Intravenous morphine reduces plasma endothelin 1 concentration through activation of neutral endopeptidase 24.11 in patients with myocardial infarction.

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摘要

Abstract

Study Objective: Morphine has multiple cardiovascular effects, but its action on hydrolysis of endothelin 1 (ET-1) has not been investigated. Methods: We measured plasma levels of ET-1, C-terminal degradation products of ET-1, and neutral endopeptidase 24.11 (NEP) in 68 patients with acute Q-wave myocardial infarction and 29 control subjects. All the patients underwent blood sampling at initial presentation and 10 minutes later. Thirty-six of those with Q-wave myocardial infarction intravenously received 3 mg of morphine immediately after the first sampling (group 1), and the other 32 received the same after the second sampling (group 2). Twenty-four of the control subjects (group 3) were randomized to the protocol of group 1, and the remaining 5 subjects (group 4) were randomized to the protocol of group 2. Results: The plasma ET-1 levels were significantly higher in groups 1 and 2 than in groups 3 and 4 (control groups). In group 1, the ET-1 level decreased significantly at second blood samplings (2.5±0.4 pmol/L versus 1.7±0.6 pmol/L, P<.001), whereas there were no definite changes of ET-1 levels in group 2 (2.5±0.5 pmol/L versus 2.6±0.6 pmol/L, P=not significant). However, the C-terminal degradation products increased significantly at second blood samplings in group 1 (0.8±0.2 pmol/L versus 1.3±0.4 pmol/L, P<.001), whereas there were no definite changes in group 2 (0.9±0.3 pmol/L versus 0.9±0.4 pmol/L, P=not significant). There was no significant difference in baseline NEP activities between groups 1 and 2 (5.02±1.30 nmol/mg protein versus 5.06±1.48 nmol/mg protein, P=not significant). However, the NEP activities at second blood samplings declined significantly in group 1 (9.76±1.76 nmol/mg protein, P<.001 versus baseline), whereas no definite changes were observed in group 2 (5.09±1.62 nmol/mg protein, P=not significant versus baseline). Conclusion: Intravenous morphine may increase NEP activities to accentuate hydrolysis of ET-1. [Wang T-L, Chang H. Intravenous morphine reduces plasma endothelin 1 concentration through activation of

neutral endopeptidase 24.11 in patients with myocardial infarction. Ann Emerg Med. May 2001;37:445-449.]