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IMAGES IN INTERVENTION

Colocalization of Low and Oscillatory Coronary Wall Shear Stress With Subsequent Culprit Lesion Resulting in Myocardial Infarction in an Orthotopic Heart Transplant Patient

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Low and oscillatory wall shear stress (WSS) have been implicated in the pathogenesis of atherosclerosis (1,2). Furthermore, cardiac allograft vasculopathy (CAV) has the highest rate of progression opposite flow dividers, suggesting a role of regional fluid dynamics (3). However, to our knowledge, the association between regional WSS and clinically manifest CAV has not previously been described.

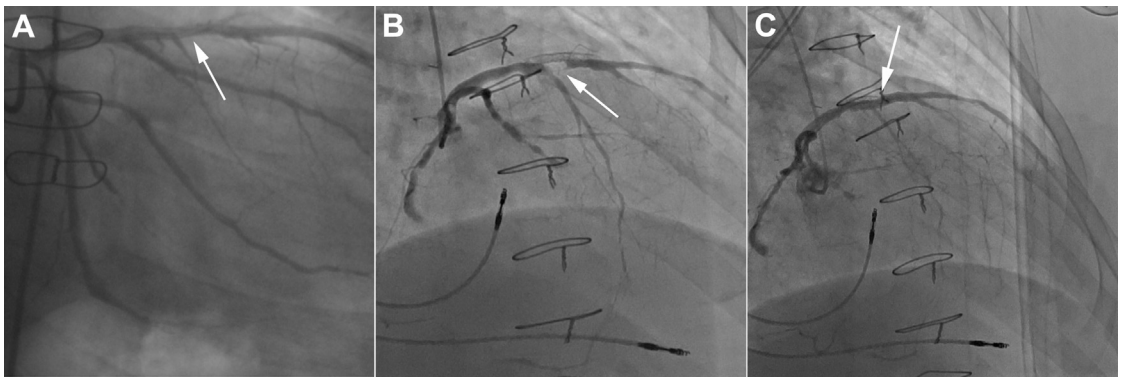

Figure 1. Angiographic Image Data

(A) Angiographically normal coronary arteries (before cardiac allograft vasculopathy formation, 7 years post-heart transplant) utilized to create retrospective computational fluid dynamics (CFD) model (right anterior oblique [RAO]-caudal view); **arrow** indicates the site of subsequent culprit lesion. **(B)** Angiographic presentation at time of non-–ST-segment elevated myocardial infarction (RAO-cranial); **arrow** indicates 99% stenosis in mid-left anterior descending coronary artery (LAD); note the 1st diagonal (D1) had been stented and subsequently occluded in the interim between **A and B**. **(C)** Angiogram following percutaneous coronary intervention (PCI) with the LAD reconstituted; **arrow** indicates the site of PCI (RAO-cranial) (Online Video 1).

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We present images from a 45-year-old man, 15 years post-heart transplant (HTx), who has undergone annual surveillance coronary angiography. His angiogram 7 years post-HTx demonstrated no significant CAV (Fig. 1A). In subsequent years, he developed significant CAV, underwent percutaneous coronary intervention (PCI) in the 1st diagonal and 1st obtuse marginal arteries, and was treated with standard immunosuppressive therapy, statins, and aspirin. He recently presented

