

Thrombosis modeling in stented cerebral aneurysms with Lattice Boltzmann method

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Summary. This contribution presents the results of numerical simulations of thrombosis in a patient specific cerebral aneurysm, deployed with stents of different porosity. The thrombosis models are based on low wall shear stress constraint and high residence time of fluid. Simulations have been performed with a massively parallel Lattice Boltzmann solver on 8192 cores of Intel Sandy Bridge processors. Our studies show that the treatment of a cerebral aneurysm with a flow diverter stent induces thrombosis in the aneurysm bulge, which is even more intense with a less porous stent, due to enhanced flow diversion.

Key words: cerebral aneurysm, stent, thrombosis, Lattice Boltzmann method

Introduction

A cerebral aneurysm is a weak balloon like bulging of the wall of a brain artery. Rupture of an aneurysmal sac, termed subarachnoid hemorrhage (SAH) is a lethal condition and endovascular treatments have proved to be a good alternative over surgical options for their cure. Deployment of a flow diverter stent is one such treatment, the goal of which is to trigger the process of thrombosis inside the aneurysm by changing the local flow properties. Configuration and porosity of the stent plays a significant role on the aneurysmal fluid dynamics and consequently on thrombosis. Thrombosis modeling is a multiscale problem and its application in a patient specific case requires the use of efficient numerical techniques together with high performance computing resources [1]. We employ the Lattice Boltzmann method (LBM) with thrombosis models based on flow properties to qualitatively observe the changes in clotting inside an aneurysm bulge after the deployment of a stent.

Numerical Method

The Lattice Boltzmann Method

The Lattice Boltzmann method, which is based on the mesoscopic representation of fictional particle movements, is a numerical technique to simulate incompressible flows. The particles collide and stream on a fixed grid and in fixed directions, each of which have discrete velocities, to relax towards a thermodynamic equilibrium. Evolution of these particles over time is described by the Lattice Boltzmann equation with the BGK collision operator:

$$f_i(\mathbf{r} + \mathbf{c}_i \Delta t, t + \Delta t) = f_i(\mathbf{r}, t) + \Omega(f_i^e(\mathbf{r}, t) - f_i(\mathbf{r}, t)) \quad (1)$$

where f_i denotes the probability of finding a particle with discrete velocity \mathbf{c}_i at a position \mathbf{r} at time t . The indices which run from $i = 1 \dots Q$ denote the links per element i.e. the discrete

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directions, depending on the chosen stencil (D3Q19 in our case). The BGK collision operator Ω implies a single relaxation time at which distributions f_i relax towards the equilibrium f_i^e :

$$f_i^e = w_i \rho \left(1 + \frac{\mathbf{c}_i \cdot \mathbf{u}}{c_s^2} - \frac{\mathbf{u}^2}{2c_s^2} + \frac{1}{2} \frac{(\mathbf{c}_i \cdot \mathbf{u})^2}{c_s^4} \right) \quad (2)$$

where w_i are the weights for each discrete link, c_s is the speed of sound in vacuum and \mathbf{u} is the fluid velocity.

Flekkøy model for convection-diffusion in LBM

The Flekkøy model for passive scalar transport [2] is analogous to the transport of distributions within the LBM and is used in our thrombosis models to trace the age of the fluid. The transport counterparts of eqns. 1 & 2 for the Flekkøy model read:

$$\Delta_i(\mathbf{r} + \mathbf{c}_i \Delta t, t + \Delta t) = \Delta_i(\mathbf{r}, t) + \Omega_D(\Delta_i^e(\mathbf{r}, t) - \Delta_i(\mathbf{r}, t)) \quad (3)$$

$$\Delta_i^e = w_i \rho \left(1 + \frac{\mathbf{c}_i \cdot \mathbf{u}}{c_s^2} \right) \quad (4)$$

where Δ_i represents the probability distributions for the species, \mathbf{u} is taken from the underlying fluid and the relaxation parameter Ω_D is now dependent on the diffusion coefficient. For the species, a reduced D3Q6 stencil is used. Since the convection-diffusion equation itself does not contain any quadratic \mathbf{u} terms, they are allowed to vanish in the equilibrium distribution Δ_i^e as well. The kinematic viscosity and the diffusion coefficient are determined by:

$$\nu = \frac{1}{6} \left(\frac{2}{\Omega} - 1 \right) \quad D = \frac{1}{6} \left(\frac{2}{\Omega_D} - 1 \right) \quad (5)$$

