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Difference in hemodynamic and wall stress of ascending thoracic aortic aneurysms with bicuspid and tricuspid aortic valve

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ABSTRACT

The aortic dissection (AoD) of an ascending thoracic aortic aneurysm (ATAA) initiates when the hemodynamic loads exerted on the aneurysmal wall overcome the adhesive forces holding the elastic layers together. Parallel coupled, two-way fluid–structure interaction (FSI) analyses were performed on patient-specific ATAAs obtained from patients with either bicuspid aortic valve (BAV) or tricuspid aortic valve (TAV) to evaluate hemodynamic predictors and wall stresses imparting aneurysm enlargement and AoD. Results showed a left-handed circumferential flow with slower-moving helical pattern in the aneurysm's center for BAV ATAAs whereas a slight deviation of the blood flow toward the anterolateral region of the ascending aorta was observed for TAV ATAAs. Blood pressure and wall shear stress were found key hemodynamic predictors of aneurysm dilatation, and their dissimilarities are likely associated to the morphological anatomy of the aortic valve. We also observed discontinues, wall stresses on aneurysmal aorta, which was modeled as a composite with two elastic layers (i.e., inhomogeneity of vessel structural organization). This stress distribution was caused by differences on elastic material properties of aortic layers. Wall stress distribution suggests AoD just above sinotubular junction. Moreover, abnormal flow and lower elastic material properties that are likely intrinsic in BAV individuals render the aneurysm susceptible to the initiation of AoD.

1. Introduction

Aortic dissection (AoD) is defined as the progressive separation of the layers of the thoracic aortic wall. Specifically, an intimal tear typically originates above the sinotubular junction (STJ) by permitting the blood to enter the aortic wall and progressively separating the medial plane along the axial direction of the aorta. Although AoD is pathologically distinct from ascending thoracic aortic aneurysm (ATAA), thoracic aneurysms are prone to developing dissection (Davies, 1998; Ince and Nienaber, 2007). A cause of difficulties in diagnosis, reported rates of 3–4 cases per 100,000 persons per year are probably underestimates of the true incidence of AoD. The morbidity risk for emergent surgery remains 24% worldwide according to data from the International Registry of Acute Aortic Dissection (Rampoldi et al., 2007).

Patients with ATAA frequently have bicuspid aortic valve (BAV) that is the most common heart defect (Ward, 2000) compared with the morphological, normal tricuspid aortic valve (TAV), even when matched for the degree of aortic stenosis or regurgitation (Hahn et al., 1992). An association between ATAA and BAV has been confirmed in numerous studies (Cripe et al., 2004; Keane et al., 2000). Furthermore, BAV individuals (1–2% of the population) with thoracic aneurysms have 9-fold higher risk of AoD (Davies et al., 2002).

The most relevant theory on the pathogenesis of ATAA associated to BAV suggests that a genetic or developmental defect in the proximal aortic tissue leads to weakness of aortic wall, thus imparting the risk of aneurysm formation (Milewicz et al., 2008). However, a flow-mediated mechanism for aneurysm dilatation should not be ruled out. Indeed, there are echocardiographic observations of increased blood systolic velocities at the aneterolateral region of the ascending aorta (AoA) in patients with BAV (Bauer et al., 2006) and specific segments of ATAA varing with the type of aortic valve leaflet fusion (Fazel et al., 2008) that cannot be easily explained by a genetic theory alone. Additionally, BAV ATAA

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typically bulge asymmetrically toward the right, greater curvature of the aorta, and this is possibly explained by abnormal flow patterns developing in individuals with BAV as observed by time-resolved three-dimensional phase-contrast MR imaging (also know as four-dimensional (4D) flow MR imaging). Hemodynamic disturbances may therefore engender elevated wall stresses and promote aortic dilatation (den Reijer et al., 2010; Hope et al., 2010, 2011; Weigang et al., 2008).

From a biomechanical prospective, AoD is a separation of the elastic layers of the degenerated aortic wall that occurs when the hemodynamic loads exerted on the aneurysmal wall exceed bonding forces that normally hold the mural layers together (Pasta et al., 2012; Rajagopal et al., 2007). Biomechanical studies demonstrated that maximum aortic diameter fails to predict rupture or dissection especially for small-sized ATAAs (McGloughlin and Doyle, 2010); therefore, predictors other than aortic size are needed to prevent aneurysm disease. Thus, computational fluid–solid interaction (FSI) analysis represents a valid finite element (FE) technique to investigate simultaneously the relevant hemodynamic and mechanical forces underlying the mechanics of AoD in ATAAs. FSI was used in several patient-specific simulations to study aortic aneurysms (Khanafer and Berguer, 2009; Molony et al., 2009).

The purpose of the present investigation was therefore to assess if difference exists on hemodynamic and wall stress arising in ATAAs. We speculate that the difference in both elastic properties of aortic layers and aortic valve morphology (BAV vs. TAV) influence directly the risk of AoD in ATAAs.

2. Material and methods

2.1. Geometry

Electrocardiogram (ECG)-gated computed tomography angiography (CTA) scans were used to reconstruct ATAA geometries identified from radiologic records of Mediterranean Institute for Transplantation and Advanced Specialized Therapies (IsMeTT). The study was approved by the local research ethics committee. Specifically, we segmented ATAAs of three patients with TAV and two patients with BAV which demographic data, BAV type, history of hypertension and presence of aortic stenosis or aortic insufficiency are reported in Table 1.

