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Coronary Events in Patients Presenting for Repair of Acute Type A Aortic Dissection

Paul C. Tang, MD, PhD*, Shahab A. Akhter, MD, Satoru Osaki, MD, PhD, Lucian Lozonschi, MD, Takushi Kohmoto, MD, PhD, Nilto C. De Oliveira, MD

Department of Surgery, Division of Cardiothoracic Surgery, University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin, USA

Abstract

Background: Preoperative coronary angiography is often not performed in acute Type A dissection. We examined differences in the incidence of pre-existing coronary disease and subsequent coronary events between patients undergoing acute Type A dissection repair and patients undergoing elective proximal aortic aneurysm repair.

Methods: From 2000 to 2015, there were 154 acute Type A dissection repairs and 457 elective proximal aortic aneurysm repairs. We performed a retrospective review to evaluate preoperative coronary disease and postoperative coronary interventions such as percutaneous coronary intervention (PCI) and coronary bypass grafting (CABG).

Results: A total of 31 (20%) dissection patients and 123 (27%) elective surgery patients had preoperative evidence of coronary artery disease (p = 0.094). All elective surgery patients but only six (4%) dissection patients had preoperative coronary catheterization. More CABGs were performed in the elective surgery group (19%) than in the dissection group (3%, p < 0.001). There were no differences in the incidence of prior PCI, CABG, or myocardial infarction between groups. Following dissection repair, four patients required coronary interventions. Of these, two (1.3%) experienced chest pain and underwent PCI at 4.7 and 4.3 months postoperatively, respectively, and another two experienced symptoms and required PCI at 5 and 7 years, respectively. The 30-day and 14-year mortality rates after dissection repair were 13% and 24%, respectively. Although the dissection group had poorer survival than the elective surgery group (p < 0.001), there was no difference in conditional survival after aortic-related deaths over the first year were censored (p = 0.104).

Conclusions: Given the low incidence of missed significant coronary disease (1.3%), it is reasonable to perform Type A dissection repair without coronary angiography.

Key Words

Aorta • Thoracic • Aortic dissection • Coronary artery disease • Coronary artery imaging • Coronary syndromes • Myocardial ischemia

Introduction

Acute Type A dissection is an emergent condition that requires timely operative intervention. While aortography allows the diagnosis of concomitant native coronary artery disease during evaluation for dissection, the diagnosis of acute aortic dissection has largely been supplanted by computed tomographic angiography and sometimes magnetic resonance imaging [1], largely due to the widespread accessibility and reproducibility of these modalities. Although these techniques may identify proximal coronary artery involvement, they are unable to evaluate more distal disease. However, transesophageal echocardiography during the course of dissection evaluation...
can identify regional wall motion abnormalities in addition to ostial coronary obstruction that may indicate coronary compromise.

The incidence of acute myocardial ischemia due to coronary malperfusion is around 5–6% [2-4]. In an autopsy series, Larson and Edwards found critical (i.e., grade 4) coronary artery disease in 27 out of 121 (22%) patients who had Type I or II aortic dissection [5]. Rizzo et al. found high operative mortality among patients with Type A dissection who had preoperative angiography, whereas there were no deaths due to aortic rupture or coronary artery disease complications among patients taken directly to surgery following noninvasive diagnosis of acute Type A dissection [6]. By contrast, Penn et al. found that angiographic delineation of coronary anatomy did not negatively impact operative survival, although this study did not include patients who died while waiting for angiography and subsequent surgery [7].

At our institution, we prefer the expeditious direct transfer of patients to the operating room. Following repair of Type A dissection, coronary evaluation is not usually routinely performed unless the patient is symptomatic. In the present study, we evaluated the short- and long-term outcomes of this approach and subsequent postoperative coronary interventions. Our control group consisted of patients who underwent elective ascending aortic aneurysm repair in which preoperative coronary catheterization was mandatory. The validity of this group as a control is based on the assumption that a significant portion of Type A dissections result from pre-existing ascending aortic aneurysms.

Materials and Methods

Patients

This study was approved by the University of Wisconsin-Madison Institutional Review Board and conforms to the provisions of the Declaration of Helsinki. We analyzed the records of 154 patients who underwent consecutive acute Type A aortic dissection repair and 457 patients who underwent elective proximal aortic aneurysm repair at University of Wisconsin hospitals and clinics between January 2000 and July 2015. Stanford Type A dissection was defined as acute if the onset of symptoms was less than 14 days from the time of surgery. A retrospective review was performed for preoperative coronary disease based on prior coronary catheterization, stress tests, and history of coronary events such as myocardial infarction (MI). We also analyzed patient age, gender, comorbidities, creatinine, left ventricular ejection fraction (LVEF), operative variables, postoperative complications, and survival. Postoperative coronary studies and interventions such as stress tests, coronary catheterization, percutaneous coronary interventions (PCI), and coronary artery bypass grafting (CABG) were examined. All elective surgery patients and 6 out of 154 (4%) dissection patients received preoperative coronary evaluation (p < 0.001). For patients in the dissection group who did not undergo preoperative coronary angiography, coronary evaluation was performed intraoperatively by visually inspecting the coronary ostia after opening the aorta. For patients in the dissection group who did undergo preoperative coronary angiography, two patients had no coronary disease; one patient had prior CABG, with catheterization demonstrating all patent grafts; one patient had right coronary malperfusion from the dissection flap; and two patients had significant underlying atherosclerotic coronary disease.

Follow-up

Survival data were available for all patients and were obtained through detailed clinical follow-up. Follow-up is expressed in years using mean and standard deviation. Maximum follow-up was 14.04 years, with a total follow-up of 1948.48 patient years. Aortic-related deaths are defined as those that occurred as a complication of the initial surgery for aortic-related pathology or from any residual aortic disease (e.g., residual Type B dissection).

Statistical Analysis

Pearson Chi-square or Fisher’s exact tests were used to analyze categorical variables. Kaplan-Meier survival curves with Mantel-Cox statistics were used to analyze survival data. Student’s t-tests were used to analyze continuous variables. Statistical analysis was performed using SPSS software (SPSS Inc., Chicago, IL).
Results

Patient Demographics and Coronary Findings

There were no differences in patient age, gender, LVEF, or other comorbidities between the dissection and elective surgery groups (p > 0.05, Table 1). The lack of difference in a history of cancer within 5 years of surgery suggests that any differences in survival were unlikely due to cancer. However, creatinine was higher in the dissection group (1.2 ± 0.6) than in the elective surgery group (1.1 ± 0.5, p = 0.018). Coronary evaluation (Table 2) demonstrated no difference in history of previous PCI, MI, or CABG (p > 0.05). There was no difference in the number of patients with known preoperative coronary artery disease between the dissection (31 out of 154, 20%) and elective surgery (123 out of 457, 27%) groups (p = 0.094). In the elective surgery group, 85 out of 457 (19%) patients required CABG for atherosclerotic coronary disease. Of these, 57 out of 457 (12%) required a graft to the left anterior descending artery. Most patients (44 out of 457, 10%) in the elective surgery group needed one coronary graft, whereas 26 (6%) needed two grafts. In the dissection group, four concomitant CABGs were performed. Of these four patients, one was found to have a right coronary artery ostial stenosis noted visually upon opening the ascending aorta; one patient had preoperatively identified left anterior descending artery disease on coronary angiography; one received three-vessel CABG for dissection of the left main and right coronary artery; and one received empiric bypass grafts to the left anterior descending and obtuse marginal arteries due to unexplained poor anterolateral wall function on cardiac reperfusion. This last patient had no direct visual evidence of coronary dissection or technical issues with the coronary button, so poor cardiac function after cross clamp removal may have resulted from undiagnosed underlying atherosclerotic coronary artery disease. This patient’s cardiac function improved after CABG, and the patient ultimately survived.

Following Type A dissection repair, four patients required subsequent coronary interventions. Of these, two (1.3%) experienced chest pain following discharge and underwent coronary catheterization with stent placement at 4.7 and 4.3 months after the operation, respectively. Stents were placed in the left anterior descending artery in the first patient and in the left anterior descending and left main arteries in the second patient. Another two patients

Table 1. Patient demographics.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Type A Dissection Repair (n = 154)</th>
<th>Elective Proximal Aortic Surgery (n = 457)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>61.4 ± 14.3</td>
<td>59.8 ± 14.1</td>
<td>0.229</td>
</tr>
<tr>
<td>Sex (male)</td>
<td>106 (69%)</td>
<td>335 (73%)</td>
<td>0.284</td>
</tr>
<tr>
<td>Body mass index</td>
<td>29.2 ± 6.5</td>
<td>29.1 ± 6.0</td>
<td>0.867</td>
</tr>
<tr>
<td>Preoperative creatinine (mg/dL)</td>
<td>1.2 ± 0.6</td>
<td>1.1 ± 0.5</td>
<td>0.018</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>58.2 ± 9.3</td>
<td>59.0 ± 10.3</td>
<td>0.403</td>
</tr>
<tr>
<td>Hypertension</td>
<td>110 (71%)</td>
<td>305 (67%)</td>
<td>0.281</td>
</tr>
<tr>
<td>Dialysis</td>
<td>2 (1%)</td>
<td>4 (1%)</td>
<td>0.645</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>13 (8%)</td>
<td>32 (7%)</td>
<td>0.554</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>38 (25%)</td>
<td>81 (18%)</td>
<td>0.060</td>
</tr>
<tr>
<td>Lung disease</td>
<td>29 (19%)</td>
<td>102 (22%)</td>
<td>0.362</td>
</tr>
<tr>
<td>Liver disease</td>
<td>2 (1%)</td>
<td>2 (0.4%)</td>
<td>0.252</td>
</tr>
<tr>
<td>Diabetes</td>
<td>12 (8%)</td>
<td>35 (8%)</td>
<td>0.957</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>70 (45%)</td>
<td>224 (49%)</td>
<td>0.444</td>
</tr>
<tr>
<td>Cancer within 5 years of surgery</td>
<td>3 (2%)</td>
<td>8 (2%)</td>
<td>0.873</td>
</tr>
</tbody>
</table>

