Prediction of blood pressure and blood flow in stenosed renal arteries using CFD

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Abstract. In the present work an attempt is made to develop a diagnostic tool for renal artery stenosis (RAS) which is inexpensive and in-vitro. To analyse the effects of increase in the degree of severity of stenosis on hypertension and blood flow, haemodynamic parameters are studied by performing numerical simulations. A total of 16 stenosed models with varying degree of stenosis severity from 0-97.11\% are assessed numerically. Blood is modelled as a shear-thinning, non-Newtonian fluid using the Carreau model. Computational Fluid Dynamics (CFD) analysis is carried out to compute the values of flow parameters like maximum velocity and maximum pressure attained by blood due to stenosis under pulsatile flow. These values are further used to compute the increase in blood pressure and decrease in available blood flow to kidney. The computed available blood flow and secondary hypertension for varying extent of stenosis are mapped by curve fitting technique using MATLAB and a mathematical model is developed. Based on these mathematical models, a quantification tool is developed for tentative prediction of probable availability of blood flow to the kidney and severity of stenosis if secondary hypertension is known.

1. Introduction
Renal artery stenosis (RAS) has been known to be leading cause of Renovascular Hypertension (RVH) and kidney failure in human. Moreover, a patient suffering from renal artery stenosis is likely to have coronary artery disease which is leading cause of death and morbidity in the world. RAS reduces the blood supply to the kidney which can result in atrophy (shrinking in the size) or may ultimately lead to the failure of kidney. RAS also results in increasing the peripheral resistance to the blood flow thereby increasing the RVH also called as secondary hypertension or high blood pressure related to renal artery disease [1]. Physiological significance of criticality of RAS cannot be confirmed only on the basis of geometrical information obtained from modern imaging diagnostic techniques like intravascular ultrasound (IVUS), magnetic resonance angiography (MRA), angiography etc. specially for moderate area stenosis (AS – reduction in lumen cross-sectional area due to stenosis). These imaging techniques help the doctors in knowing the exact region & criticality of stenosis. But, these imaging techniques are pricey and not accurate in deciding the severity if lumen diameter stenosis (DS - reduction in lumen diameter due to stenosis) is in the range of 40 - 70 \% [2-4]. Hence, physiological parameters like flow velocity & blood pressure should be assessed around the region of stenosis [5-7]. Additionally, in an arterial stenosis hemodynamic parameters play an important role in further disturbing the blood flow which results into progression of stenosis at same and multiple locations [8].
From the perspective of a doctor, evaluation of physiological parameters that possibly evaluate the criticality of stenosis is crucial. Therefore, the main aim of this work is to evaluate the availability of blood flow to kidney and RVH due to RAS using numerical analysis of stenosed models of artery. The relation between RVH and available blood flow with % AS are also correlated.

In many of the earlier works on arterial stenosis, blood flow is assumed to steady having constant inlet and outlet velocity or pressure \([9,10]\) and blood is assumed as Newtonian fluid \([9-11]\). However in the present work for the simulation of blood flow in the arterial stenosed models, non-Newtonian model of blood, pulsatile flow and unsteady velocity profile at the inlet are considered. To perform CFD analysis of a bigger portion of renal artery tree is a very time squandering process. Therefore, to save computational time the numerical analysis can be restricted to a 3D region of interest like region of renal artery tree having blockage without any compromise with accuracy. The main aim of the present work is to formulate a mathematical model which determines the criticality of RAS on the basis of blood pressure in the form of a handy chart which may help the doctors in preliminary diagnosis.

2. Methodology

2.1. Modelling of renal artery

Sixteen types of 3-dimensional models of renal artery are constructed so that a mathematical model can be developed which correlates the %AS in renal artery with renal hypertension (RVH) and decrease in available blood flow to kidney. In the present work, the 3-D renal artery models are generated assuming it to be symmetrical and cylindrical as mentioned in \([12,13]\). The length and diameter of healthy renal artery model is 16 mm and 6 mm respectively as per the available clinical data.

In clinical terminology, stenosis in any artery is referred in the form of % DS. But, physiologically the haemodynamic parameters are functions of % AS and they are having different values depending on the orientation and shape of deposited plaque for the same percentage of DS (refer table 1). Therefore, for the same % DS physiological parameters may behave different. Hence, in the present study stenosis based on reduction in lumen area only is followed. Three types of stenosed model: (1) asymmetric blockage (AB), (2) Eccentric blockage (EB) and (3) Concentric blockage (CB) as shown in figure 1 are considered. Five different cases of %DS i.e. 25%, 50%, 66.67%, 75% and 83.3% are modelled for each stenosed model. A circular shape plaque is assumed for the induction of blockage in all the three types of stenosed models. Although geometry of all the three types of stenosed models are designed with same five cases of %DS, their %AS values are different. Hence, these renal artery models resulted into fifteen different stenosed models which varies from 14.41- 97.22% of AS as shown in table 1.

