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## Effects of ACE Inhibition and AT1 Receptor Blockade on Cardiac Ischaemia-Reperfusion Induced Mortality and Cardiac Markers in Rats

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**Abstract:** Many studies have established the therapeutic benefits of angiotensin-converting enzyme (ACE) inhibitors such as reducing reperfusion arrhythmias, and angiotensin II type 1 (AT1) blocker may have similar effects to ACE inhibitors. In this study, it was aimed to compare the effects of an ACE inhibitor captopril and AT1 receptor blocker losartan on death from arrhythmias and biochemical markers such as cardiac troponin T and I (cTnT, cTnI), myoglobin, creatin kinase (CK), creatine kinase-MB isoenzyme (CK-MB) and aspartate aminotransferase (AST) after cardiac ischemia/reperfusion in an in vivo rat model. Study design and methods: sixty four male rats were divided into four groups: Control, captopril (3 mg/kg), losartan (2 mg/kg) and sham. The drugs were administered intravenously 10 min before ischemia under anesthesia. Except for the sham group, the left coronary artery was occluded for 7 min and followed by 10 the min of reperfusion. Blood pressure, heart rate and ECG were monitored throughout the experiment. Biochemical markers were evaluated from the blood samples obtained at the 10th min of reperfusion. Captopril significantly decreased total ventricular fibrillation (VF) and death due to irreversible VF, while losartan did not. cTnT, myoglobin, total CK and CK-MB levels were higher in the control and drug administered groups than in the sham group. cTnT and cTnI levels were significantly increased after captopril administration in comparison with the control group, while losartan administration had no effect. In conclusion, captopril is more effective than losartan, especially for decreasing death from irreversible VF. In addition, captopril may increase the biochemical cardiac markers in the blood during early reperfusion.

**Key Words:** Myocardial Ischemia-Reperfusion, Arrhythmia, Cardiac Marker, Troponin, Captopril, Losartan

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