Published in final edited form as:

Ann Biomed Eng. 2013 April ; 41(4): 725–743. doi:10.1007/s10439-012-0719-9.

Virtual Treatment of Basilar Aneurysms Using Shape Memory Polymer Foam

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Abstract

Numerical simulations are performed on patient-specific basilar aneurysms that are treated with shape memory polymer (SMP) foam. In order to assess the post-treatment hemodynamics, two modeling approaches are employed. In the first, the foam geometry is obtained from a micro-CT scan and the pulsatile blood flow within the foam is simulated for both Newtonian and non-Newtonian viscosity models. In the second, the foam is represented as a porous media continuum, which has permeability properties that are determined by computing the pressure gradient through the foam geometry over a range of flow speeds comparable to those of *in vivo* conditions. Virtual angiography and additional post-processing demonstrate that the SMP foam significantly reduces the blood flow speed within the treated aneurysms, while eliminating the high-frequency velocity fluctuations that are present within the pre-treatment aneurysms. An estimation of the initial locations of thrombus formation throughout the SMP foam is obtained by means of a low fidelity thrombosis model that is based upon the residence time and shear rate of blood. The Newtonian viscosity model and the porous media model capture similar qualitative trends, though both yield a smaller volume of thrombus within the SMP foam.

Keywords

aneurysm; shape memory polymer foam; computational fluid dynamics; post-treatment hemodynamics

1 Introduction

Over the last decade, the treatment of choice for intracranial aneurysms has become endovascular coiling. Within the United States, approximately 25,000 patients are treated each year using coils [8, 37]. Despite the widespread use of this technique, a significant fraction of these cases have a suboptimal outcome [29]. Most often, the reasons for these treatment failures are the protrusion of the coils into the parent artery, incomplete occlusion of the aneurysm, or coil compaction over time. In light of these shortcomings, a number of

alternative endovascular techniques have been developed or are being investigated to either supplement or replace the use of coils. Some of these include three-dimensional coil designs and supplementary stents, which span the aneurysm neck and better retain additional coils inside the aneurysm [7, 11]. Others include liquid embolic agents and, as is being investigated by our research group, shape memory polymer (SMP) foam [38, 42, 43].

SMP foam was first used to treat aneurysms in a study performed by Metcalfe *et al.* [42], who invasively inserted the foam into canine model aneurysms. The results of the study demonstrate that the open cellular structure of the foam favors an in-growth of the cells involved in neointima formation. Recently, our research group developed an endovascular technique to deliver SMP foam to an aneurysm [38]. During the treatment procedure, a compressed piece of foam (Fig. 1) is delivered through a catheter to the aneurysm where it is heated by laser or thermal energy from a radially-diffusing fiber optic cable or electrical wires, respectively, embedded within the foam. The energy deposition releases the foam from its secondary compressed shape, allowing it to expand to its primary shape and, thereby, fill the aneurysm. Since the primary shape into which the foam is cut is arbitrary, patient-specific shapes can be produced from pre-operation CT or MRI scans of the aneurysm to be treated. Predetermined shapes and sizes can also be mass produced for more general, non-patient-specific use. Lastly, the small recovery stresses of the SMP foam allow for the delivery of an oversized device that can completely fill the aneurysm and not pose a risk of exceeding the maximum aneurysm wall strength [30].

An aneurysm treated with SMP foam will subsequently fill with thrombus, leading to the occlusion of the aneurysm. This complex thrombus formation process has been characterized by Anand *et al.* [1], Biasetti *et al.* [6], Bedekar *et al.* [4], Einav and Bluestein [16], Fogelson and Guy [22], Goodman *et al.* [24], Harrison *et al.* [27], Sorensen *et al.* [56, 57], Tamagawa and Matsuo [60], Wootton *et al.* [63], and others. These models use techniques that couple the flow field to the cascade of biochemical reactions governing the activation of platelets due to injury or high shear and the subsequent transport, interactions, and adhesion of cellular products to one another, as well as to the vessel wall. While these high fidelity approaches have proven to be relatively successful in modeling thrombus formation within rather simple flow geometries, their complexity is especially prohibitive for highly three-dimensional shapes, such as the SMP foam. Therefore, an entirely different approach is needed at the present time.

Such an approach can be found from a number of lower fidelity techniques that do not explicitly model these complex biochemical reactions and biomechanical processes, but rather identify relatively simple hemodynamic conditions in the flow field that favor flow stagnation and subsequent thrombus formation. When such conditions are met, blood is transformed to thrombus and the flow field is modified accordingly. As this process continues over time, a history of the thrombus shape is produced. For example, Friedrich and Reininger [23] experimentally determine that thrombus growth is associated with regions characterized by long fluid residence times, such as recirculation zones. A comparison of the transient *in vitro* thrombus shapes surrounding a catheter and those predicted computationally shows very good agreement between the two. Narracott *et al.* [44] model the development of thrombus within a lateral aneurysm filled with a representation of endovascular coils. In a manner similar to that of Friedrich and Reininger [23], Narracott *et al.* also compute the residence time using an advection-diffusion equation. However, additional criteria are implemented into the model to account for the thrombogenicity of the coil surface. Ouared *et al.* [47] study thrombus growth in lateral aneurysms, giving particular attention to the effect that the shear rate has upon thrombus formation. In their simulations, thrombus is allowed to form when the fluid shear rate decreases below 100 s^{-1} .

Harrison *et al.* [26] include both residence time and shear stress criteria into their thrombus formation model, which is developed to investigate the surface deposition of enzyme-activated milk used as a blood analogue. Thrombus is predicted to occur when the residence time exceeds the clotting time of milk and when the fluid shear stress decreases below a predefined value. This approach predicts thrombus patterns similar to those observed within an *in vitro* experiment of a three-dimensional stenosis. Bernsdorf *et al.* [5] also find qualitative agreement between the computational and experimental results of *in vitro* flow through a three-dimensional stenosis, though only the clotting time criterion is considered for thrombus formation. Corbett *et al.* [12] characterize the size of thrombus within recirculation zones surrounding a stenosis. Numerical simulations of their experiment, which uses blood having a clotting time of 200–220 s, demonstrate that thrombus forms when the shear rate is less than 49 and 54 s⁻¹ for Newtonian and non-Newtonian viscosity models, respectively. Lastly, Rayz *et al.* [49] model the flow within patient-specific, fusiform basilar aneurysms, which became occluded with thrombus over the course of the patients' medical history. A correlation analysis shows a significant relationship between the *in vivo* thrombus deposits and those regions within the simulations characterized by both low wall shear stress and long fluid residence times. Due to the simplicity of these lower fidelity approaches, they can be readily extended to the flows about more complex geometries, such as that within a foam-filled aneurysm. This, in turn, can lead to a better understanding of the SMP foam treatment technique.

