analyzed by unpaired t-test. Categorical variables were analyzed by the Fisher's exact test.

RESULTS

The subject characteristics showed a preponderance of men (Table 1). The mean age was approximately 60 years and there were no significant differences in age between TAD cases with or without renal artery involvement for each sex. The majority of patients in Group 1 had Crawford Type 1 (38.1%) or Type 2 (38.1%) TAAD's. There was no significant difference in the presumed site of origin of the aortic dissection between the two patient groups. There was also no statistically significant difference in the type of TAD suffered by patients in the two Groups when categorized using the Stanford classification system; 66.7% of those in Group 1 had Type A dissections compared to 71.4% of cases in Group 2. The majority of individuals in both Groups had surgery or TEVAR for their TAD.

A history of coronary artery disease was significantly (p=0.04) more frequent in persons with TAD involving the renal arteries.

Table 1: Shows the patient characteristics of subjects in the study.

	TAD with Renal artery involvement (n=21)		TAD without renal artery involvement (n=21)		p value
Age (Years)	59.5±11.3		60.5±9.6		0.76
Sex (% Males)	67.70%		71.40%		1
Pre-existing hypertension	11 (52.4%)		10 (47.6%)		0.66
Crawford Classification					
1	8 (38.1%)		_		_
2	8 (38.1%)		_		_
3	1 (4.8%)		_		_
4	4 (19.1%)		_		_
Stanford Classification of TAD	Type A	14 (66.7%)	Type A	15 (71.4%)	0.33
	Type B	7 (33.3%)	Type B	6 (28.6%)	
Presumed initial site of dissection on aorta					
Aortic root/sinus	3 (14.3%)		1 (4.8%)		1
Ascending aorta	5 (23.8%)		8 (38.1%)		
Aortic arch/isthmus	4 (19.1%)		6 (28.6%)		
Descending thoracic aorta/ diaphragm	9 (42.9%)		6 (28.6%)		
Aortic Surgery or TEVAR	15 (71.4%)		17 (80.9%)		0.27
Conditions/ risk factors					
Cigarette smokers	3 (14.3%)		7 (33.3%)		0.064
Coronary artery disease	3 (14.3%)		1 (4.8%)		0.04
Dyslipidemia	9 (42.9%)		8 (38.1%)		0.65
Diabetes Mellitus	2 (9.5%)		2 (9.5%)		1

The cardiovascular risk factors assessed, including pre-existing hypertension, history of smoking cigarettes, dyslipidemia, and diabetes mellitus were not greater in the TAD with renal involvement.

The proportion of resistant hypertension (rHTN) was significantly (p=0.038) higher in Group 1 as compared to Group 2 (Figure 1). The prevalence of resistant hypertension was approximately six times greater in those with renal artery involvement or 28.6% as compared to 4.8% in those without renal artery involvement.

The medications prescribed to control BP were examined (Figure 1). Amongst patients receiving 1 antihypertensive, a beta-blocker was the drug of choice, with 83.3% of Group 1 patients and 100% of Group 2 patients receiving a beta blocker. For patients on two antihypertensive, the most common combination was a beta-blocker with a calcium-channel blocker, in 60% of patients in Group 1 and 80% of patients in Group 2. In patients on 3 antihypertensive, the most common combination was a beta-blocker combined with a calcium channel blocker (CCB) and an Angiotensin converting enzyme inhibitor (ACEi) or angiotensin receptor blocker (ARB) considered together as Renin angiotensin aldosterone system inhibitor (RAASi). The next most common combination of antihypertensive was a diuretic rather than a CCB.

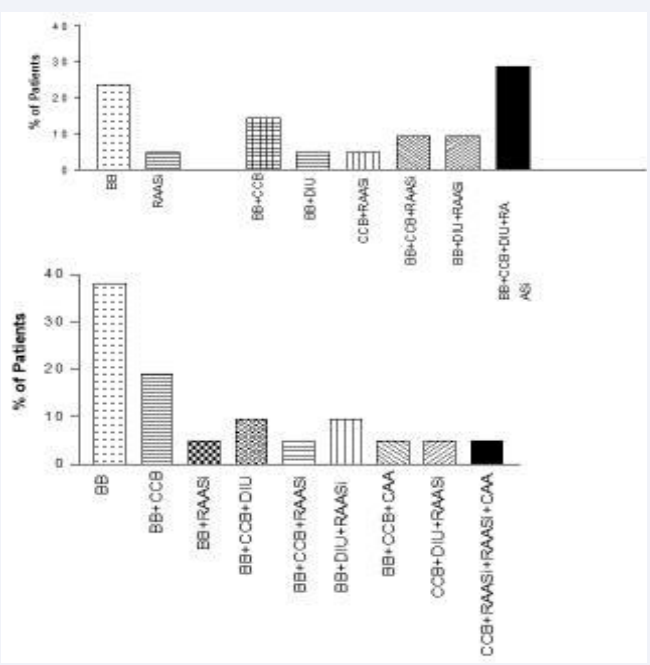

Figure 1 Show the proportion of patients in Groups 1 (upper panel) (N=21) and Group 2 (N=21) (lower panel), taking one, two, three or four antihypertensive agents and the specific combinations of antihypertensive drugs used. The antihypertensive drugs were ß-blockers (BB), calcium channel blockers (CCB), renin-angiotensin-aldosterone inhibitors (RAASi), diuretics (DIU) and centrally acting agents (CAA).