Nominal data are presented as frequency (n) and percentage of the total population and were analyzed using Pearson Chi-square or Fisher’s exact tests. Continuous data are presented as mean ± standard deviation and were analyzed using two-tailed paired Student’s t-tests. LVEF= left ventricular ejection fraction.
experienced symptoms consistent with progression of native atherosclerotic coronary disease and required coronary stent placement at 5 and 7 years after dissection repair, respectively. We identified five coronary malperfusions (3.2%) upon presentation. All four patients with malperfusion and confirmed involvement of the left coronary artery died during their hospital stay, and one patient with malperfusion involving the right coronary artery was alive at 5 years. No patient presented with new MI after 30 days postoperatively.

Operative Parameters

There was no difference in median operative year, which was 2010 (Table 3), suggesting that operative techniques as well as pre- and postoperative management were likely comparable between groups. There were more reoperative procedures in the elective surgery group (15%) than in the dissection group (6%, p = 0.007). No group difference in concomitant mitral or tricuspid valve surgery was found (p > 0.05). There were more hemi- and total arch replacements in the dissection group (p < 0.001) and more ascending aortic replacements without arch repair in the elective surgery group (p < 0.001). There were more composite valve graft root and aortic valve replacements in the elective surgery group (p < 0.001) and more aortic valve repairs (mostly aortic valve resuspensions) in the dissection group (p < 0.001). The cross-clamp time was higher in the elective surgery group (130 ± 50 min) than in the dissection group (104 ± 47 min, p < 0.001), probably due to the higher number of composite valve graft root and aortic valve replacements in the elective surgery group. However, cardiopulmonary bypass time was higher in the dissection group (258 ± 92 min) than...
likely because of the longer cooling and rewarming time needed for circulatory arrest in order to perform an open distal anastomosis. Given the emergent and challenging nature of acute dissection repairs, the dissection group had a higher incidence of complications, including neurological events, pneumonia, prolonged ventilation, gastrointestinal complications, acute renal failure, and new dialysis (Table 4, p < 0.05). Length of hospital stay was also longer in the dissection group (10.8 ± 15.1 days) than in the elective surgery group (6.4 ± 5.1 days, p < 0.001).

Survival
Mean follow-up was 2.78 ± 3.61 years for the Type A dissection repair group and 3.32 ± 3.23 years for the elective ascending aortic aneurysm surgery group.

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The 30-day and 14-year mortality rates were 13% and 24%, respectively, for patients with acute Type A dissection and 1.5% and 8.3%, respectively, for patients who underwent elective ascending aortic aneurysm surgery. Although the dissection group had poorer survival than the elective surgery group (Figure 1, \( p < 0.001 \)), there was no group difference in conditional survival when aortic-related deaths over the first year were censored (Figure 2, \( p = 0.104 \)).

**Discussion**

Although coronary angiography was not routinely performed prior to Type A dissection repairs at our institution, we found a very low incidence (2 out of 154 patients, 1.3%) of missed coronary artery disease that became symptomatic soon after surgery and required subsequent coronary intervention. We found another two (1.3%) patients who likely had coronary artery disease in evolution at the time of dissection repair, with lesions not becoming symptomatic until 5 and 7 years postoperatively. Although there likely was atherosclerotic coronary disease that was missed during the acute surgical management of an acute Type A dissection, these lesions seemed to remain mostly silent.

In our Kaplan-Meier survival analysis, there was no difference in long-term conditional survival when early aortic deaths were censored. Given the 1–2% per hour early mortality from acute Type A dissection [8], the mortality risk of surgical delay for coronary catheterization outweighs the 1.3% risk of missed significant native coronary artery disease requiring early postoperative intervention. Our two patients with early postoperative PCI for missed atherosclerotic coronary artery disease presented with stable angina and shortness of breath on exertion. No patient presented with MI after the initial dissection repair, suggesting that any missed coronary artery disease was likely well collateralized or not severe enough to cause significant hemodynamic issues.

Delays in surgery leading to patient demise in Type A dissection have been well documented by several clinicians. Glower et al. found that 10 out of 91 (11%) patients with DeBakey Type I or II aortic dissections died before an operation could be done [9]. Rizzo et al. found that preoperative coronary angiography led to delays in surgical intervention and...
increased acute Type A dissection mortality [6]. In an Italian multicenter study where routine preoperative coronary angiography was performed, 23 out of 242 (10%) patients who were considered surgical candidates died before the operation [10]. However, with increasing availability of hybrid operating rooms in the endovascular era, there are greater opportunities in many facilities for intraoperative angiographic coronary evaluation. The negative impact of contrast agents on postoperative renal function needs to be justified. Furthermore, even without routine preoperative coronary angiography, we found a greater baseline incidence of postoperative acute renal failure and new dialysis in the dissection group. Impaired pre- and postoperative renal function can occur from renal malperfusion due to dissection flap obstruction or systemic hemodynamic instability.

However, there may be subpopulations of dissection patients who could benefit from coronary angiography. Indeed, a history of coronary artery disease is associated with increased risk for in-hospital death following Type A dissection repair [11]. Kern et al. found a potential survival benefit of preoperative coronary angiography in patients with Type A dissection with significant clinical history suggestive of coronary artery disease [12]. Interestingly, patients with prior open heart surgery presenting with acute Type A dissection infrequently have tamponade or hemodynamic collapse, which may be due to scar tissue providing support for mediastinal structures. Therefore, coronary angiography may be justified in the preoperative management of these patients, particularly if evaluation of previous CABGs is needed [13].

Although instrumentation of the dissected aorta for coronary angiography may increase the risk of further aortic complications, we have not experienced any complications from this procedure. Penn et al. found that concomitant CABG at the time of emergent aortic surgery had no effect on in-hospital mortality, and defining coronary anatomy before surgical intervention had no effect on overall CABG rate, likely because 74% of CABGs at the time of surgical repair were for known coronary dissection and not chronic atherosclerotic coronary artery disease [7]. Of the
coronary malperusions in our cohort, four out of five patients died from cardiogenic shock due to myocardial ischemia and MI. This is consistent with a previous finding that a need for concomitant CABG for an evolving myocardial infarct is predictive of postoperative mortality [14].

The arguments for preoperative coronary angiography include the opportunity to graft a critical stenosis while the patient is on cardiopulmonary bypass during aortic repair to improve the likelihood of successful weaning, to avoid perioperative MI, and to improve survival. However, we had only five (3.2%) cases of perioperative MI, of which four were thought to be due to coronary dissection and malperfusion. Although coronary malperfusion associated with Type A dissection is uncommon, its outcome is often fatal [4, 8, 15]. No MIs were confirmed to be the result of underlying atherosclerotic coronary artery stenosis. Therefore, routine preoperative coronary angiography to address this disease process is of questionable value in improving postoperative survival.

Creswell et al. reported that the prevalence of atherosclerotic coronary artery stenosis >50% was 34.8% in patients with acute ascending aortic dissection [1]. As ascending aortic aneurysm diseases are precursors to Type A aortic dissection, we believe that electively operated ascending aneurysm patients constituted a reasonable control for the coronary disease in the dissection group. Although routine coronary catheterization was not performed for our dissection patients, we found a 19% concomitant CABG rate in our elective ascending aneurysm surgery population. This percentage is lower than that of Creswell et al., as our criterion for CABG was >70% stenosis for all vessels except for left main disease, where we graft >50% stenosis. Of the 34.8% of patients with coronary artery disease, Creswell et al. found a 75% incidence of single vessel disease and 25% incidence of triple vessel disease in the preoperative angiography group of his acute dissection series [1]. This finding is similar to that in our elective surgery group, in which 82% of CABGs involved two or fewer grafts. Assuming this is representative of the dissection population, the low incidence of multivessel disease may explain the high rate of successful cardiopulmonary bypass weaning in dissection patients despite likely missed underlying significant coronary artery disease. Alternatively, vascular collateralization with chronic coronary compromise may maintain adequate myocardial perfusion and contractility without causing myocardial ischemia or symptoms.

