Multicenter Study Heart Rhythm. 2017 Dec;14(12):1849-1855.
doi: 10.1016/j.hrthm.2017.07.028. Epub 2017 Jul 27.

Association between oxidative stress and atrial fibrillation

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PMID: 28757307 PMCID: PMC5817893 DOI: 10.1016/j.hrthm.2017.07.028

Abstract

Background: Oxidative stress (OS) may be a key mechanism underlying the development of atrial fibrillation (AF) in experimental studies, but data in humans remain limited.

Objective: Systemic OS can be estimated by measurements of circulating levels of the aminothiols including glutathione, cysteine, and their oxidized products. We tested the hypothesis that the redox potentials of glutathione (E_hGSH) and cysteine will be associated with prevalent and incident AF.

Methods: Plasma levels of aminothiols were measured in 1439 patients undergoing coronary angiography, of whom 148 (10.3%) had a diagnosis of AF. After a median follow-up of 6.3 years, 104 of 917 patients (11.5%) developed incident AF. Multivariate logistic regression and Cox regression models were used to determine whether OS markers were independent predictors of prevalent and incident AF after adjustment for traditional risk factors, heart failure, coronary artery disease, and high-sensitivity C-reactive protein level.

Results: For each 10% increase in E_hGSH, the odds of prevalent AF was 30% higher (odds ratio [OR] 1.3; 95% confidence interval [CI] 1.1-1.7; P = .02) and 90% higher (OR 1.9; 95% CI 1.3-2.7; P = .004) when the median was used as a cutoff. The E_hGSH level above the median was more predictive of chronic AF (OR 4.0; 95% CI 1.3-12.9; P = .01) than of paroxysmal AF (OR 1.7; 95% CI 1.1-2.7; P = .03). Each 10% increase in E_hGSH level was associated with a 40% increase in the risk of incident AF (hazard ratio 1.4; 95% CI 1.1-1.7; P = .01).

Conclusion: Increased OS measured by the redox potentials of glutathione is associated with prevalent and incident AF. Therapies that modulate OS need to be investigated to treat and prevent AF.

Keywords: Atrial fibrillation; Glutathione; Inflammation; Oxidative stress; Redox potentials.

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