Amongst patients on 4 or more antihypertensives, a beta-blocker, CCB, diuretic, and ACEi/ARB was the choice, with 100% of Group 1 patients receiving such a combination. A greater proportion of patients in Group 1 as compared to Group 2 were found to be on ARB therapy (23.8% versus 4.8%).

The mean SBP over the first 24 months post-TAD was not significantly different between the two patient groups (Figure 2). A similar finding was evident for the mean DBP as there was no significant different in DBP between the two groups. Over the first 24 months after-TAD, BP in those in Group 1 was 125.0/74.4 mmHg, compared to 123.7/70.9 mmHg in Group 2. Blood pressure was also not significantly different between Groups 1 and 2 if men and women were examined separately.

Renal blood supply could originate from the true or false aortic lumen (Figure 3). Examining the affected renal arteries involved subdividing Group 1 into four different combinations since the renal artery might perfuse either the right or left kidney. Group 1a patients (n=6) were those having both left and right renal artery blood supply from the true lumen. Group 1b patients had both left and right renal artery supplied by the false lumen (n=2). Patients in Group 1c received right renal artery blood supply from a true lumen and left from a false lumen (n=6). Group 1d had the left renal artery supplied by a true lumen and right by a false lumen (n=7). Patients in Group 1d had the greatest mean SBP (128.8 mmHg) over the first 12 months post-TAD, with the next highest mean SBP of 125.2 mmHg found amongst Group 1a patients. Resistant hypertension was 3 times more prevalent in Group 1d than in any other group (Figure 4). Group 1d also contained the majority of patients taking 3 antihypertensive agents. The mean and median number of antihypertensive agents were found to be 3 and 3.1, respectively. These values were greater than those found in Groups 1a (mean= 1.6, median= 1.5) or 1c (mean= 2.3, median= 2.5) – Group 1b was not considered for this analysis due to insufficient number of patients. There was a significantly greater proportion of patients on three (p=0.024)

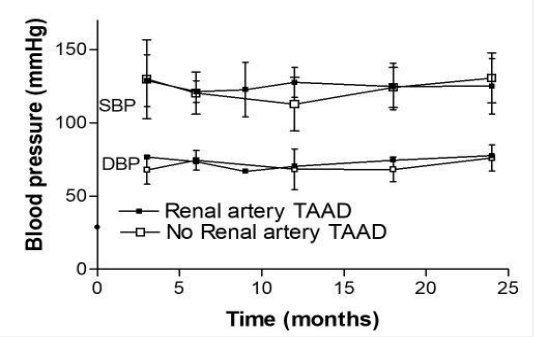

Figure 2 shows the mean systolic and diastolic BP values in follow-up after the occurrence of TAD for Group 1 and 2. Patients were seen in the clinic on average 3 months after the occurrence of TAD. There was no significant difference in BP values between the two groups over the first 24 months after TAD.).

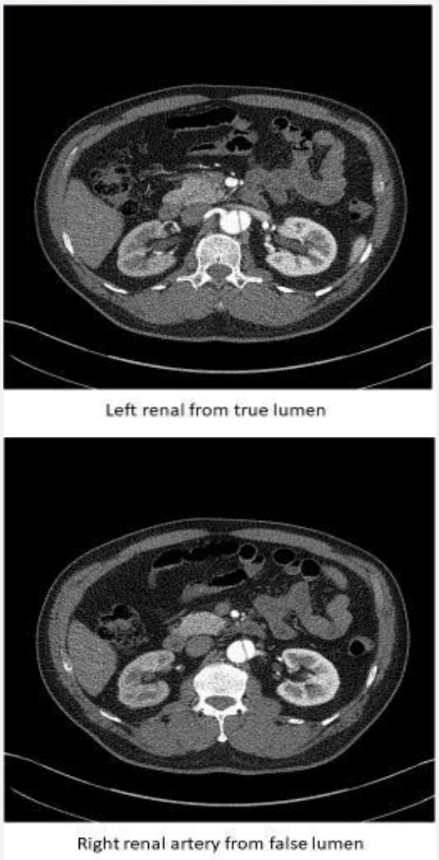

Figure 2 shows a CT angiogram of a case with the left renal artery from the true lumen (top panel) and the right renal artery from the false lumen (bottom panel).

