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LETTER TO THE EDITOR

N-Acetylcysteine is ineffective on short-term neuron-specific enolase levels following coronary artery bypass graft surgery

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To the Editor:

The incidence of neurological complications related to cardiac surgery has remained unchanged, despite increasing age and comorbidity [1]. One of the most important methods for stopping this oxidant-mediated damage is the use of agents containing glutathione. The most investigated among these agents is *N*-acetylcysteine (NAC), which is a precursor of glutathione. Serum neuron-specific enolase (NSE) level was shown to reach its peak during cardiopulmonary bypass (CPB) at the end of rewarming and started to decrease immediately after the end of operation, down to near-normal levels by the second day after surgery, in neurologically intact patients [2]. We aimed to investigate the indirect neuroprotective effects of NAC on NSE levels in patients undergoing elective CPB surgery. Approved by the hospital ethics committee, 40 patients, of ages 30–70 years between March and October 2010 were enrolled in this study. Patients were randomly divided into two groups: group N, NAC infusion group ($n = 20$) and group C, saline infusion group ($n = 20$).

In group N, 20 mg/kg/h NAC infusion was initiated intravenously at 100 ml/h. In group C, intravenous infusion of saline at 100 ml/h was started. Blood samples were collected three times from the jugular bulb catheters of the patients at prespecified times: T_0, before CPB; T_1, at the end of CPB surgery; and T_2, in the sixth postoperative hour. Neurological examination results were recorded on the postoperative third day. However, these results revealed no abnormality.

Patient demographics were similar (Table 1). There was no significant difference between the NSE concentrations of groups at T_0, T_1, and T_2 ($P > 0.05$) (Table 2). A correlation was found between the increase in NSE values at T_2 and CPB time in patients in group N (Pearson: $r = 0.47$, $P < 0.01$).

NSE seems to be the most useful biochemical marker for the evaluation of cognitive disorders after CPB [3]. For the detection of NSE values, we could not find a study in which the blood samples were taken from the jugular bulb, which is anatomically closest to the cerebral circulation. NSE levels of the NAC group were not significantly decreased when compared to those of the control group after CPB. Therefore, it is thought that NAC does not have a neuroprotective effect in the early period after CPB. According to the results of our study, differences between the blood samples collected from the jugular bulb and peripheral blood should be considered, and comparative studies should be done with the blood samples collected simultaneously from jugular bulb and peripheral blood.

In conclusion, it was shown that levels of NSE increased at the end of CPB. According to the results of our study, NAC was not found to be neuroprotective in the early period after CPB surgery. The administration of NAC in

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Table 1 Patient demographics and peroperative parameters (values are means)

Parameter	Group C	Group N	*P* values
Age	52.12 ± 6.95	55.80 ± 8.72	0.299
BSA (m^2)	1.6 ± 0.07	1.8 ± 0.03	0.185
CPB time (min)	77.50 ± 33.57	89.70 ± 18.46	0.186
XCT (min)	52.37 ± 26.96	58.10 ± 28.72	0.56
Preoperative Hct (%)	41.96 ± 3.73	39.45 ± 2.63	0.112
CPB minimum Hct (%)	24.75 ± 3.66	24.59 ± 3.58	0.647

BSA Body surface area, *CPB* cardio-pulmonary bypass, *XCT* cross clamp time, *Hct* hematocrit

Table 2 Neuron-specific enolase (NSE) (ng/ml) and *P* values of the jugular bulb blood samples (values are means)

Time	Group C	Group N
T_0	8.45 ± 3.36	9.12 ± 4.68
T_1	25.46 ± 10.81	26.85 ± 13.16
T_2	13.51 ± 5.23	17.10 ± 4.98

T Time

patients who have undergone CPB should be investigated in a larger series of patients.

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