Thrombosis models based on flow properties

The thrombosis models used in this study are based on local flow properties namely *Wall shear stress* and *Residence time* of fluid. An additional *proximity condition* is imposed on both these models, which allows only those fluid elements to turn into solid which are already attached to wall or previously formed clot. This condition ensures that no isolated clots appear in the center of the domain or near the in/outlets.

Wall shear stress constraint

Thrombosis is believed to occur in areas of low wall shear stress (WSS) and being locally driven by the blood shear rate near the vessel walls, which is controlled by a threshold [3]. This threshold level is influenced by factors like platelets, tissue, physiology etc. [3]. The occurrence of thrombosis in areas of low WSS is further supported by the fact that high shear can nevertheless wash away the onset of thrombosis. Numerically, WSS is computed for each element in the vessel to allow the decision for solidification together with the proximity condition.

Residence time model

Medical literature illustrates the coagulation of blood as a time dependent process [4]. The reason is that the activated platelets and procoagulants which are present in the blood need sufficient time to aggregate and form clots. This inspires the use of a residence time model as an additional constraint to the WSS for thrombosis modeling [5]. The residence time is computed by injecting a passive scalar, convection of which traces the age of the underlying fluid. The decision for solidification of elements in this model thus depends on the residence time and WSS thresholds along with the proximity condition.

Simulation Parameters

The patient specific aneurysm and the stent geometry were provided as surface meshes in STL format. Simulations were performed with our fully parallel Multiphysics framework *Apes* [6]. The mesh generation tool *Seeder* [7] created a voxelized mesh that was used by the LBM solver *Musubi* [8] for computations, and finally visualized by the post-processing tool *Harvester*.

The stented aneurysm geometry is shown in fig. 1(a). For a reasonable resolution of the stent, the mesh was discretized with cubical elements of $\delta x = 65\mu\text{m}$ which resulted in nearly 45 million fluid elements. Stent pores which are located towards aneurysmal sac are $\sim 585\mu\text{m} \times 295\mu\text{m}$ which gave us roughly 55 fluid elements between the pores, whereas the struts were resolved with nearly 1 element. The parent artery diameter was $D_{\text{artery}} \sim 3.96\text{mm}$. The simulation time step was $\delta t = 28.9\mu\text{s}$ and inlet velocity $u_{\text{in}} = 9.435 \times 10^{-2}\text{m/s}$. Density and kinematic viscosity of the blood were respectively set to $\rho = 1025\text{kg/m}^3$ and $\nu = 3.8 \times 10^{-6}\text{m}^2/\text{s}$. The parent artery Reynolds number was 50 based on D_{artery} , $u_{\text{mean}} = u_{\text{in}}/2$ and the prescribed blood properties. The simulations were performed using 8192 cores of the *SuperMUC* x86 cluster, at LRZ Munich.

Simulation Results

Thrombosis with wall shear stress constraint

Figure 1(b) shows the onset of thrombosis in the stented aneurysm with an upper WSS threshold of $1.037 \times 10^{-2}\text{Pa}$. The clotting was initiated after the simulation achieved a steady state and the clots continued to grow up to nearly 8000 iterations after that.