Although aortic dissection patients had a higher early mortality than elective ascending aortic aneurysm surgery patients due to the challenging pathology and emergent nature of the disease, we found no group difference in long-term survival when aortic-related deaths in the first year were censored from the analysis. This suggests that missed coronary artery disease at the time of acute dissection repair does not limit long-term survival. Indeed, the four patients with coronary artery disease in the dissection group who needed subsequent intervention were managed successfully with PCI in a semi-elective manner. Therefore, as angiography is not needed to establish the diagnosis of acute Type A dissection given the advantages of modern tomographic imaging, the benefit of angiography to evaluate coronary artery disease is likely not worth the considerable risks and delay associated with performance of the test, for which no survival benefit can be demonstrated.

The conclusions of this study are limited by its retrospective nature, which has inherent limitations and biases. The lack of routine preoperative coronary angiography in the dissection group precludes accurate assessment of underlying atherosclerotic coronary artery disease. Inferences made for dissection patients by extrapolating the burden of coronary artery disease in the elective ascending aortic aneurysm surgery group may not be valid. Without comprehensive follow-up of the entire patient population, it is possible that patients received subsequent coronary intervention without our knowledge. Causes of intraoperative myocardial ischemia in some cases were unclear and may have been due to coronary button complications, coronary dissection, or underlying atherosclerotic disease. We also do not have data on the clinical presentation or myocardial function of patients with Type A dissection who did not undergo surgery because they were not surgical candidates, refused surgery, or died prior to operation. Patient age and incidence of significant underlying atherosclerotic coronary artery disease may have
been higher in this population that did not undergo surgery. Although we found no difference in conditional survival after censoring aortic-related mortalities in the first year, it is possible that our dissection population was too small to detect increased mortality due to complications of residual Type B dissections or missed coronary artery disease at the time of surgery.

Conflict of Interest

The authors have no conflict of interest relevant to this publication.

References


Abstract

Background: Chronic descending thoracic aortic dissection (CDTAD) following surgical repair of ascending aortic dissection requires long-term imaging surveillance. We investigated four-dimensional (4D)-flow magnetic resonance imaging (MRI) with a novel multi-velocity encoding (multi-VENC) technique as an emerging clinical method enabling the dynamic quantification of blood volume and velocity throughout the cardiac cycle.

Methods: Patients with CDTAD (n = 10; mean age, 55.1 years; standard deviation (SD) 10.8) and healthy volunteers (n = 9; mean age, 37.1 years; SD 11.4; p < 0.01) underwent 3T MRI, and standard views and 4D-flow data were obtained. Flow measurements were made in selected regions of interest within the ascending and descending thoracic aorta.

Results: The overall flow profile at peak systole was reduced in the false lumen (FL) compared with the true lumen (TL) and normal aortas (p < 0.05 for velocity < 0.4 m/s). Peak systolic flow rate per aortic lumen area (mL/s/cm²) was lower in the FL than in the TL (p < 0.05), and both rates were lower than that of control aortas (p < 0.05). Blood flow reversal was higher in the FL than in the TL throughout the descending aorta in CDTAD patients (p < 0.05). A derived pulsatility index was elevated in the TL compared with that in the FL in CDTAD patients. Generated pathline images demonstrated flow patterns in detail, including sites of communication between the true and FL.

Conclusions: 4D-flow MRI revealed FL blood flow and reduced blood flow velocity and flow rate in the TL of CDTAD patients compared with normal aortas of healthy participants. Thus, multi-VENC 4D-flow MRI could serve as an adjunct in the long-term assessment of CDTAD following surgical repair of ascending aortic dissection.

Key Words
Aorta • Thoracic • Aortic dissection • Magnetic resonance imaging

Introduction

Patients with chronic descending thoracic aortic dissection (CDTAD) following surgical repair of as-
cending aortic dissection require continued surveillance throughout their lifetime, as some will develop progressive aortic dilatation [1]. The risk of complications can be difficult to predict and appears to be independent of the initial location of dissection (Stanford Type A vs. Type B) or initial medical or surgical therapy [2]. Adverse remodeling of the chronically dissected descending aorta can result in an increased overall diameter > 55 mm [1], enlargement of the false lumen (FL) [2], and residual blood flow [3] or partial thrombosis of the FL [4], which are associated with later complications.

Four dimensional (4D)-flow magnetic resonance imaging (MRI) is an emerging imaging tool that permits accurate quantification of blood flow velocity and volume through the aorta, as well as flow dynamics over time, to be represented as velocity-encoded pathlines [5, 6]. The use of a multi-velocity encoding (multi-VENC) approach additionally improves pathline tracking and streamline estimation [7]. Its capability of quantifying bulk flow and measuring flow patterns suggests that 4D-flow MRI may be a useful tool to evaluate CDTAD, particularly for assessment of FL blood flow and intimal flap integrity, providing information beyond current measurements of aortic diameter. Unlike conventional phase-contrast flow MRI, 4D-flow MRI data allow post-acquisition analysis of any region of interest (ROI) in the aorta.

The aim of this study was to assess the potential utility of 4D-flow MRI in measuring true lumen (TL) and FL blood flow in patients with persistent dissection of the descending thoracic aorta following previously surgically repaired ascending aortic dissection. Aortic blood characteristics, including peak velocity, forward flow, reverse flow, and a derived pulsatility index (PI) within the TL and FL, were quantified with 4D-flow MRI and compared with characteristics of healthy control participants without aortic pathology.

Materials and Methods

Patients

CDTAD patients were recruited from the Marfan and Aortic Diseases Clinic at Royal Prince Alfred Hospital (RPAH, Sydney, Australia) from January 2014 to June 2015. Patients were included if they were over 18 years of age and had aortic dissection at least 6 months previously. Exclusion criteria were any contraindication to MRI. Healthy control participants were recruited via a flyer advertisement at the hospital and screened by interview for the absence of known aortic or cardiovascular disease.

MRI Acquisition

Brachial sphygmomanometry was performed immediately following each scan. Data were acquired using a Siemens 3T Skyra MRI (Erlangen, Germany) with electrocardiographic and respiratory gating. All images were analyzed by a radiologist and cardiologist who were accredited in cardiovascular MR. Intravenous contrast was not utilized. All participants underwent a routine cardiac MRI protocol consisting of anatomical and time-resolved (cine) steady-state free precession sequences to confirm the absence of additional cardiac disease or abnormality and to enable placement and acquisition of four-chamber and two-chamber views used in post-processing. Left ventricular ejection fraction (LVEF) was calculated using the Simpson disk summation method. 4D-flow MRI was previously validated against traditional time-resolved phase contrast MRI techniques [8, 9]. Scans were obtained using a multi-VENC 4D-flow protocol at three different VENC values of 150, 60, and 20 cm/s covering the entire thoracic aorta [10]. All three scans were isotropic with a spatial resolution of 2.5 mm, and temporal resolution was 16–23 phases per cardiac cycle. Other parameters were a repetition time (TR) of 5.1–5.8 ms and a echo time (TE) of 2.8–3.6 ms. Scan time was approximately 10 min for a VENC of 150 cm/s and 5–6 min for a VENC of 60 and 20 cm/s. The three different VENC datasets were combined on a per-point basis using custom software written in C++, Python, and the VTK Imaging Visualization Toolbox (Kitware Inc., New York). Other acquisition parameters were a flip angle of 8 degrees, acquisition matrix of 144 × 78, and a field of view of 250 × 360 mm. Techniques used for processing the multi-VENC dataset were previously described in detail [7].

Data Analysis

Four manually placed transverse planes along the short axis of the thoracic aorta were isolated during post-scan analysis at the levels of the mid ascending...
aorta (native or prosthetic; AscAo) and the midpoints of the proximal, middle, and distal third of the descending thoracic aorta (ProxAo, MidAo, and DistAo, respectively). Flow measurements were acquired from manually created intra-luminal ROIs within these planes. Total forward and reverse blood flow volumes through the aortic ROIs were calculated for the entire cardiac cycle (using the R-R interval) as well as peak systolic blood velocity (m/s) and maximal blood flow rate (mL/s). The percentage of flow reversal was determined as reversed volume over total volume. A PI of blood flow was calculated using the following formula [11]:

\[ PI = \frac{\text{maximum blood flow (mL/s)} - \text{minimum blood flow (mL/s)}}{\text{mean blood flow (mL/s)}}. \]

Images demonstrating blood flow patterns, represented by pathlines (i.e., the path traveled by massless source particles originating from the aortic ROI over a cardiac cycle), were displayed using the Paraview Scientific Visualization Program (Kitware Inc., New York). For CDTAD participants with evident TL and FL fenestrations, additional cross-sectional ROIs were placed perpendicular to these fenestrations for the purpose of flow qualification.

Statistical Analysis

Statistical analysis was performed using SPSS version 22.0 (IBM, New York). Participant body surface area was calculated as \( m^2 = \sqrt{\text{height (cm) \times weight (kg) / 3600}} \) [12]. Normality of continuous data was determined by Shapiro-Wilk tests. Continuous variables are shown as a mean ± standard deviation (SD) when normally distributed or as median and interquartile range (IQR) otherwise. Categorical variables are described as absolute and relative frequencies (percentage). Group differences in baseline data were analyzed using Student’s t-tests, Kruskal-Wallis tests, or Chi-squared tests, as appropriate. Group differences in 4D-flow data (i.e., blood flow velocity, rate, PI, and reversal) were analyzed using Mann-Whitney U tests. The relationship between PI and aortic lumen cross-sectional area was assessed using Spearman’s rank correlation coefficients. A two-tailed \( p < 0.05 \) was considered statistically significant.

