Finite Element Analysis of Patient-Specific Heart Model with Simulated Aortic Stenosis

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***Abstract*—The main aim of this study was to evaluate the impact of simulated aortic stenosis on velocity and shear stress distribution within the patient-specific heart model by using computational Finite Element (FE) method. The three-dimensional (3D) patient-specific model of heart, including surrounding arterial and vein structures, was reconstructed based on Computed Tomography (CT) scan images in order to obtain the 3D FE mesh. Computational Fluid Dynamics (CFD) analysis was performed, with applied equivalent material characteristics and boundary conditions. Using one patient-specific heart model with clinically confirmed hypertrophic cardiomyopathy, three different cases were simulated: (i) without aortic stenosis, (ii) with 30% of aortic stenosis (mild aortic stenosis), and (iii) with 70% of aortic stenosis (severe aortic stenosis). The initial results of the study (velocity and shear stress distribution) were quantified concerning anatomical patient's structures and simulating different degrees of aortic stenosis to analyse the blood flow patterns, as well as the correlation between shear stress and aortic and left ventricular remodelling. It was found that gradient of shear stress distribution increases with stenosis degree, especially in the ascending aorta which can lead to different aortopathies and endothelial diseases. Due to the difficulties in obtaining such characteristics *in vitro* or *in vivo*, the performed computational analysis gave better insight into the biomechanics of the heart and aortic stenosis that is needed to achieve improvements in surgical repair techniques and presurgical planning.**

Keywords—patient-specific heart model, aortic stenosis, computational analysis, finite element method

I. INTRODUCTION

In case of heart malformations, complex changes in left ventricular geometry are in most cases caused by continuous exposure to cardiovascular risk factors and/or hemodynamic conditions, which usually start as a physiological response [1]. In contrary to anatomically and physiologically normal heart function, there are various inherited and acquired heart diseases that lead to serious cardiovascular complications and heart failure. One of the most common causes of heart failure is cardiomyopathy. According to the guidelines of the European Society of Cardiology [2], cardiomyopathies are defined as structural and functional abnormalities of the ventricular myocardium.

In addition to cardiomyopathy, there are other relatively common reasons for the heart failure, whereby aortic stenosis, which represents a progressive narrowing of the aortic valve opening and leads to remodelling of the left heart ventricle and hypertrophy, is certainly one of them. When it comes to the associated diseases of cardiomyopathy, as well as congenital heart diseases that affect its normal function, although they have been intensively investigated in previous years, many questions related to the mechanisms of the origin and development of these diseases are still open.

Analysis of valvulopathies and aortopathies using different computer methods represent an important segment in understanding their cause-and-effect relationships with cardiomyopathy and determining characteristic biomechanical parameters that cannot be determined by *in vivo* experiments. The studies of the aortic valve attract a lot of attention from different scientific fields such as biology, pharmacy, medicine and engineering. Structural Finite Element (FE) analysis has been performed in recent years in the dynamic analysis of aortic valves in order to calculate the stress distribution in open valves, and then to determine areas of abnormally high stress concentrations, their correlation with the degree of stenosis, calcification zones, etc., as well as possible mechanical failure [3,4]. Also, various dynamic FE analyses were performed to simulate the complex movement of the valves (cusps) during the cardiac cycle and to identify zones of increased stress concentrations in the valvular structure [4, 5-7], as well as for the analysis of different degrees of aortic stenosis [8]. Fluid-Solid Interaction (FSI) has been performed in case of cardiomyopathy analysis using simplified models of left heart ventricle [9], as well as for the analysis of valve stenosis [10]. Computational Fluid Dynamics (CFD) has been applied to analyse blood flow in aortic stenosis and the correlation between shear stress and aortic and left ventricular remodelling [11,12].

The creation of a 3D patient-specific model, based on Computed Tomography (CT) scans is included in the first part

of the study, as well as the material properties and boundary conditions that were used for the FE analysis. The second part covers discussion of results (velocity and shear stress distribution) for this single patient-specific case. In the last part, main conclusions and plans for further improvements of presented analysis are given.

II. Materials and Methods

A. 3D Reconstruction of Patient-Specific Model

The complex anatomy of the heart requires detailed segmentation in order to capture all substructures of patient-specific geometry, as altered geometry affects the biomechanical parameters. In the purpose of computer simulation of associated diseases of cardiomyopathy, such as aortopathy (aortic root stenosis), segmentation and 3D reconstruction of patient-specific heart, aorta, surrounding arteries and vena cava have been performed including different software.

