Hemodynamic effect of calcifed plaque on blood flow in carotid artery disease: A preliminary study

Hemodynamic effect of calcified plaque

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***Abstract*—The purpose of the study is to investigate the hemodynamic effect of calcified carotid plaque on blood flow in patients diagnosed with carotid artery disease. Two carotid artery models were generated based on a sample patient data, with normal and calcified carotid artery appearances. Circular calcified carotid plaque was found at the carotid bifurcation based on 3D computed tomography images. A computational fluid dynamics was performed to analyze the changes of blood flow in different situations. Our results showed that apparent turbulence was found in the diastolic phase at the carotid bifurcation in normal carotid artery geometry. In the presence of the calcified plaque, the flow velocity was increased to some extent, indicating the effect of plaque on hemodynamic changes. Wall shear stress was noticed to decrease at the aortic branches, and this indicates the potential risk of developing stenosis at this area. Our preliminary study demonstrates fluid structure interaction between calcified plaque and artery in terms of flow changes and wall shear stress.**

Keywords-carotid artery disease; calcification; blood flow, computed tomography, model

I. INTRODUCTION

It is estimated that 80% of all strokes that occur annually in the Western countries are ischemic, and about 30% of these are caused by thromboemboli arising from atherosclerotic lesions leading to an abnormal narrowing (stenosis) at the carotid artery bifurcation [1, 2]. Therefore, a leading index for assessing stroke risk for patients with symptoms of minor ischemic stroke is the degree of stenosis of the carotid artery. In addition, the unique geometry of each person's carotid artery can affect the blood flow pattern through the artery, which in turn, affects the risk of having plaque build-up and atherosclerosis [3]. Understanding the blood flow patterns in different carotid geometries can therefore lead to the identification of people vulnerable to atherosclerosis.

The causative factors that contribute to the formation of atherosclerotic lesions have been studied extensively. Hemodynamic factors are found to be important determinants of the local distribution of atherosclerosis and the development of plaques at the vascular wall. However, the precise role played by hemodynamics in the development and progression of vascular disease is not fully understood, and findings have been reported to be sometimes contradictory [4]. More direct studies, in which presence or absence of disease (carotid artery stenosis or occlusion) can be compared with hemodynamic analyses in individual vessels, since subtle changes in vessel geometry can affect the flow field. Thus, the aim of our study was to investigate the fluid structure interaction in both normal and diseased carotid arteries. Specifically, we are interested in the relationship between carotid plaque (in the carotid bifurcation) and its effect on the blood flow before surgical treatment when compared to the analysis performed in a normal situation.

II. MATERIALS AND METHODS

A. Selection of Sample Patient Data

A sample patient with carotid artery disease was selected for inclusion in the study. The patient was performed with a multislice CT scanner 64x0.625 mm beam collimation (High-Speed Advantage scanner, GE Medical Systems, Milwaukee, WI, USA), and scanning protocol was section thickness 0.625 mm, pitch 0.5, reconstruction interval of 0.4 mm and gantry rotation time was 0.33 second.

B. 3D Reconstruction and Postprocessing

Original CT DICOM data (digital imaging and communication in medicine) were transferred to a workstation equipped with Analyze V 7.0 (AnalyzeDirect, Inc., Lexana, KS, USA) for generation of 3D reconstructed images.

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Segmentation of volume data was performed semi-automatically with a CT number thresholding technique [5, 6]. Manual editing was applied in some slices to remove unwanted structures such as carotid veins or soft tissues. 3D object of the carotid artery model was saved as STL (stereolithography), a common format for computer-aided design and rapid prototyping. The 'STL' file was converted into the CAD (computer aided design) model files using the CATIA V5 R17 (Dassault Systèmes, Inc., Suresnes Cedex, France).