While much progress has been made over the past several years in developing the SMP foam, a number of important questions still remain, three of which are as follows. What hemodynamic changes occur to the flow within the aneurysm following the treatment? How effective is the foam geometry at stagnating the blood within the aneurysm and promoting hemodynamic conditions that will increase the likelihood of thrombus formation and subsequent healing? And, what improvements can be made to the SMP foam device to further enhance its effectiveness? The purpose of this study is to provide additional answers to these questions. To do so, we virtually treat two patient-specific basilar aneurysms with SMP foam and investigate the resulting hemodynamics using computational fluid dynamics (CFD) simulations.

2 Computational Setup

In early CFD studies, the virtually treated aneurysm is modeled using a low fidelity approach, in which the coils are represented as random, solid computational cells located throughout the aneurysm [25]. Later studies model the coil geometry, though with increased computational costs as a result of having to resolve the complex flow details surrounding each coil [9]. Other studies, such as Jou *et al.* [32], strike a compromise between these low and high fidelity approaches by modeling the coil-filled aneurysm as a porous media continuum, in which the geometric details of the coils are not resolved in order to produce a pressure gradient as blood passes through the treated aneurysm. This significantly reduces the required computational resources, but two critical assumptions must be made in order to execute this third modeling approach. First, the permeability of the coils is often obtained from analytical expressions based upon canonical shapes, such as flow through parallel tubes or perpendicular to circular cylinders. Lacking experimental data to confirm this representation of the coils, it is difficult to determine both the accuracy of the porous media model and its applicability to the complex coil mass, which may very well have an anisotropic, heterogeneous permeability. Second, the porous media models that have been utilized within the literature do not account for the non-Newtonian viscosity of blood. While this assumption can be justified for higher speed flow within the larger parent artery, it is uncertain as to whether or not it is applicable to the slower moving blood passing through the treated aneurysm.

2.1 Modeling of the SMP Foam

For the present study, the SMP foam is modeled within the aneurysm using the two higher fidelity approaches. We first simulate the SMP foam within the aneurysm using the actual foam geometry (geometric approach) with both Newtonian and non-Newtonian viscosity models. The shape of the foam is obtained by means of a micro-CT scanner (Skyscan 1172, Micro Photonics Inc.). The foam is virtually reticulated by applying an intensity threshold to the micro-CT images, thereby removing the faces that exist between individual foam pores and generating an open-cell structure. The resulting foam geometry has a porosity of $\varepsilon = 0.985$ and a surface area per unit bulk volume of $1300 \text{ m}^2/\text{m}^3$ (Fig. 2a–b). Since the high resolution micro-CT scan of the foam limits the sample volume to a value less than that of the aneurysms to be treated, multiple copies of the foam geometry are arranged to form an assembly that is larger than the aneurysm volume. This assembly is delivered to two patient-specific basilar aneurysms that are obtained from computed tomography angiography (CTA). The foam at the aneurysm neck is sculpted under the guidance of an interventional neuroradiologist (J. Hartman), such that none of the aneurysm wall is exposed at the intersection of the foam and the parent artery (Fig. 2c–d). While it is possible to further optimize the shape of the SMP foam in order to achieve improved post-treatment hemodynamics at the bifurcation (unpublished results), we select a convex, spherical representation that smoothly directs the flow from the parent artery into the two PCAs. This shape approximates the spherical foam devices used within our *in vitro* and *in vivo* treatments of terminal and lateral aneurysms [38, 52]. Following this virtual treatment, the total surface area inside the aneurysm (patient 1) from which thrombus can adhere to and grow from is increased from $2.3 \times 10^{-4} \text{ m}^2$ (pre-treatment aneurysm wall surface area) to $7.6 \times 10^{-4} \text{ m}^2$ (pre-treatment aneurysm wall surface area plus the SMP foam surface area). For patient 2, the corresponding values are $3.0 \times 10^{-4} \text{ m}^2$ and $9.7 \times 10^{-4} \text{ m}^2$, respectively.

The second method of modeling the SMP foam is through the use of a porous media model (continuum approach). While such models exist for non-Newtonian fluids [53], none have been developed for the generalized power law viscosity model of blood [3, 21, 31],

$$\mu = \lambda |\dot{\gamma}|^{n-1} \quad (1)$$

utilized in the present study, where λ is the power law consistency index,

$$\lambda(\dot{\gamma}) = \mu_0 + \Delta\mu \exp \left[- \left(1 + \frac{|\dot{\gamma}|}{a} \right) \exp \left(\frac{-b}{|\dot{\gamma}|} \right) \right] \quad (2)$$

n is the power law index,

$$n(\dot{\gamma}) = n_o - \Delta n \exp \left[- \left(1 + \frac{|\dot{\gamma}|}{c} \right) \exp \left(\frac{-d}{|\dot{\gamma}|} \right) \right] \quad (3)$$

$n_o = 1.0$, $\mu_0 = 0.0035 \text{ Pa}\cdot\text{s}$, $\Delta\mu = 0.025 \text{ Pa}\cdot\text{s}$, $\Delta n = 0.45$, $a = 50$, $b = 3$, $c = 50$, $d = 4$, $\dot{\gamma}$ is the fluid shear rate,

$$\dot{\gamma} = \sqrt{2\mathbf{S}:\mathbf{S}} \quad (4)$$

and $\mathbf{S} = \frac{1}{2}(\nabla \mathbf{u} + \nabla \mathbf{u}^T)$ is the symmetric portion of the velocity gradient tensor, $\Delta \mathbf{u}$. This viscosity model predicts a shear-thinning behavior for very small shear rates and a constant value of 0.0035 Pa·s for shear rates greater than approximately 200 s⁻¹.

