

Serum Cortisol in Acute Myocardial Infarction

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There were several prognostic indicators in acute myocardial infarction included: clinical and electrocardiographic features, hemodynamic parameters, elevation of cardiac biomarkers and combination of the above parameters.

An early study by Forssman et al in 1952 showed that in many patients with myocardial infarction (MI), the hormone output from the adrenal cortex is increased¹. Serum levels of cortisol also were reported to be increased². Prakash et al suggested that the generalized metabolic stress of acute myocardial infarction results in elevation of cortisol, free fatty acid and catecholamines as measurable biochemical indicators and / or predictors of the severity of the infarction³. It also was revealed that adrenal cortex activity occurring in the first days of MI is much more pronounced in complicated cases than in subjects with a mild clinical course³. The causal relationship of these findings remains uncertain⁴.

Logan and Murdoch² suggested that myocardial tissue necrosis was the stimulus to adrenocortical secretion, but such direct relationships can not be drawn with any certainty. Probably a number of factors are involved, including other hormonal and biochemical changes, haemodynamic disturbances and emotional stimuli⁵.

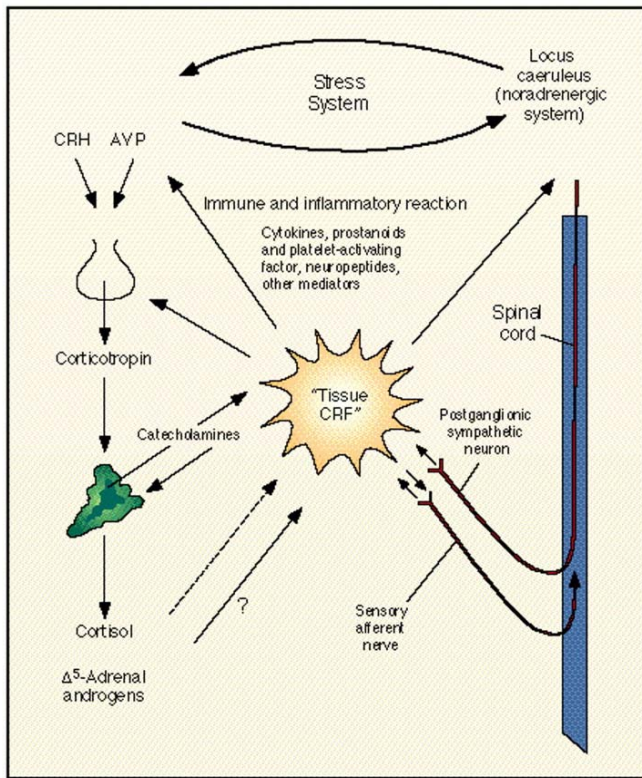
In this issue of the Journal, Nito and colleagues⁶ report the relationship of cortisol levels with myocardial infarction and the correlation of cortisol levels elevation with the outcome of myocardial infarction. In the deceased patient, the cortisol levels were higher significantly and duration of elevation cortisol levels were longer from survived ones. This result is consistent with the study by Wiener⁵ that reported the cortisol levels significantly higher in the group who died. In the patients

who received thrombolytic therapy, cortisol levels lower significantly and the duration of cortisol elevation were shorter as compared to patients without thrombolytic therapy. Although, the groups with large infarct size and myocardial infarction complications had higher cortisol levels, it's not significantly different from the groups with small infarct size and patients without complications. This result is inconsistent with the study by Prakash³ that reported a correlation between the level of cortisol and the presence and subsequent development of myocardial infarction complication (left ventricular failure, arrhythmias, shock, or death). This difference may be due to inadequate number of samples. What can we learn from this study about the pathophysiology of acute myocardial infarction and its complication?

The past decade has been characterized by a growing interest in the idea that atherosclerosis is an inflammatory disease⁷ and by the finding that serum levels of markers of inflammation in acute coronary syndrome can be used to predict the risk of cardiovascular events⁸. Figure 1 shows disturbances of the interaction between the hypothalamic-pituitary-adrenal (HPA) axis and immune mediated inflammation. An excessive HPA response to inflammation can mimic the state of stress or hypercortisolemia⁹. The generalized metabolic stress of acute myocardial infarction can stimulate HPA axis. Tumor necrosis factor α , interleukin-1, interleukin-6, and perhaps other mediators of inflammation collectively called tissue corticotrophin releasing factor stimulate the secretion of corticotrophin releasing hormone (CRH) and arginine vasopressin (AVP) from hypothalamic CRH and AVP neurons; at high concentrations they stimulate the secretion of corticotrophin from the pituitary corticotroph and glucocorticoid from the adrenal cortex.

There is still much to be learned about the mechanisms that link hormonal activation (cortisol) and inflammatory markers to the risk of cardiovascular complications. Progress in this field enhances our ability to predict the risk of such complications, allow clinicians

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Figure 1. Interactions Between the Stress System and Immune-Mediated Inflammation

to administer preventive therapies, provides potential new target for the treatment of acute myocardial infarction and promises to contribute to a new era of preventive cardiovascular medicine.

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