C. Generation of Carotid Model With/Without Calcification

Carotid artery models were produced with demonstration of the common carotid artery, bifurcation and internal and external branches, as shown in Figure 1. In addition, we also simulated the calcification which is formed in the carotid bifurcation with circular appearance based on CT images (Fig 2). This aims to make our analysis reflect the actual condition of carotid artery stenosis caused by the plaque formed at the carotid bifurcation.

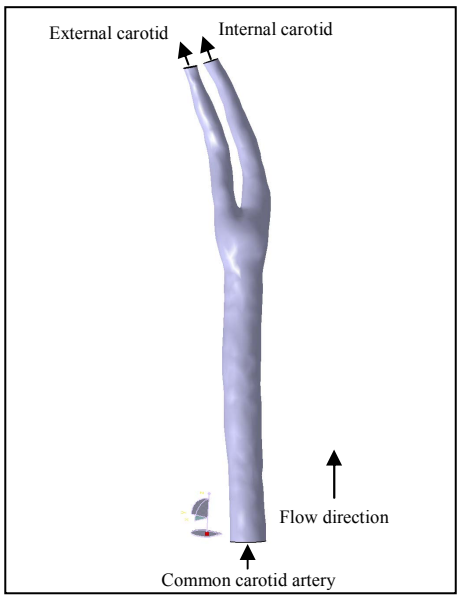

Figure 1. Segmented normal carotid artery model.

D. Computational Fluid Dynamics (CFD)

The fluid and materials properties for different entities were referenced from a previous study [7]. In the current study, we compared the flow velocity at the common carotid artery and internal and external carotid arteries. The blood flow was simulated at different cardiac phases (Fig 3) using the Ansys CFX 11 (Ansys, Inc., Canonsburg, PA, USA). The mesh model of the blood was defined by tetrahedral volume mesh using the Ansys ICEM CFD 11 (Ansys, Inc., Canonsburg, PA, USA) (Fig 4). The boundary conditions are time-dependent. The velocity inlet (the common carotid artery) boundary conditions are taken from the referenced value showing measurement of the aortic blood velocity.

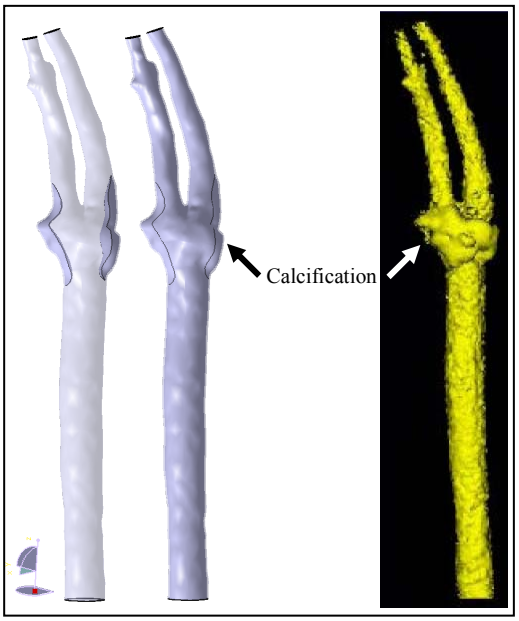

Figure 2. Segmented carotid artery model with calcification at the bifurcation.

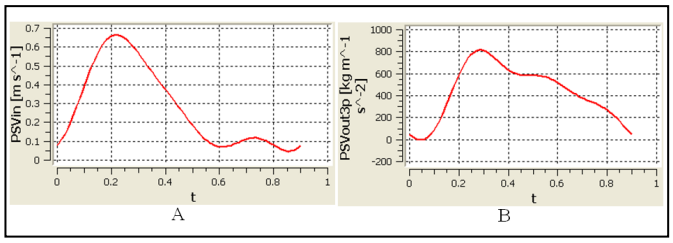

Figure 3. Flow pulsatile in different cardiac cycles at the common carotid artery (A), Time-dependent pressure at external carotid artery and internal carotid artery (B).