Lacking a more representative non-Newtonian porous media model for blood, we employ the Forchheimer-Hazen-Dupuit-Darcy (FHDD) equation for a Newtonian fluid,

$$-\nabla p = \frac{\mu_0}{\mathbf{K}} \mathbf{u} + \rho \mathbf{C} |\mathbf{u}| \mathbf{u} \quad (5)$$

which lumps the complex geometric details of the media into an expression relating the pressure gradient, ∇p , across a differential element of the porous media to the fluid velocity, $\mathbf{u}$, fluid density, ρ , permeability tensor, $\mathbf{K}$, and form factor tensor, $\mathbf{C}$, where ρ is taken to equal 1060 kg/m³ for blood [35]. To compute the values of $\mathbf{K}$ and $\mathbf{C}$ that will be used in the continuum approach, we solve for the flow field within the actual foam geometry (geometric approach), which is centered in a hexahedral-shaped computational domain that has the dimensions 15L×1L×1L, where L is the length of one side of the cube-shaped foam sample (Fig. 2a–b). At the inlet to the computational domain, a steady, uniform velocity boundary condition is specified and the pressure is extrapolated from the inside of the domain. The inlet velocity, u_0 , ranges up to 1.2 m/s, which is slightly greater than the maximum fluid velocity in a healthy basilar artery (BA) [34]. A slip velocity boundary condition is specified on the four walls and the computed variables are extrapolated at the outlet face of the computational domain. On the surface of the foam, a no-slip velocity boundary condition is specified. We assign a rigid boundary condition to the surface of the foam since our previous *in vitro* treatment of a terminal basilar aneurysm showed no significant foam deformation due to flow impingement [38]. Since the foam geometry may be anisotropic, it is oriented along each of the three coordinate directions in separate simulations. Blood is modeled as a continuum since the red blood cell diameter ($\approx 8 \times 10^{-6}$ m) is approximately 0.8% of the nominal foam pore size [17].

The flow field for the geometric approach is subsequently obtained by solving the conservation of mass,

$$\frac{\partial}{\partial t} \int_V \rho dV + \oint_A \rho \mathbf{u} \cdot d\mathbf{A} = 0 \quad (6)$$

and momentum,

$$\frac{\partial}{\partial t} \int_V \rho \mathbf{u} dV + \oint_A \rho \mathbf{u} \mathbf{u} \cdot d\mathbf{A} = - \oint_A p \mathbf{I} \cdot d\mathbf{A} + \oint_A \mu (\nabla \mathbf{u} + \nabla \mathbf{u}^T) \cdot d\mathbf{A} \quad (7)$$

equations for an incompressible fluid using a finite-volume code (STAR-CCM+ v. 6.02.007, CD-Adapco), where V and A are the computational cell volume and face areas, respectively, and $\mathbf{I}$ is the identity matrix. The code solver uses a co-located variable arrangement and a Rhie-and-Chow-type pressure-velocity coupling combined with a SIMPLE-type algorithm [13, 14, 20, 40, 41, 48]. The convective fluxes in Eqs. 6–7 are evaluated with a second-order upwind scheme. These equations are solved within the computational domain on an unstructured, hexahedral cell mesh, which is comprised of a Cartesian core mesh and several layers of hexahedral prismatic cells extruded from the foam surface. Between these two meshes are transitional, polyhedral cells. For inlet velocities that require the solution of an unsteady flow, the time derivatives in Eqs. 6–7 are discretized using a second-order implicit

scheme with a time step of 1×10^{-3} s and approximately 20 to 40 iterations are required for the solver to converge at each time step.

2.2 Solution of the Intra-Aneurysmal Flow

Using the geometric and continuum approaches, we solve for the flow fields within the foam-filled aneurysms. For the geometric approach, Eqs. 6–7 are computed throughout the aneurysm and parent artery for both the Newtonian and non-Newtonian viscosity models, whereas in the continuum approach, only the Newtonian model is employed. Within the continuum representation of the foam, the conservation of mass and momentum equations are

$$\frac{\partial}{\partial t} \int_V \rho \varepsilon dV + \oint_A \rho \mathbf{u} \cdot d\mathbf{A} = 0 \quad (8)$$

$$\frac{\partial}{\partial t} \int_V \rho \varepsilon \mathbf{u} dV + \oint_A \rho \mathbf{u} \mathbf{u} \cdot d\mathbf{A} = - \oint_A p \mathbf{I} \cdot d\mathbf{A} + \oint_A \mu_0 (\nabla \mathbf{u} + \nabla \mathbf{u}^T) \cdot d\mathbf{A} - \int_V \left(\frac{\mu_0}{\mathbf{K}} + \rho C |\mathbf{u}| \right) \cdot \mathbf{u} dV \quad (9)$$

To properly apply the boundary conditions to the computational domain, circular, cylindrical extensions are lofted from the basilar artery, the two posterior cerebral arteries (PCA), and the two superior cerebellar arteries (SCA) (Fig. 3a–b). Drawing from previous work, we assume that the distensibility of the aneurysm and artery walls is minimal and therefore model them as rigid [15, 19, 59, 64].

A pulsatile velocity boundary condition is specified at the inlet to the basilar artery. Using the *in vivo* measurements of Kato *et al.* [34] (Fig. 3c), we compute the Womersley solution [62] for the pulsatile velocity profile, $U(r, t)$, that is applied to the inlet (Fig. 3a–b), where r is the radial distance from the centerline of the inlet. A typical resting pulse frequency of $f = 1.17$ Hz (70 beats/minute) is chosen, yielding a Womersley number, $Wo = 0.5d_0 \sqrt{2\pi f \rho / \mu_0}$, of 3.0, where $d_0 = 4.0 \times 10^{-3}$ m is the diameter of the inlet circular cylinder to the basilar artery. To complete the specification of the boundary conditions, fractional outlet flowrates are assigned to the two PCAs and two SCAs. From our previous study of the basilar aneurysm of patient 1, we specify that 38.5% of the basilar artery inflow exits through each of the PCAs and that 11.5% of the remaining inflow exits through each of the SCAs (see Ortega *et al.* [46] for the justification of these outlet flowrates).

To generate an initial condition for the unsteady solver, an estimate of the flow field is first obtained by solving the steady-state formulation of the conservation of mass and momentum equations for a few thousand iterations with a steady inlet velocity profile of $U(r, t = 0)$. The unsteady solver is then initialized with this “steady-state” solution and then run for three cardiac cycles to reduce any start-up transients. After the third cardiac cycle, the unsteady solution is computed for seven additional cycles over which the variables are analyzed and the results presented. The solution is advanced with a time step of 1×10^{-3} s, which is sufficiently small for resolving the periodic vortex shedding within the aneurysm [46]. Depending upon the modeling approach, approximately 25 to 100 solver iterations are required at each time step for the normalized conservation of mass and momentum equation residuals [58] to decrease to at least $\approx 1 \times 10^{-4}$.

