THE ROLE OF HEMODYNAMICS IN ARTERIOVENOUS FISTULA REMODELLING AND FAILURE

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Introduction

Despite its clinical relevance, the mechanism responsible for stenosis development in arteriovenous fistula (AVF) for haemodialysis (HD) remains still largely unknown [1]. This study aims at investigating the relationship between altered hemodynamics and vessel remodelling in a native AVF that failed due to stenosis formation 1.5 years after surgery.

Methods

Non contrast-enhanced MRI [2] and Doppler Ultrasound (US) examinations were acquired in a 72-year male at 3 days, 40 days, 6 months, 1 year and 1.5 years after surgery at the Unit of Nephrology and Dialysis of Bergamo Hospital. Three-dimensional AVF surface models were reconstructed using the Vascular Modeling Toolkit (VMTK). High fidelity computational fluid dynamic simulations were performed using pimpleFoam (OpenFOAM suit), with a mean timestep of 0.1 ms, setting patient-specific boundary conditions derived from US at the proximal and distal artery. Blood was modelled as Bird-Carreau non-Newtonian fluid and walls were assumed to be rigid.

Vascular lumen cross-sectional areas (CSAs) along the models' centreline were obtained at 0.1 mm intervals in each model to characterize vessel remodelling. Turbulent kinetic energy (TKE) was calculated as

$$TKE = \frac{1}{2}\rho\,(\underline{u'^2} + \underline{v'^2} + \underline{w'^2}) \qquad (1)$$

where ρ is the blood density and u', v', w' are the fluctuating components of the velocity field [3].

Results

The AVF was characterized by a successful maturation, with a massive arterial and venous dilatation within the 6 months after surgery and a corresponding increase in blood flow volume.

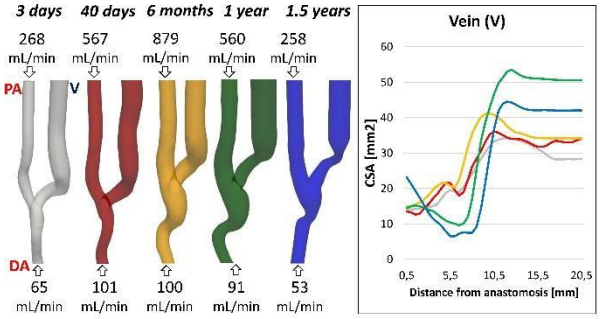

Figure 1: 3D geometrical AVF models, pertinent blood flow rates and vein's CSAs evolution.

Between 6 months and 1 year, a stenosis of the juxta-anastomotic vein caused a CSA reduction of 62.5%, becoming very significant at 1.5 years (-78.8% in CSA). Vessel lumen changes were accompanied by a decrease of the blood flow rate, that did not allow HD.

The development of the stenosis in the venous segment was paralleled by the regularization of blood flow and consequent decrease in the disturbed flow metrics (see **Figure 2**).

Figure 2: Volume rendering of the mean TKE.

Discussion

In this longitudinal study, performed on a AVF that developed stenosis after 1.5 year from surgery, we showed the presence of a focal remodelling process in the vein that was paralleled by progressive disturbed flow regularization. Our results, albeit on a single patient, show that local flow instabilities may have induced progressive intimal hyperplasia during time and suggest that inward remodelling could act as a protective mechanism to induce blood flow stabilization.

Future longitudinal studies with an increased number of patients will be required to elucidate the role of disturbed flow in stenosis development, with the final aim of improving AVF clinical outcomes.

References

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