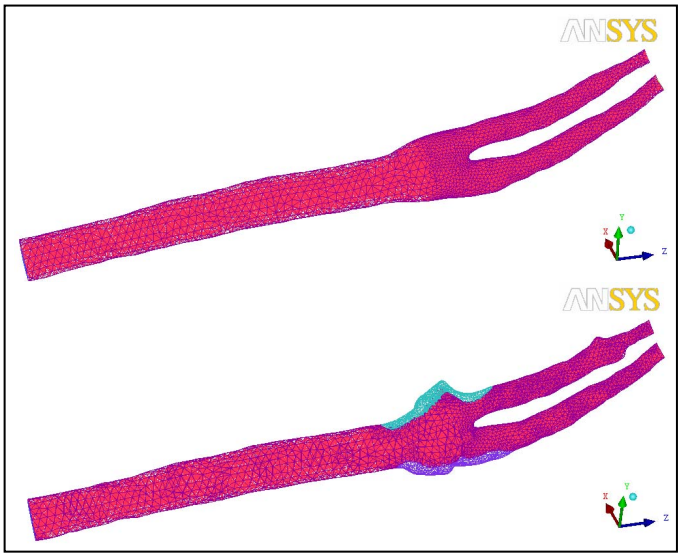

Figure 4. Carotid artery meshes for normal artery (top) and the calcified plaque present at the carotid bifurcation (bottom).

The fluid is (blood) assumed to behave as a Newtonian fluid, as this was known for the larger vessels of the human body. The density was set to 1060 kg/m^3 and a viscosity of 0.0027 Pa s, corresponding to the standard values. Given these assumptions, the fluid dynamics of the system are fully governed by the Navier-Strokes equations. The flow was assumed to be incompressible and laminar. The arterial wall was assumed to be deformable during flow analysis. The calcium is assumed to be non-material properties.

III. RESULTS

CFD analysis was successfully performed in both normal and calcified carotid models at different cardiac cycles. The velocity reached the highest at systolic phase, approximately at 0.2-0.3 sec (Fig 5). Similarly the wall pressure was noticed to be the highest at systolic phase at 0.225 sec (Fig 6).

Wall shear stress was found to increase in the carotid branches when compared to that noticed in the common carotid artery, and this is especially apparent in both internal and external carotid arteries at systolic phase, as shown in Figure 7. However, in the presence of carotid stenosis, wall shear stress was decreased in these arteries.

Our results also showed that with presence of calcification at the carotid bifurcation, the flow velocity was increased significantly when compared to the normal situation, as shown in Figure 8. This is mainly due to the increased flow rate when the blood flows through relatively narrowed artery caused by the severe calcification in the artery wall.

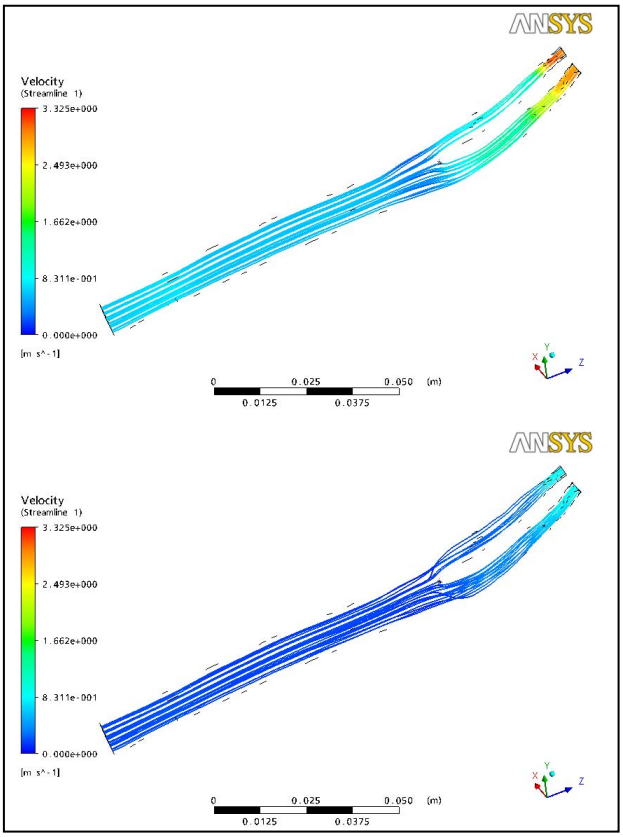

Figure 5. Velocity measured at 0.2 sec and 0.6 sec in normal carotid artery.