Ethics

The study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki as reflected by a priori approval by the Human Research Ethics Committee of RPAH (Protocol No. X14-0106 and HREC/14/RPAH/129). All participants provided written informed consent.

Results

Participant Characteristics

Participants were 10 patients with CDTAD and 9 healthy controls. Demographic characteristics are shown in Table 1. CDTAD patients were older than control participants (55.1 ± 10.8 years vs. 37.1 ± 11.4 years, \( p < 0.05 \)). CDTAD patients had experienced dissection of the ascending aorta or aortic arch between 19 months and 16 years prior to study enrollment. All CDTAD patients were on maximal tolerated doses of a beta blocker (\( n = 3 \)) or combination therapy with an angiotensin II blocker (\( n = 7 \)), whereas no control participants were on these medications. In CDTAD patients, the underlying aortic pathologies were non-syndromal thoracic aortic aneurysm and dissection (\( n = 6 \)), hypertension/atherosclerosis (\( n = 2 \)), Marfan syndrome (\( n = 1 \)), and iatrogenic aortic dissection secondary to a diagnostic coronary angiogram (\( n = 1 \)). Both control and CDTAD groups had normal LVEF.

The maximum diameter of the descending thoracic aorta was larger in CDTAD patients (39.5, IQR 30.0–43.8 mm) than in control participants (19.0, IQR 17.0–20.0 mm, \( p < 0.001 \); Table 2). There were no significant differences in intra-luminal area between control participants and the TL of CDTAD patients at any aortic plane. Among CDTAD patients, intra-luminal area was significantly larger in the FL than in the TL.

Velocity Flow Profile

In the ASCAo (without aortic dissection), there was a greater proportion by area of low velocity flow (< 0.4 m/s) in the TL of CDTAD patients than in the normal aortas of control patients (\( p < 0.001 \); Figure 1A). The proportions of velocities at mid-range (0.4–0.8 m/s) were similar between groups throughout the descending aorta, whereas the fraction of high velocity flow (> 0.8 m/s) was greater in control participants than in CDTAD patients (\( p < 0.05 \) throughout the aorta).
Table 1. Characteristics of CDTAD patients and summary of control group.

<table>
<thead>
<tr>
<th>ID</th>
<th>Age (Years)</th>
<th>Gender</th>
<th>Diagnosis</th>
<th>LVEF (%)</th>
<th>Beta-Blocker Therapy</th>
<th>Ang II Receptor Blocker Therapy</th>
<th>BSA (m²)</th>
<th>Prior Aortic Surgery</th>
<th>Time to Imaging</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>36</td>
<td>Female</td>
<td>MFS</td>
<td>60</td>
<td>Yes</td>
<td>Yes</td>
<td>1.69</td>
<td>AVR and ascending aorta</td>
<td>9 y</td>
</tr>
<tr>
<td>2</td>
<td>40</td>
<td>Male</td>
<td>ns-TAAD</td>
<td>60</td>
<td>Yes</td>
<td>No</td>
<td>2.05</td>
<td>AVR and ascending aorta</td>
<td>9 y</td>
</tr>
<tr>
<td>3</td>
<td>52</td>
<td>Male</td>
<td>Iatrogenic dissection</td>
<td>55</td>
<td>Yes</td>
<td>Yes</td>
<td>2.31</td>
<td>Ascending aorta</td>
<td>19 mo</td>
</tr>
<tr>
<td>4</td>
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<td>55</td>
<td>Yes</td>
<td>Yes</td>
<td>2.06</td>
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<td>4 y</td>
</tr>
<tr>
<td>5</td>
<td>57</td>
<td>Male</td>
<td>ns-TAAD</td>
<td>55</td>
<td>Yes</td>
<td>Yes</td>
<td>2.32</td>
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<td>16 y</td>
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<tr>
<td>6</td>
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<td>ns-TAAD</td>
<td>60</td>
<td>Yes</td>
<td>Yes</td>
<td>2.07</td>
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<td>3 y</td>
</tr>
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<td>Female</td>
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<td>55</td>
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<td>No</td>
<td>1.99</td>
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<tr>
<td>8</td>
<td>69</td>
<td>Male</td>
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<td>60</td>
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<td>Yes</td>
<td>1.90</td>
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<td>9</td>
<td>56</td>
<td>Male</td>
<td>ns-TAAD</td>
<td>50</td>
<td>Yes</td>
<td>No</td>
<td>2.22</td>
<td>AVR and ascending aorta</td>
<td>14 y</td>
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<tr>
<td>10</td>
<td>64</td>
<td>Male</td>
<td>ns-TAAD</td>
<td>55</td>
<td>Yes</td>
<td>Yes</td>
<td>1.91</td>
<td>AVR, ascending aorta and aortic arch</td>
<td>5 y</td>
</tr>
</tbody>
</table>

CDTAD (n = 10) | 55.1 (SD 10.8) | 2 female, 8 male | - | 55.0 (55.0–60.0) | - | - | 2.1 (SD 0.2) | - | 7.1 y (SD 4.9) |
Control (n = 9) | 37.1 (SD 11.4)* | 2 female, 7 male | - | 55.0 (55.0–60.0) | None | None | 1.9 (SD 0.2) | - | - |

*P < 0.05 vs. CDTAD. Ang II = angiotensin II; AVR = aortic valve replacement; BSA = body surface area; CDTAD = chronic descending thoracic aortic dissection; MFS = Marfan syndrome; ns-TAAD = non-syndromal thoracic aortic aneurysm and dissection; SD = standard deviation.

For the Proxₐₐ₀, Midₐ₀, and Distₐ₀ in CDTAD patients, the percentage of velocity < 0.4 m/s was significantly higher in the FL than in the TL (all p < 0.05). For both mid-range and high velocities (≥ 0.4 m/s), the relative proportion in the TL was consistently higher than that in the FL, with significant differences at several velocities and aortic locations (Figure 1B, 1C, and 1D).

**Proportional Flow and Pulsatility**

Maximal blood flow rate was not significantly different between the normal aortas of control participants and the TL of CDTAD patients at the Ascₐ₀ (P = 0.15; Figure 2). Maximal flow rate was significantly less in the TL of CDTAD patients (Figures 2E, 2F, 2G, and 2H) than in that of control participants (Figures 2A, 2B, 2C, and 2D) for all ROIs at the descending thoracic aorta. Within CDTAD patients, proportional maximal blood flow rate in the TL was significantly greater than that in the FL (Figures 2I, 2J, and 2K).

A derived PI was compared between groups (Figure 3). Between aortic planes, there were no significant differences in PI within groups (all P > 0.05). PI was significantly less in the FL than in the TL of CDTAD patients. Across all measured ROIs, PI decreased as aortic lumen area increased, although the correlation was not significant (ρ = -0.4, P = 0.7). In only control ROIs, however, this correlation was significant (ρ = -0.36, P = 0.03).

**Flow Reversal**

At all aortic planes, there were no differences in

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Multi-VENC 4D-Flow MRI in Chronic Dissection
the percentage of flow reversal between the normal aortas of control participants and the TL of CDTAD patients (AscAO: 0.9% vs. 2.0%, \( P = 0.3 \); ProxAO: 1.3% vs. 2.2%, \( P = 0.9 \); MidAO: 1.3% vs. 5.7%, \( P = 1.0 \); and DistAO: 1.6% vs. 6.0%, \( P = 0.6 \); respectively). Comparisons between the TL and FL in CDTAD patients revealed significantly lower flow reversal in the TL than in the FL (ProxAO: 2.2% vs. 32.4%, \( P < 0.01 \); MidAO: 5.7% vs. 28.6%, \( P < 0.05 \); and DistAO: 1.6% vs. 60.0%, \( P < 0.001 \); respectively).

### Pathlines

Exemplar illustrations of the use of pathline visualization to better understand abnormal flow dynamics at an individual level are shown in Figures 4, 5, 6, and 7. As a normative comparator, the aorta of a control participant is shown in Figure 4A. In Figure 4B, aortic blood flow is shown for CDTAD patient ID 1, who had an entry tear at the proximal descending thoracic aorta into the FL. For CDTAD patient ID 7 (Figure 5), non-laminar blood flow could be visualized at the distal aortic arch at the commencement of a large FL. Within the FL, pathline blood flow did not travel the distance between consecutive aortic planes within one cardiac cycle secondary to low blood velocity. In CDTAD patient ID 10 (Figure 6), pathlines isolated from the ascending aorta and traced into the TL alone similarly demonstrated a relatively large FL. For CDTAD patient ID 5 (Figure 7), at least three distinct communication points were detected and visualized as isolated TL pathline blood flows into a larger FL.

### Discussion

In this study, we demonstrated the potential utility of 4D-flow MRI as a tool in the clinical evaluation of blood flow parameters for patients with CDTAD. 4D-flow MRI was able to quantify differing blood flow.

