Influence of tear configuration on false and true lumen haemodynamics in type B aortic dissection

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Abstract—The management and follow-up of chronic type B aortic dissections continues being a clinical challenge. Patients with chronic type B dissection have high mid/long term mortality mainly due to progressive aortic dilatation and subsequent rupture.

To predict further dilatation, guidelines suggest follow-up of the total aortic diameter. However, dilatation is triggered by haemodynamic parameters (intra-luminal pressure and flow conditions/wall shear stresses), and geometric factors such as the communication between false (FL) and true lumen (TL). The aim of this study is to assess whether TL and FL haemodynamics and its determinants (such as the tear size, location and number) will allow us to define risk markers of further aortic enlargement.

For this, we performed in-silico studies on idealized dissected aortic geometries. A type B aortic dissection was created in which FL has double the diameter of TL. Three different circular tear configurations are studied: only a 10 mm proximal tear, and a 10 mm proximal tear with either a 4 mm or a 10 mm distal tear. Resulting flow volume, pressure, and wall shear stress (WSS) profiles of TL and FL were analyzed.

Preliminary results show that the presence of an adequate outflow in the distal tear is associated with an important increase in diastolic pressure and wall stress and diastolic retrograde flow, putting FL at high risk for dilatation.

We have constructed a model of a type B dissection which allows studying dissected anatomic configurations and their resulting haemodynamics. It is expected that in-silico models will show the influence of tear configuration, thus providing ways for a better understanding of the haemodynamic conditions, as observed in clinical practice, and related evolution in patients with a chronic aortic dissection.

I. INTRODUCTION

ORTIC pathologies are an important subgroup within cardiovascular diseases and are associated with a very high morbidity and mortality. Despite improved diagnostic and therapeutic techniques, the management and follow-up of aortic dissections continue being a challenge in clinical practice.

Acute type B dissection is less lethal than type A. However, a type B dissection persists after correcting an acute type A dissection in more than 60% of cases [1,2], and patients with type B dissection have high mid/long term mortality during the chronic phase, mainly due to the progressive dilatation of the aorta and subsequent rupture.

In current clinical practice, prediction of outcome in type B chronic aortic dissections is mainly based on maximum total aortic diameter, which is compared with guidelines for deciding the best therapeutic approach. However, previous work has shown that maximum diameter is not a reliable determinant of rupture and progression [3-5]. In addition to it, haemodynamic parameters (intra-luminal pressure and flow conditions/wall shear stresses), geometric factors such as the communication between false (FL) and true lumen (TL), and intrinsic wall properties seem to be potential markers of dilatation and rupture risk.

Several features have been suggested to aid in the prediction of dilatation. The patency of the descending aorta false lumen may be responsible for progressive aortic dilation [2] and partial thrombosis of the false lumen has been found as a predictor of post-discharge mortality in patients with type B acute aortic dissection [6].

It was also observed that prognosis of patients with open communication between true and false lumina is poorer than in those without such communication, and free communication with high flow rates carries a higher risk for reoperation because of the high flow pressure and wall stress. Nevertheless, complete obliteration of the false lumen can occur despite open communication and is possibly related to the size of communication [7]. Poor inflow in the true lumen and lack of outflow in the false lumen may have impact in false lumen dilation and rupture during follow-up period [8]. Moreover, preliminary work of our group also suggests that when the flow is retrograde in the false lumen during diastole, it is because of an inadequate outflow in the distal tear. This data could be a good marker of high diastolic pressures in the false lumen and consequent higher wall stress and progressive aortic dilatation. Therefore, from clinical observations, the importance of tear size and location is clear. However, the contradictory findings on which situations are leading to further dilatation of the FL show that there is still a lack of understanding of the interplay of all variables.

Another factor that could affect the dilatation of the false lumen is the compliance or mechanical strength of the dissected aortic wall. It is known that all the mechanisms that weaken the aortic media layer will eventually lead to
higher local wall stress, which can induce aortic dilation and aneurysm formation, and finally result in aortic rupture [3]. Arteries respond to changes in blood pressure and flow conditions by remodeling. Wall shear stress (WSS) is the tangential force resulting from the friction that the flowing blood exerts on the luminal surface. It has been shown that WSS can change the morphology and orientation of the endothelial cell layer [9]. Prolonged high WSS is known to cause vessel dilation and internal elastic lamina fragmentation, and may be the responsible for dissection initiation [10]. On the other hand, inflammatory and atherosclerotic pathways triggered by low WSS could be also implicated in dissection pathogenesis. Excessively low WSS could lead to atherosclerotic inflammatory infiltration and thereby cause deterioration of the aortic wall that could lead to rupture [11].

Therefore, it is expected that better aortic morphologic and hemodynamic analysis will be much more predictive for aortic dilatation and will improve the clinical stratification of the risk of these patients, facilitating a better therapeutic management.