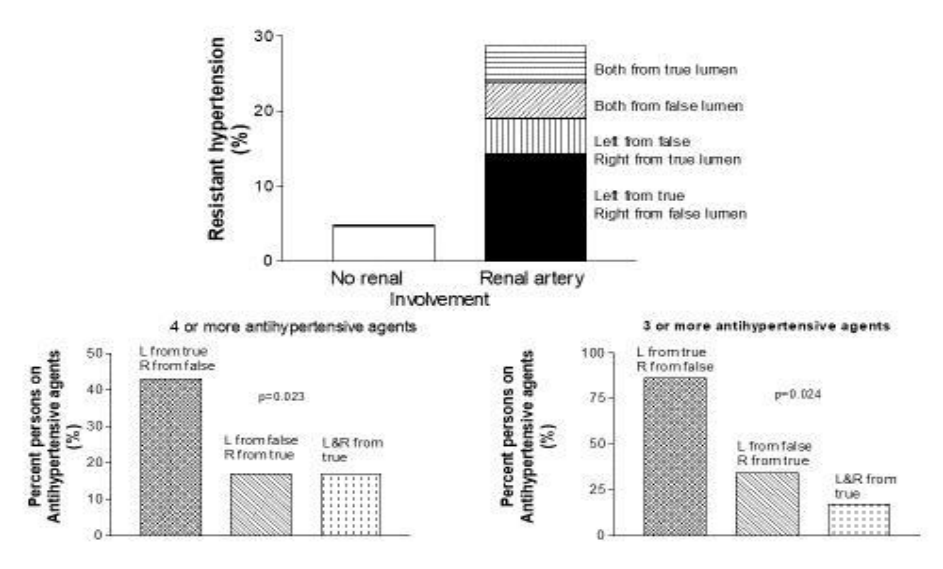

Figure 4 Displays the proportion of patients with resistant hypertension in thoracic aortic dissection without and with renal arteries as part of the dissection (upper panel). The percentage or rHTN is shown according to the type of renal artery involvement. Patients with renal artery involvement were considered according to source of renal blood supply for individuals taking 3 or more antihypertensive agents (lower right panel) or 4 or more antihypertensive agents (lower left panel).

or four (p=0.023) antihypertensive agents when the left renal artery originates form the true lumen and the right renal artery arises from the false lumen.

DISCUSSION

This study systematically examined BP and its pharmacological management in patients with TAAD's involving the renal arteries. The results of this study offer insights into the differing blood pressure as well as the pharmacological management required to maintain acceptable values in patients with TAAD, especially in dissections involving the renal arteries.

We found a greater prevalence of resistant hypertension (rHTN) amongst patients with TAD extending to the renal arteries as compared to patients without renal artery involvement. The definition of rHTN includes attaining a normal BP only upon use of 4 or more antihypertensive drugs [13-15]. In the management of patients with TAD's involving the renal arteries, our findings suggest that an expedited usage of 4 or more antihypertensive drugs should be considered. This is consistent with the concept that the greater the number of antihypertensive drugs the greater the probability of achieving adequate blood pressure control in patients with potential rHTN [10,16]. Indeed delay in controlling BP to within acceptable limits is associated with adverse health outcomes [13,14]. This may be especially relevant for patients with conditions such chronic TAD that have increased mortality [2]. The pathophysiology may involve a role for increased blood pressure to produce greater wall stress in TAA [17] with a greater probability of expansion.

With the use of a more intensive BP management strategy in patients with TAAD involving the renal arteries, BP was controlled to the same levels of patients who had TAD without renal artery involvement. On average, BP's were within an acceptable range and there was no discernable difference in either SBP or DBP over time after TAD between patients having TAD with or without renal artery involvement.

Although the advantages of multiple antihypertensive agents in controlling BP amongst patients with TAD's has been discussed, the drawbacks of polypharmacy should not be overlooked—namely the predisposition to adverse drug interactions and the associated costs patients incur [18]. The potential high mortality in chronic TAD, however, should justify the use of effective medication combinations. We found effective BP control with combinations of antihypertensive agents that include ARBs. Based on this observation, we speculate that the earlier inclusion of ARB's into treatment regimes in the subset of patients with TAD's involving the renal arteries may be advantageous.

Our finding that rHTN was 6 times more prevalent amongst patients in with renal artery involvement compared to those without renal artery involvement raises several possible explanations. TAD with renal artery involvement may invoke an altered physiological state requiring more pharmacological interventions to control blood pressure. Reduced renal perfusion should activate the renin-angiotensin-aldosterone system (RAAS) [19]. Another explanation is that changes in aortic wall structure in TAD may be a factor in producing rHTN [20]. The greater proportion of patients with renal artery involvement on ARB therapy suggests a potentially specific role for ARB's in managing BP in patients with TAAD involving the renal arteries. This is consistent with the concept of RAAS activation in a situation of impairment in renal blood flow [19]. Our data that blood pressure is controlled in Group 1 patients suggests that RAAS blockade is advantageous in this condition. This was also suggested in a case report of TAD's with false lumen extension to the renal arteries [21].