2.3 Solution of the Passive Scalar Flow

By applying a low fidelity thrombus formation model to the post-treatment aneurysms, we can gain information about the initial development of thrombus within the SMP foam. The

residence time, t_r , of blood is modeled as a passive scalar using an advection-diffusion equation,

$$\frac{\partial}{\partial t} \int_V \rho \varepsilon t_r dV + \oint_A \rho t_r \mathbf{u} \cdot d\mathbf{A} = \oint_A \frac{\mu}{Sc} \nabla t_r \cdot d\mathbf{A} + \int_V \varepsilon T dV \quad (10)$$

where $Sc = \nu/D$ is the Schmidt number, $\nu = \mu/\rho$ is the diffusivity of momentum, D is the diffusivity of t_r , and T is a source term. For the geometric modeling approach, $\varepsilon = 1$, while for the continuum approach, $\varepsilon = 0.985$ (the porosity of the SMP foam) within the foam-filled aneurysm. A value of zero is assigned to t_r at the inlet to the computational domain, while at the outlets, t_r is extrapolated from the interior of the domain. Along the walls of the artery and foam, a zero gradient boundary condition is specified for t_r . The residence time field is initialized to zero at $t = 0$ and, over the first three cardiac cycles, $T = 0$. At the end of the third cardiac cycle at which time the initial flow field transients have subsided, $T = \rho$, such that t_r increases by one unit for each unit of simulation time. To minimize diffusion, such that the value of t_r remains closely associated with a single fluid parcel over the course of the entire simulation, the Schmidt number is set to 1×10^6 . A simple scaling analysis shows that with this value of Sc , the diffusion length scale of t_r over the seven cardiac cycles of data analysis is $\sqrt{7D/f} = \sqrt{7\mu_0/(\rho f Sc)} = 4 \times 10^{-6}$, which is $\approx 0.4\%$ of the nominal pore size. As such, this value of Sc is suitable for the present study. The amount of time required for thrombus to begin forming in the domain is taken to be 240 s, which is based upon the measurements of Evans *et al.* [18] for the incipient clot formation time of whole human blood. Since the computing resources required to model the flow field for time scales greater than the clotting time are quite substantial, we solve Eq. 10 for seven cardiac cycles and then extrapolate the transient behavior of t_r to future times. These trends in t_r are then combined with the shear rate thresholds of Corbett *et al.* [12] to establish the criteria for thrombus formation. Obviously, this approach cannot capture the transient evolution of thrombus because the computed values of the residence time and shear rate are limited to the original thrombus-free foam. However, the locations at which thrombus is likely to begin forming can be estimated, which is useful for assessing the effectiveness of the treatment procedure.

An additional advection-diffusion equation,

$$\frac{\partial}{\partial t} \int_V \rho \varepsilon C_a dV + \oint_A \rho C_a \mathbf{u} \cdot d\mathbf{A} = \oint_A \frac{\mu}{Sc} \nabla C_a \cdot d\mathbf{A} \quad (11)$$

is simultaneously solved to model the transport of a virtual contrast agent with concentration, C_a , an approach which is similar to that of Cebal *et al.* [10], Larrabide *et al.* [36], and others. The boundary conditions for the contrast agent at the outlets and at the artery and foam walls are identical to those of t_r and Sc is likewise set to 1×10^6 . At $t = 0$, C_a is initialized to zero throughout the entire computational domain. To model the transient injection of contrast agent into the basilar artery, the inlet concentration boundary condition, $C_{a0}(t)$, is set to one over the fourth and fifth cardiac cycles and zero otherwise. Virtual angiography is performed on the pre- and post-treatment cases by interpolating C_a onto Cartesian planar slices made in the z -direction (see Fig. 2d for the coordinate system definition). The values of C_a at each (x, y) location are then summed along the z -direction to generate a two-dimensional projection of the contrast agent field.

3 Results

In the following sections, the porous media properties of the SMP foam are determined and applied to the continuum modeling approach of the post-treatment aneurysms. The mesh-convergent results from both the continuum and geometric modeling approaches are then assessed and correlated with the conditions that will lead to the development of thrombus within the SMP foam.

3.1 Porous Media Properties of the SMP Foam

The pressure drop, Δp , across the SMP foam geometry is measured as a function of u_0 (Fig. 4a) and is seen to exhibit a transverse anisotropy, in which Δp is about equal in the x - and z -directions, but is distinctly larger in the y -direction. This anisotropy is produced by the foam forming process, whereby the foam is expanded vertically in the y -direction against gravity and is confined in the x - and z -directions by the walls of a cylindrical container [55]. Consequently, the pores are relatively smaller in the $x-z$ plane (Fig. 2b) and present a greater resistance to blood passing through the foam in the y -direction. This anisotropy is utilized during the virtual aneurysm treatment by orienting the foam such that the y -direction is aligned to the parent artery axis (Fig. 2c-d). By doing so, the foam is more effective at both decelerating the blood entering the aneurysm and establishing conditions that are favorable for thrombus formation. The values of the diagonal terms for $\mathbf{K}$ and $\mathbf{C}$ are then obtained by fitting the FHDD equation (Eq. 5) to the $\Delta p/L$ versus u_0 data. It should be noted that the x -, y -, and z -directions are nearly aligned to the principle axes of the $\mathbf{K}$ and $\mathbf{C}$ tensors due to both the cylindrical, asymmetric shape of the container in which the foam is formed and the alignment of the y -axis of the foam sample to the axis of the cylindrical container. Therefore, the off-diagonal terms in $\mathbf{K}$ and $\mathbf{C}$ are taken to equal zero.

The FHDD equation, which utilizes a constant viscosity, μ_0 , captures the pressure gradient for the geometric approach with the non-Newtonian viscosity model (Fig. 4a), indicating that the average shear rates within the foam (≈ 400 to 10000 s^{-1}) are large enough for blood to behave as a Newtonian fluid over this range of inlet flow speeds (0.1 to 1.2 m/s, respectively). As a result, $\mathbf{K}$ and $\mathbf{C}$ are nearly identical for both viscosity models. At the largest values of u_0 , recirculation zones form downstream of the pore struts (Fig. 4b), as inertial forces represented by the second term in the FHDD equation (Eq. 5) become more prominent. The larger value of C_{yy} relative to C_{xx} or C_{zz} (see the inset of Fig. 4a) indicates that the transverse anisotropy of the foam arises from inertial forces. Or in other words, the cell struts are less streamlined in the y -direction than in the x - and z -directions. For smaller flow speeds, *i.e.* those less than 0.1 m/s, the non-Newtonian viscous effects could become more prevalent and differences may arise between the geometric approach with the non-Newtonian viscosity model and the continuum approach.

