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ARVO Annual Meeting Abstract | June 2023

Analysis of vessel tortuosity and its impact on hemodynamics in retinopathy of prematurity

Brent Koogler; Benjamin Young; J. Peter Campbell; Giovanna Guidoboni

— Author Affiliations & Notes

Brent Koogler
Mathematics, University of Missouri, Columbia, Missouri, United States

Benjamin Young
Ophthalmology, Oregon Health & Science University School of Medicine, Portland, Oregon, United States

J. Peter Campbell
Ophthalmology, Oregon Health & Science University School of Medicine, Portland, Oregon, United States

Giovanna Guidoboni
Electrical Engineering and Computer Science, University of Missouri, Columbia, Missouri, United States
Mathematics, University of Missouri, Columbia, Missouri, United States

Footnotes

Commercial Relationships **Brent Koogler** None; **Benjamin Young** None; **J. Peter Campbell** Boston AI Lab, Code C (Consultant/Contractor), Genentech, Code F (Financial Support), Siloam, Code O (Owner), Boston AI Lab, Code R (Recipient); **Giovanna Guidoboni** Foresite Healthcare LLC, Qlaris, Code C (Consultant/Contractor), Gspace LLC, Code I (Personal Financial Interest)

Support NIH R01EY034718, NSF-DMS 2108711/2108665, NSF-DMS 1853222/2021192

Investigative Ophthalmology & Visual Science June 2023, Vol.64, 1237. doi:

Abstract

Purpose : Vessel tortuosity is a prominent biomarker of Plus disease in retinopathy of prematurity (ROP). The centerline of tortuous vessels is longer than straight vessels. We provide a theoretical, computationally efficient model to study how vessel tortuosity impacts local hemodynamics and wall shear stress (WSS) within the retinal circulation.

Methods : We developed a reduced computational fluid dynamics (rCFD) model to simulate blood flow through a vessel network. Two networks extracted from fundus images of the same ROP patient are considered, one

before the development of Plus disease (Fig.1a) and one after (Fig.1b). rCFD simulations are performed to quantify the relative importance of increased length and increased curvature on the distribution of pressure and WSS along the network. To this end, two scenarios are simulated and compared, one in which both actual length and curvature are considered (S1), and one in which only the actual length is considered (S2).

Results : All rCFD simulations have been performed setting the blood pressure at network inlet and outlet equal to 40mmHg and 15mmHg, respectively. Vessel diameters were set at the values extracted from the images and were found to be approximately 20% larger after Plus disease development. Comparing the results obtained under S1 and S2 evidenced no remarkable differences in the pressure distributions along the networks, both before and after Plus disease development. Conversely, when comparing the WSS distributions, we found marked differences between S1 and S2 in the Plus disease network (Fig. 1b) but not in

the one prior to Plus disease (Fig. 1a). Larger WSS differences are observed where the local curvature is higher.

Conclusions : Our rCFD simulations suggest that the centerline curvature of tortuous vessels leads to marked changes in the WSS distribution. Higher local WSS is known to impact vascular remodeling, and this may play a role in endothelial cell damage, vascular leakage, and neovascularization. In the future, this work may lead to better insights into the hemodynamic significance of plus disease in ROP, and the role of WSS on the aberrant vasculogenesis and neovascularization seen in ROP.

This abstract was presented at the 2023 ARVO Annual Meeting, held in New Orleans, LA, April 23-27, 2023.

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Figure 1. Simulated curvature-induced WSS differences in vascular networks of the same ROP patient before (Fig. 1a) and after (Fig. 1b) development of Plus disease.