The 3D reconstruction of the patient-specific geometry included three phases. The first phase was related to the segmentation and generation of the heart geometry (including surrounding arteries and veins) from DICOM images. The set of 2D DICOM images was obtained as part of CT scans, one of the most usual diagnostic procedures. The segmentation of 2D images and calculation of initial 3D patient-specific model was performed in segmentation software (Materialise Mimics) [13]. This was enabled through visualization of cross-sections in three different planes: coronal, sagittal and transversal (Fig. 1).

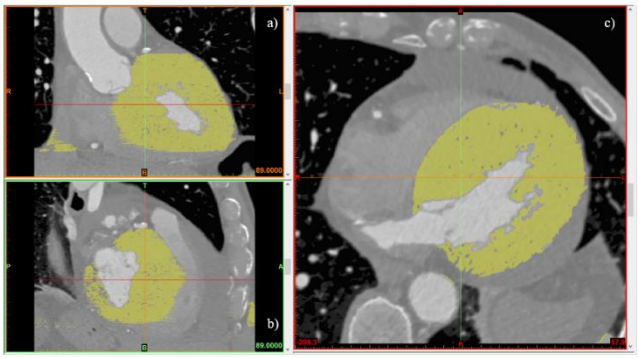

Fig. 1. Coronal (a), sagittal (b) and transversal (c) cross-section of heart model.

The initial model mainly consisted of triangular surface mesh not suitable for further computational analysis due to mesh overlapping and distorted elements. For this reason, in the second step the model was processed to further optimization in order to create smooth surface mesh with uniformly distributed FEs (Fig. 2).

Finally, third step included generation of a 3D volumetric tetrahedral mesh. The volumetric FE model was generated using Femap software [14] in conjunction with our own tool for conversion of tetrahedral elements to eight-node brick elements [15]. The total number of 3D eight-node elements was 381980. The analysed model of patient-specific heart-model, created from eight-node brick elements, was imported into the in-house FE PAK-F solver [16] for further computational simulations.

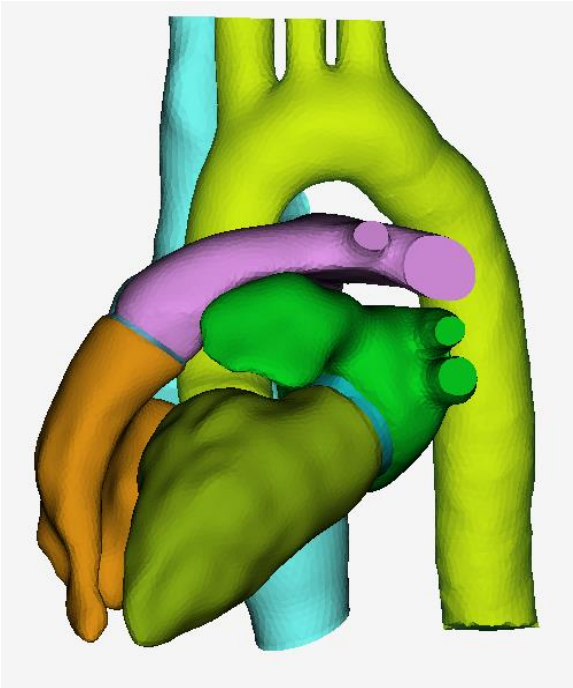

Fig. 2. Final 3D FE mesh of the patient-specific surface heart model with surrounding venous and arterial structures.

B. Numerical Simulation

In order to perform CFD simulation for a patient-specific heart model, including the aorta together with the arterial branches of the aortic arch, the proximal part of the thoracic aorta, as well as the vena cava and the pulmonary artery, the PAK-F solver was used. Due to the complex geometry and non-structural FE mesh, only the fluid domain was observed, i.e. the blood flow through the atria, ventricles, arterial and venous structures was simulated.