### Table 2. Hemodynamic and aortic data for CDTAD patients and summary of control group.

<table>
<thead>
<tr>
<th>ID</th>
<th>HR (Beats/Min)</th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
<th>Maximum Aortic Diameter (mm)</th>
<th>TL Area (cm²)</th>
<th>FL Area (cm²)</th>
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<td>CDTAD (n = 10)</td>
<td>61.5 (SD 13.4)</td>
<td>132.1 (SD 19.7)</td>
<td>81.3 (SD 15.6)</td>
<td>39.5 (SD 43.8)</td>
<td>7.81 (SD 2.91)</td>
<td>3.99 (SD 1.89)</td>
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<tr>
<td>Control (n = 9)</td>
<td>64.9 (SD 8.8)</td>
<td>118.3 (SD 9.7)</td>
<td>75.6 (SD 7.3)</td>
<td>19.0 (SD 20.0)</td>
<td>6.18 (SD 1.95)</td>
<td>3.65 (SD 0.98)</td>
</tr>
</tbody>
</table>

* \( P < 0.001 \) vs. CDTAD, † \( P < 0.05 \) vs. CDTAD (TL). AscAO = mid ascending aorta; CDTAD = chronic descending thoracic aortic dissection; DBP = diastolic blood pressure; FL = false lumen; HR = heart rate; ProxAO, MidAO, and DistAO = midpoints of the proximal, middle, and distal third of the descending thoracic aorta, respectively; SBP = systolic blood pressure; SD = standard deviation; TL = true lumen.
Figure 1. Percentage of total velocity at peak systole stratified by velocity levels and aortic locations. Panel A. Asc_{AO}. Panel B. Prox_{AO}. Panel C. Med_{AO}. Panel D. Dist_{AO}. *p < 0.001 vs. chronic descending thoracic aortic dissection (CDTAD) true lumen (TL), †p < 0.05 vs. CDTAD TL.

Figure 2. Blood flow rate per aortic lumen area (mL/s/cm²) curves standardized by one cardiac cycle. Panels A-D. Control participants. Panels E-H. TL of CDTAD patients. Panels I-K. False lumen (FL) of CDTAD patients. *p < 0.01 vs. control (peak systole), †p < 0.05 vs. control (peak systole), ‡p < 0.05 vs. CDTAD TL (peak systole).
characteristics between CDTAD patients and healthy participants. Our unique multi-VENC approach allowed an accurate assessment of FL and TL flow. Current guidelines do not include aortic hemodynamic and flow characteristics as indicators for intervention in CDTAD [1], as independent of their method of derivation, their relationship with aortic disease progression and physiology remains unclear [13].

Figure 3. PI of control participants and CDTAD patients. *P < 0.05.

Figure 4. Sagittal pathline views at the isolated thoracic aorta during peak systole within one cardiac cycle. Aortic planes are demonstrated and color-coded by blood flow velocity. Panel A. Control participant. Panel B. CDTAD patient ID 1 with TL and false lumen FL pathlines isolated (TL sits along the inner curvature of the aortic arch).
Figure 5. Pathline image at the descending thoracic aorta in CDTAD patient ID 7 demonstrating peak systole within one cardiac cycle. The TL (along the inner curvature of the aortic arch) and FL pathlines are isolated.

Figure 6. Isolated TL pathline image of the thoracic aorta in CDTAD patient ID 10 at peak systole within one cardiac cycle. The bare volume within the descending aorta represents the extent of the FL. Panel A. Sagittal ‘candy cane’ view. Panel B. View from caudal aspect along longitudinal plane.
Computational fluid dynamic modeling has shown that increased flow and greater wall shear stress are associated with aortic aneurysm expansion in the setting of Type B aortic dissection [14]. Assessment of pulse wave velocity and wall shear stress are among the novel applications of MRI for the measurement of aortic pulsatile flow [15, 16].

**PI as a Predictor of Adverse Events**

We used a PI derived from 4D-flow data to characterize flow dynamics within the TL and FL. Although not prognostic for CDTAD, abnormal PI is predictive of aneurysm expansion in porcine models of abdominal aortic aneurysm [17] and in carotid artery aneurysms [11]. PI is inversely proportional to wall shear stress in the vasculature of hypertensive patients [18], and low wall shear stress is associated with sites of atherogenesis in the aorta as measured by 4D-flow MRI [19]. Elevated PI correlates with increased downstream vascular resistance at other arterial locations, including the pulmonary artery [20] and renal arteries [21]. In CDTAD, greater PI within the FL may be indicative of elevated downstream resistance secondary to thrombosis formation or aortic branch occlusion. We found that PI was reduced throughout the FL, consistent with a chronically dilated lumen with minimal thrombosis and multiple distal exit sites. Among healthy participants, greater PI was associated with reduced aortic lumen area. Although not assessed in our study, the use of after-load reduction medication may also influence the PI. Thus, a derived PI from 4D-flow MRI data may serve as an adjunct to existing predictors of future adverse events in CDTAD.

**Velocity and Flow Profiles in CDTAD**

**Figure 7.** Isolated TL pathline image of the thoracic aorta in CDTAD patient ID 5 at peak systole within one cardiac cycle. The bare volume within the descending aorta represents the extent of the FL. Inset highlights TL and FL communication.
The velocity profile within the FL was markedly dampened compared with that of the TL. Overall blood flow via the FL was significantly less than that via the TL in our CDTAD group, and the transit time of blood via the FL was markedly prolonged with significant blood flow reversal. This was observed despite correction for individual aortic lumen short axis area. These results highlight a particular application of 4D-flow MRI whereby the assessment of aortic blood flow within the TL and FL can be performed separately. This is particularly valuable for the surveillance of CDTAD, in which the distinction between FL thrombosis and slow flow can influence future risk of adverse events [4].

We found that the percentage of blood flow reversal was significantly higher in the FL than in the TL in CDTAD patients, consistent with previous reports [6]. Additionally, communication between the FL and TL was detected in CDTAD patients. Unlike with conventional MRI-based blood flow assessment, the acquisition of these properties when utilizing 4D-flow is potentially available anywhere within the acquired field of view during post-scan processing.

At the ascending aorta, overall blood velocity was reduced in the control and CDTAD groups despite no significant difference in cross-sectional area. The maximal blood flow rate was not significantly different between groups and likely reflects their normal cardiac function (as measured by LVEF). Patients in the CDTAD group had previously undergone graft replacement of the ascending aorta as well as aortic valve replacement. In our cohort, such prior intervention may have influenced blood velocity but did not appear to influence maximal blood flow rate. Previous investigators of 4D-flow MRI show that wall shear stress and non-laminar blood flow are elevated in this setting following more proximal aortic or valvular surgical intervention [22].

Utility of Pathline Analysis

Previous reports using 4D-flow MRI to assess aortic blood flow have included semi-qualitative assessment of blood flow helicity, defined as corkscrew-like movement of encoded pathlines [6, 23]. Due to our clinically heterogeneous CDTAD group, we did not formally assess flow helicity. Although helical blood flow is positively correlated with aortic enlargement, its use as a prognostic marker is yet to be confirmed [5, 16]. However, our generated pathline images demonstrate additional potential prognosticators, including quantifiable FL blood flow, TL and FL communication, and localized differences in blood flow velocity between normal and chronically dissected aortas. Thus, a multi-VENC approach can allow the differentiation of fast and slow flow domains of the TL and FL.

Study Limitations

This study has several limitations. CDTAD patients were older than healthy participants, which may have contributed to some of the differences in observed blood flow characteristics. This difference may impact hemodynamics as a result of decreased aortic wall compliance with age. Additionally, our sample sizes were small. Furthermore, CDTAD patients presented with a Stanford Type A aortic dissection, and such patients show a different natural history than patients who initially presented with a Stanford Type B dissection [24].

Conclusion

We demonstrate that 4D-flow MRI allows identification of detailed compartmental quantitative blood flow values, including pulsatility, velocity, flow rate, and flow direction, within the TL and FL of CDTAD patients that differ significantly from those of healthy participants. The addition of pathline visualization may allow an improved appreciation of TL and FL hemodynamics, particularly when using a multi-VENC 4D-flow approach. As reliance upon aortic diameter alone as an indicator of intervention is insufficient [25], 4D-flow MRI could serve as a useful adjunct to the risk stratification of these patients. Longitudinal studies are required to determine the clinical relevance of this imaging modality.

Acknowledgements

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Conflict of Interest

The authors have no conflict of interest relevant to this publication.

References


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Abstract

We present the case of a 64-year-old male who presented to the emergency department with bilateral limb ischemia and paralysis for approximately 1 hour. Computed tomographic angiography demonstrated occlusion of the infrarenal aorta beginning just above the patient’s known abdominal aortic aneurysm (AAA) and extending into both common iliac arteries. He was emergently treated via open repair of the AAA with a Gore-Tex tube graft, bilateral common iliac thrombectomies, and bilateral lower extremity four-compartment fasciotomies. Post-operatively, he had monophasic signals in both posterior tibial arteries, neither of which was present before the operation. During recovery, he developed an ileus but otherwise did not have complications. He was discharged to rehabilitation on post-operative day 15.