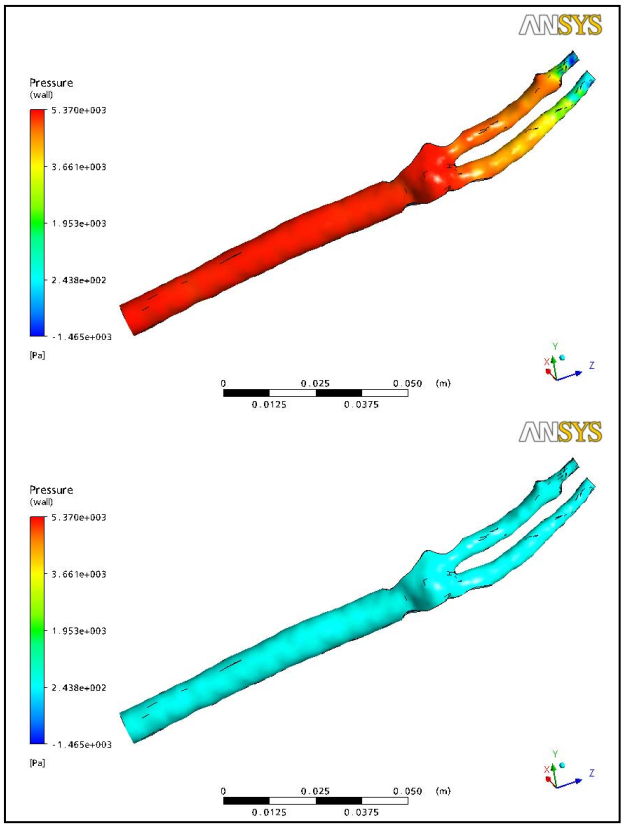

Figure 6. Wall pressure measured at 0.2 sec and 0.6 sec with calcification.

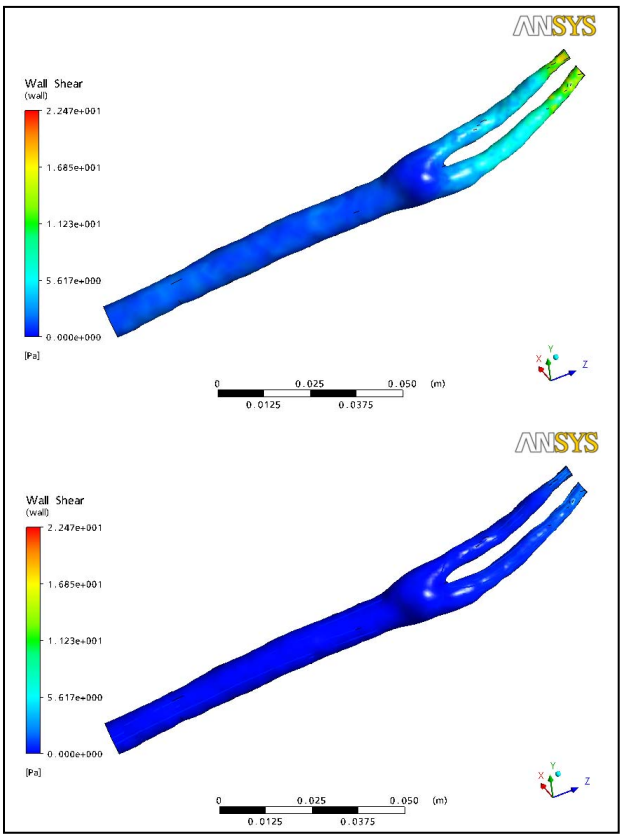

Figure 7. Wall shear measured at 0.2 sec and 0.6 sec in normal carotid artery.