3.2 Mesh Refinement

To demonstrate a mesh-convergent trend, the solutions are repeated on multiple meshes. For the simulated flow through the cube-shaped foam sample (Fig. 2a-b), these meshes are increased in size from 30×10^6 to 300×10^6 cells and are run on 120 to 240 2.8 GHz Intel CPUs. The relative changes in the permeabilities and form factors decrease as the meshes are refined (see the inset of Fig. 4a), demonstrating that the finite-volume solver is converging towards a mesh-independent solution. The terms for $\mathbf{K}$ and $\mathbf{C}$ to be used in the continuum modeling approach are chosen from the results of the Newtonian model simulations corresponding to mesh 1, which places at least ≈ 60 cells around the circumference of each SMP foam strut.

Similarly, we refine the meshes of the pre- and post-treatment aneurysms. For the cases in which the foam-filled aneurysm is modeled using the geometric approach, the spatial resolution is refined to yield meshes of 50×10^6 and 190×10^6 cells, which place at least ≈ 30 and ≈ 60 cells, respectively, around the circumference of each SMP foam strut. The resulting simulations are run on 360 to 480 2.8 GHz Intel CPUs and require approximately 180 to 280 seconds of wall clock time per solution time step, respectively. The pre-treatment cases and those post-treatment cases modeled with the continuum approach lack the finer details of the foam geometry and, consequently, the meshes are refined from only 2×10^6 to 13×10^6 cells. These simulations are run on 60 to 480 2.8 GHz Intel CPUs and require approximately 40 to 130 seconds of wall clock time per solution time step, respectively. A comparison of the instantaneous fluid speed at a given location within the treated aneurysms demonstrates a mesh-convergent trend for both patients (Figs. 5a, 6a). A similar trend is indicated in the transient evolution of the volume-averaged fluid residence time, $\bar{t}_{rV}$, within the aneurysms (Figs. 5b, 6b). Likewise, the planar-averaged shear rate, $\bar{\gamma}_p$, within the post-treatment aneurysms (geometric approach) exhibits minimal sensitivity to mesh refinement.

At systole, the maximum relative differences of $\bar{\gamma}_p$ between the two mesh resolutions are 3.6% and 4.7% for the Newtonian and non-Newtonian viscosity models, respectively (Fig. 7). At diastole, the corresponding maximum relative differences are both 3.7%. Given the results of this mesh refinement exercise, we conclude that the selected mesh resolutions are adequate for the present investigation.

3.3 Pre-Treatment Hemodynamics

Prior to treatment, the parent artery flow for patient 1 enters the aneurysm neck in a manner similar to that of a confined jet. After impinging upon the dome, the flow produced by this jet sweeps back along the aneurysm wall and exits around the periphery of the neck (Fig. 8a). This constant flux of blood within the pre-treatment aneurysms is evident in the virtual angiograms, as less than one cardiac cycle is required for the contrast agent to be washed out (Fig. 9). The volume-averaged fluid residence time, $\bar{t}_{rV}$, within the aneurysms reflects a similar observation. For patients 1 and 2, $\bar{t}_{rV}$ periodically oscillates between 0.20–0.34 s and 0.22–0.43 s, respectively, a significant portion of which arises simply from the time it takes blood to travel from the inlet of the computational domain to the aneurysm neck (Figs. 5b, 6b). The larger values of $\bar{t}_{rV}$ for patient 2 are caused by blood becoming trapped within the predominant swirling flow. Due to the relatively large shear rates, both the Newtonian and non-Newtonian viscosity models yield similar values of $\bar{t}_{rV}$.

3.4 Post-Treatment Hemodynamics

Following the foam deployment, a number of distinct changes arise within the aneurysms. When the parent flow artery flow now impinges upon the foam, it undergoes a rapid deceleration. Both the geometric and continuum modeling approaches capture this effect, though differences are immediately apparent between the two. For the simulations employing the geometric approach, the impinging flow breaks apart into small “finger-lets,” which form as high speed blood preferentially travels through channels produced by interconnected pores (Fig. 8b,e). When the SMP foam is modeled with the continuum approach, these “finger-lets” no longer form (Fig. 8c,f). Depending upon the patient and the specific location within the aneurysm, this lack of fidelity of the continuum approach can lead to discrepancies between the resulting velocity magnitude and that predicted using the geometric approach (Fig. 5a).

3.4.1 Virtual Angiography—By decelerating the parent artery flow, the foam causes blood to stagnate within the aneurysms (Fig. 10). For patient 1, blood tagged with contrast agent impinges upon the foam and slowly spreads throughout the aneurysm over the two

cardiac cycles of contrast agent injection. However, due to the reduced blood speed within the foam, the aneurysm does not entirely fill with contrast agent over this period of injection (Fig. 10a). In fact, the upper 0.9×10^{-3} m of the dome consists of contrast agent-free blood at the end of the second cycle. Once the injection ceases, contrast agent-free blood fills the aneurysm from below, generating an interface that travels towards the dome. Both the continuum and geometric approaches predict a similar sized region ($\approx 2 \times 10^{-3}$ m wide in the y -direction) of contrast agent that remains within the dome at the end of the fifth cardiac cycle (Fig. 10e,j). If a qualitative assessment of the SMP foam treatment is the only information needed, the virtual angiography provided by the continuum approach is more than adequate to represent that derived from the actual foam geometry (Figure 10f-j). Similar trends are evident in the virtual angiograms of patient 2, though the slower moving blood within this treated aneurysm increases the amount of time for the contrast agent to travel throughout the foam. In fact, by the seventh cardiac cycle, a region of contrast agent still remains in the upper-left corner of the dome (Fig. 10o,t).

3.4.2 Fluid Residence Time—This increase in the amount of time required for blood to travel through the foam is quantified by computing the transient behavior of the volume-averaged fluid residence time, $\bar{t}_{rV}$, within the treated aneurysms. Unlike the pre-treatment case of patient 1 in which $\bar{t}_{rV}$ rises very quickly and then oscillates about a final value, the treated aneurysm for this patient requires a greater amount of time for $\bar{t}_{rV}$ to achieve its final oscillatory behavior (Fig. 5b). Depending upon the modeling approach, differences arise between the various trends of $\bar{t}_{rV}$, such that the results of the continuum approach fall between those of the two viscosity models of blood used with the geometric approach. Similar trends are evident for patient 2, though much larger values of $\bar{t}_{rV}$ are achieved following the treatment procedure (Fig. 6b). In addition, the values of $\bar{t}_{rV}$ for the geometric approach with the non-Newtonian viscosity model are distinctly greater than those with the Newtonian viscosity model and those of the continuum approach, both of which have values of $\bar{t}_{rV}$ that are nearly equal to one another.