Key Words:
Thrombosis • Abdominal aortic aneurysm • Paralysis

Introduction

Aneurysms are defined as focal dilatations at least 50% larger than the expected normal arterial diameter [1]. A practical working definition of an abdominal aortic aneurysm (AAA) is a transverse diameter of ≥ 3 cm and of a common iliac aneurysm is a transverse diameter > 1.8 cm based on average values for normal individuals. The normal aortic diameter gradually decreases from the thorax (28 mm in men) to the infrarenal location (20 mm in men) [2]. Rather than being termed atherosclerotic, AAAs are more accurately referred to as degenerative or non-specific in etiology. Degenerative aneurysms account for more than 90% of all infrarenal AAAs [3].

In contrast to peripheral aneurysms, acute thrombosis of an AAA is a rare sequela [4]. Peripheral aneurysms have an inherent risk of acute thrombosis, which is commonly described for aneurysms of the popliteal artery. An intraluminal thrombus is present in approximately 70–80% of patients with an AAA. This thrombus is usually of no significance to blood flow and may even have some protective effects against the wall stress associated with an AAA [5]. Occlusion of the AAA from an intraluminal thrombus is an extremely rare pathology with an accompanying high morbidity and mortality. This entity was first described in a case report by Schumaker in 1959 and again by Janetta et al. in 1961 [4, 6], and the first case series was published by Johnson et al. in 1974 [7]. As described by Criado, there are three proposed mechanisms that precipitate acute occlusion of an AAA [8]. The first proposed mechanism stems from occlusive iliac artery disease, leading to aneurysm outflow obstruction as the most common causative
factor. The second proposed mechanism occurs after cardioaortic embolization, leading to distal aortic occlusion by a saddle embolus. The third and least common etiology is progression of intrasaccular mural thrombus, which becomes obstructive to blood flow if a sudden change in position occurs, such in a patient fall. Furthermore, in the age of endovascular aortic aneurysm repair (EVAR), one may argue that a fourth potential mechanism may be the clot being provoked by foreign graft material.

Clinical presentations of acute thrombosis of an AAA are often relatively easy to diagnose secondary to acute onset of severe symptoms. In Criado’s review, mottling was described to the level of the iliac crest or umbilicus in 14 out of 26 cases [8]. Also, in Johnson’s et al. review, paraplegia was described at the time of presentation in 10 out of 17 patients [1].

Case Presentation

A 64-year-old man presented to the emergency room (ER) complaining of sudden onset bilateral lower extremity pain and numbness that had progressed to paralysis, paraesthesias, and stabbing back pain at the time of presentation, which was approximately 1 hour after onset. He did not have a significant past medical history; although, he was a smoker (30 pack-years). His initial vital signs while in the ER were a blood pressure of 139/88, heart rate of 102 bpm, respiratory rate of 20, and oxygen saturation of 94% on 4-L nasal cannula. Physical exam demonstrated mottled bilateral lower extremities with the right being more mottled than the left. Motor strength was diminished in the left (3/5) and absent on the right (0/5). The ER physician was unable to palpate or appreciate a doppler signal bilaterally in the popliteal, posterior tibial, and dorsalis pedis arteries. The patient was alert and oriented but diaphoretic and in obvious distress. Neurological exam was significant for complete sensory loss in the right lower extremity and diminished in the left. Cardiovascular, pulmonary, and abdominal exams were within normal limits. Duplex exam was ordered, but it was unable to be completed secondarily to low flow in the common femoral arteries bilaterally. Velocities ranged from 5.4–6.9 cm/sec and 9.6–11.6 cm/sec on the right and left side, respectively. The patient subsequently underwent a computed tomography angiography (CTA) scan of his chest, abdomen, and pelvis. This revealed a 5-cm infrarenal aortic aneurysm that was acutely thrombosed with extension into the bilateral common iliac arteries (Figures 1 and 2). There was reconstitution in both extremities at the level of the internal and external iliac arteries. Complete blood count and chemistries were drawn, and all were within normal limits.

Consultation with vascular surgery led to a recommendation of emergent operative intervention. The patient gave informed consent and was transferred to a nearby hospital for definitive management. He remained hemodynamically stable during this time; although, the weakness in his left

Figure 1. Computed tomography angiography coronal reformat showing thrombosed abdominal aortic aneurysm.
lower extremity progressed to paralysis at the time of transfer.

The patient underwent open repair of his AAA with a Gore-Tex tube graft, bilateral common iliac thrombectomies, left common femoral artery exploration, open thrombectomy with repair, and bilateral lower extremity four-compartment fasciotomies. Upon completion of the surgery, he had monophasic signals in the posterior tibial arteries bilaterally that were not appreciated at the start of the surgery. He remained intubated and was taken to the cardiothoracic intensive care unit in stable condition. On post-operative day 1, he was extubated and was able to move both lower extremities. The posterior tibial signals remained intact. He remained in the cardiothoracic intensive care unit for 2 days before being transferred to the floor. Despite low output from his nasogastric tube, which was removed on post-operative day 3, his recovery was prolonged by a post-operative ileus, necessitating reinsertion of the nasogastric tube as well as parenteral nutrition. The patient continued to work with physical therapy and maintained perfusion and function of his bilateral lower extremities. He was ultimately discharged to an acute rehabilitation facility on post-operative day 15. Upon discharge, he maintained signals bilaterally in the posterior tibial arteries and ambulated with a walker. He has since followed up as an outpatient and is doing well. Three-month CTA confirmed graft patency (Figure 3).

Discussion

Unlike peripheral aneurysms, acute occlusion of an AAA is a rare entity that carries high morbidity and mortality when diagnosis and treatment is delayed [1, 2, 4, 6-9]. Mortality rates can reach as high as 46%, which approaches that of aneurysmal rupture [8]. Regardless of etiology, both direct consequences of acute ischemia as well as those associated with revascularization subject the patient to considerable risk [9]. Skeletal muscle injury often occurs within 2 hours of ischemia, and in the presence of paralysis, ultimate limb loss is likely within 6 to 8 hours after onset of acute arterial occlusion [9, 10]. Our patient presented with signs of severe lower limb ischemia that began as severe abdominal pain accompanied by paresthesia of the lower extremities. This rapidly progressed to mottling of the lower extremities up to the iliac crest level, and his paresthesia rapidly progressed to complete paralysis of the bilateral lower extremities. The initial response to sudden occlusion of an artery is distal spasm in the main channels and collaterals, which is thought
to prevent further thrombosis distal to the occlusion [7, 9]. The motor deficit in the lower extremities can be explained in part by anterior spinal cord syndrome secondary to acute occlusion of lumbar arteries [9, 11, 12]. The motor deficit can also theoretically be attributed to sudden acute ischemia to the lower extremity muscles, ultimately leading to neuronal and muscle ischemia. Therefore, we postulate that the paralysis was multifactorial in nature, from both an anterior spinal cord syndrome as well as prolonged skeletal muscle ischemia. Although the diagnosis of a thrombosed aorta by clinical signs and symptoms is not difficult, the clinician must differentiate thrombosed AAA from a saddle embolus [11]. This is paramount because the therapeutic intervention for a saddle embolus is a transfemoral embolectomy, whereas an abdominal aortic occlusion requires revascularization [10-12]. To prevent further clot propagation, it is vital to initiate systemic heparinization after the diagnosis has been established in either situation [1, 3, 4, 6-8, 10, 11]. Emergent surgical repair is mandatory and usually requires thrombectomy and replacement of the aneurysm with graft; however, if this is not feasible, an axillobifemoral bypass should be performed [11, 13].

Although thrombosis of an AAA is uncommon, its mortality approaches that of a ruptured AAA. There appears to be no correlation between size and the likelihood of thrombosis, although most reported thromboses have been in smaller aneurysms [12, 13]. Once the diagnosis has been established, systemic heparinization is crucial for preventing further propagation of the clot and should be followed by emergent revascularization.

**Conflict of Interest**

The authors have no conflict of interest relevant to this publication.

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**References**


7. Johnson JM, Gaspar MR, Movius HJ, Rosenthal JJ. Sudden complete thrombosis of...


Case Report

Thoracic Endovascular Aortic Repair for Aortoesophageal Fistula after Covered Rupture of Aortic Homograft
A Durable Option?

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Abstract

A 63-year-old woman underwent replacement of the aortic root, ascending aorta, and partial arch due to Type A aortic dissection. Shortly thereafter, a replacement of the distal aortic arch and descending aorta was performed. Three years later, the patient developed an aortoesophageal fistula (AEF) resulting in re-replacement of the distal aortic arch and proximal descending aorta with a cryopreserved aortic homograft. Six weeks post-discharge, the patient was readmitted due to recurrent AEF. A thoracic endovascular stent graft was implanted to cover the aortic rupture, followed by correction of an esophageal lesion. The patient was monitored closely over time.

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Key Words

TEVAR • Aortoesophageal fistula • Graft infection

Introduction

Although it is a rare clinical condition, aortoesophageal fistula (AEF) presents problems to therapy because of the high rates of morbidity and mortality associated with surgical management. Therefore, less invasive approaches that reduce perioperative mortality have been evaluated, with special attention given to thoracic endovascular aortic repair (TEVAR). However, this technique has important limitations in treating AEF, mainly due to a high risk of graft contamination. Here, we describe a case of recurrent AEF treated with TEVAR stenting of a cryopreserved aortic homograft replacement of the aortic arch and Hemashield prosthesis replacement of the descending aorta.

