4727-A-2204

Effect of glutamate infusion on NT-proBNP after coronary artery bypass grafting in high-risk patients (GLUTAMICS II): A randomized controlled trial

Jonas Holm[1], Gabriele Ferrari[2], Anders Holmgren[3], Farkas Vanky[1], Örjan Friberg[2], Mårten Vidlund[2,4], Rolf Svedjeholm[1]

[1] Department of Thoracic and Vascular Surgery, Dept of Health, Medicine and Caring Sciences, Unit of Cardiovascular Medicine, Linköping University, Sweden.
[2] Department of Cardiothoracic and Vascular Surgery, Faculty of Medicine and Health, Health Care Research Center, Örebro University, Örebro, Sweden
[3] Heart Center and Department of Public Health and Clinical Medicine, Medicine, Umeå University, Sweden
[4] Department of Cardiothoracic Surgery, Sahlgrenska University Hospital, Gothenburg, Sweden

Background: Glutamate plays a key role for the recovery of myocardial metabolism after ischemia. The first GLUTAMICS-trial suggested that glutamate mitigated myocardial dysfunction in high-risk patients after CABG, except in patients with diabetes. We investigated whether glutamate infusion can mitigate rises of NT-proBNP in high-risk patients after CABG.

Methods: A prospective, randomized, double-blind study at four academic hospitals. Patients underwent CABG ± valve procedure and had LVEF 0.30 or EuroSCORE II 3.0. Intravenous infusion of 0.125M L-glutamic acid or saline at 1.65mL/kg/h was started before releasing the aortic cross-clamp and continued for 150 minutes. The primary endpoint was the difference between preoperative and day-3 postoperative plasma NT-proBNP.

Results: We studied 303 patients (age 74±7 years; females 26%, diabetes 47%), 148 receiving glutamate group and 155 controls. Glutamate was associated with a trend towards a reduced primary endpoint (5390 5396 ng/L vs. 6452 5215 ng/L; p=0.086). One patient died 30 days in the glutamate group compared to six controls (0.7% vs 3.9%; p=0.12).

A significant interaction between glutamate and diabetes was found (p=0.03). Among patients without diabetes the primary endpoint (4503 ± 4846 ng/L vs. 6824 ± 5671 ng/L; p=0.007), and the incidence of acute kidney injury (11% vs 29%; p=0.005) was reduced in the glutamate group. These associations remained significant after adjusting for differences in baseline data.

Conclusions: Diverging results in patients with and without diabetes agree with previous observations and suggest that the concept of enhancing post-ischemic myocardial recovery with glutamate merits further evaluation.