Are Decreased Levels of Cystatin-C an Indicator of Weak Arterial Walls in Abdominal Aortic Aneurysms?

Chandra, Rayna

Yearly, more than 15,000 people die from aneurysms, making it the 13th leading cause of death in the United States. Today, many risk factors have been identified however no single cause has been narrowed down as to what is causing the weakness in arterial walls from which is generating the development of abdominal aortic aneurysms. In my experiment, I wanted to identify what was causing the weakness in arterial walls by measuring certain quantities of proteins that indicate likely causes. I wanted to determine if the elimination of hemodynamic stress on arterial walls (placing a stent graft in the aneruysm) reduces the levels of certain proteins (Specifically Cathepsin-S) and consequently changes the level of Cystatin-C (An inhibitor of Cathepsin-S). Cathepsin S is an example of an elastolytic enzyme, which breaks down the elastin in the wall of the artery. Because Cystain-C is an inhibitor, it works to slow down the process of Cathepsin-S which slow down the breakdown of arterial walls. By measuring quantities of Cystatin-C and determining an increase or decrease in the levels, after a stent graft has been placed in the aneurysm, quantity levels of Cystatin-C can further suggest what is really causing weak arterial walls. After comparing the two quantites before and after surgery, the Cystatin-C levels decreased showing that aneurysmal disease is still present in the body even after treatment which eliminated hemodynamic stress. Levels of Cystatin-C continued to decrease because elastolytic enzymes are still present. This information shows that patients are likely to have another aneurysm within the next 2-10 years.

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