Subsets of CD8+ T Cells Are Expanded in Stable Coronary Artery Disease

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Background: There is evidence for a systemic immune response, involving continuous T cell activation, in stable coronary artery disease (CAD). So far, the type of this T cell response is not fully understood. In this study we examined certain phenotypical properties of CD8+ T cells in stable angina patients.

Methods: Forty-four men (< 60 years of age) with stable angina and angiographically verified CAD were included as well as 70 healthy controls. T cell phenotypes in peripheral blood were determined by 3-colour flow cytometry using triple monoclonal antibodies against the following surface antigens and activation markers: CD3, CD4, CD8, CD57, CD28 and HLA-DR.

Results: The total number of CD8+ cells did not differ significantly between patients and controls (577±230 vs 476±95 cells/mm^3). However, the proportions of CD8+ cells expressing HLA-DR and CD57 were significantly increased in the patient group while the percentage of CD8-negative cells was decreased. Data are presented in the table.

Conclusions: CD8+ cells are activated in patients with stable angina and there is an expansion of a certain CD8+ phenotype expressing CD57 but not CD28. This phenotype has been associated with suppressive activity and its role in stable CAD may be to control an antigen-driven immune response.

Patients Controls p
CD8+/CD3+ cells/mm^3 317±234 220±258 0.04
% of total CD8+ 47 37 0.005
CD8+/CD4+ cells/mm^3 365±264 262±282 0.06
% of total CD8+ 52 45 0.05
HLA-DR+/CD8+ cells/mm^3 152±111 101±73 0.003
% of total CD8+ 25 20 0.01

Impact of New Diagnostic Criteria for Myocardial Infarction in an Unselected United Kingdom Cohort With Suspected Cardiac Chest Pain

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Background: The Joint European Society of Cardiology/American College of Cardiology Committee's revised definition of myocardial infarction (MI) proposes the primacy of the cardiac troponin in the diagnosis of MI, with CK-MB a recommended alternative. These recommendations may increase the incidence of MI and reduce case fatality, but this has not been examined prospectively. We therefore assessed the diagnostic impact of these changes in an unselected UK cohort with suspected cardiac chest pain.

Methods: We enrolled all patients admitted with suspected cardiac chest pain over 6 months, with usual care provided by the attending physician with serial ECgs and creatine kinase (CK)/aspartate transaminase (AST). Additional blinded measurements of CK-MB mass and troponin T (cTNT) were made. After discharge a blinded panel reviewed the case notes and reached a World Health Organisation diagnosis utilising electrocardiograms/CKAST, a CK-MB diagnosis using CK-MB as the gold-standard cardiac marker at 2 discriminator values (5 and 10µg/L), and a cTNT diagnosis in accordance with the new recommendations.

Results: 401 patients were enrolled. In comparison to the WHO diagnosis, cTNT-CK at 5µg/L diagnosed a further 23.1% (36 of 155) MI. 11 patients with high risk unstable angina pectoris (UAP) and excluded MI in 4 patients, with overall 51 (12.7%) patients having a significant diagnostic adjustment. At 10µg/L, cTNT diagnosed 126 patients as MI, 35 as high risk UAP, with a diagnostic alteration in 34 (5.3%) patients. The revised criteria utilising cTNT diagnosed a further 26.1% (46 of 181) MI, and excluded MI in 4 patients, with 11.5% of patients having a significant diagnostic alteration. Serial CK and AST measurements had a diagnostic sensitivity of 70.5% and 69.0% respectively for MI.

Conclusion: Introduction of the revised diagnostic criteria for MI diagnoses an additional 26.1% of patients with MI, CK-MB diagnoses a broadly similar cohort as MI at a lower discriminator value, but not at the higher threshold. Serial CKAST and ECGs mis-diagnose 11.5% of all chest pain admissions when compared with the new definition for MI.