![Figure 1. 75% diameter stenosed 3-D renal artery models for (1) asymmetric blockage (AB), (2) Eccentric blockage (EB) and (3) Concentric blockage (CB) ](image-url)
The sixteen arterial models comprising of a healthy and fifteen stenosed arteries are generated in SolidWorks 2013. Then after these CAD models are imported to ANSYS fluent v 14.5 for mesh generation and post processing. Tetrahedral mesh with linear elements are generated for all the geometries. To eliminate any computational error and to reduce the computational time and cost, grid independency test is also performed.

Table 1. The values of % Area Stenosis corresponding to their % Diameter Stenosis for three different types of stenosed models.

| Stenosed Model | AB  | EB  | CB  |
|----------------|-----|-----|-----|
| 25             | 14.4| 19.54| 43.75|
| 50             | 38.45| 49.97| 75  |
| 66.67          | 58.35| 70.82| 89.44|
| 75             | 68.5 | 80.45| 93.75|
| 83             | 78.87| 88.88| 97.22|

2.2. Fluid properties and boundary conditions

To analyse the effects of RAS on flow conditions in the renal artery, blood is considered as a fluid having constant density of 1060 kg/m$^3$ and homogeneous as in [14]. Since, in the presented study, the average Reynolds number ranges from 654 to 1900 therefore flow is assumed as laminar only [15,16]. The viscosity of blood decreases with increase in shear rate, hence it is a shear thinning non-Newtonian fluid. Therefore, to incorporate this non-Newtonian behaviour of blood in the flow simulation, Carreau model is employed in the study as suggested in many previous literatures [17, 18].

As per Carreau non-Newtonian model [19],

$$\frac{\mu - \mu_\infty}{\mu_0 - \mu_\infty} = [1 + (\lambda \gamma)^2]^{n-\frac{1}{2}}$$  

(1)

Here, $\mu$ represents the effective viscosity while $\mu_0$ and $\mu_\infty$ are the dynamic viscosities at zero and infinite shear rate respectively. Also $n$, $\lambda$, and $\gamma$ are representing power law index, characteristic viscoelastic time of fluid and shear rate respectively. The values of $\lambda$, $n$, $\mu_0$ and $\mu_\infty$ for blood are 3.313 s, 0.3568, 0.056 kg/m·s and 0.0035 kg/m·s respectively.

The continuity equation

$$\nabla \cdot \mathbf{V} = 0$$  

(2)

and Navier-Stokes equation

$$\rho \left( \frac{\partial \mathbf{V}}{\partial t} + \mathbf{V} \cdot \nabla \mathbf{V} \right) = -\nabla \tau - \nabla p$$  

(3)

governs the flow of incompressible blood in renal artery.

Here, $\mathbf{V}$, $\rho$, $t$, $\tau$ and $p$ is 3-D velocity vector, density, time, stress and pressure respectively. These governing equations are therefore solved numerically in ANSYS fluent v 14.5.

A set of boundary conditions are required to solve these governing equations. On the solid arterial walls, no-slip boundary condition is imposed. In order to simplify the simulation, the walls of the renal artery models are assumed rigid as it do not affect the output results significantly [20,21]. A constant pressure of 100 mm Hg is imposed at the outlet of the model since it is mean of systolic (120 mm of Hg) and diastolic (80 mm of Hg) blood pressure of a healthy person. Physiologically, blood flow inside vascular system is pulsatile in nature. Therefore to include this nature of blood flow in the analysis, a uniform time- varying velocity profile (shown in figure 2) is imposed at the inlet. The sinusoidal wave profile of blood flow during systole [21,22] is taken as an approximation to hemodynamic pulse. The heart cut – off the supply of blood in the diastolic phase. Hence, it is assumed that blood is flowing with a fixed velocity of 0.1 m/s during diastole. This applied velocity
profile yields a maximum inlet velocity of 0.5 m/s at \( t = 0.109 \) s (peak systole) with a rapid heart-beat of 120 / minute. The duration of this whole cardiac cycle is 0.5 s comprising of systolic phase for \( 0 s < t \leq 0.218 \) s and diastolic phase for \( 0.218 s < t \leq 0.5 \) s. The assumed velocity profile represents cardiac cycle during exercise state of human body. For the implication of the assumed velocity profile at inlet, a user defined function (UDF) is written in C language and interpreted in the fluent. In the present analysis, it is observed that results are period independent after second cycle. Hence, for the development of mathematical model results obtained from the simulation of the second cycle are used.

