

High-pressure distention of the saphenous vein during preparation results in increased markers of inflammation: a potential mechanism for graft failure

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Background: Coronary artery disease is the single leading cause of death in the United States. Commonly it is treated with coronary bypass grafting using the saphenous vein (SV) or internal mammary artery (IMA) as a conduit. Unfortunately, the SV has much lower patency rates compared with the IMA. Several hypotheses exist as to why occlusion occurs more commonly in SV grafts than in IMA grafts. However detailed studies in this area have been limited. This study investigates the effects of pressure distention on inflammation in SV conduit used in coronary artery bypass grafting (CABG).

Methods: Saphenous vein distention pressure was measured intraoperatively during 48 CABG procedures. A segment of SV was excised from the conduit before distention. Because the vein was used for coronary artery grafting, sequential pieces were archived for evaluation. Real-time polymerase

chain reaction (RT-PCR) and immunohistochemical analyses were performed to investigate a change in the expression of biomarkers.

Results: Upregulation of various biomarkers occurred. These biomarkers included scavenger receptors A and B (SR-A, SR-B), toll-like receptors 2 and 4 (TLR2, TLR4), platelet endothelial cell adhesion molecule (PECAM), vascular cell adhesion molecule (VCAM), and intercellular cell adhesion molecule (ICAM) in segments of SV that were subjected to distention. Immunohistochemical results mirrored RT-PCR findings. A significant correlation was observed between biomarkers and pressure values.

Conclusion: These studies demonstrate that markers of inflammation are upregulated in response to SV distention. The data suggest that the pressure used in graft preparation procedures should be regulated to avoid inflammation and its potential to induce graft failure.

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