

Cardioplegic solutions and nitric oxide in coronary artery bypass surgery

Koroner arter baypas cerrahisinde nitrik oksit ve kardiyoplejik solüsyonları

In the study by Karaca et al. (1), published in the current issue of the journal authors have shown that use of blood cardioplegia in the reperfusion period after aortic cross clamp in patients with diabetes mellitus type II undergoing coronary artery bypass grafting surgery was superior to crystalloid solution in terms of myocardial protection as assessed by the degree of nitric oxide (NO) release.

Vascular tone is regulated by vasodilators and vasoconstrictors. Nitric oxide is the primary vasodilator peptide that causes relaxation of vascular smooth muscle, whereas endothelin-1 (ET-1) is the predominant vasoconstrictor peptide that constricts vascular smooth muscle (2).

During coronary artery bypass grafting, the heart is arrested and subjected to ischemia-reperfusion injury. The injury may involve coronary endothelium and NO mechanisms. Many studies have shown that an important feature of ischemia-reperfusion injury is the post-ischemic endothelial dysfunction, which impairs NO release (3, 4). It has been experimentally and clinically shown that this harmful effect can be alleviated by L-arginine administration, which is a nitric oxide precursor (5, 6). However, some studies have shown that the release of NO increases after tepid or normothermic cardiopulmonary bypass showing that NO release is affected by the temperature used (7). Hypothermia decreases NO release whereas tepid or normothermic cardioplegia increases NO release. In this study (1), although the cardiopulmonary bypass temperature was constant, the crystalloid cardioplegia temperature was usually at +4 °C. Because the blood cardioplegia prepared in the other group was warmer than this temperature, this might be the factor affecting differences of NO release between the two groups.

In diabetic patients the endothelial function and mediator release, which affect this function (NO and ET) are different from non-diabetics (8). Sharma et al. (9) found that diabetic patients appear to differ significantly from the non-diabetic population in that there is a significant increase in coronary affluent ET-1 during reperfusion periods after coronary artery bypass grafting without concomitant increases in NO concentrations. On the other hand, Donatelli et al. (10) did not find any difference in ET-1 concentrations between diabetic and non-diabetic patients with coronary artery disease.

Despite developments in surgery, anesthesia, and myocardial protection Type II diabetes, requiring treatment with insulin or oral antidiabetic drugs, is associated with an increased

early and long-term risk of death or acute myocardial infarction after coronary artery bypass grafting (11). It is important that this study showed blood cardioplegia protects endothelial functions better than crystalloid cardioplegia through protection of NO release.

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