Case Presentation

A 63-year-old woman underwent emergency replacement of the aortic root with a biological valve conduit (Medtronic Freestyle-Aortic-Root Modell 995, Gr. 23 mm, Medtronic Inc., Minneapolis, MN) as well as ascending aorta and partial arch replacement (Hemashield prosthesis, 26 mm) due to Type A aortic dissection. Two months later, the patient presented with a rapidly enlarging false lumen of the remaining aortic arch and proximal descending aorta. A replacement of the distal aortic arch (one-branch Hemashield prosthesis; 30 mm) and descending aorta (Hemashield prosthesis, 22 mm) was performed under circulatory arrest via a left thoracotomy.
The postoperative course was uneventful, and the patient was discharged on postoperative day 21.

Three years later, the patient presented with recurrent hematemesis and anemia. Esophagogastroduodenoscopy (EGD) identified an esophageal ulcer with evidence of slight ongoing bleeding. Computed tomography (CT) examination confirmed the presence of an AEF (Figure 1A). An emergency re-replacement of the distal aortic arch and proximal descending aorta was performed with a cryopreserved aortic homograft (CryoLife, Kennesaw, GA; 25 mm). The adjacent esophageal lesion was closed during circulatory arrest with interrupted suture. An endoluminal esophageal stent (28/10 mm, Leufen-Medical-GmbH) was inserted at the end of the procedure. The stent was removed 2 months later, and control esophagogastroduodenoscopy demonstrated ulcerations with no visible fistula.

Six weeks post-discharge, the patient was readmitted due to massive hematemesis. Recurrent AEF was suspected, and the patient underwent emergent esophagogastroscopy. Active bleeding at the primary AEF location was observed, and a Segstaken-Blake more tube was inserted. A CT scan was suspicious for a new aortic rupture at the previous homograft site (Figure 1B). An interdisciplinary team meeting resulted in the decision to place a TEVAR stent over the aortic rupture (32/32/180, Valiant-Captivia, Medtronic, Santa Rosa, CA; Figure 2). After stabilization: mediastinal debridement, esophageal resection, and a gastric pull-up procedure with a cervical anastomosis (end-to-end, double-row suture with 3-0 Vicryl, Ethicon, Somerville, NJ). During the recovery phase, bronchoscopy revealed a 5-mm perforation of the trachea and a large amount of surrounding pus. Mediastinitis (secondary to Enterococcus faecalis and Streptococcus anginosus) was diagnosed and treated with local debridement, vacuum-assisted closure therapy, and broad-spectrum antibiotics.

The table below provides a summary of the patient’s admission dates, indications, and treatment strategies:

<table>
<thead>
<tr>
<th>Admission Date</th>
<th>Indication</th>
<th>Treatment</th>
</tr>
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| Dec 2009       | Type A aortic dissection                        | • Full sternotomy, central cannulation, 26°C  
|                |                                                | • Aortic root replacement with a biological valve conduit (Medtronic Freestyle-Aortic-Root Modell 995, Gr. 23 mm, Medtronic Inc, Minneapolis, MN)  
|                |                                                | • Ascending aorta and partial arch replacement (Hemashield prosthesis, 26 mm)  
|                |                                                | • Selective antegrade cerebral perfusion (sACP, 1000 mL/min)  
| Feb 2010       | Rapidly enlarging aneurysma dissectans of the remaining aortic arch and proximal descending aorta | • Left lateral thoracotomy, femoro-femoral bypass, left ventricular venting, circulatory arrest at 25°C, antegrade cardioplegia administration using a Foley catheter (1800 mL Brettschneider solution), sACP (750 mL/min)  
|                |                                                | • Replacement of distal aortic arch (one-branch Hemashield prosthesis, 30 mm) and descending aorta (Hemashield prosthesis, 22 mm)  
| June 2013      | Aortoesophageal fistula (Figure 1A)            | • Full sternotomy, right axillary artery cannulation, sACP (750 mL/min), 25°C  
|                |                                                | • Re-placement of aortic arch and proximal descending aorta with a cryopreserved aortic homograft (Cryolife, Kennesaw, GA, 25 mm)  
|                |                                                | • Closing of esophageal lesion with interrupted suture  
|                |                                                | • Insertion of endoluminal esophageal stent (28/10 mm, Leufen-Medical-GmbH)  
| Oct 2013       | Recurrent aortoesophageal fistula, new aortic rupture at the previous homograft site (Figure 1B) | • Thoracic endovascular aortic repair stent over the aortic rupture (32/32/180, Valiant-Captivia, Medtronic, Santa Rosa, CA; Figure 2)  
|                |                                                | • After stabilization: mediastinal debridement, esophageal resection, and a gastric pull-up procedure with a cervical anastomosis (end-to-end, double-row suture with 3-0 Vicryl, Ethicon, Somerville, NJ)  
| Aug 2014       | Infection of the stent graft (Figure 3)        | • Patient refused aggressive treatment and was discharged on analgesia and broad-spectrum antibiotics for palliative care  

Nozdrzykowski, M. et al Minimally Invasive Approach for AEF
The patient recovered well and was put on long-term antibiotics to prevent recurrent septicemia. A CT angiogram 5 months later confirmed satisfactory position of the implanted stent graft and showed no signs of endoleak or infection. Therefore, antibiotic therapy was discontinued.

Ten months after the last admission, the patient experienced a persistent high-grade fever. A blood test revealed an elevated leukocyte count and highly elevated C-reactive protein level. CT revealed air around the stent grafts, suggesting infection (Figure 3). After being informed that any
further operative treatment (consisting of removal of the infected stent grafts and replacement of the descending aorta or extra-anatomic bypass grafting) was likely to prove fatal; the patient and her family decided to refuse the aggressive treatment. The woman was discharged on analgesia and broad-spectrum antibiotics for palliative care.

Discussion

Patients who undergo surgery for aortic dissection often have residual dissected aortic tissue that may become a source of late complications. Repeat surgery is required in approximately 12–30% of patients, usually due to extension of dissection, aneurysm formation, or infection [1]. The very rapid progression of the distal arch and proximal descending aorta in our patient was caused by patent false lumen. CT scanning and intraoperative findings excluded infection of the prosthesis or presence of septic false aneurysm.

During follow-up, our patient developed an AEF at the site of the anastomosis between the original aortic arch replacement graft and the subsequent distal arch/descending aortic replacement graft. Secondary AEF following surgery is uncommon (4.8%), with 50% of cases occurring after aortic surgery [2]. The mechanism of AEF after conventional surgery involves rupture of the prosthesis, dehiscence of the repair, direct erosion of the graft into the esophagus, or local infection. Our intraoperative findings suggested that the cause of the AEF was dehiscence of the repair due to infection, which caused aortic rupture and secondary penetration into the esophagus. On the other hand, previous replacement of the descending aorta implicates the occlusion of esophageal arteries arising directly from the aorta with impaired tissue healing and possible esophageal ischemia [3].

To treat the first AEF, we used a cryopreserved homograft to replace the distal aortic arch. Recent studies of AEF and aortoenteric fistulae demonstrate the superiority of cryopreserved aortic allografts because they are more resistant to infection [4]. However, homografts are not always immediately available in emergency situations. An alternative solution for orthotopic vascular reconstruction is use of self-made xenopericardial tube grafts constructed from a patch [5]. Recently, investigators from the European Registry of Endovascular Aortic Repair Complications presented the results of different treatment strategies for AEF following TEVAR at the 27th European Association for Cardio-Thoracic Surgery Annual Meeting in Vienna and concluded that radical esophagectomy and extensive aortic reconstruction is the only durable approach for this fatal complication.

When our patient re-presented with recurrent AEF, our interdisciplinary team decided against a fourth aortic arch operation because of the patient’s generally poor condition and the excessive operative risk. We therefore opted to perform TEVAR as a life-saving intervention, which was followed by esophageal resection and a gastric pull-up procedure in one stage with a cervical anastomosis and long-term antibiotic therapy. This treatment strategy resulted in immediate control of aortic bleeding and a complete regression of the recurrent AEF, but may have increased the subsequent risk of infection. Despite correction of the esophageal lesion, the efficacy of our therapy was limited by mediastinal infection, which required multiple surgical interventions. Prolonged postoperative antibiotic therapy is advocated as a key component for success, but there is currently no consensus on the appropriate duration of antibiotics in this group of patients [6]. Most commonly parenteral antibiotics are given for 2 to 8 weeks post-procedure, but whether lifelong oral antibiotics are necessary is debatable [6]. Most recently, Canaud et al. reviewed the outcomes of TEVAR for AEF and reported that prolonged antibiotic treatment (i.e., greater than 4 weeks) was associated with significantly lower aortic mortality [7].

In our opinion, TEVAR for AEF can be used only as a bridge to definitive open aortic surgery or as combined treatment with mediastinal debridement, mediastinal drainage, and/or esophageal resection, particularly in patients in poor general condition. For long-term durability, it is necessary to resect the aorta and esophagus simultaneously to prevent prosthesis re-infection [8]. Based on our experience, stent graft infection can occur many months after the procedure. Thus, prolonged antibiotic therapy and life-long surveillance are mandatory in these patients regardless of symptoms or clinical signs of infection. However, additional clinical reports exclusively focusing on recurrent AEF are required to determine the optimal management strategy for this challenging problem.
Conflict of Interest

The authors declare no conflict of interest relevant to this publication.

