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Computational study of the fluid-dynamics in carotids before and after endarterectomy

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ABSTRACT

In this work, we provide a computational study of the effects of carotid endarterectomy (CEA) on the fluid-dynamics at internal carotid bifurcations. We perform numerical simulations in real geometries of the same patients before and after CEA, using patient-specific boundary data obtained by Echo-Color Doppler measurements. We analyze four patients with a primary closure and other four where a patch was used to close arteriotomies. The results show that (i) CEA is able to restore physiological fluid-dynamic conditions; (ii) among the post-operative cases, the presence of patch leads to local hemodynamic conditions which might imply a higher risk of restenosis in comparison with the cases without patch.

1. Introduction

Carotids are a preferential site of development of atherosclerotic plaques, which leads to vessel stenosis and, possibly, to the formation of blood clots and breakage (Falk, 2006; Rauch et al., 2001; Timsit et al., 1992; Wootton and Ku, 1999). For these reasons, carotid endarterectomy (CEA), consisting in the surgical removal of the plaque, is routinely performed in the clinical practice. Several studies have highlighted a significant reduction of the stroke risk in patients with severe carotid stenosis who underwent CEA (Barnett et al., 1998; European Carotid Surgery Trial, 1998; Executive Committee for the Asymptomatic Carotid Atherosclerosis Study, 1995). In particular, Doppler measurements and radiological evidences showed that CEA induces a reduction of the peak blood velocity and pressure gradients across the plaque (Harloff et al., 2013a; Steinke et al., 1991), restoring the physiological hemodynamic conditions.

After the complete removal of the atheromatous plaque, the arteriotomy can be closed through a *direct suture* or with the interposition of a synthetic or vein *patch*. Although patch angioplasty is recommended by randomized prospective trials (Bond et al., 2004), the updated meta-analysis showed only a weak benefit for patching in terms of procedural risk of stroke and death (Rerkasem and Rothwell, 2009).

Some authors consider that patch closure benefits could be more apparent in selected case: distal extension of the plaque in the internal carotid artery (ICA), small calibre vessel, in women, and in patients who have previously undergone ipsilateral carotid surgery (Archie, 2000). In any case, no definitive evidences are nowadays available to support the clinical decision. For this reason, a comparison of the fluid-dynamics after CEA between these two scenarios (direct suture vs patch) could provide useful information to drive the clinical decisions.

Computational methods with patient-specific data provide an effective tool to investigate quantitatively and non-invasively the fluid-dynamics in carotid arteries since at most two decades. We mention, among the others, (Gao et al., 2009; Groen et al., 2010; Milner et al., 1998; Perktold and Resch, 1990; Tang et al., 2004; Younis et al., 2004).

In this work, we provided a computational study of the fluid-dynamics in carotids before and after CEA for eight patients. For four of them, CEA was characterized by a primary closure, whereas for the other four a patch was inserted. The aim of the present study was twofold. First, we assessed the restoration of physiological conditions in terms of systolic velocity, wall shear stresses, and vorticity produced by the plaque removal, comparing the

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results obtained before and after CEA. Second, we provided a comparison of the fluid-dynamics after CEA between patients with a primary closure and with a patch insertion, to highlight the different responses in terms of hemodynamic conditions which could promote the risk of restenosis.

To obtain these goals, we performed numerical simulations based on the Finite Element Method with patient-specific geometries and boundary conditions. This was possible thanks to the acquisition of MRI images and Echo-Color Doppler signals for the eight patients before and after CEA.

These comparisons allowed us (i) to quantify the reduction of peak velocity, viscous forces, and vorticity induced by CEA, and (ii) to highlight the difference behavior in terms of well-known hemodynamic factors potentially involved in restenosis development, between the cases with and without the patch insertion.

2. Methods

2.1. Patients recruitment and carotid endarterectomy

In this study, we considered eight carotids with a degree of stenosis greater than 70% who underwent elective CEA. For four of them (N1,N2,N3,N4) a primary closure was performed, whereas for the remaining four (P1,P2,P3,P4) the arteriotomies were closed with a Dacron patch.

All the patients were followed at the Vascular Surgery Operative Unit of Fondazione IRCSS Ca' Granda, Ospedale Maggiore Policlinico, Milan, for the Echo-Color Doppler (ECD) acquisitions and for the surgical treatment, and at the Operative Unit of Radiology for the MRI acquisitions. The surgeons followed the recommendations of the Carotid Artery Stenosis Consensus conference (Grant et al., 2003) for grading carotid stenosis.

2.2. Acquisition of Echo-Color Doppler signals and boundary conditions

For each patient we have at disposal velocity signals from ECD at the CCA and ICA both before and after CEA, see the Appendix. The ECD signal represents the histograms of the velocities measured at the center of a given section, where the gray-scaled intensity of pixels is proportional to the number of blood-cells moving at a certain velocity. We first estimated for each time instants the maximum velocity using the 95-th quantile of the velocity histogram as a robust estimator. Then, we used a Fourier smoothing of the time signal along three heartbeats (Azzimonti, 2013; Buratti, 2011). Once we had at disposal the velocity at the center of the cross section, we assumed that it corresponds to the (spatial) maximum velocity over the section. Then, owing to the formula proposed in Ponzini et al. (2006) and validated in Ponzini et al. (2010) and Vergara et al. (2010) (see the Appendix) we estimated the flow rates $Q_{CCA}(t)$ and $Q_{ICA}(t)$ to be used as boundary conditions in the numerical simulations.

2.3. Acquisition of radiological images and mesh generation

A surface model of the interface between the blood and the arterial wall for each of the 16 carotids (eight before and eight after CEA) was obtained from MRI images (see the Appendix), using a level-set segmentation technique (VMTK, http://www.vmtk.org). The surface models were successively turned into volumetric meshes of tetrahedra in view of computational fluid-dynamics simulations.

2.4. Numerical simulations

Unsteady numerical simulations have been performed by using the Finite Element library LifeV developed at Politecnico di Milano, EPF de Lausanne, INRIA Paris, Emory University (http://www.lifev.org). We considered the blood as Newtonian, homogeneous, and incompressible, and, accordingly, we used the Navier–Stokes equations (Formaggia et al., 2009). For the space discretization, we used P1bubble finite elements for the velocity (where "bubble" basis functions are added to the classical linear ones) and linear finite elements for the pressure. This choice avoids the generation of spurious pressure modes (Formaggia et al., 2009). As for the time discretization, we used the backward Euler method with a semi-implicit treatment of the convective term. The time step was set equal to 0.01 s (see the Appendix). The vessel wall was considered rigid and we did not use any turbulence model (see Section 4).

For the prescription of the flow rates Q_{CCA} and Q_{ICA}, in absence of any radiological information about the spatial velocity distribution, we used the *Augmented Formulation*, based on the introduction of Lagrange multipliers, proposed in Formaggia et al. (2002) and in particular the algorithm introduced in Veneziani and Vergara (2005). This formulation is based on adding to the Navier–Stokes system two more scalar unknowns (the Lagrange multipliers, one for each flow rate condition) and two more scalar equations (the flow rate conditions). This allows to prescribe the flow rates implicitly, without any assumption on the velocity profile. As for the boundary condition at ECA we prescribed zero traction.

2.5. Comparison with available measurements

To assess the accuracy of the results reported in this work, we compared the systolic velocity field computed by our simulations with available internal ECD measures not used in the numerical experiments. In view of the comparison, we selected in the computational domains a cylinder of 2 mm of height with center located in the measurement point, and we took the maximum computed velocity within this cylinder.

3. Results

The computational meshes were obtained after a refinement study, see the Appendix. A local refinement was performed at the stenosis level in the cases before CEA, see Fig. 1.

In the first set of results, we compared the fluid-dynamics in the eight cases before and after CEA. In Fig. 2 we plotted the flow rates $Q_{CCA}(t)$ and $Q_{ICA}(t)$ both before and after CEA, generated from the ECD signals and prescribed as boundary conditions in our numerical simulations, whereas in Table 1 we reported the systolic Reynolds numbers for each cases.

In Fig. 3 we depicted the streamlines of the computed velocity field at systole for all the eight cases, both before and after CEA. We observe that the velocity obtained before CEA is for all the cases higher than the one obtained after CEA, due to the presence of the stenosis. This is also confirmed by the values of the peak systolic velocity (PSV), i.e. the maximum value reached inside the domain at systole, reported in Table 1. From Fig. 3 we also observe that the non-physiological morphology of the pre-CEA carotids caused the development of systolic disturbed/swirling patterns, particularly visible at the ICA, which are completely absent in the post-CEA ICAs. To better investigate this point, in Fig. 4 we reported the systolic vorticity at the bifurcation and at the ICA and in Table 1 the maximum-in-space systolic vorticity. These results highlight a big reduction of the vorticity after CEA in all the cases.

In Fig. 5 we showed the WSS at systole at the bifurcation and at the first tract of the ICA, whereas in Table 1 we reported the maximum systolic WSS in these regions. Again, we observe significantly higher values featured by the configurations before CEA with respect to the ones after CEA, although for case N3 the value of the maximum WSS after CEA is quite great.

To assess the reduction of PSV, vorticity, and WSS after CEA, for each of these quantities we performed a one-sided statistical non-parametric test for coupled data (coupled one-sided Wilcoxon test) with a 5% significant level. The results of these tests showed a significant reduction of all the quantities after CEA since all the p-values were lower than 1%.

Looking at Fig. 2, we observe that for cases P1, P2, and N1 the flow rates obtained by ECD measures after CEA are lower than the ones measured before CEA. In order to evaluate the contributions of the geometry change and of the flow reduction on the WSS and vorticity reduction, we ran for these three cases additional numerical simulations where the pre-CEA flow rates were used as boundary conditions for the post-CEA geometries. From Figs. 4 and 5 we observe that for P1 and N1 the reduction of these quantities is still notable. This is confirmed also by the results reported in Table 1. For case P2 the decrease of WSS and vorticity is less remarkable, but still present.

In the second set of results, we compared the fluid-dynamics after the plaque removal in the cases with and without patch insertion.