INCREASED EXPRESSION OF OXIDATION-SPECIFIC EPITOPES AND APOPTOSIS ARE HAPTOGLOBIN GENOTYPE DEPENDENT: IMPLICATIONS FOR PLAQUE PROGRESSION IN HUMAN ATHEROSCLEROSIS

ACC Poster Contributions
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Background: We have previously reported that Haptoglobin (Hp) 2-2 genotype may be a significant marker for plaque progression in human atherosclerosis. In this study we tested the hypothesis that increased expression of oxidation-specific epitopes may trigger apoptosis in Hp 2-2 atherosclerotic plaques.

Methods: Twenty-six human atherosclerotic plaques (26 aortas) were genotyped for Hp gene using corresponding liver samples by PCR. Hp 2-2 plaques (n=13) were compared to control plaques (n=13). The expression of oxidation-specific epitopes was quantified by immunostaining using EO6 and IK-17 antibodies. Morphological features of apoptosis including nuclear fragmentation, chromatin condensation, margination, percentage of apoptosis, cytoplasmic blebs and eosinophilia were randomly counted in 20 oil immersion fields and expressed as percentage of total cells. DNA fragmentation and activated caspase-3 were quantified by in-situ end labeling (ISEL), and immunohistochemistry respectively.

Results: See table and figure. Binary logistic regression analysis identified correlation between density of oxidation-specific epitopes (EO-6, IK-17) and percentage of apoptotic cells (r =0.78; p= 0.0001).

Conclusions: Increased expression of oxidation-specific epitopes is associated with increased apoptosis in Hp2-2 plaques. These observations have important implications for understanding genotype-dependent mechanisms in the progression of human atherosclerosis.

![Image of table and figure showing apoptosis and histological features]