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Cardiac Troponin – Friend or Foe?

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Despite the widespread use of cardiac troponin as a specific biomarker for the acute coronary syndrome, little is known about the clinical consequences of its release into the systemic circulation. Since cardiac troponin I is perceived as not only being strictly confined to the intracellular compartment but is also present on the surface of cardiomyocytes, autoantibodies and autoreactive lymphocytes specific to cardiac troponin I, recognized as foreign entities by the immune system, could possess the potential to interfere with heart functioning in terms of an autoimmune reaction.

The present work was intended to study these immunologic facets following a myocardial infarction. For this purpose patients were being investigated for the presence of autoantibodies against cardiac troponin I in relation to changes of their heart function: in a first prospective trial, 56 patients with myocardial infarction were assessed through echocardiography, electrocardiography and the measurement of NTproBNP reflecting heart insufficiency. In a second clinical study, cardiac magnetic resonance imaging served to examine the course of the left ventricular function in further 108 patients. Additionally, the proliferative response of T lymphocytes to cardiac troponin I was measured in subjects in order to explore the involvement of the cellular response specific to the cardiac troponin I release.

Autoantibodies detected in patients were associated with a worse clinical outcome manifested by high NTproBNP levels and decreased left ventricular ejection fractions over the study period. On the other hand, in patients with myocardial infarction, T lymphocytes proliferating in response to cardiac troponin I could be depicted, suggesting the existence of potentially self-reactive T cells to cardiac troponin I.

The immune system has been shown to play a substantial role in the ventricular remodeling process secondary to a myocardial infarction. The present results suggest the contribution of both the humoral (autoantibodies) as well as the cellular (lymphocytes) immune response specific to cardiac troponin I to the progression toward heart failure, a common and worrisome sequela of the acute coronary syndrome. These findings emphasize the importance of the immune system in the patient suffering from myocardial infarction. A better understanding of the pathophysiological mechanisms of autoimmunity is seminal on the way to novel treatment options. Whether patients can benefit from interventions either by means of plasmapheresis, anti-autoantibodies or immunosuppressive drugs by anticipating progression to heart failure, should be the focus of future clinical studies. Finally, as a friend of the cardiologist, cardiac troponin aids in the diagnosis of the acute coronary syndrome. But conversely, as a potential stimulus for the immune system with detrimental effects precipitating heart insufficiency, cardiac troponin may represent a foe to the patient as well.