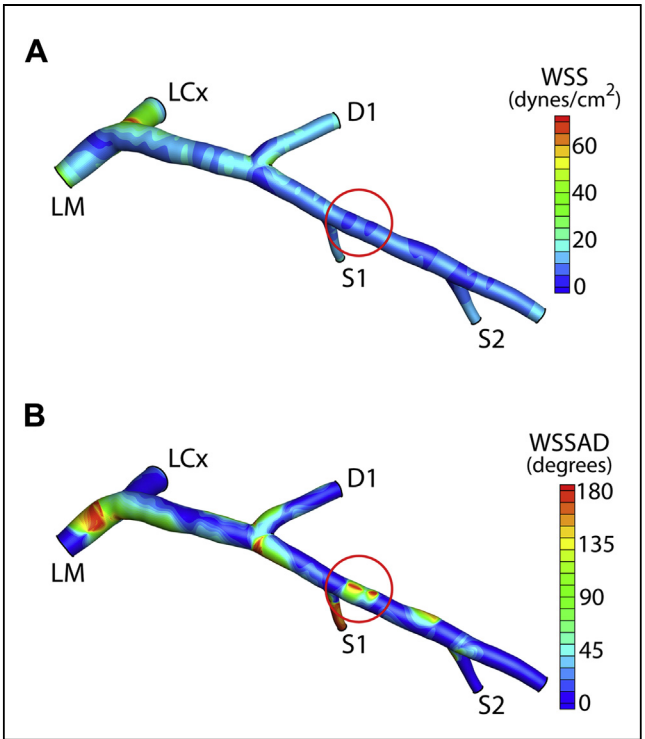

Figure 2. Results of CFD in 3D Coronary Reconstructed Geometry

(A) Time-averaged WSS distribution. **(B)** WSS angle deviation (WSSAD) distribution (WSSAD >180° indicates complete flow reversal). The location of the subsequent culprit lesion **(red circle)** occurred in an area of low, oscillatory WSS before cardiac allograft vasculopathy formation. 3D = 3-dimensional; LM = left main coronary artery; LCX = left circumflex coronary artery; S1 = 1st septal coronary artery; S2 = 2nd septal coronary artery; other abbreviations as in Figure 1 (Online Video 3).

with a large anterior non–ST-segment elevated myocardial infarction (NSTEMI), and emergent angiography revealed a 99% stenosis in the mid-left anterior descending coronary artery (LAD) with Thrombolysis In Myocardial Infarction flow grade 2 (Fig. 1B, Online Video 1).

In order to calculate WSS, 3-dimensional geometric reconstruction and computational fluid dynamics (CFD) simulation were performed from the patient's 7-year post-HTx angiogram (when he had no significant CAV). CFD utilizes computational methods to quantify velocity and pressure data throughout the domain of interest (e.g., vessel). Post-processing of these data allows for quantification of various fluid dynamics parameters (e.g., WSS [2]).

Results revealed low velocity (Online Video 2) and low WSS, defined as the frictional force exerted on the luminal surface by flowing blood, magnitudes (Fig. 2A, Online Video 3) opposite the flow divider at the site of the subsequent culprit lesion that persisted throughout the cardiac cycle. Furthermore, oscillatory WSS was observed at the site of subsequent culprit lesion formation as demonstrated by the WSS angle deviation (Fig 2B) and reversal of WSS vectors during the cardiac cycle (Fig. 3, Online Video 4).

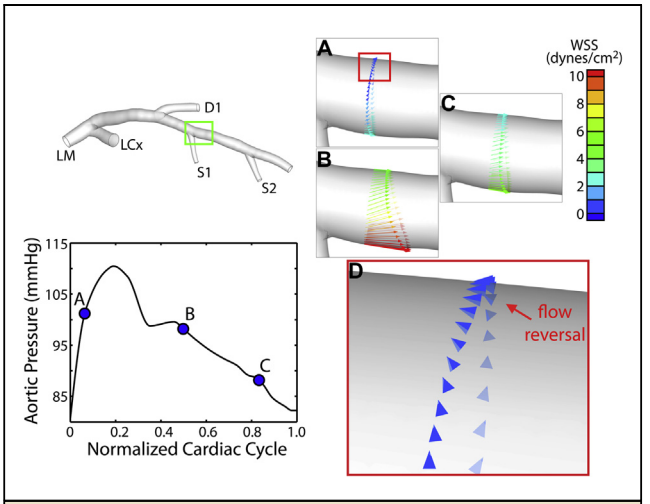

Figure 3. Instantaneous WSS Vectors at Culprit Lesion Site

Dynamic WSS vectors were examined at the site of the subsequent culprit lesion in the 3D reconstructed geometry **(top left; green rectangle)**. The **panel at the bottom left** shows the corresponding time points in the cardiac cycle during which **A** to **C** were examined in the CFD simulation. WSS vectors in early systole demonstrate flow reversal opposite the flow divider **(A: red rectangle, D: zoomed-in)**, whereas WSS vectors in the remaining panels exhibit no flow reversal **(B and C)** in aggregate, demonstrating oscillatory WSS during the cardiac cycle. Note that low WSS magnitudes **(color scale)** exist throughout the cardiac cycle. Abbreviations as in Figures 1 and 2 (Online Video 4).

These images introduce a hypothesis-generating association between low and oscillatory WSS and subsequent development of a focal, clinically manifest CAV, that warrants future investigation.

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Key Words: cardiac allograft vasculopathy ■ computational fluid dynamics ■ hemodynamics.

APPENDIX

For accompanying videos, please see the online version of this paper.