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Study of Newtonian and Non-Newtonian Effect of Blood Flow in Portal Vein in Normal and Hypertension Conditions using CFD Technique

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Abstract

The hemodynamic simulation studies have been frequently used to gain a better understanding of functional, diagnostic and therapeutic aspects of blood flow. Many fundamental issues of blood flow, like effects associated with pressure and viscous force field are not fully understood or entirely explained through mathematical formulation and characterization of blood flow is still a challenging task. The study of behaviour of blood flow in the blood vessels also provides understanding of relation of blood flow and development of associated diseases to the cardiovascular experts. The understanding of the flow dynamics past prosthetic devices such as heart valves, vascular grafts and artificial heart will improve the design of implant and exhibit the better scenario for remedial option of cardiac surgery i.e. open heart surgery, bypass surgery etc.

In this paper the Newtonian effect of blood flow in the portal vein has been simulated for normal flow condition and considering portal hypertension case (with the formation of Thrombosis) and compared and validated with the results (with non-Newtonian effect) of C.C. Botar et al (2010). The 3-D geometry is prepared using 3D CAD software Pro-E and simulation has been carried out using commercial CFD code Ansys FLUENT. It has been observed that considering blood as Newtonian or non-Newtonian has no significant effect on the results for measurement of wall shear stress as the primary consideration.

Keywords: Blood flow, Hemodynamics, Bio-CFD, Portal vein, CFD simulation, Newtonian.

1. Introduction

The study of blood flow characteristics in the blood vessels in normal and diseased case has been a tremendous source of attraction for the medical practitioners and CFD analysts for past few decades. Various experimentations and simulations have been carried out to reveal the undisclosed facts associated with the blood flow behaviour. With the image based techniques from MRI and CT scans to reconstruct the real geometries of the human organs which otherwise was not possible, has given a new amplitude to the bio-CFD experts to produce the actual visualization of the hemodynamic properties. This has reduced the complexity of 3-D visualization of the blood flow behaviour in order to understand various diseases and their remedial measures.

People all over the world suffer from a disease called liver cirrhosis, a disease that can by itself cause an increase in the mortality risk and reduce the patient's quality of life. Cirrhosis is scarring which accompanies the healing of liver injury caused by hepatitis, alcohol or other less common causes of liver damage. Other causes of portal hypertension include blood clots in the portal vein, blockage of the veins that carry the blood from the liver to the heart, a parasitic infection called schistosomiasis and focal nodular hyperplasia. The problem associated with this disease is the rupture and bleeding of small blood vessels, due to the reduction of blood flow in the portal vein, which makes the pressure rise on the wall, forcing blood coming from the intestine around small vessels. These blockages originate blood recirculation within the portal vein increasing the wall shear stress as their size increases. The main reason causing the obstructed blood flow in the portal vein is 'portal hypertension'. Portal hypertension is an increase in the blood pressure within a system of veins called the portal venous system. Veins coming from the stomach, intestine, spleen and pancreas merge into the portal vein, which then branches into smaller vessels and travels through the liver. If the vessels in the liver are blocked due to liver damage, blood cannot flow properly through the liver. As a result, high pressure in the portal system develops. This increases pressure in the portal vein may lead to the development of large swollen veins (Varices) with the oesophagus, stomach, rectum or umbilical area belly button. Varices can rupture and bleed, resulting in potentially life-threatening complications.

C Van Steenkiste et al (2010) carried out a quantitative analysis of the WSS in the portal vein in casts of different animal models of portal hypertension (PHT) and cirrhosis using computational fluid dynamics (CFD). Microvascular changes in the splanchnic, hepatic and pulmonary territory of portal hypertensive and cirrhotic mice are described in detail with stereomicroscopic examination and scanning electron microscopy. WSS was significantly different between sham/cirrhotic/pure PHT animals with the highest values in the latter. This study showed that vascular casting has an important role not only in the morphological evaluation of animal models of PHT and cirrhosis, but also in defining the biological response of the portal vein wall to hemodynamic changes.

Although the blood is considered to be a non –Newtonian fluid and best described by power law model [4] as well as by casson velocity model [6], however Barbara M. Johnson et al (2006) studied the blood flow behaviour with a non-Newtonian blood

model (the Generalised Power Law), as well as the usual Newtonian model of blood viscosity to study the wall shear stress in the arteries over the entire cardiac cycle. The difference between Newtonian and non-Newtonian blood models was also studied over the whole cardiac cycle. The study showed that, considering the wall shear stress distribution for transient blood flow in arteries, the use of a Newtonian blood model is a reasonably good approximation. However, to study the flow within the artery in greater detail, a non-Newtonian model is more appropriate.