The aim of this study is to assess whether true and false lumen haemodynamics and its determinants (such as the tear size, tear location and tear number) will allow us to define risk markers of severe aortic enlargement. In silico studies were performed to investigate the impact of morphological characteristics on the haemodynamics of the false and true lumen.

II. MATERIALS AND METHODS

A. Simulated idealized geometries

Computational 3D models of synthetic type B aortic dissections (Fig. 1(a)) were constructed with the CAD software GID (CIMNE, Barcelona). The dimensions of the models were selected based on anatomical measurements from previous clinical and experimental studies.

The dimensions of the models are: aortic diameter: 20 mm; dissected segment diameter: 40 mm; false lumen length: 160 mm; true lumen thickness: 3 mm; dissection flap thickness: 2 mm; and false lumen thickness: 1 mm. A thickness transition was modeled between the dissection flap and the true lumen wall, with a 2.5 mm thickness in the middle section of the true lumen wall. The ratio of different thickness was taken from previous studies, where a dissection plane is created within the media separating the intima from the overlying adventitia in a way that two-third of the full thickness remains in the dissection flap and one-third of the thickness remains in the false lumen wall.

Our model includes the aortic arch and the ascending aorta. Part of the aortic arch curvature occurs in a single plane and is formed from a tubular section that extends 165°. The ascending aorta extends through 55° in a plane angled at 50° with respect to the main arch plane and the outlet curves through 15° in a mirrored plane before forming the descending aorta.

Proximal and distal tears are simulated with sizes of 4 mm and 10 mm diameter, which correspond to 10% and 25% of the dissected segment diameter, respectively. The proximal tear is located at 20 mm from the aortic arch and the distal tear was placed at the end of the dissected segment.

The models studied differed in tear size, tear number, and tear location. Two different anatomic configurations (Fig. 1 (b)) were studied: I) proximal tear only and II) both proximal and distal tears. We considered three cases: only a 10 mm proximal tear (1), and a 10 mm proximal tear with either a 4 mm (2) or a 10 mm distal tear (3).

B. Computational fluid dynamic simulations

Computational meshes consisting of approximately 1.1 million tetrahedral elements with a size range of 0.5-1.0 mm were created for the dissection models with GID (CIMNE, Barcelona).

Computations were performed using the commercial CFD-Tdyn (CompassIS, Barcelona) code based on the stabilized finite element method FIC/FEM for solving the Navier Stokes equations.

The no-slip wall of the dissection models was assumed to be rigid. This assumption is consistent with some studies that suggest that the difference in flow induced pressure variations and consequent wall stress between fluid structure interaction (FSI) and static structural (SS) models is negligible, and justify the use of SS models instead of FSI models [12,13]. Additionally, most patients with a chronic dissection show reduced flap motion, so that a rigid flap is a good first approximation.

Velocity and pressure waveforms (Fig. 2) were applied at the inlet and outlet of the fluid domain respectively. These
time dependent velocity and pressure waveforms were adapted from [14].

The blood was assumed to be incompressible and Newtonian [15], with viscosity of 0.0035 kg/(m·s) and density of 1040 kg/m³.

C. Haemodynamic parameters

We assessed intra-luminal pressure and flow volume in the FL and TL at the distal and proximal descending aorta, respectively (Fig. 1 (b)) to examine the possible effects of dissection anatomy on flow characteristics within the dissection.

To compare the influence of the different anatomic configurations on the FL wall, we analyzed the WSS distribution at its surface.

To analyze flow patterns in the FL and TL, we plotted the velocity vectors at the midplane of the dissected geometries.

III. RESULTS

The presence of a distal tear results in significantly more FL flow. However, in the absence of outflow (case 1) at the distal tear, there is still some FL flow and this is associated with a slight increase in diastolic pressure compared to case 2. The highest diastolic pressure was observed for the case 3 (Fig. 3). Additionally, in case 2 and 3, there is a high early systolic flow, changing the flow dynamics in the FL (Fig. 4).

From the assessment of the flow pattern in the dissected region (Fig. 5), we observed a bidirectional flow in the FL in all cases and a more prominent retrograde flow in case 3 during peak diastole.

The most significant elevation in WSS and the highest variability in WSS direction were registered for case 3, potentially promoting endothelial remodeling and weakening of the wall together with high WSS where the incoming jet hits the vessel wall, potentially stimulating dilatation (Fig. 6).

IV. CONCLUSION

We evaluated how hemodynamic parameters in the false and true lumen can be modified by different anatomic configurations of aortic dissection. The influence of tear configuration on the false and true lumen haemodynamics was significant in the presence of an adequate outflow at the distal part of the dissection flap, where the highest diastolic pressures were observed in the false lumen and the variability in WSS direction was more pronounced. Hence, an important distal outflow could be a risk marker of progressive dilation and rupture.

A future extension of this study will be the realization of in-vitro experimental models using silicone phantoms and explanted aortas, and the correlation of experimental results with clinical data obtained during the follow-up of patients with type B aortic dissection.
References


