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Role of C-reactive protein in a murine model of vein graft disease

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C-reactive protein (CRP), an acute phase reactant plasma protein, plays a key role in modulating the innate immune system. Elevated plasma levels of CRP are independently associated with increased risk of thrombosis. In coronary artery bypass grafting (CABG), leg veins are grafted around a coronary artery in order to divert blood flow around blocked areas of the artery. However, vein grafts can develop thrombosis, intimal hyperplasia, and atherosclerosis leading to vein graft disease (VGD). Published studies suggest that increased plasma levels of CRP are a risk factor for developing VGD. Microparticles (MP), submicron cell fragment released from apoptotic cells, promote thrombosis by increasing available surface area for clotting factor assembly. As increased plasma CRP levels can cause vascular cells to apoptose and release MP, we studied the relationship between increased plasma CRP levels, vein graft intimal hyperplasia, and MP formation in the mouse. CRP transgenic and wild type (WT) mice were used to assess the effect of CRP on VGD. Vein grafts harvested from donor mice were grafted into recipient mice and one month after surgery vein grafts were excised and processed for analysis. Planemetry was performed to measure intimal hyperplasia and samples were stained with antibodies against CRP to view the levels of the protein in each graft. MPs were separated from mouse whole blood and quantified by flow cytometry. No significant difference in neointima formation was seen between groups. CRP immunostaining suggests that CRP present in vein grafts is primarily of systemic origin. There was no significant difference in MP concentration between groups, although there was a trend for higher concentrations in CRP-Tg mice. CRP does not significantly increase intimal hyperplasia in vein graft segments or plasma MP concentration. However, sample sizes were small and further studies need to be performed to elucidate.