A diseased artery with plaques was demonstrated by T Chaichana et al (2011) to study the effect of plaque formation to the blood flow in the arteries which reveals the insight of the atherosclerosis, the most common cause of coronary artery disease inside the artery wall leading to blockage of the blood supply to the heart muscle. The results demonstrated a large circulation region at the left coronary bifurcation, and the velocity through bifurcation was increased. In contrast, a smooth flow pattern was observed in the non-calcified regions and flow velocity was low at the bifurcation. Low wall pressure was present in the coronary artery with a simulated coronary plaque whereas there was high wall pressure in the normal coronary artery. The simulated plaques resulted in high wall shear stress when compared to the low wall shear stress present in the normal coronary artery.

2. Methodology

The 3-D reconstruction of the portal vein geometry has been done from MRI scan, the dimension of which is obtained by C.C. Borat et al (2010) as given in Table 1. The geometrical model is prepared in the modelling software Pro-E and meshing is done in GAMBIT. The surfaces mesh of the right and left branch and main portal vein has been generated with quad mesh. The prism mesh has been adopted for the whole volume and exported to commercial CFD software Ansys Fluent for numerical modelling.

Table 1: Portal vein model dimensions (C.C. Botar et al 2010)

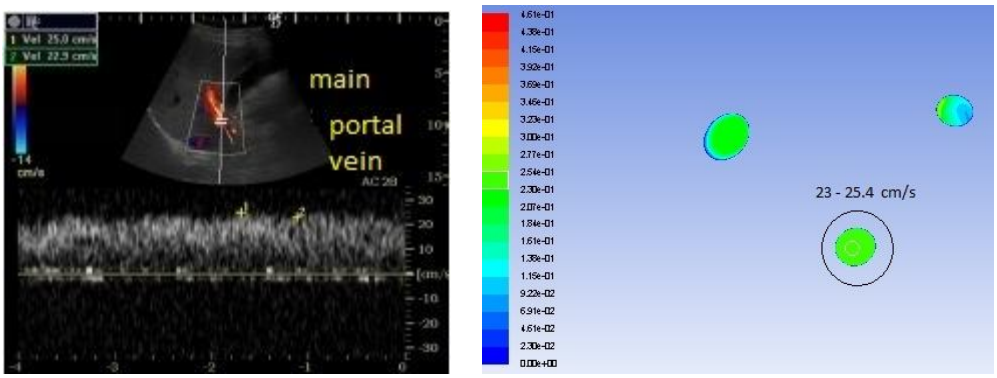
Description	Dimensions (mm)
Inlet main branch diameter	10.52
Middle main branch diameter	9.91
Distal main branch diameter	9.41
Main branch length	45.02
Left branch Inlet diameter	10.14
Left branch middle diameter	8.53
Left branch distal diameter	7.69
Left branch length	29.66
Right branch Inlet diameter	7.45
Right branch middle diameter	8.62
Right branch distal diameter	7.48
Right branch length	27.15

The flow model is considered to be incompressible and 3-D Navier-Stokes equations were solved for the characteristics of the blood. The viscosity of the blood is defined by Newton's law of viscosity which has been taken a value of 0.0037 kg/m-s. The turbulence model implemented to describe the hemodynamics was the realizable k-ϵ, which satisfies certain mathematical constraints on the Reynolds stresses, consistent with the physics of turbulent flows. It is also likely to provide superior performance for flows involving rotation, boundary layers under strong adverse pressure gradients, separation, and recirculation [7]. The CFD simulation of portal vein is initialized with a static pressure 2737.1 Pa and inlet velocity of 0.25 m/s [4]. The outlet of right and left branch is defined as outflow boundary with a flow rate weighing 0.7 for left branch and 0.3 for right branch. A steady state model has been used to reduce the time dependency of the blood flow in portal vein system. The young's modulus of elasticity of the vein is neglected considering the wall as rigid. The accuracy of the solution is of the second order with a convergence criteria being 1e-04.