We found that patients with TAD involving the renal arteries were more likely to have coronary artery disease. This finding is consistent with epidemiologic data [8]. While not exactly similar, our finding can be supported by the observation that patients with abdominal aortic aneurysms also exhibit an increased prevalence of coronary artery disease [22]. The explanation for this association is uncertain. Elkalioube *et al.* suggested that this is due to a the higher prevalence of smoking and hypertension in patients with CAD [22].We did not find a difference in the prevalence of smoking and hypertension in the two types of TAD. Rather we suggest that other factors may be operative to account for the association of coronary artery disease with this more severe kind of TAD.

An additional observation that stemmed from the analysis of patients separated by the nature of the involvement of the renal arteries was the finding that those with a left renal artery supplied by a true lumen and right by a false lumen had the highest proportion of rHTN—with 42.9% taking 4 or more antihypertensive agents—and a greater mean SBP over the first year post-TAD. In particular, the proportion of rHTN was significantly (p=0.023) higher in Group 1d as compared to other Group 1 patients. The proportion of rHTN with individuals taking 3 or more antihypertensive medications was also significantly (p=0.024) greater in such persons with left renal artery supplied by a true lumen and right by a false. There are several potential explanations for this novel finding. It may be consistent with the suggestion of Siegelmen *et al.* [23] that the anatomic origin of the left renal artery makes it more likely to be narrowed by an aortic dissection compared to the right renal artery. Alternatively the right renal artery supplied from the false lumen is the one compromising renal blood flow. We contend that the nature of renal perfusion may then account for some of the cases of rHTN. Compromised renal artery blood flow can increase blood pressure through activation of RAAS, as well as through the sympathetic nervous system [19]. This may also be one explanation for the greater mean SBP over the first 12 months post-TAD amongst patients with this kind of dissection. A clinical extrapolation would be to initiate more vigorous antihypertensive drug therapy in patients with TAAD once left renal artery involvement is identified. Finally, given the superior aortic origin of Type A dissections, it was expected that the more distally originating Type B dissections would have a propensity for false lumen extension past the renal arteries and would thus be more prevalent amongst Group 1 patients [24]. However this was not the case and in fact Type A dissections exhibited a majority in both Groups, in line with their expected 60-70% constitution of all aortic dissections [24].

STUDY LIMITATIONS

There are several issues that warrant attention. First, the small sample size is an issue. TAAD, however, is a less common type of TAD so that the number of cases with this condition is usually small. Second, the retrospective nature of this analysis constitutes problems with data collection [25]. Data such as serum renin and aldosterone were not available. The use of archival data often lends itself to having to work with missing information, which in this study meant variability in sample size for BP measurements at some time points after TAD. Missing data reflects variance in information recorded by medical professionals and have the potential to create a challenge for retrospective studies [26,27]. These are the standard concerns for retrospective case control studies. In studying conditions with small prevalence with the need for longitudinal follow-up, however, retrospective case control studies, such as the current study, are both essential and may provide the only available data on the subject. Importantly there is a greater emphasis now on the collection of non-clinical trial data to learn from 'real world' experience. Third, manual data abstraction is a source of bias in retrospective studies. However, the potential for inaccuracy is small and is usually expected to be less than 5% [28]. Furthermore we minimized bias by using a predesigned data extraction methodology. Fourth, the number of antihypertensive drugs was used as the definition of rHTN rather that an approach that sought to systematically identify *a priori* the optimal antihypertensive treatment through a standard management protocol. This by its nature is the standard issue with retrospective studies. However, the use of a high number of antihypertensive drugs is an accepted definition of rHTN [14,15]. Lastly, several other cardiovascular risk factors such as BMI, low physical activity, or ethnicity that were not considered in this study may be of value in future an investigation in order to discern any correlations with blood pressure following TAD's involving the renal arteries.

In summary, TAD involving the renal arteries is associated with a six-fold and meaningful increased prevalence of rHTN. This study supports the notion that TAD's involving the renal arteries does not compromise the ability to maintain acceptable BP's when combination therapy across the spectrum of antihypertensive agents especially ARBs are used. The concomitant presence of coronary artery disease shows a predilection for TAAD's involving the renal artery that warrants further research. Lastly it should be emphasized that further sub-classification of TAAD's based on source of renal blood supply specifically the right renal artery supplied by the false lumen and the left renal artery by the true lumen identifies a subset with rHTN and the need for additional antihypertensive drug therapy.

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