For the calculation of reduction in availability of blood flow to kidney, another set of simulations are performed for all the arterial models with pressure wave profile at the inlet having maximum pressure of 13599.3 Pa at peak systole and 13352.4 Pa during diastolic phase. The imposed pressure wave profile is constructed based on the results obtained from the simulations of healthy artery model for velocity inlet boundary condition.

![Uniform time varying velocity profile](image)

**Figure 2. Inlet velocity profile**

### 3. Results and discussion

The CFD simulation results for both the set of boundary conditions are obtained for velocity and pressure distributions in all the sixteen 3-D renal artery models for two cardiac cycle with a time step size of 0.01 s that results into a huge amount of data and is hard to present here. Hence, the velocity and pressure distribution results of healthy artery & only of the 75% DS of all the 3 types of stenosed models (AB, EB and CB) for \( t = 0.55 \) s are shown in figures 3 & 4 respectively as an illustration. It can be observed from figures 3 & 4 that there are large variations in maximum pressure and velocity of the three stenosed models in spite of same %DS. It is because of the fact that % area stenosis of these models are non-identical. For all the arterial models, it is observed that blood flow velocity reaches its peak at the medial region of stenosis as shown in figure 3. Generally, large pressure drop is observed post stenosed region and at the inlet, pressure is maximum as shown in figure 4.

The obtained numerical results are then utilized to calculate the rise blood pressure i.e. RVH as well as reduction in availability of blood flow to kidney during systolic as well as diastolic phase because of rise in severity of stenosis. In order to calculate the RVH during systole, the maximum value of pressure at peak of systole \( (t = 0.61 \) s) is obtained for the healthy and all the 15 stenosed artery models. After that, from the obtained values of maximum pressure in healthy and stenosed models, the corresponding percentage rise in pressure to healthy artery case is calculated. Now, considering 120 mm of Hg as standard baseline systolic blood pressure of healthy human, RVH
because of rise in severity of stenosis can be computed easily by adding percentage pressure rise to 120 mm of Hg baseline pressure. It can be described from the case of 93.75% AS (75% DS of CB stenosed model) as an example (refer row 7 of Table 2). For this case, the maximum pressure during peak systole is 359 mm of Hg which is approximately 72% more than that of a healthy artery. After adding this 72% pressure rise to baseline pressure of 120 mm Hg, systolic blood pressure or RVH at systole becomes 206 mm of Hg.

**Figure 3.** The velocity vector for healthy (a) & 75% DS of asymmetric blockage (b), Eccentric blockage (c) and concentric blockage (d)
Figure 4. The pressure distribution contour for healthy (a) & 75% DS of asymmetric blockage (b), Eccentric blockage (c) and concentric blockage (d)

In the similar way, the obtained maximum pressure at the end of cardiac cycle i.e. for t = 1 s are used to calculate RVH during diastole. During diastolic phase, 80 mm of Hg is taken as standard baseline pressure. The computed and obtained values from CFD analysis for rising extent of stenosis severity and its corresponding systolic and diastolic blood pressure (RVH/secondary hypertension) for the healthy and CB stenosed models are presented in table 2 as a demonstration.

Table 2. The computed and obtained values from CFD analysis for rising extent of stenosis severity and its corresponding systolic and diastolic blood pressure (RVH/secondary hypertension) for the healthy and CB stenosed models

| AS (%) | Systolic | Diastolic |
|--------|----------|----------|
|        | Maximum Pressure (mm of Hg) | % Pressure Rise | Blood Pressure (120 mm of Hg Baseline) | Maximum Pressure (mm of Hg) | % Pressure Rise | Blood Pressure (80 mm of Hg Baseline) |
| 0      | 102      | 0        | 120     | 100      | 0        | 80      |
| 43.75  | 103      | 1.36     | 122     | 100      | 0.08     | 80      |
| 75     | 118      | 13.44    | 136     | 101      | 0.76     | 81      |
| 89.44  | 183      | 44.26    | 173     | 105      | 5.11     | 84      |
| 93.75  | 359      | 71.59    | 206     | 113      | 11.54    | 89      |
| 97.11  | 1388     | 92.65    | 231     | 165      | 39.21    | 111     |

Another consequence of RAS is loss of blood flow to part of kidney with rise in percentage of stenosis. Hence to calculate the reduction in availability of blood flow to kidney, blood flow rate are obtained for a healthy and 15 stenosed artery models at peak systole (t = 0.61 s) and diastole (t = 1 s) using second set of boundary conditions in flow simulation. Considering the rate of blood flow obtained for the healthy artery model as 100% flow to kidney, corresponding reduction in rate of flow for all the cases of stenosed models are calculated. This consequently furnish the percentage availability of blood flow to kidney post stenosis. Table 3 shows the available blood flow to kidney for healthy and CB stenosed models during systolic and diastolic phase as a demonstration.