3. Results & Discussion

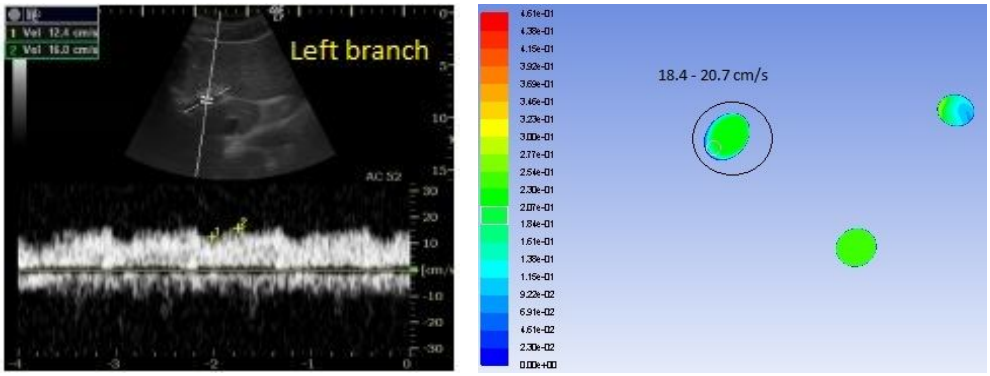
The simulated results for normal portal vein have been shown in figure 1,2 & 3 which are compared with experimental measurement i.e. vivo Eco-Doppler measurement from C.C. Botar et al (2010), where blood was considered as non-Newtonian. The results obtained from the present CFD simulation for Newtonian blood are very well agreed with those obtained by eco-Doppler measurement.

The velocity in the main portal vein obtained by the simulation is 23-25.4 cm/s and that obtained by medical investigation is 25 cm/s. Similarly left and right branches have the velocity 18.4-20.7 cm/s and 11.5-13.6 cm/s respectively obtained from the CFD simulation which is in the approximation of medical investigated measurements i.e. 12.4-16 cm/s and 10.8-13.2 cm/s for the left and right branches respectively Figure 1, 2 & 3. Further strain rate and wall shear stress distribution is shown in the portal vein. We can see the wall shear stress is almost constant in most of the flow path in portal vein and having the value of 0.1-0.2 Pa however it rises slightly near bifurcation but overall range remains within 0.1-0.45 Pa approximately which is well agreed with the wall shear stress obtained by C.C. Botar et al (2010) where blood was considered as non-Newtonian in his study and defined by power law viscosity model. Besides the experimental validation, the simulated results are also with the great agreement of the work carried out by M. Johnson et al (2006), where the results shown that considering the wall shear stress distribution for blood flow in arteries, the use of a Newtonian blood model is a reasonably good approximation.



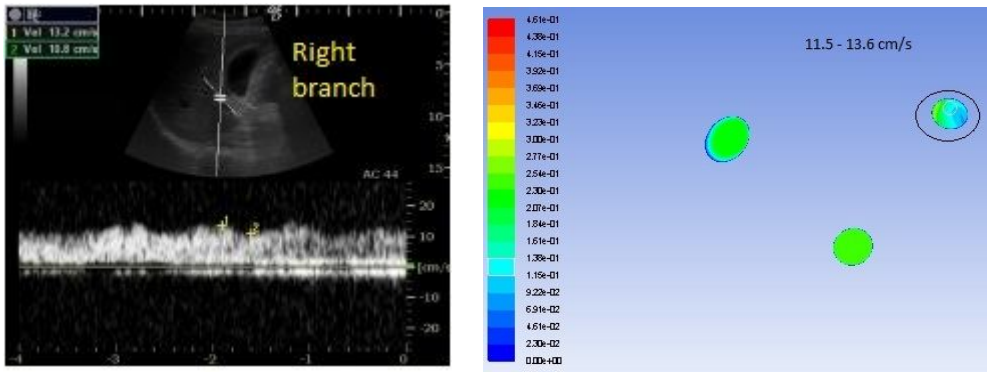
(a) **(b)**

Figure 1: Eco-Doppler measurement of velocity in main portal vein, courtesy of C.C. Botar, 2010 (a) Simulated result (b).



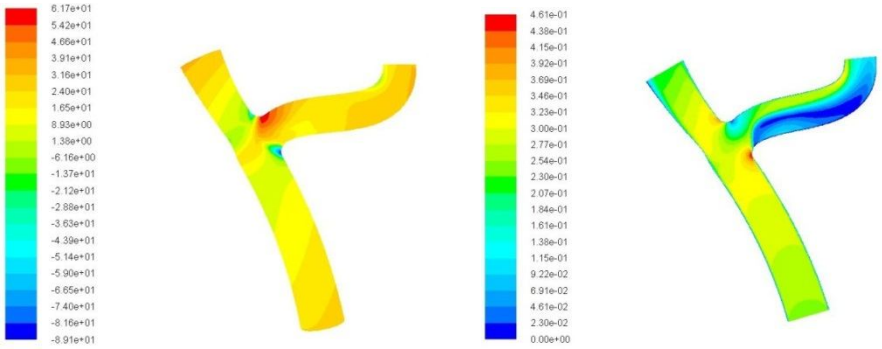
(a) **(b)**

Figure 2: Eco-Doppler measurement of velocity in left portal vein branch, [C.C. Botar, 2010] (a), Simulated result (b).



