Increased Levels of Matrix Metalloproteinase-9 in Patients With Acute Myocardial Infarction: No Correlation With C-Reactive Protein

Joan Luu Buob, Daniela M. Zähringer, Eric J. Tschirhart, Malou Glaesener, Georges Girod, Daniel R. Wagner, Centre de Recherche Publica de la Santé, Luxembourg, Luxembourg, Centre Hospitalier, Luxembourg, Luxembourg

Background: Matrix metalloproteinases (MMPs) appear to play an important role in the development of plaque rupture and acute myocardial infarction (AMI). Small series have shown that peripheral levels of MMP-2 and MMP-9 are elevated in patients with acute coronary syndrome. The aim of the study was to determine the relationship between MMP-9, MMP-2, and high-sensitivity C-reactive protein (hs-CRP), a classical marker of inflammation, in patients with AMI. Methods: We measured MMP-2, MMP-9, and hs-CRP in peripheral leukocyte samples from 357 consecutive patients undergoing coronary angiography. The activities of MMP-2 and MMP-9 were determined using zymography and densitometry analysis (area under the curve). The levels of hs-CRP were measured with sandwich enzyme immunoassay. Results: Of 397 consecutive patients, 49 had AMI and the remaining had unstable angina, stable angina, cardiomyopathy or valvular disease. MMP-2 activity was not elevated in patients with AMI (45.24 ± 49.10 pixel/s, p<0.0005), and was highest during the first 24 post-AMI (52.270 ± 85.93 pixel/s, p<0.00001). MMP-2 activity appeared to be correlated with the severity of AMI, peak activities being observed in 2 patients with AMI (+_00,78 ± 0.05 pixel/s) and a single patient with AMI (50.10 ± 50.10 pixel/s). Levels of hs-CRP were also elevated in patients with AMI (3.19±0.6 ± 0.8 ± 0.1 mg/dL, p=0.0001) but did not correlate with the rise in MMP-9 activity. Conclusions: MMP-2 activity is elevated early after the onset of acute ischemia and hs-CRP is also elevated in patients with AMI. However, no correlation between these two biomarkers was observed in patients with AMI.

Short-Term Atorvastatin Pretreatment Reduces Myocardial Infarct Size in Normcholesterolemic Rats

Yogiw Ramberg, Taras Ashton, Barry F. Uretsky, Scott Ballinger, Massoud Motamedi, University of Texas Medical Branch, Galveston, TX

Background: Previous studies have suggested that HMG-CoA reductase inhibitors (statins) attenuate ischemia-reperfusion injury. We assessed whether short-term treatment with atorvastatin reduces myocardial infarct size. Methods and Results: We treated rats with atorvastatin (80 mg/kg, n=10), (8 mg/kg, n=8), or (2 mg/kg, n=8) water (n=10) administered once daily for 3 d and induced myocardial infarction by ligation of a coronary artery for 30 min followed by 3 h of reperfusion. Ischemic area at risk was assessed with blue dye and infarct size by triphenyltetrazolium chloride. Infarct size was expressed as a percentage of the area at risk. Area at risk was comparable among groups. In contrast, infarct size, expressed as a percentage of the area at risk was significantly smaller in the atorvastatin groups (80 mg/kg) and water (n=10) administered once daily for 3 d and induced myocardial infarction by ligation of a coronary artery for 30 min followed by 3 h of reperfusion. Ischemic area at risk was assessed with blue dye and infarct size by triphenyltetrazolium chloride. Infarct size was expressed as a percentage of the area at risk. Area at risk was comparable among groups. In contrast, infarct size, expressed as a percentage of the area at risk was significantly smaller in the atorvastatin groups (80 mg/kg) and water (n=10) administered once daily for 3 d and induced myocardial infarction by ligation of a coronary artery for 30 min followed by 3 h of reperfusion. Ischemic area at risk was assessed with blue dye and infarct size by triphenyltetrazolium chloride. Infarct size was expressed as a percentage of the area at risk. Area at risk was comparable among groups. In contrast, infarct size, expressed as a percentage of the area at risk was significantly smaller in the atorvastatin groups (80 mg/kg) and water (n=10) administered once daily for 3 d and induced myocardial infarction by ligation of a coronary artery for 30 min followed by 3 h of reperfusion. Ischemic area at risk was assessed with blue dye and infarct size by triphenyltetrazolium chloride. Infarct size was expressed as a percentage of the area at risk. Area at risk was comparable among groups. In contrast, infarct size, expressed as a percentage of the area at risk was significantly smaller in the atorvastatin groups (80 mg/kg) and water (n=10) administered once daily for 3 d and induced myocardial infarction by ligation of a coronary artery for 30 min followed by 3 h of reperfusion. Ischemic area at risk was assessed with blue dye and infarct size by triphenyltetrazolium chloride. Infarct size was expressed as a percentage of the area at risk. Area at risk was comparable among groups. In contrast, infarct size, expressed as a percentage of the area at risk was significantly smaller in the atorvastatin groups (80 mg/kg) and water (n=10) administered once daily for 3 d and induced myocardial infarction by ligation of a coronary artery for 30 min followed by 3 h of reperfusion. Ischemic area at risk was assessed with blue dye and infarct size by triphenyltetrazolium chloride. Infarct size was expressed as a percentage of the area at risk. Area at risk was comparable among groups. In contrast, infarct size, expressed as a percentage of the area at risk was significantly smaller in the atorvastatin groups (80 mg/kg) and water (n=10) administered once daily for 3 d and induced myocardial infarction by ligation of a coronary artery for 30 min followed by 3 h of reperfusion. Ischemic area at risk was assessed with blue dye and infarct size by triphenyltetrazolium chloride. Infarct size was expressed as a percentage of the area at risk. Area at risk was comparable among groups. In contrast, infarct size, expressed as a percentage of the area at risk was significantly smaller in the atorvastatin groups (80 mg/kg) and water (n=10) administered once daily for 3 d and induced myocardial infarction by ligation of a coronary artery for 30 min followe...