The spatial variations of the fluid residence time can be readily determined by computing the transient behavior of t_r averaged within planes of constant y (Fig. 11a–b). At the aneurysm neck, the resulting quantity, $\bar{t}_{rP}$, has a magnitude that is comparable to that within the entire volume of the pre-treatment aneurysms (see Figs. 5b, 6b). However, deeper into the foam-filled aneurysms, $\bar{t}_{rP}$ assumes much larger values. Furthermore, at the aneurysm dome where blood is moving the most slowly, $\bar{t}_{rP}$ increases in a near linear fashion. To compute the final residence time would entail running the simulations over hundreds, if not thousands, of cardiac cycles, an effort that would take a considerable amount of time given the large computational requirements for these meshes. Yet, it is possible to estimate the final residence time, $\bar{t}_{rP\infty}$, by fitting $\bar{t}_{rP}(t)$ for each y -plane with the expression,

$$\bar{t}_{rP}(t) = \bar{t}_{rP\infty} (1 - e^{-\alpha t}) \quad (12)$$

where α is the rise-time constant. Doing so demonstrates that Eq. 12 follows the low frequency behavior of $\bar{t}_{rP}$ and predicts final residence times of approximately 600 s at both aneurysm domes (Fig. 11c–d). Near the neck, there is relatively good agreement in the $\bar{t}_{rP\infty}$ values obtained from the two modeling approaches and the two viscosity models. However, farther away from the neck where blood travels more slowly, $\bar{t}_{rP\infty}$ obtained from the non-Newtonian viscosity model is considerably larger than that from the Newtonian viscosity model and that from the continuum approach. For patient 2, this is especially true throughout the upper two-thirds of the treated aneurysm.

3.4.3 Fluid Shear Rate—These differences reveal that the shear rate, $\dot{\gamma}$, locally decreases below the limit ($\approx 100 \text{ s}^{-1}$) for which non-Newtonian effects can be ignored. To quantify the shear rate within the aneurysms, $\dot{\gamma}$ is averaged across planes of constant y . The resulting values of $\bar{\dot{\gamma}}_p$ show substantial variation over the cardiac cycle and along the height of both aneurysms (Fig. 12a–b). While $\bar{\dot{\gamma}}_p$ is quite large at the neck, it decreases in a monotonic fashion towards the dome as the blood flow is decelerated by the foam. For the geometric approach with the non-Newtonian viscosity model, the time-averaged values of $\bar{\dot{\gamma}}_p$ are less than 100 s^{-1} throughout the upper 5.2×10^{-3} and $7.1 \times 10^{-3} \text{ m}$ of the foam for patients 1 and 2, respectively. As might be expected, both of these regions encompass the slow moving blood highlighted by the virtual angiography (see Fig. 10).

For the continuum approach, the shear rate can also be determined from the velocity field, though not by means of Eq. 4 due to the fact that $\Delta \mathbf{u}$ is not directly accessible from the FHDD equation. Rather, it is necessary to compute a volume-averaged shear rate magnitude, $\bar{\dot{\gamma}}_v$, as a function of the local blood speed using an analytical or empirical expression. In the case of the former, a first-order approximation to the shear rate can be derived from a hydraulic radius model, which, for example, in the principal x -direction gives $u_0 K_{xx}^{-1/2}$ [50]. However, this approximation only accounts for viscous forces and neglects the inertial forces that clearly make an important contribution to the flow within the foam (see Fig. 4a). It is therefore necessary to employ an empirical expression, which can be determined by computing $\bar{\dot{\gamma}}_v$ within the cube-shaped sample as a function of the flow speed (see Fig. 2a–b). Performing these calculations for Newtonian flow in all three coordinate directions yields the data shown in Fig. 12c. These data are then fit with an empirical expression, which in the x -direction is

$$\bar{\dot{\gamma}}_{v,x} = c_0 u_0 + c_1 u_0^2 \quad (13)$$

Similar expressions are used in the y - and z -directions. It is clearly evident that Eq. 13 follows the general behavior of the volume-averaged shear rate much better than the hydraulic radius model. The coefficient, c_1 , in the y -direction is an order of magnitude larger than those in the other two directions, an effect due to the transverse anisotropy of the foam. With the dependence of the volume-averaged shear rate now known as a function of the local Newtonian flow speed, we apply Eq. 13 to the intra-aneurysmal velocity field computed using the continuum approach and estimate the shear rate magnitude from

$$\sqrt{\bar{\dot{\gamma}}_{v,x}^2 + \bar{\dot{\gamma}}_{v,y}^2 + \bar{\dot{\gamma}}_{v,z}^2} \quad (14)$$

which is then averaged across each y -plane. Doing so indicates that the shear rate from the continuum approach qualitatively follows the trends from the geometric approach, though at a much smaller magnitude (Fig. 12a–b). This difference is not too surprising given the fact that the continuum approach does not resolve the higher speed and, therefore, higher shear flow through the interconnected pores (see Fig. 8b,e).

3.4.4 Initial Thrombus Formation within the SMP Foam—Having calculated the planar-averaged fluid residence time, $\bar{t}_{r,p\infty}$, and shear rate, $\bar{\dot{\gamma}}_p$ (time-averaged over the cardiac cycle), we estimate the y -locations of where thrombus may initially form within the foam-filled aneurysms. These locations for thrombus formation are denoted as “initial”

because the flow field within the treated aneurysms is computed only for thrombus-free SMP foam. Hence, any subsequent thrombus growth and its impact upon the flow field cannot be evaluated. To do so would require the inclusion of the thrombus volume into the simulations, a step that is not taken in the present study.

Recall from Sections 1 and 2.3 that thrombus is estimated to occur when the fluid residence time is greater than 240 s and when the average shear rate is less than 49 or 54 s^{-1} for the Newtonian or non-Newtonian viscosity models, respectively. For patients 1 and 2 simulated with the geometric approach and the non-Newtonian viscosity model, these conditions are simultaneously met within the upper 240×10^{-6} and 280×10^{-6} m of the aneurysms, respectively (Figs. 11c–d, 12a–b). Likewise, for the geometric approach with the Newtonian viscosity model, the respective values are 70×10^{-6} and 20×10^{-6} m and, for the continuum approach, 80×10^{-6} and 20×10^{-6} m. Interestingly, it is the fluid residence time, not the shear rate, which is the limiting factor for thrombus formation within the SMP foam.