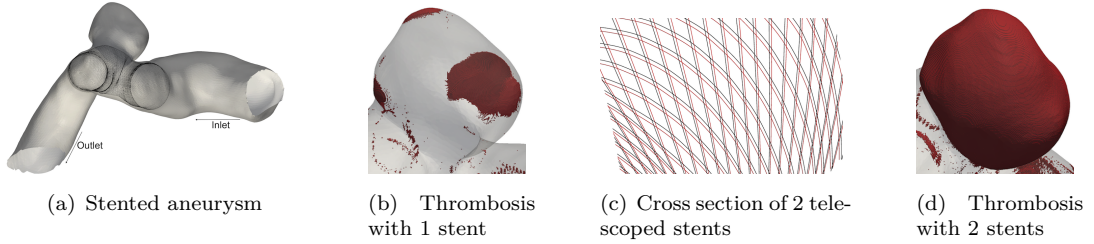


Figure 1: Thrombosis with a wall shear stress threshold of $\text{WSS} \leq 1.037 \times 10^{-2}\text{Pa}$.

Telescoping stents

A common treatment for wide neck aneurysms is multiple stent-in-stent deployments which are surgically carried out in a telescoping fashion [9]. Fig. 1(c) shows such an overlay of stents which decreased the porosity of the original stent by $\sim 1.5\times$, and consequently increased the clotting in the bulge dramatically for the same shear threshold (fig. 1(d)).

Thrombosis with residence time model

Figure 2 shows thrombosis growth in the aneurysm, post deployment of a single stent with thresholds of $t_{\text{thr}} \geq 3.88\text{s}$ & $\text{WSS} \leq 1.037 \times 10^{-1}\text{Pa}$. Unlike the WSS model, the clots cultivated

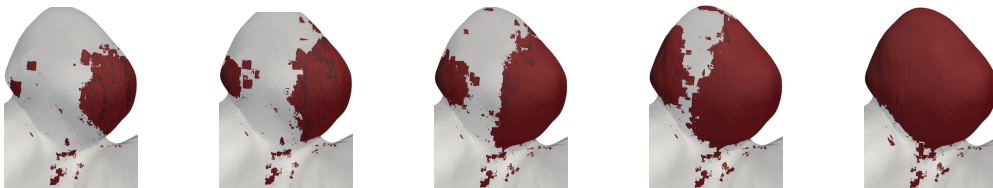


Figure 2: Thrombosis progression with residence time model: 10^5 , $2 \cdot 10^5$, $3 \cdot 10^5$ and $4 \cdot 10^5$, $6.6 \cdot 10^5$ time steps after clotting initiation. Thresholds are $t_{\text{thr}} \geq 3.88\text{s}$ & $\text{WSS} \leq 1.037 \times 10^{-1}\text{Pa}$. Complete bulge obliterated after $1.3 \cdot 10^6$ time steps.

in layers with this model to eventually obliterate the complete aneurysm bulge. *Telescoping* of stents with this model showed similar results, but the complete occlusion was ~ 1.4 times faster

as the less porous stent did not only decrease the shear stress, but it also increased the residence time of fluid inside the bulge markedly.

Conclusions

These results qualitatively show that stent deployment sparks the onset of thrombosis in a side wall aneurysm and the technique of telescoping stents looks promising for treatment of such aneurysms. The use of LBM seems very suitable for such simulations due to its ease in handling complex boundary conditions and resolving a stent. The residence time model puts an additional constraint to shear stress and mimics biology more meticulously as the clots grow in layers over time. It is however too early to quantify the performance of different stents with the use of time independent flow and assuming blood as a Newtonian fluid. Our future work will cover investigations with pulsating flow due to cardiac cycle and non-Newtonian models on different classes of aneurysms like those with bifurcation, with physiologically realistic parameters.

Acknowledgements

The geometries used for these studies were provided by the *THROMBUS* project, funded by the EU in the FP7 in the area of VPH (ICT-2009.5.3, PR 269966). Computer resources for this project were provided by the Leibniz Supercomputing Center under grant *pr45du*.

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