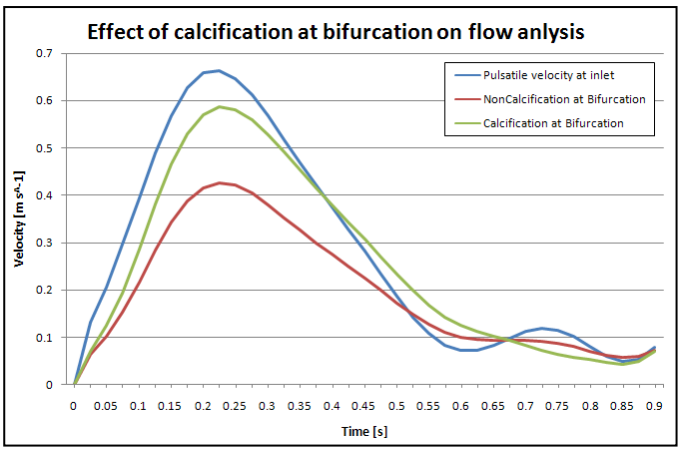

Figure 8. Flow velocity measured at normal and calcified arteries.

IV. DISCUSSION

We have simulated the hemodynamic factors in realistic human carotid artery models based on different cardiac phases. The artery models were generated using patient's CT data which reflect the realistic situation. Our results showed that circular calcification in the carotid bifurcation led to increased flow velocity and shear stress (particularly in the internal and external carotid arteries), however, no significant effect on the wall pressure.

Curvatures and bifurcations of large and medium sized arteries are severely affected by atherosclerosis. Numerous investigations point out that there is a relationship between the genesis and the progression of the disease with the locally irregular flow field occurrence in these zones. It is generally believed that local hemodynamic conditions are directly related to the initiation, promotion, and development of atherosclerotic plaque and thrombus formation [3, 8, 9]. Thus, the stenosed carotid geometry or the induced flow patterns may provide insight into the plaques that are vulnerable to enhanced thromboembolic potential or increased risk of stroke (9).

The decreased wall shear stress observed in the internal and external carotid artery when there is a plaque formed at the bifurcation should draw clinical attention, as low wall shear stress is associated with neointimal hyperplasia in either bypass graft or stent [10]. Further studies are needed, especially with implantation of carotid stents to verify our initial results.

Obviously, flow patterns in the arterial tree are changed considerably by alterations of pulsatility [11, 12]. Previous studies on the non-stenosed physiologic geometry showed that the borderlines of flow separation and the local values of shear stress changed considerably with changing amplitudes in the mean flow pattern [11, 12]. Our simulation models are rigid without taking into account of the pulsatility of the artery wall during each cardiac cycle. However, we do perform CFD analysis at different cardiac cycles, indicating that our results are valid and could be used for further studies with regard to the investigation of development of atherosclerosis at the carotid artery.

Flow patterns are known to be highly dependent on the exact geometry [13]. Ideally, an in vivo imaging method would be able to cope with complex geometry and extreme stenoses such as studied in a 2D computational model by Stroud et al [14]. Our model was generated using patient's CT data, thus it represents the complex anatomical structures and geometry, although further studies are needed to investigate the flow changes along the inner and outer arterial wall with regard to wall stress and flow velocity.

In conclusion, we have performed a simulation of flow analysis in both normal and diseased carotid artery models using a patient's data. With presence of calcification at the bifurcation, flow pattern was affected to a greater extent. Further studies with inclusion of pulsatility and more focused investigation of the wall shear stress are necessary to identify potential risk of atherosclerosis.

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