4 Discussion

The porous media properties of a treated aneurysm are critically important for subsequent healing. When filled with endovascular coils, an aneurysm must receive a dense packing that will result in a decreased permeability capable of promoting the conditions necessary for thrombus formation. Since a single coil cannot produce this level of packing, multiple coils must be delivered. The permeability of the resulting coiled mass can be compared to that of SMP foam by considering the work of Kakalis *et al.* [33]. In that particular study, the permeability of an anterior communicating artery aneurysm (volume of $4.22 \times 10^{-7} \text{ m}^3$) is computed as it is gradually filled with 15 coils that each have a diameter of 2.54×10^{-4} m. Interestingly, the permeability of the coil-filled aneurysm does not decrease to a value comparable to that of the SMP foam until the ninth coil (22% packing, permeability of $2.47 \times 10^{-8} \text{ m}^2$) is delivered to the aneurysm.

In addition to decreasing the aneurysm permeability, the SMP foam geometry also eliminates the high frequency velocity fluctuations that characterize the flow of blood within the pre-treatment aneurysms (Figs 5a, 6a), an effect which would likely be produced by coils, as well. Before the treatment procedure, a shear layer instability develops around the circumference of the flow entering the aneurysm, producing a train of periodic vortices (see Fig. 5a of Ortega *et al.* [46]) that yields a significant level of unsteadiness and smaller scale flow structures. This type of unsteadiness has also been observed in a number of previous studies [19, 45, 54]. In the presence of such an unsteady flow field, the aneurysm and artery walls can vibrate or resonate, which may lead to increased wall stresses, subsequent bleeding, or even rupture [28, 51]. However once the aneurysm is filled with SMP foam, the shear layer instability and the accompanying flow unsteadiness is mitigated since any vortex formed at the parent artery bifurcation is prevented from advecting upwards into the aneurysm (see Figs. 4–5 of [46]). The slower moving blood within the foam-filled aneurysms now assumes a transient behavior that closely resembles that of the inlet velocity waveform (see Fig. 3c).

Given the extent of this slower moving blood (see Fig. 10), we might expect the conditions for thrombus formation to locally exist even when they are not met on average across the aneurysm. To determine if this is the case, we repeat the calculations for the final fluid residence time and the shear rate time-averaged over the cardiac cycle for points located throughout the entire post-treatment aneurysms. Doing so indicates that thrombus is likely to arise over a much larger portion of the treated aneurysms (Fig. 13a–b). For the geometric approach with the non-Newtonian viscosity model, complete occlusion of the aneurysm cross-section is estimated to occur within the upper 130×10^{-6} and 140×10^{-6} m of the

aneurysms for patients 1 and 2, respectively. Away from the dome, the thrombus is distributed along one side of the aneurysm in a layer that is at most $\approx 600 \times 10^{-6}$ m thick (Fig. 13c,e). The fraction of the aneurysm cross-section that is occupied by thrombus diminishes towards the neck as the fluid residence time decreases and the shear rate increases. The volumetric fraction of thrombus is approximately 1.2% and 5.1% for patients 1 and 2, respectively.

While these results provide additional insight into the post-treatment hemodynamics, there are several limitations in the computational approach that are clearly evident. Namely, the continuum approach assumes a constant viscosity, which is invalid across the upper portion of the foam-filled aneurysms. Since blood is a shear thinning fluid, the viscosity is underestimated within this low shear region and, therefore, so is the pressure gradient (see Eq. 5). This, in turn, has a significant effect upon the results of the thrombus formation model, which predicts substantially less thrombus than that of the geometric approach with the non-Newtonian viscosity model (see Fig. 13a–b). As a result, neither the continuum approach nor, for that matter, the geometric approach with the Newtonian viscosity model yields a complete occlusion at the aneurysm dome. And furthermore, even the non-Newtonian viscosity model (Eq. 1) suffers from a deficiency in that it does not reproduce the yield stress of blood, an effect which could be important in the regions of lowest shear [17].

The most critical limitation of the low fidelity thrombus formation model is that it does not include the foreign body response to the SMP foam or any long-term healing processes. Immediately following the treatment procedure, numerous biochemical reactions, which can be influenced by medication or other factors, are initiated within the aneurysm that lead to the adhesion of fibrinogen, proteins, and red blood cells to the surface of the foam [2, 39, 61]. Additionally, an assortment of cell types are produced as the body transitions through various healing stages, which can have time scales ranging from minutes to months. The end result is a very complex, heterogeneous, anisotropic distribution of healing byproducts throughout the foam-filled aneurysm [52]. The present thrombus formation model neglects these stages of healing and, therefore, significant modeling improvements must be made in order to accurately predict this post-treatment behavior. Yet, as these improvements are made and accompanied by increases in computer speed, it may become possible to model the evolution of a foam-filled aneurysm over the course of weeks to months and possibly even years.

5 Conclusions

We have investigated the hemodynamics of two patient-specific basilar aneurysms that are virtually treated with SMP foam, which is modeled using both geometric and continuum approaches. Immediately following the procedure, a number of changes occur to the intra-aneurysmal flow fields. An unstable confined jet, which drives the velocity field within both pre-treatment aneurysms, is rapidly decelerated when the parent artery flow impinges upon the SMP foam. Consequently, the foam acts as a low-pass filter that damps out the high-frequency velocity fluctuations within the aneurysm. Away from the aneurysm neck, the slower moving blood within the treated aneurysms has residence times that are several orders of magnitude greater than that prior to treatment. This increase in residence time, coupled with a decrease in the fluid shear rate, establish conditions conducive to thrombus formation by means of a low fidelity model. For both patients, a layer of thrombus is likely to occur within the aneurysm dome and along one side of the aneurysm wall within minutes following the treatment procedure.

Keeping in mind that the low fidelity thrombus formation model does not include any physiological healing processes or transient thrombus growth, the initial layer of thrombus

comprises a relatively small fraction of the aneurysm volume. This indicates that further modifications can be made to the SMP foam in order to enhance its occlusion effectiveness. Presently, the limiting factor for thrombus formation is the residence time. An increase in this quantity can be achieved by decreasing the permeability of the foam while still maintaining enough open pores to promote blood penetration and thrombus development throughout the entire treated aneurysm. For future studies, we can determine the impact that the permeability has upon the hemodynamic flow within the SMP foam. In the meantime, we rely upon other techniques to assess the outcome of the treated aneurysms. An example of this is a recent animal model investigation of aneurysms filled with SMP foam [52], which has a smaller permeability than the sample considered herein. The results of this investigation demonstrate that by 90 days after the treatment procedure, a complete re-endothelialization occurs across the neck of the foam-filled aneurysms. Positive histologic findings such as these suggest that the SMP foam may offer a viable option for the endovascular treatment of aneurysms.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The authors thank R. Cook, W. Small, and T. Wilson of Lawrence Livermore National Laboratory for their assistance in this study. This work was supported by the National Institutes of Health/National Institute of Biomedical Imaging and Bioengineering Grant R01EB000462 and partially performed under the auspices of the U.S. Department of Energy by Lawrence Livermore National Laboratory under Contract DE-AC52-07NA27344. LLNL-JRNL-564718.