ECG-gated CTA scans were retro-reconstructed to obtain images at cardiac phase with the largest aortic valve opening area, which frequently occurs at 50–100 milliseconds after the R peak (Abbara et al., 2007). Reconstruction was therefore performed with the shape of aortic valve fully opened. ATAA geometries were reconstructed using the vascular modeling toolkit VMTK (http://www.vmtk.org). Images were segmented from the aortic valve, through the ascending aorta, the aortic arch and supra-aortic vessels and the descending aorta, ending at the level of the diaphragm. ATAA models were then exported to GAMBIT v2.3.6 (ANSYS Inc., Canonsburg, PA) for meshing both fluid (lumen) and structural (aneurysm wall) domains.

2.2. Fluid–structure interaction

Parallel coupled two-way FSI analyses were performed using the commercial software MpCCI v4.2 (Fraunhofer SCAI, Germany) to couple the structural component, ABAQUS v6.12 (SIMULIA Inc, Providence, RI), and the fluid solver, FLUENT v14.0.0 (ANSYS Inc., Canonsburg, PA). Fluid time step was set to 0.0068 s and data exchange occurred every time step with FLUENT sending the fluid-induced wall forces to ABAQUS, and ABAQUS sending the deformed nodal coordinates to FLUENT. Wall forces also include the frictional component induced by the fluid on the aortic wall, which causes the origin of the fluid shear stress. Transient simulations were accomplished after a total time of 0.68 s (i.e., 100nd time steps).

Both codes share a common boundary surface where the data exchange occurs. The MpCCI algorithm, which allows for non-matching meshes, identifies nodes or elements near each other based on an association scheme, and data are then transferred from one node to the other (Fraunhofer, 2008).

2.3. Structural model

ATAA wall was modeled as a hyperelastic, layered, incompressible and isotropic material, whose mechanical properties were derived from previously published experimental data (Pasta et al., 2012). Following delamination of ATAA tissue samples, tensile tests on the delaminated halves were performed to evaluate the elastic stress-stretch response of the outer (adventitia and dissected media) and inner (intima and dissected media) layers of the dissected aortic wall. Thus, the data sets were fit using the constitutive model developed by Raghavan and Vorp (2000) for modeling the human aorta:

$$W = \alpha(I_B - 3) + \beta(I_B - 3)^2$$

In these formulation, W represents the strain energy, I_B is the strain invariant of Left Cauchy-Green tensor while α and β are the model material parameters of the mechanical properties of ATAA wall layers.

Wall thicknesses were 1 mm and 0.7 mm for outer layer and inner layer, respectively, as measured experimentally (Pasta et al., 2012). Material nonlinearities due to large deformation were considered using Dynamic/Implicit formulation in ABAQUS FE software. ATAAs were meshed with triangular elements whereas tie contact conditions were used between outer and inner layers. For the aneurysmal wall, the structural density was 1120 kg/m^3. For the aorta to deform in a physiological way, the distal ends of supra-aortic vessels, aortic valve and descending aorta were fixed in all directions. The luminal surface of inner layer was used to exchange data with FLUENT. The aortic valve leaflets were assumed rigid which geometry was obtained from ECG-gated CTA images at cardiac phase with the largest valve opening area.

2.4. Fluid model

Transient-time solver with 2nd order implicit time advanced scheme was used for fluid dynamic simulations. The blood flow was assumed laminar, incompressible and Newtonian with density of 1060 kg/m^3 and viscosity of 0.00371 Pa $\times$ s. Continuity equation and linearized momentum equations were solved sequentially by a segregated algorithm (Pekkan et al., 2008). Pressure-implicit with splitting of operators (PISO) and skewness correction was set as pressure–velocity coupling algorithm to improve the convergence of the transient calculations in close vicinity of distorted cells. To eliminate numerical diffusion in calculations, 2nd order upwind scheme is applied to discretize the convective terms in momentum equations. Pressure staggering option (PRESTO) scheme as pressure interpolation method was set with 2nd order accurate discretization. Convergence was enforced by reducing the residual of the continuity equation by 10^{-5} at all time steps.

The total cardiac output was assumed at 5 L/min, and this flow was distributed between the supra-aortic vessels and the descending aorta with a ratio of 40/60 (Pekkan et al., 2008). Based on these assumptions, inlet aortic flow velocity and pressure boundary conditions were calculated at inlet and outlets, which were also extended six diameters normal to the vessel cross-section.

Dynamic mesh with smoothing and remeshing options were set at luminal ATAA wall surface to allow data exchange with ABAQUS. In FLUENT, remeshing of cells was adopted to handle with deforming mesh. Specifically, tetrahedral cells were remeshed based on whether they violate a user specified size and skewness criteria (i.e., 3% of the initial size or skewness lower than 0.75).

3. Results

Representative tensile stress-stretch responses of both inner and outer layers of ATAAs are shown for both BAV ATAA and TAV

Table 1
Clinical data of patients used for FSI analysis. Type 1 R/N BAV indicates fusion of right and non-coronary aortic leaflets while Type 0 BAV indicates a purely bicuspid with two symmetric leaflets.

Patient ID	Valve	Leaflet fusion	Aneurysm diameter (mm)	Age (year)	Sex	History of hypertension	Aortic stenosis	Aortic insufficiency
(A)	BAV	Type 1 R/N	57	48	Male			
(B)	BAV	Type 0	41	68	Male	Yes		
(C)	TAV		39	62	Male			
(D)	TAV		44	68	Female			
(E)	TAV		45	76	Female		Severe	Severe