FLUID DYNAMICS OF PATENT FORAMEN OVALE: INSIGHTS INTO SHEAR-MEDIATED RED BLOOD CELL DYSFUNCTION

Fabio Morelli (1), Valeria Guadagno (1), Tatiana Mencarini (1), Silvia Bozzi (1), Marta Brambilla (2), Laura Fusini (2), Daniela Trabattoni (2), Marina Camera (2,3), Alberto Redaelli (2)

1. Politecnico di Milano, Italy; 2. IRCCS Centro Cardiologico Monzino, Italy; 3. Università degli Studi di Milano, Italy

Introduction

Patent Foramen Ovale (PFO) is a congenital cardiac anomaly involving incomplete closure of the opening between the right and left atria after birth. Patients with PFO are more likely to experience migraine with aura (MHA) than the general population, suggesting a potential link between the two conditions [1]. However, the mechanisms remain unclear.

During right-to-left shunting through a PFO, blood experiences high shear stress, potentially damaging red blood cell (RBC) membranes. This mechanical stress may cause hemolysis or structural changes in RBCs, triggering systemic inflammation and oxidative stress [2]. These processes might contribute to vascular dysfunction linked to MHA.

This study investigates PFO fluid dynamics and quantifies mechanical stress on RBCs across different PFO morphologies to clarify how PFO hemodynamics affect RBC dysfunction and MHA.

Methods

A cardiac model developed by collaborating academic institutions [3] was used to construct atrial geometries and a simplified conduit connecting the atria was created to represent the PFO channel. Eight patient-specific PFO geometries were generated, each characterized by different PFO lengths, inlet diameters, and outlet diameters.

Transient computational fluid dynamics simulations were performed on all geometries to simulate a shunting event with the PFO open. Steady-state particle tracking simulations were then carried out to quantify the mechanical stresses on RBCs passing through the PFO. The scalar stress tensor at each particle position was calculated to determine stress accumulation (the integral of stress along the RBC trajectory) and stress rate (the time-dependent variation of stress).

Results

The results show that the volumetric flow rate and wall shear stress through a PFO vary significantly, depending on the anatomical characteristics of the defect, ranging from 0.05 to 0.6 l/min and from 25 to 106 Pa, respectively. Additionally, peak shear stresses are observed at the edges of the septum primum and secundum driven by the high-velocity blood jets passing through narrow or irregularly shaped PFOs (Figure 1).

Mechanical stress on red blood cells varies notably across different PFO geometries, with average stress accumulation along RBC trajectories ranging from 0.02 to 0.49 Pa·s (Figure 2). Peak scalar stresses and stress rate also differ by nearly one order of magnitude among the analysed PFO geometries.

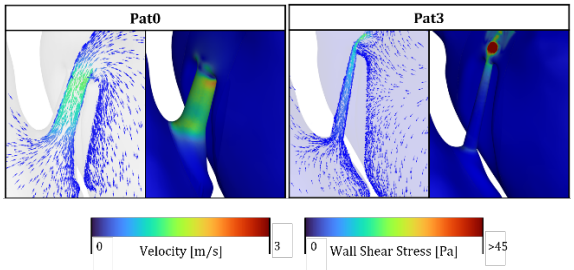

Figure 1: Comparison of velocity vectors (left) and wall shear stress (right) between two patients: Pat0 with a large, short PFO and Pat3 with a narrow, long PFO.

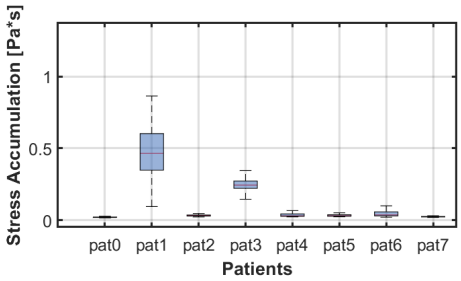

Figure 2. Stress accumulation along red blood cells trajectories in the different patient-specific PFO.

Discussion

This study highlights substantial variability in PFO fluid dynamics, which may have important clinical implications: larger defects with higher flow rates could be linked to an increased risk of embolic events, while narrower PFOs may cause red blood cell damage due to intense mechanical stress.

Future research will focus on experiments using a microfluidic system that mimics the heart defect, aiming to determine whether mechanical stresses on RBCs can lead to their dysfunction, potentially triggering MHA events.

Acknowledgments

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References

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