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Format: Abstract



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N-acetylcysteine administration prevents nonthyroidal illness syndrome in patients with acute myocardial infarction: a randomized clinical trial.

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Author information

Abstract

CONTEXT: The acute phase of the nonthyroidal illness syndrome (NTIS) is characterized by low T3 and high rT3 levels, affecting up to 75% of critically ill patients. Oxidative stress has been implicated as a causative factor of the disturbed peripheral thyroid hormone metabolism.

OBJECTIVE: The objective of the study was to investigate whether N-acetylcysteine (NAC), a potent intracellular antioxidant, can prevent NTIS in patients with acute myocardial infarction.

DESIGN: This was a randomized, multicenter clinical trial.

SETTINGS: Consecutive patients admitted to the emergency and intensive care units of two tertiary hospitals in southern Brazil were recruited. Patients and intervention included 67 patients were randomized to receive NAC or placebo during 48 hours. Baseline characteristics and blood samples for thyroid hormones and oxidative parameters were collected.

MAIN OUTCOME: Variation of serum T3 and rT3 levels was measured.

RESULTS: Baseline characteristics were similar between groups (all P > .05). T3 levels decreased in the placebo group at 12 hours of follow-up (P = .002) but not in NAC-treated patients (P = .10). Baseline rT3 levels were elevated in both groups and decreased over the initial 48 hours in the NAC-treated patients (P = .003) but not in the control group (P = .75). The free T4 and TSH levels were virtually identical between the groups throughout the study period (P > .05). Measurement of total antioxidant status and total carbonyl content demonstrated that oxidative balance was deranged in acute myocardial infarction patients, whereas NAC corrected these alterations (P < .001).

CONCLUSIONS: NAC administration prevents the derangement in thyroid hormone concentrations commonly occurring in the acute phase of acute myocardial infarction, indicating that oxidative stress is involved in the NTIS pathophysiology.

TRIAL REGISTRATION: ClinicalTrials.gov NCT01501110.

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