Table 3. The computed and obtained values from CFD analysis for rising extent of stenosis severity and corresponding availability of blood flow for the healthy and CB stenosed models.
The evaluated CFD results for RVH and available blood flow are then mapped to develop a mathematical model which relates blood flow rate and blood pressure to % AS by applying standard curve fitting techniques of MATLAB. The graph and equation of correlation equation for systolic secondary hypertension (RVH) and cross-sectional area stenosis is shown in figure 5. A four degree polynomial with coefficients having 95% confidence bounds fits the data appreciably (R-square = 0.992). Figure 6 depicts the graph and equation of correlation for diastolic secondary hypertension and cross-sectional area stenosis. A significant and good correlation exists for derived diastolic blood pressure and % AS (R-square = 0.9625, p < 0.05).

![Graph](image_url)

**Figure 5.** The correlation between % AS and systolic secondary hypertension.
Figure 6. The correlation between % AS and diastolic secondary hypertension

Estimated blood flow available to kidney are appreciably correlated with the % AS at systole (R-square = 0.9941, p < 0.05) and diastole (R-square = 0.9874, p < 0.05) as shown in figures 7 and 8 respectively. The cross-sectional area stenosis and available blood flo are non-linearly related. It can be visualized from figures 7 and 8 that blood flow is critically lower in part of kidney where blood is supplied by renal artery with stenosis more than 80 % in comparison to those regions where blood is supplied by artery having stenosis lesser than 50 %.

Figure 7. The correlation between available blood flow during systole and cross-sectional area stenosis
Figure 8. The correlation between available blood flow during diastole and cross-sectional area stenosis

Using developed mathematical models, decrease in blood flow and increase in blood pressure due to variation in extent of stenosis can be interpolated and can be reproduced in the form of graphs as shown in figures 9 and 10 respectively. These graphs can be employed as ready utility tools for the prediction of probable percentage of renal artery stenosis (RAS) and probability of blood flow available to kidney on the basis of measured blood pressure. These utility tools can be described by taking an example of 90% AS. The blood pressure for this case is 181/83 mm of Hg and for the corresponding %AS blood flow available to part of kidney during systole is 10.2% while that for diastole is only 7.2%. Hence, with the help of this simple and handy chart, a doctor/clinician can have a preliminary idea of severity of stenosis even by only measuring blood pressure.
Figure 9. A ready utility graph to diagnose probable reduction in blood flow based on cross-sectional area stenosis

Figure 10. A ready utility graph to diagnose probable renal artery Stenosis based on measured blood pressure

Various works based on experiment suggested that variations in pressure gradients or flow of blood are clearly visible if artery’s cross-sectional area gets decreased by 80% [23-26]. Sadeghi et al [27] in their work concluded that the average inlet and outlet pressure is having less difference for %AS < 70 and a visible change is observed for cross-sectional area stenosis of 80%. Therefore, in their work they suggested that the stenosis is severe beyond 70% area stenosis. In the current work too, it can be visualized that the rise in secondary hypertension and reduction in flow of blood is severe after 80% AS which is in accordance.

The results shown in the present study comprised of 3 types of stenosed artery models with some limitations and assumptions. The geometrical features that impacts the characteristics of haemodynamic variables like vessel bending [28,29], plaque curvature and vessel wall roughness are not included in the present work. The section of renal artery having plaque deposition at one location are only analysed for this work. Therefore, the formulated diagnostive tool may not remain valid for the case of multiple stenosis at different regions of renal artery. Even though, the prediction of RAS severity on the basis of this diagnostive tool can assist in inexpensive and early diagnosis, the proposed tool should not be considered as an alternative to Fractional Flow Reserve (a gold standard technique) while making decision for intercessional procedure planning.

4. Conclusion
The present work propounds use of a graph/chart formed diagnostive method to assess the criticality of stenosis. The propounded handy diagnostive graph is prepared after performing CFD analysis of blood flow in 3 types of stenosed artery models of renal artery having same %DS with variation in %AS. The chart is inexpensive and simple for use and can assist doctors in preliminary diagnosis while dealing with patients of high blood pressure.
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