Numerical simulation performed in this study included CFD analysis in order to examine biomechanical parameters of patient-specific heart model with clinically confirmed hypertrophic cardiomyopathy, as well as the influence of aortic stenosis on flow conditions. Computational simulations included simplifications and assumptions of geometrical and material properties due to complex biological structure of heart. The average material characteristics of blood were adopted from the literature, which is a common practice due to the lack of experimental data for a specific patient [17,18]. In order to perform analysis which reflects the realistic human blood flow we used average blood properties: a dynamic viscosity (μ) of 0,0035 Pa·s and a density (ρ) of 1050 kg/m^3 [17]. Flow was considered to be laminar, homogeneous (Newtonian) and viscous incompressible. The 3D flow was governed by the Navier-Stokes equations and the continuity equation, respectively:

$$\rho\left(\frac{\partial v_i}{\partial t}+v_j\frac{\partial v_i}{\partial x_j}\right)=-\frac{\partial p}{\partial x_i}+\mu\left(\frac{\partial^2 v_i}{\partial x_j\partial x_j}+\frac{\partial^2 v_j}{\partial x_j\partial x_i}\right) \quad (1)$$

$$\frac{\partial v_i}{\partial x_i}=0 \quad (2)$$

where v_i denotes the blood velocity in direction x_i, p is the pressure, ρ is the fluid density and μ is the dynamic viscosity. Summation is assumed on the repeated indices i,j=1,2,3. Eq.

(1) represents the balance of linear momentum, while (2) expresses the incompressibility condition. The boundary conditions consisted of: prescribed inlet velocity, zero-velocity at the rigid walls and physiological resistance pressure at the outlets. At the inlet, a parabolic flow velocity profile was used together with a pulsatile waveform. As the analysis included only the fluid flow, the myocardial tissue, aortic wall, as well as the valves' leaflets were not included in the model.

In the zone of simulated aortic valve stenosis, the valve parameter percentages for different degrees of aortic stenosis (0%, 30%, 70%) were prescribed. Numerical simulations were performed with 40 steps with a duration of 0.025 s. The simulations were performed for three different cases:

1. Heart model without aortic stenosis;
2. Heart model with 30% of aortic stenosis (mild aortic stenosis);
3. Heart model with 70% of aortic stenosis (severe aortic stenosis).

After applied appropriate boundary conditions and material characteristics, the in-house FE PAK-F solver [16] for CFD analysis was used, which calculated the velocity and shear stress distribution.

III. Results and Discussion

In the last years, different computational studies have demonstrated that *in silico* approach and computational tools are very useful for investigating the biomechanical characteristics of the both heart and surrounding arterial structures which are difficult to obtain by *in vitro* or *in vivo* experiments.

After employing the in-house FE PAK-F software for CFD analysis, the obtained results included velocity and shear stress distribution. The resulted parameters are shown at the maximum of systole, taking into account that the left heart ventricle and the aortic valve are under the greatest load during the cardiac cycle at that moment. The performed computational simulations allowed assessment of different aortic stenosis severities on biomechanical implications for this specific patient.

Figs. 3-5 represent the velocity distributions for 0%, 30% and 70% of aortic stenosis, respectively. During each individually simulated cardiac cycle, the velocity is maximal at the moment of maximum systole. The maximal velocity is within the limits corresponding to different degrees of stenosis [9]. Comparing the model without aortic stenosis (Fig. 3) and the models with simulated 30% and 70% of aortic stenosis (Fig. 4 and Fig. 5, respectively) it can be noted that at the maximum of systole blood velocities are the highest in the left heart ventricle and aortic root. Observing the whole model, the velocities are the highest for 70% of aortic stenosis, which is consistent with the real clinical picture. For all three simulated cases, the velocities gradually decrease going towards the veins, pulmonary artery and aortic arch where the initial segments of the brachiocephalic arterial tree are located, the common carotid artery and the left subclavian (subclavian) artery, and then towards the thoracic segment of the aorta.

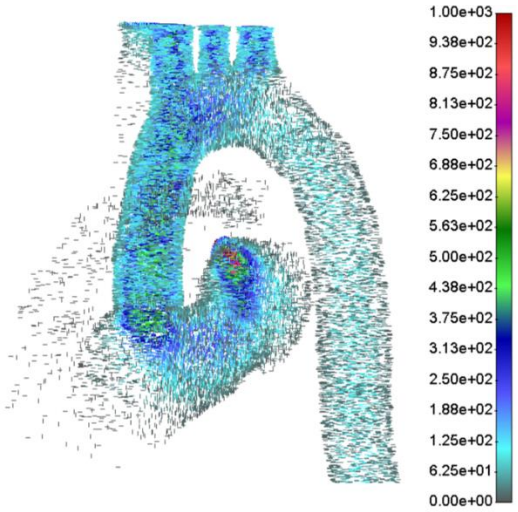

Fig. 3. Velocity distribution for case without aortic stenosis (peak systole, units: mm/s).