Abbreviations

BA	basilar artery
CFD	computational fluid dynamics
FHDD	Forchheimer-Hazen-Dupuit-Darcy
GDC	Guglielmi detachable coil
PCA	posterior cerebral artery
SCA	superior cerebellar artery
SMP	shape memory polymer

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Figure 1.

(a) A sample of SMP foam in a primary, expanded shape. (b) Catheter delivery of a compressed piece of SMP foam to the aneurysm. (c) Fully expanded SMP foam within the post-treatment aneurysm.

Figure 2.

(a) Side and (b) bottom views of the SMP foam geometry obtained from a micro-CT scan. Geometries of basilar aneurysms from patients (c) 1 and (d) 2 that have been filled with SMP foam.

Figure 3. Computational domains for patients (a) 1 and (b) 2. (c) Mean inlet velocity boundary condition through a basilar artery as measured *in vivo* by Kato *et al.* [34].

Figure 4.

(a) Pressure gradient through the SMP foam geometry as a function of the blood flow speed in all three coordinate directions for the non-Newtonian (NN, Eq. 1) viscosity model of blood. The curves denote least-square fits of the FHDD equation (Eq. 5) to the computational data. The results for $\mathbf{K}$ and $\mathbf{C}$ (inset) are shown for multiple mesh resolutions: mesh 0 (30×10^6 cells), mesh 1 (100×10^6 cells), and mesh 2 (300×10^6 cells). (b) Iso-surfaces of reversed flow ($v = -1 \times 10^{-6}$ m/s) downstream of the SMP foam struts for $u_0 = 1.2$ m/s.

Figure 5.

(a) Velocity magnitude at a point within the pre- (inset) and post-treatment aneurysm for patient 1. The results for the non-Newtonian viscosity model (Eq. 1) are denoted by NN. The only simulation condition with a significant difference in the velocity magnitude is that of mesh 0_{post} FHDD. (b) Volume-averaged fluid residence time over the entire aneurysm volume for the pre- and post-treatment cases of patient 1. The gray line shows the nominal amount of time required for blood to travel from the inlet of the basilar artery to the aneurysm neck.

Figure 6.

(a) Velocity magnitude at a point within the pre- (inset) and post-treatment aneurysm for patient 2. The results for the non-Newtonian viscosity model (Eq. 1) are denoted by NN. The only simulation condition with a significant difference in the velocity magnitude is that of mesh 0_{post} NN. Note that the legend for (a) is the same as that for (b). (b) Volume-averaged fluid residence time over the entire aneurysm volume for the pre- and post-treatment cases of patient 2. The gray line shows the nominal amount of time required for blood to travel from the inlet of the basilar artery to the aneurysm neck.

Figure 7.

Planar-averaged shear rate across the height of the post-treatment aneurysm (patient 1) for two different mesh resolutions using the geometric approach with the non-Newtonian (Eq. 1) and Newtonian (inset) viscosity models of blood.

Figure 8.

Iso-surfaces of velocity magnitude ($|\mathbf{u}| = 0.289$ m/s, which is the time-averaged mean inlet velocity over the cardiac cycle; see Fig. 3c) for patients (a–c) 1 and (d–f) 2 at systole. (a,d) Pre-treatment, (b,e) post-treatment for the geometric approach with the non-Newtonian viscosity model for blood (Eq. 1), and (c,f) post-treatment with the FHDD continuum approach (Eq. 5). The $\rightarrow$'s in (b,e) denote the "finger-lets" of flow that form along interconnected pores.

Figure 9.

Virtual angiography of the pre-treatment aneurysms for patients (a–d) 1 and (e–h) 2. $C_{a0}(t)$ is the concentration of the virtual contrast agent at the inlet to the basilar artery. The $\rightarrow$'s highlight the confined jet entering the aneurysms.

Figure 10.

Virtual angiography of the post-treatment aneurysms for patients (a–j) 1 and (k–t) 2. (a–e, k–o) Geometric approach with the non-Newtonian viscosity model for blood (Eq. 1), (f–j, p–t) FHDD continuum approach (Eq. 5). $C_{a0}(t)$ is the concentration of the virtual contrast agent at the inlet to the basilar artery. The $\rightarrow$'s highlight the residual contrast agent within the foam at the end of the fifth and seventh cardiac cycles for patients 1 and 2, respectively.

Figure 11.

Planar-averaged fluid residence time at various y -values within the post-treatment aneurysms of patients (a) 1 and (b) 2 for the geometric approach with the non-Newtonian viscosity model of blood (Eq. 1). Final fluid residence time across the height of the post-treatment aneurysms for patients (c) 1 and (d) 2. The vertical dashed lines indicate the values of y for which $\bar{t}_{r_{P\infty}} = 240$ s.

Figure 12.

Planar-averaged shear rate across the height of the post-treatment aneurysms for patients (a) 1 and (b) 2. The gray lines denote the shear rate magnitude estimated from the FHDD continuum approach (Eq. 5) using Eqs. 13–14. The ● and ○ indicate the y -values at which $\bar{\gamma}_p = 54$ and 100 s^{-1} , respectively, for the geometric approach with the non-Newtonian viscosity model of blood (Eq. 1). (c) Volume-averaged shear rate magnitude for Newtonian blood flow through the cube-shaped SMP foam sample (see Fig. 2a–b). The gray lines indicate the shear rate predicted by the hydraulic radius model.

Figure 13.

Predicted initial locations for thrombus formation within the foam-filled aneurysms. Cross-sectional area of thrombus, A_{throm} , normalized by the aneurysm cross-sectional area, A_P , as a function of the aneurysm height, y , for patients (a) 1 and (b) 2. The Δy value is the distance for which thrombus completely fills the aneurysm cross-sectional area ($A_{throm}/A_P = 1$). The % values indicate the fractional volume of the aneurysm filled with thrombus. Virtual histology images for patients (c-d) 1 and (e-f) 2 for the (c,e) geometric approach with the non-Newtonian viscosity model (Eq. 1) and the (d,f) FHDD continuum approach (Eq. 5). The four y -slices in (c-f) correspond to the white lines across the aneurysms in (a-b, inset). Black—thrombus, white—cross-sections of the SMP foam struts, gray—unclothed blood.