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# Atorvastatin restores the balance between pro-inflammatory and anti-inflammatory mediators in rats with acute myocardial infarction

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## Abstract

**– Background: Pro- and anti-inflammatory cytokines play a major role in the development of acute myocardial infarction (AMI). This paper tests the hypothesis that atorvastatin may attenuate the severity of myocardial ischemic injury by restoring the balance between pro-inflammatory and anti-inflammatory mediators.**

**Materials and Methods: Sixty adult male albino rats were used. Experimental AMI was induced by subcutaneous injection of isoprenaline. Atorvastatin was given for five days,then, it was combined with isoprenaline in the last two days of treatment protocol. Rats without any treatment were used as controls. Rats were subjected to ECG tracing, assessment of Creatine phosphokinase (CPK) and CPK-MB, easurements of C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α), interleukin- 10 (IL-10), and plasminogen activator inhibitor-1 (PAI-1).**

**Results: Induction of AMI by isoprenaline resulted in a significant elevation of ST segment, elevation of CPK and CPK-MB. CRP, TNF-α and plasma PAI-1 were significantly elevated in the AMI rats compared to the control groups. On the other hand, the level of the anti-inflammatory cytokine IL-10 was significantly reduced. Treatment with atorvastatin prior to induction of AMI was associated with a significant reduction of serum CRP, TNF-α, plasma PAI-1 and an increase of serum IL-10.**

**Conclusions: This study suggests the usefulness of atorvastatin as an attenuating agent against AMI. Atorvastatin restores the balance between the pro-inflammatory and the anti-inflammatory mediators and modulates the fibrinolysis by reducing the levels of PAI-1.**