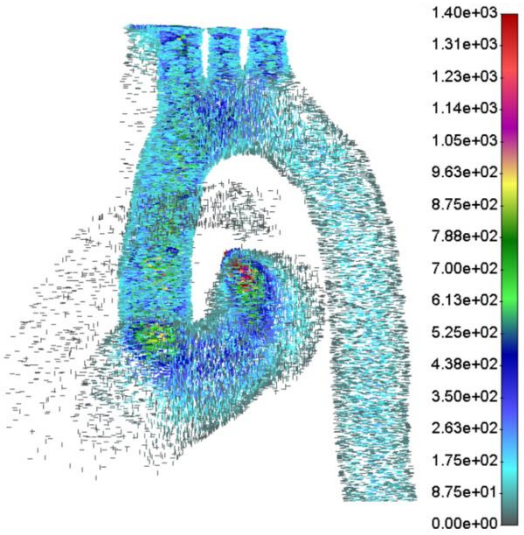

Fig. 4. Velocity distribution: simulated 30% of aortic stenosis (peak systole, units: mm/s).

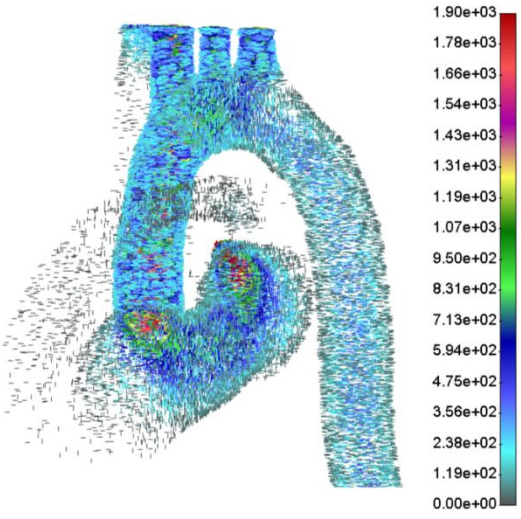

Fig. 5. Velocity distribution: simulated 70% of aortic stenosis (peak systole, units: mm/s).

Figs. 6-8 represent the shear stress distributions for 0%, 30% and 70% of aortic stenosis, respectively. During each individually simulated heart cycle, the shear stresses have

maximal values at the peak systolic moment. Comparing the model without aortic stenosis (Fig. 6) and the models with simulated 30% and 70% of aortic stenosis (Fig. 7 and Fig. 8, respectively), the maximal values of shear stresses are present in the zone of the aortic root and in the ascending aorta, which is in accordance with applied boundary conditions. Observing the entire model, the zones of high shear stresses are the highest for simulated 70% of aortic stenosis. Also, increased shear stresses are present in the arteries of the aortic arch. For all three simulated cases, the rest of surrounding venous and arterial structures are not under high shear stresses.

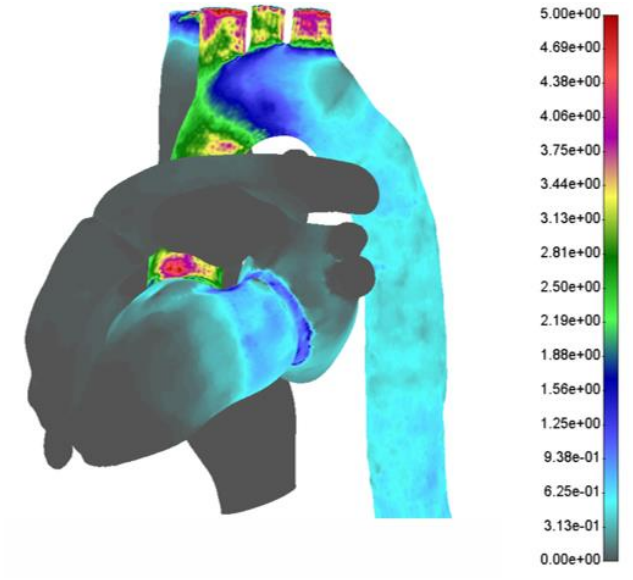

Fig. 6. Shear stress distribution for case without aortic stenosis (peak systole, units: Pa).

