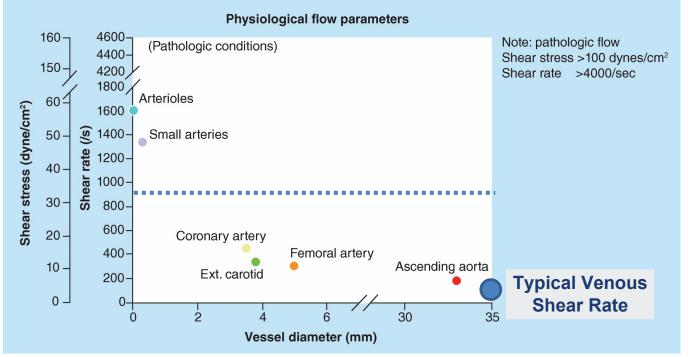
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Introduction Background *Iliac Vein Compression Syndrome (IVCS)* • Formerly known as May-Thurner Syndrome • Anatomical variant in which the **right common** iliac artery compresses the left common iliac **vein** (LCIV) against the lumbar spine. • Present in over 20% of the population. [1] Associated with increased risk of Deep Vein Thrombosis (DVT). [2] **Before LCIV Stenosis After LCIV Stenosis** 14 In IVCS, the narrowing increases LCIV velocity by as much as 10x. [3] L. Oğuzkurt, Diagn Interv Radiol, 2007. Shear Activation of Platelets • High velocities in narrow vessels leads to high shear rates. • High shear rates unravel von Willebrand protein and enable platelet adherence via the glycoprotein lb receptor. [4]

• Shear activation of platelets begins to occur around **1000** s⁻¹. [5] • Thrombus initiation via platelet shear activation is typically thought to only occur in the arteries.



K.S. Sakariassen, Future Sci. OA, 2015.

Hypothesis

Elevated shear rates in IVCS patients trigger platelet activation and thus initiates thrombus formation.

Approach

• We employed the simulation framework CRIMSON: a multi-scale modeling software that combines a 3D geometric model, inflow waveform boundary conditions, and Windkessel models to evaluate IVCS hemodynamics.

• Patient-specific models were informed by retrospective CT data and duplex ultrasound measurements (IRB-HUM00212189) of the Inferior Vena Cava (IVC), Left/Right Common Iliac Veins (LCIV/RCIV), and Left/Right External Iliac Veins (LEIV/REIV).



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Computational Analysis of Altered Hemodynamics in Iliac Vein Compression Syndrome

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