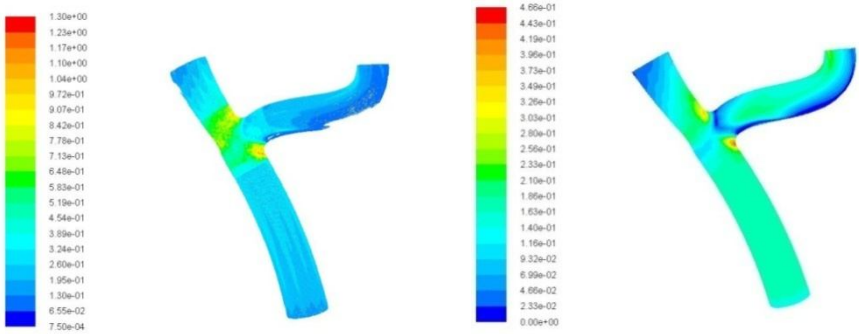
(a) **(b)**

Figure 3: Eco-Doppler measurement of velocity in right portal vein branch, [C.C. Botar, 2010] (a), Simulated result (b)



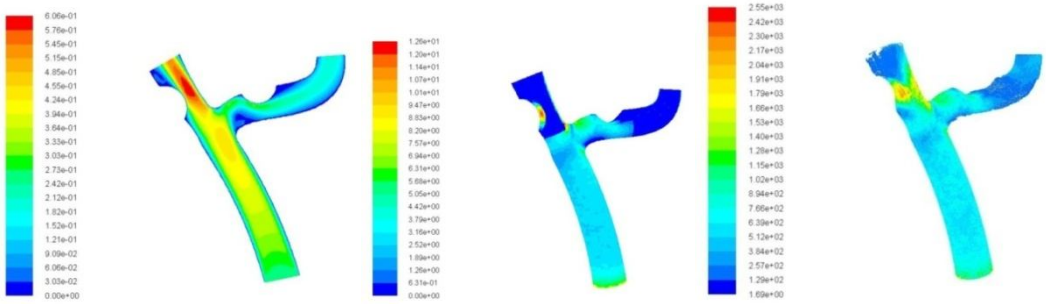
(a) **(b)**

Figure 4: Static Pressure (a), Contour of velocity on x-y plane (b) cutting through the middle of the geometry.



(a) **(b)**

Figure 5: Strain rate (a), Wall Shear Stress (b)



(a) **(b)** **(c)**

Figure 6: Velocity profile with Thrombosis on x-y plane cutting through the middle of the geometry (a) , Wall Shear Stress with Thrombosis (b), Strain rate with Thrombosis (c).

Further the case of portal hypertension has been considered with the formation of thrombosis at each branch as shown in Figure 6 and same methodology has been applied keeping the pressure and inlet velocity same as in normal case. Figure 6 (a), (b)

& (c) shows velocity, wall shear stress and strain rate profile in the obstructed blood flow condition. The velocity of the blood is increased up-to 50 cm/s and 24 cm/s in left and right branch which is far more than in normal case. The high velocity in the branch near the thrombus formation area results a low static pressure and thus shrinkage of the elastic wall of the vein which causes obstruction and even complete blockage of the blood flow. Also the wall shear stress is very high in thrombus formation area, Figure 6 (b) which can cause rupture and/or bleeding of the vein. The strain rate is high (of the order 1e+03) near the obstructed area, Figure 6 (c) which is low and uniform in case of normal blood flow (of the order 1e+00) Figure 5 (a). Due to reduced area of the vein near the thrombus formation, produces higher strain rate which eventually results high wall shear stress near the blockage area.

4. Conclusion

The simulation has been carried out for the blood considering Newtonian fluid and validation is done with eco Doppler measurements. The simulation has a very good agreement with the experimental results as well as with previous work done by M. Johnson et al (2006). The above simulation represents an insight of the obstructed blood supply in the portal vein due to injury or diseased liver, which might be helpful to understand the behaviour, effect and cause of the obstruction and further remedial actions can be suggested. The thrombosis is considered for the blockage inside the vein, similar to a portal hypertension condition. In future we will study the elasticity of the vein to predict the shrinkage effect due to low pressure in the obstructed area. Moreover species transport phenomenon, considering the blood as a combination of 3 species i.e. platelets, white blood cells and red blood cells is another aspect of study. The above approach is efficient and capable to study the flow parameters based on the patient's specific condition and can predict the medical practitioners the degree of risk and chances of liver damage.

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