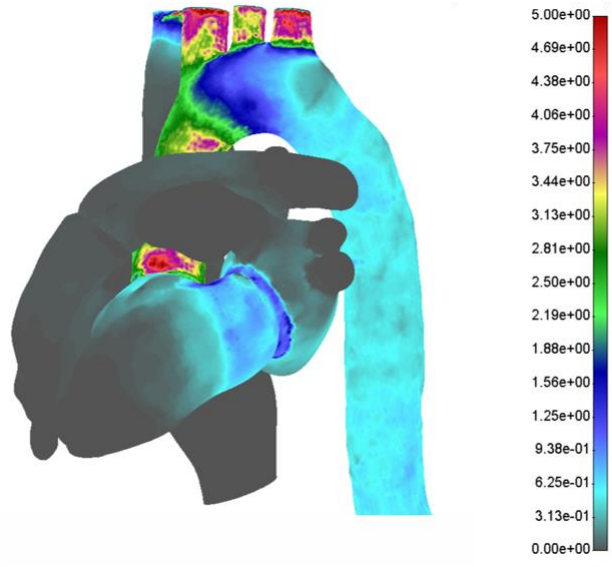

Fig. 7. Shear stress distribution: simulated 30% of aortic stenosis (peak systole, units: Pa).

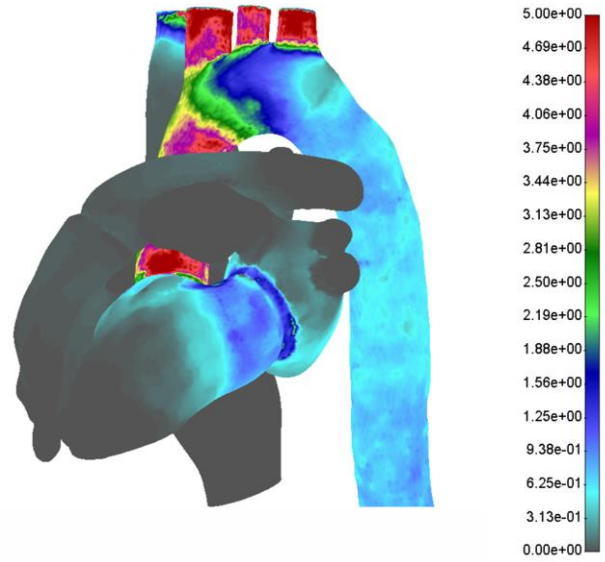

Fig. 8. Shear stress distribution: simulated 70% of aortic stenosis (peak systole, units: Pa).

Increased values of shear stresses in the area of the aortic root and in the ascending aorta create a precondition for further degradation of the intima layer of the aortic wall, a degradation of mechanical characteristics, and therefore a precondition for the further progression of aortic root stenosis and the appearance of comorbidities (atherosclerosis of the aortic root, dilatation of the aortic root, etc.). Recent studies have shown that the visualization of shear stress distribution, as an important parameter in the initialization of aortopathy, can suggest the direction of clinical monitoring of patients. Shear stress has the potential to be a biomarker to guide surgical strategies and patient care after aortic root surgery, as well as patient monitoring and prediction of disease progression [19, 20]. It can be seen from Figs. 6-8 that gradient of shear stress distribution increases with stenosis degree, especially in the ascending aorta which can lead also to different endothelial diseases (such as infective endocarditis).

IV. Conclusions

The presented study gave the insight into the model-based FE simulation of the patient-specific heart model including surrounding arterial and vein structures, and employing the in-house FE PAK-F software. The presented approach aimed to propose an advanced assessment of biomechanical characteristics based on computational modelling. The FE analysis included CFD and comparison of blood velocity and shear stress distribution in case of simulated different aortic valve stenosis severities (0%, 30% and 70%). The obtained results are in correlation with previous studies, and can be a predictor for different diseases caused by high gradient of shear stress. This *in silico* approach is a useful method which may enhance our understanding for better insight into the biomechanical characteristics which cannot be obtained *in vitro* or *in vivo*. Moreover, in case of malformed aortic roots, it may contribute to the advancements in surgical repair techniques and presurgical planning.

Considering that the analysis included only the fluid flow – the myocardial tissue, aortic wall, as well as the valves' leaflets were not included in the model, which is one of the limitations of the study. Next studies will include more advanced and complex 3D models, as well as analysis for coupled FSI which is challenging considering the complexity

of the heart. Also, numerical simulations will be improved in terms of eliminating the current modelling limitations so that new models can more closely replicate the physiological problem, which is our main goal.

ACKNOWLEDGMENT

This research is supported by the European Union's Horizon 2020 research and innovation programme under grant agreement No 952603 (SGABU). This article reflects only the author's view. The Commission is not responsible for any use that may be made of the information it contains. The research was also funded by Serbian Ministry of Education, Science, and Technological Development, grants [451-03-47/2023-01/200378 (Institute for Information Technologies, University of Kragujevac)] and [451-03-47/2023-01/200107 (Faculty of Engineering, University of Kragujevac)].

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