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The balance of serum matrix metalloproteinase-8 and its tissue inhibitor in acute coronary syndrome and its recurrence

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Introduction: Matrix metalloproteinase-8 (MMP-8) is involved in the breakdown of the extracellular matrix increasing the vulnerability of atherosclerotic lesions. MMPs are endogenously inhibited by the specific tissue inhibitor of metalloproteinases (TIMP-1).

Methods: Sera were collected from 343 patients with ACS [including108 unstable angina pectoris and 235 acute myocardial infarctions] within 24 hours after the diagnosis and three to six months after the acute event during the recovery period (n=157). Serum MMP-8 concentrations were determined by a time-resolved immunofluorometric assay. 326 age- and sex-matched subjects served as controls.

Results: The ACS patients had high IgG-class antibody levels to chlamydial heat shock protein 60 and the levels correlated with serum TIMP-1 concentrations (r=0.225, p=0.001). High serum MMP-8 and TIMP-1 concentrations (4th quartile vs. 1st) associated with ACS. The trend from quartile 1 to 4 was very significant (p<0.001). A strong direct association was found between TIMP-1 concentration and CVD death during the follow-up. Acute phase and recovery period TIMP-1 concentrations associated with cardiovascular death with hazard ratios 4.31 (2.00–9.26, p<0.001) and 4.69 (1.10–20.01, p = 0.037), respectively.

Conclusions: A persistent inflammation existed before the acute event. The increase of serum MMP-8 and TIMP-1 concentrations may reflect plaque instability and tissue damage. TIMP-1 may exert a continuous damaging stimulus promoting inflammation. If TIMP-1 is "over produced" as a response to acute phase MMP-8 expression or if its production is not suppressed enough during the recovery, the prognosis is poor. The fndings may have practical implications in both diagnostics and therapeutics.

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