MCB, vol.16, supplemental 2, pp.16-17, 2019

Numerical Simulation of Myocardial Bridging in Patients with Hypertrophic Cardiomyopathy

Mohammadali Sharzehee[1], Yuan Chang[2], Jiang-ping Song[2] and Hai-Chao Han[1,*]

[1]Department of Mechanical Engineering, The University of Texas at San Antonio, San Antonio, TX 78249, USA.

[2]Department of Cardiac Surgery, State Key Laboratory of Cardiovascular Disease, Fuwai Hospital, National Center for Cardiovascular Diseases, Beijing, China.

[*]Corresponding Author: Hai-Chao Han. Email: hchan@utsa.edu.

Abstract: A myocardial bridge (MB), a congenital anomaly of the coronary artery, occurs when a segment of the epicardial coronary artery goes underneath the myocardium. MBs are often observed in the middle part of the left anterior descending (LAD) artery. MB squeezes the vessel wall periodically and induces hemodynamic abnormalities which are correlated with angina and myocardial ischemia. The level of hemodynamics disturbances induced by MB depends on the myocardial bridge length, the degree of myocardial contractility, thickness, and location [1]. Hypertrophic cardiomyopathy (HCM), characterized by abnormal thickening of the heart wall, is a leading cause of death in patients of all ages [2]. The prevalence and severity of myocardial bridging are higher among patients with HCM, up to 30% [3]. Therefore, understanding the effects of MB in HCM patients is of clinical significance. The purpose of this study was to determine the role of MB on coronary hemodynamics in HCM patients with various level of MB using computational fluid dynamics (CFD) modeling. We analyzed three HCM patients with MB in the middle LAD who underwent coronary angiography at Fuwai hospital (Tab. 1). All patients had a significant MB (diameter stenosis ≥ 50%) as diagnosed by arteriography.

Table 1: HCM Patients' information

Patient no.	Age (yrs)/sex	Area stenosis (%)	Diameter stenosis (%)	MB length (mm)	LAD length (mm)	Vessel compression duration/cardiac cycle
1	59M	87	65.01	38.56	110.62	0.45
2	14F	94	76.19	31.14	161.91	0.46
3	49F	82.51	58.18	26.21	121.38	0.44

Biplane angiographic images were recorded at 15 frames/s using Artis zee III floor (Siemens Ltd. China) with a pixel spacing of 154μm. 2D vessel centerlines of two different oblique views were extracted from angiographic data and then combined to create a 3D vessel centerline in SolidWorks®. Lumen diameter of LAD at both end-diastole and end-systole were measured to reconstruct the LAD model (Fig. 1). Linear interpolation was used to predict vessel wall movements during the cardiac cycle using a user-defined function (UDF). The arbitrary Lagrangian-Eulerian (ALE) method was employed for the moving boundaries. Blood was assumed to be homogenous Newtonian fluid with a density of 1050 kgm^{-3} and dynamic viscosity of 0.00316 Pa.s. To ensure fully developed flow, extended lengths of 10-diameter and 15-diameter were added to the inlet and outlet sections, respectively [4]. Intracoronary pressure waveforms were applied as the inlet and outlet boundary conditions [5]. The model was meshed with hexahedral elements with a finer mesh near the wall. The shear stress transport (SST) turbulence model was used for simulating flow in ANSYS Fluent (v 17.1). The Navier-Stokes equations were solved using semi-implicit method (Simple) with the maximum residual of less than 10^{-6}. To achieve a periodic solution, three cardiac cycles were simulated and the last cycle results were used for the interpretation.