References


Cite this article as: Nozdrzykowski M, Garbade J, Leinung S, Schmidt A, Mohr FW, Borger MA. Thoracic Endovascular Aortic Repair for Aortoesophageal Fistula after Covered Rupture of Aortic Homograft: A Durable Option? AORTA (Stamford). 2017;5(3):96-100. DOI: https://doi.org/10.12945/j.aorta.2017.16.044
A 75-year-old woman was admitted to the emergency department with severe and sudden chest pain. Transthoracic echocardiogram showed an unusual case of aortic dissection Stanford Type A with complete circumferential detachment of the ascending aorta intima. An intussusception of the intima flap into the left ventricular outflow tract was also observed. This case presents a very rare surgical treatment involving root repair using tissue adhesives for a left ventricular intimal flap.

Abstract

A 75-year-old woman was admitted to the emergency department with severe sudden chest pain. Transthoracic echocardiogram showed an unusual case of aortic dissection Stanford Type A with complete circumferential detachment of the ascending aorta intima. An intussusception of the intima flap into the left ventricular outflow tract was also observed. This case presents a very rare surgical treatment involving root repair using tissue adhesives for a left ventricular intimal flap.

Key Words

Dissection • Aorta • Intimal flap • Intussusception

A 75-year-old woman was admitted to the emergency department with severe sudden chest pain, signs of malperfusion of the right arm, and left-sided hemiplegia. Transthoracic echocardiogram showed an acute aortic dissection Stanford Type A. Preoperative transesophageal echocardiography (Video 1; see supplemental Video 1 at https://doi.org/10.12945/j.aorta.2016.16.048.vid.01) demonstrated a complete circumferential dissection of the ascending aorta with remaining attachment in close proximity to both coronary ostia. An invagination of the complete intimal flap across the aortic valve into the left ventricle was seen during diastole (Figure 1A). During systole, the flap was pumped into the left ventricular outflow tract (Figure 1B). As a result of this intussusception, the patient showed severe aortic insufficiency. Furthermore, a dissection of the brachiocephalic trunk with occlusion of the true lumen was observed. Despite severe aortic insufficiency and repetitive occlusion of both coronary ostia, the patient showed no periods of hypotension. There was no need for inotropes or intubation.

Due to impending hemodynamic collapse, emergency surgery was performed. After opening the pericardium, the floating intimal membrane was visible under a transparent adventitial layer. The
The aortic root was repaired using tissue adhesives, and replacement of the supracommissural ascending aorta was performed. The existing intimal flap was additionally fixed with the main graft to the root suture line. Subsequently, the brachiocephalic trunk and aortic hemiarch were replaced under deep hypothermic circulatory arrest. Postoperative computed tomography scanning after surgery confirmed adequate perfusion of the supra-aortic vessels and complete occlusion of the false lumen. The patient was transferred to the admitting clinic after 4 days. Preoperatively existing aphasia improved significantly after 3 weeks of rehabilitation.

**Conflict of Interest**

The authors have no conflict of interest relevant to this publication.

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**Figure 1.** Panel A. Long-axis echocardiogram showing intussusception of an intimal flap into the left ventricle. Panel B. Short-axis echocardiogram showing circumferential dissection of the ascending aorta with the intimal flap in the left ventricular outflow tract.
Page for the General Public

Anneke Damberg, MD
(on behalf of the Editorial Office)

The following pages summarize and review this issue’s articles for an audience without a background in medicine or research.

Original Research Articles

Paul C. Tang et al.: “Coronary Events in Patients Presenting for Repair of Acute Type A Aortic Dissection”

When a patient undergoes emergent surgery for type A aortic dissection, in which a tear occurs in the layers of the body’s main artery, there is usually no time to perform an additional study of the state of the patients coronary arteries, the vessels which supply the heart muscle with blood. The authors of this study analysed how often these patients also have disease of their coronary arteries and how often this causes problems. Studying 31 patients with aortic dissection and 123 patients who underwent planned surgery for aortic aneurysm (dilation), they came to the conclusion that coronary heart disease was missed only in very few patients, and that not screening for it before surgery did not decrease survival due to non-aortic causes. They therefore conclude that it is not necessary to delay emergent surgery for aortic dissection to perform coronary angiography to screen for coronary heart disease. However, due to the design of the study and limited patient numbers, further and larger investigations are necessary.

Andrew G. Sherrah et al.: “Multi-Velocity Encoding Four-Dimensional Flow Magnetic Resonance Imaging in the Assessment of Chronic Aortic Dissection”

Patients who suffered aortic dissection, in which a tear occurs in the layers of the body’s main artery, need to undergo follow-up imaging after surgery to see how the disease progresses. The authors investigated a new imaging method using MRI (magnetic resonance imaging), in which they do not only use steady images but also visualize blood flow dynamics. Blood flow patterns and pulsations were studies in 10 patients with dissection and 9 healthy subjects. There were significant differences in blood flow patterns between healthy patients and patients with aortic dissection. This imaging technique could be helpful in the follow-up of patients with aortic dissection, even though its significance and usefulness needs to be further studied.

Case Reports

T. Joseph Watson et al. “Acute Bilateral Lower Extremity Paralysis Secondary to Acute Thrombosis of an Infrarenal Abdominal Aortic Aneurysm”

Watson et al. Describe a case of a patient with an abdominal aortic aneurysm, a dilation of the body’s main artery in the abdomen, which was suddenly occluded by blood clots which led to paralysis of the legs. The patient underwent open surgery to replace the aneurysm and reopen the clotted vessels. Overall, it is common that blood clots develop in an abdom-
inal aneurysm but they rarely lead to occlusion of blood flow. The paralysis is mainly due to impaired blood flow to the spinal cord. Patients need to receive medication to impair blood coagulation and undergo urgent surgery to restore blood flow.

Michal Nozdrzykowski et al: “Thoracic Endovascular Aortic Repair for Aortoesophageal Fistula After Covered Rupture of Aortic Homograft: A Durable Option?”

The authors report a case of a patient who had to undergo emergent surgery to replace parts of her aorta, the body’s main artery. Three years later, she developed an aortoesophageal fistula. In this rare but serious complication, a connection between the aorta and the esophagus develops, which can cause life threatening bleeding. Most likely, the complication was due to an infection. The patient was operated on again and her aorta replaced with a piece of donor aorta. When the complication recurred, it was treated with a stent graft, a prosthesis inserted into the vessel via a vessel in the groin, and repair of the esophagus. The postoperative course was complicated, and when an infection recurred one year later surgery was deemed too dangerous and the patient discharged on palliative care. The authors discuss several treatment options for an esophageal fistula. With regard to using stent grafts, they only see it as a bridging option to definitive surgery or in patients with poor general condition, mainly due to the high risk of infection.

What I Did

Kaufeld et al.: “Left Ventricular Intussusception of an Intimal Flap in an Aortic Dissection Type A.”

Kaufeld et al. describe the case of a patient who had a type A aortic dissection, in which a tear occurs in the layers of the body’s main artery very close to its origin at the heart. In this case, the tear involved the whole circumference of the aorta. In this rare case, the flap of the inner layer even protruded into the heart itself, causing severe leakage of a heart valve, the aortic valve. The patient underwent surgical repair and recovered quite well, even though a preoperative stroke caused lasting speech impairment.
Upcoming Meetings

AORTA, June 2017, Volume 5, Issue 3:105
DOI: https://doi.org/10.12945/j.aorta.2017.17.090

List of Upcoming Meetings

August 2017

1. Heart Valve Forum 2017  
   August 18-19, 2017  
   Seoul, South Korea  
   Meeting information available at: www.valveforum.org/valveforum

September 2017

1. 27th Annual Congress of the World Society of Cardiovascular and Thoracic Surgeons  
   September 1-3, 2017  
   Astana, Kazakstan  
   Meeting information available at: www.wscts.net

2. 11th Current Trends in Aortic, Cardiac and General Thoracic Surgery  
   September 8-9, 2017  
   Houston, TX, United States  
   Meeting information available at: www.cme.texasheart.org

   September 8-10, 2017  
   Boston, MA, United States  
   Meeting information available at: www.nesvs.org/annual-meeting.html

4. Focus: Valve 2017  
   September 10-13, 17  
   Innsbruck, Austria  
   Meeting information available at: www.focusvalve.org

5. 2017 Canadian Society for Vascular Surgery Annual Meeting  
   September 15-16, 2017  
   Banff, Alberta, Canada  
   Meeting information available at: canadianvascular.ca/Annual-Meeting

6. Annual Conference on Heart Diseases  
   September 18-19, 2017  
   Toronto, ON, Canada  
   Meeting information available at: heartdiseases.alliedacademics.com

7. ESVS 31st Annual Meeting  
   September 19-22, 2017  
   Lyon, France  

8. STS/EACTS Latin America Cardiovascular Surgery Conference  
   September 21-22, 2017  
   Cartagena, Colombia  
   Meeting information available at: www.sts.org/latinamerica

9. 37th Annual Cardiothoracic Surgery Symposium  
   September 28-October 1, 2017  
   San Diego, CA, United States  
   Meeting information available at: www.crefmeeting.com