Decreased Risk of Cardiac and Vascular Complications in Patients with Alpha-1-Antitrypsin Deficiency

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Background: Alpha-1-antitrypsin deficiency (AATD) is a genetic disorder characterized by low levels of the antiprotease Alpha-1-antitrypsin protein (AAT) within blood. In AATD, research has shown misfolded AAT accumulates within the liver and poorly controls protease activity in the lungs, resulting in liver and lung sequela. AATD's impact on cardiac and vascular health is far less researched and conflicting data has emerged. This study aims to determine if AATD's possible protective role against cardiac and vascular complications is consistent between the COSYCONET and RWEdataLab's databases.

Methods: The IU School of Medicine-Evansville RWEdataLab (CRC/Sidus Insights) psychiatric and cardiac databases were used to select two groups for comparison: patients with AATD and patients without AATD with a diagnosis of COPD, emphysema, or chronic bronchitis, all without a history of lobectomy or lung or liver transplant or malignancy. Differences in diagnosis history were compared using odds ratios.

Results: Comparing 879 AATD patient's diagnoses against 3489 non-AATD patients with lung condition's diagnoses within the psychiatric database shows a protective role in both vascular and cardiac health with the following odds ratios and respective 95% CIs: 0.501 (0.325-0.771) for cerebrovascular accident, 0.499 (0.430-0.579) for hypertension, 0.377 (0.289-0.494) for chronic heart failure, 0.442 (0.273-0.714) for myocardial infarction, 0.490 (0.398-0.605) for coronary artery disease, and 0.742 (0.589-0.935) for peripheral artery disease. Similar searches were performed in the cardiac database, but too few AATD patients were found to perform calculations with statistical significance.

Conclusion and Implications: These findings suggest that AATD may have a protective role in cardiac and vascular health. The mechanism behind these findings is unknown and may highlight an area of potential future study. Additionally, further research is needed to determine if AAT augmentation therapy is affecting this protective aspect.