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The effects of *N*-acetylcysteine on cisplatin-induced changes of cardiodynamic parameters within coronary autoregulation range in isolated rat hearts

Gvozden Rosic[a,*], Dragica Selakovic[a], Jovana Joksimovic[a], Ivan Srejovic[a], Vladimir Zivkovic[a], Nikola Tatalović[c], Zorana Orescanin-Dusic[c], Slobodanka Mitrovic[b], Milena Ilic[b], Vladimir Jakovljevic[a]

[a] Department of Physiology, Faculty of Medical Sciences, University of Kragujevac, Serbia
[b] