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Helical flow as fluid dynamic signature for atherogenesis risk in aortocoronary bypass. A numeric study

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Abstract

The main purpose of the study was to verify if helical flow, widely observed in several vessels, might be a signature of the blood dynamics of vein graft anastomosis. We investigated the existence of a relationship between helical flow structures and vascular wall indexes of atherogenesis in aortocoronary bypass models with different geometric features. In particular, we checked for the existence of a relationship between the degree of helical motion and the magnitude of oscillating shear stress in conventional hand-sewn proximal anastomosis. The study is based on the numerical evaluation of four bypass geometries that are attached to a simplified computer representation of the ascending aorta with different angulations relative to aortic outflow. The finite volume technique was used to simulate realistic graft fluid dynamics, including aortic compliance and proper aortic and graft flow rates. A quantitative method was applied to evaluate the level of helicity in the flow field associated with the four bypass models under investigation.

A linear inverse relationship ($R = -0.97$) was found between the oscillating shear index and the helical flow index for the models under investigation. The results obtained support the hypothesis that an arrangement of the flow field in helical patterns may elicit damping in wall shear stress temporal gradients at the proximal graft. Accordingly, helical flow might play a significant role in preventing plaque deposition or in tuning the mechanotransduction pathways of cells.

Therefore, results confirm that helical flow constitutes an important flow signature in vessels, and its strength as a fluid dynamic index (for instance in combination with magnetic resonance imaging flow visualization techniques) for risk stratification, in the activation of both mechanical and biological pathways leading to fibrointimal hyperplasia.

Keywords: Computational fluid dynamics; Helicity; Bypass graft; Atherogenesis; Intimal thickening; Vascular hemodynamics

1. Introduction

The role of fluid mechanical factors in the genesis and development of atherosclerosis in human arteries has been recognized for some time (Karino, 1986; Glagov et al., 1988). A thorough understanding of blood dynamics in human vessels is of great interest, since the local flow behavior of blood is certainly implicated in the formation of atherosclerotic plaques and of phenomena such as thrombogenesis, atherogenesis, endothelial damage, intimal thickening and hyperplasia (Giddens et al., 1993; Moore et al., 1994; Karino and Goldsmith, 1984; Ku et al., 1985). Results obtained by Karino (1986), for example, clearly emphasize the strong correlation between the sites of flow disturbance and the preferred sites for the genesis and development of vascular diseases clinically found in man (Caro et al., 1971).

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The propensity for plaque formation at sites with such characteristics as curvatures, bifurcations, tortuosity and branching (in general where flow velocity and shear stress are reduced and flow departs from unidirectional patterns) has led to conjectures that local wall shear stress (WSS) potentiates atherogenesis by inducing the vascular response of the endothelium (normal or abnormal): the fluidic forces acting on the vessels' walls are thought to be capable of stimulating the endothelium to produce several cellular factors that can inhibit or promote inflammatory events (Yoshizumi et al., 2003). In particular, the local hemodynamic factors involved in atherosclerotic plaque formation and development can be classified as follows: "disturbed flow" may induce initial endothelial lesions (in case of too high shear stress in association with excessive arterial wall strain) and inflammatory activation with possible platelet aggregation and acute thrombosis (Altman, 2003). Low and oscillating wall stresses contribute to atherosclerotic plaque growth by activating both mechanical and biological pathways (Buchanan et al., 2003). Indeed, the importance of shear stress oscillation at the wall for any change in physiological mechanical stimuli to the mechano-sensor endothelial cells has been pointed out by several authors, some of whom have provided indices to quantify these occurrences (Ku et al., 1985; Friedman et al., 1981). Specifically, atherogenesis appears to be affected more by the presence of low velocity and fluctuating shear stress rather than by the amplitude of the shear stress. In other words, unsteady blood flow characteristics, rather than the magnitude of WSS per se, may be the major determinant of hemodynamically induced endothelial cell turnover, a precursor to plaque formation (Glagov et al., 1988). Such flow characteristics presumably allow prolonged contact of the vessel wall with platelets, granulocytes, and metabolites that influence atherogenesis.

To identify the relationships between particular blood flow patterns and physiopathological events, knowledge of detailed hemodynamics data in realistic vessel geometries becomes relevant.

In recent years, there has been a lively interest in synthetic analytical methods for the quantitative assessment of the role played by the fluid dynamics in the activation of mechanotransduction pathways at the vessel wall, e.g., time-averaged WSS, oscillating shear index, WSS gradients and WSS angle deviation (see Glor et al., 2003 for a summary).

From this viewpoint, the much debated role played by blood flow helicity in vascular hemodynamics is still unclear (see Grigioni et al., 2005 for an exhaustive discussion on this argument). Helicity is the property of a moving fluid which represents the potential for helical flow, i.e. flow with a corkscrew-like pattern, to evolve. Helicity is proportional to the strength and the amount of turning in the flow (i.e. vorticity). Hypotheses on the detrimental clinical relevance of the presence of helical flow in the umbilical portion of the portal vein have been put forward by Sugimoto and colleagues (2002), who suggest that the simultaneous onset of dilation in the vessel and of helical flow are characteristic signs indicating the proximal stenosis of the portal vein. In stenosed coronary arteries, helical patterns have also been observed at sites with increased vessel wall thickness (Van Langenhove et al., 2000).

On the other hand, the beneficial role played by helical flow was assessed in studies on fluid dynamics in the aortic arch: Bogren and Buonocore (1999) observed the presence of helical flow in normal subjects, while Houston et al. (2003) found that carotid atheromatous disease is associated with a reduction in the prevalence of a systolic helical flow pattern in the aortic arch. Moreover, an absence of aortic helical flow might be a predictor of renal impairment deterioration in patients with renal artery stenosis (Houston et al., 2004).

Less recently, in vitro studies (Giddens et al., 1993; Zarins et al., 1983; Caro et al., 1971) have shown the clear helical patterns drawn by particles within arterial flow, describing the possible relationship between local fluid dynamics and known arterial disease. In the 1990s, researchers put forward hypotheses on the theoretical advantages derived from helical blood flow (Stonebridge et al., 1996), by virtue of the beneficial effect it could exert on the mechanisms of endothelial damage repair. Helical flow may also account for a significant amount of normal organ perfusion (Frazin et al., 1996) from branch vessels due to the centripetal spin induced in blood.

Our investigation attempts to determine if the helical patterns observed in both in vitro and in silico studies on bypass grafts fluid dynamics (Lei et al., 2001) play a significant role in preventing intimal thickening, and if helical flow is a signature of the blood dynamics of vein graft anastomosis. We went into great detail on the existence of a relationship between helical flow structures and oscillating shear stress, an index of atherogenesis in four conventional hand-sewn proximal anastomosis geometries (Redaelli et al., 2004). In order to accomplish this, we designed a computational approach with a finite volume technique used to simulate realistic graft fluid dynamics, including aortic compliance and proper aortic and graft flow rates. A quantitative method based on a mathematical description of the helical motion along particle traces (Grigioni et al., 2005) was applied to evaluate the level of helicity in the flow field associated with the four bypass models under investigation. This quantitative, Lagrangian analysis may be a powerful instrument to relate qualitative, in vivo observations of helical flow in vessels with the onset/presence of physiopathological events, in particular with reference to MRI flow tracking methods.