How to reduce the risk after heart attack?

A tremendous progress in the treatment of myocardial infarction is associated with reduction of in-hospital mortality. However, 5 years after myocardial infarction mortality rate still is about 20% and the rate of cardiovascular death or recurrent myocardial infarction is 25-30%. Why it is happening? This unacceptably high rate of major cardiovascular events is associated with persistent prothrombotic hazard in patients after myocardial infarction.

The main reason of myocardial infarction is plaque rupture in the coronary artery and thrombus formation, which together may lead to coronary artery occlusion. There is lack of diagnostic algorithm to determine persistent prothrombotic hazard based on clinical data, laboratory results and imaging examinations in patients after acute coronary syndrome. The principal barrier is not enough clinically sufficient prognostic accuracy of every single available clinical or laboratory parameter.

Therefore we plan to conduct a clinical research to determine individual profile associated with a persistent prothrombotic hazard after myocardial infarction. We hypothesized that there is a relationship between tendency of atherosclerotic plaque to rupture, the size and composition of intracoronary thrombus removed during coronary angioplasty and persistent prothrombotic hazard expressed as a persistent or increased value of unfavorable plasma-based laboratory parameters within the first months after myocardial infarction (Figure).

The aim of the study is to identify patient's prothrombotic profile based on clinical parameters, complex laboratory tests from blood (e.g. rheometry - it is analysis of the viscoelastic properties, aggregometry - method for the assessment of platelet activity, proteomics - method to analyze proteins in the tissue) and different imaging modalities (e.g. scanning electron microscopy to assess the intracoronary thrombus composition with large magnification, or optical coherence tomography to view the coronary artery from the inside), which first at baseline may correlate with intensity of local coronary pathology and second persists within the first six months after MI.

Additionally, for the first time in the current study we plan to develop new quantitative proteomic analysis of ex-vivo formed clots similar to that created in the coronary artery during myocardial infarction, in order to correlate changes in clot-bound protein compositions with other prothrombotic factors.

The final effect of our study should be first determination of plasma-based profile, hopefully including also proteome of plasma fibrin clot associated with persistent prothrombotic hazard after MI and second at least partial clarification of the mechanisms by which the selected parameters affect prothrombotic risk. There are at least two aspects of potential practical utility. The novel and innovative prognostic tool based on profile of persistent prothrombotic hazard may first improve identification of patients with high risk of unfavorable long-term clinical prognosis and second, opens up possibilities for new therapeutic approaches that would allow for intervention into prothrombotic mechanisms in order to reduce the rate of adverse cardiovascular events.

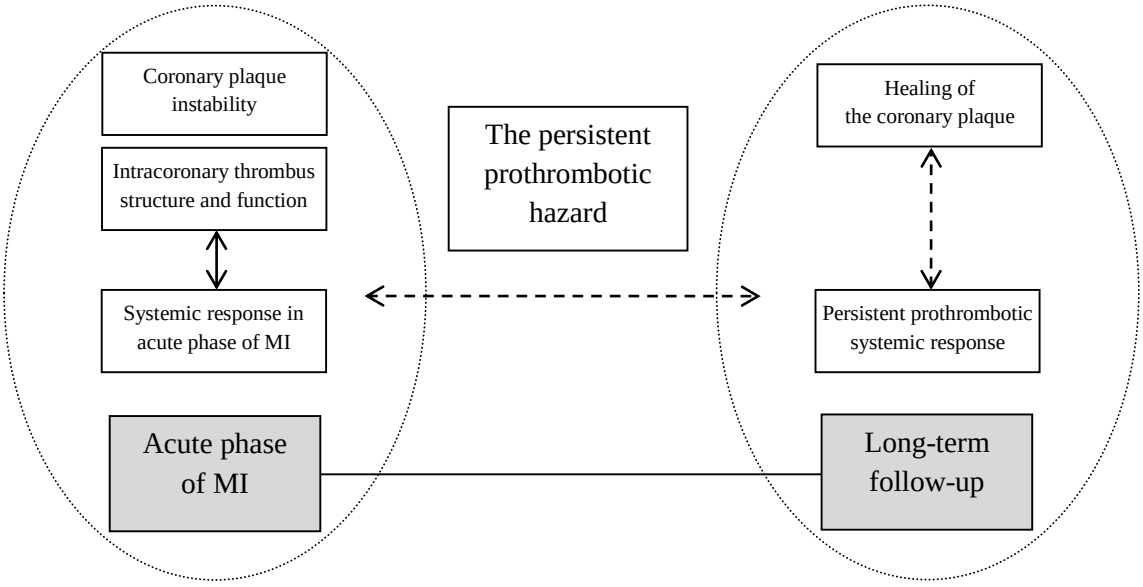

Figure. Research hypothesis (solid lines show the known mechanisms, dashed lines indicate not fully understood relationships).