Alpha-1 antitrypsin deficiency: From the lung to the heart?

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BACKGROUND AND AIMS: Alpha-1 antitrypsin (A1AT) is the most abundant serine protease inhibitor in human blood and exerts important anti-inflammatory and immune-modulatory effects. In combination with smoking or other long-term noxious exposures such as occupational dust and fumes, genetic A1AT deficiency can cause chronic obstructive pulmonary disease, a condition with elevated cardiovascular risk. The effects of A1AT deficiency on cardiovascular risk have hardly been studied today. METHODS: Using data from 2614 adults from the population-based SAPALDIA cohort, we tested associations of serum A1AT and SERPINA1 mutations with carotid intima-media thickness (CIMT, measured by B-mode ultrasonography) or self-reported arterial hypertension or cardiovascular disease in multiple regression models using a Mendelian Randomization like analysis design. Mutations Pi-S and Pi-Z were coded as ordinal genotype score (MM, MS, MZ/SS, SZ and ZZ), according to their progressive biological impact. RESULTS: Serum A1AT concentration presented a u-shaped association with CIMT. At the lower end of the A1AT distribution, an analogous, linear association between SERPINA1 score and higher CIMT was observed, resulting in an estimated 1.2% (95%-confidence interval -0.1-2.5) increase in CIMT per unit (p/=/0.060). Genotype score was significantly associated with arterial hypertension with an odds ratio (OR) of 1.2 (1.0-1.5) per unit (p/=/0.028). The association with cardiovascular disease was not significant (OR 1.3 (0.9-1.9)). CONCLUSIONS: Our results support a possible causal relationship between genetic A1AT deficiency and increased cardiovascular risk, which needs to be better taken into account for the management of affected patients and first-degree relatives.
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