DOI:10.32604/mcb.2019.07129

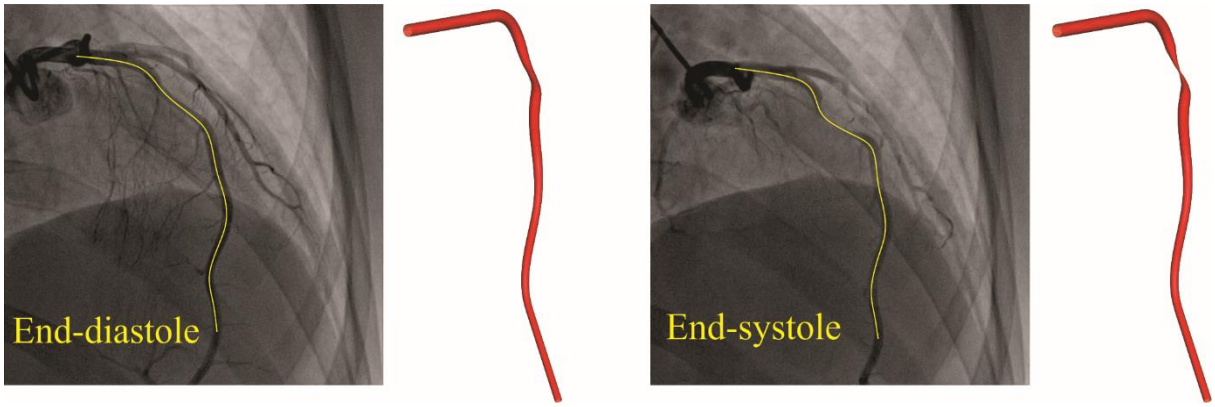

Figure 1: 3D reconstructed end-diastole and end-systole geometries from angiography in patient 2

To better understand the effect of MB, simulations were repeated for the same patient without MB (vessel compression was artificially removed). CFD results demonstrated that the presence of MB reduced the time-averaged wall shear stress (TAWSS) in the proximal (0.83 ± 0.29 Pa with MB versus 1.25 ± 0.59 without MB) and distal (2.76 ± 0.85 Pa with MB versus 3.82 ± 0.85 without MB) segments while increased TAWSS in the bridge segment (4.04 ± 1.29 Pa with MB versus 2.72 ± 1.23 without MB). The normal range of wall shear stress at arterial wall is from 1 to 7 Pa [6]. MB increased the maximum residence time of blood particles (Tab. 2), which may trigger thrombosis in the proximal segment. Increasing the degree of stenosis showed a significant decrease in the volume-averaged velocity and volumetric flow rate. Our analysis illustrated that the MB had significant effects on the distribution of wall shear stress and blood flow in HCM patients.

Table 2: Effect of MB on LAD hemodynamics in HCM patients. Values are in Mean±SD, n=3.

Patients	Maximum residence time (ms)	Volume-averaged velocity (m/s)	Volumetric flow rate (mL/s)
With MB	3.19±0.91	0.15±0.05	2.45±0.47
Without MB	2.47±1.2	0.23±0.09	3.66±1.34

Keywords: Hypertrophic cardiomyopathy; myocardial bridge; coronary artery; computational fluid dynamics

References

1. Alegria JR, Herrmann J, Holmes DR, Lerman JA, Rihal CS. Myocardial bridging. *European Heart Journal* **2005**, 26(12): 1159-1168.
2. Maron BJ. Hypertrophic cardiomyopathy: a systematic review. *Jama* **2002**, 287(10): 1308-20.
3. Kitazume H, Kramer JR, Krauthamer D, El Tobgi S, Proudfit WL et al. Myocardial bridges in obstructive hypertrophic cardiomyopathy. *American Heart Journal* **1983**, 106(1): 131-135.
4. Sharzehee M, Khalafvand SS, Han HC. Fluid-structure interaction modeling of aneurysmal arteries under steady-state and pulsatile blood flow: a stability analysis. *Computer Methods in Biomechanics and Biomedical Engineering* **2018**, 21(3): 219-231.
5. Javadzadegan A, Moshfegh A, Fulker D, Barber T, Qian Y et al. Development of a Computational Fluid Dynamics Model for Myocardial Bridging. *Journal of Biomechanical Engineering* **2018**, 140(9): 091010.
6. Papaioannou TG, Stefanadis C. Vascular wall shear stress: basic principles and methods. *Hellenic Journal of Cardiology* **2005**, 46(1): 9-15.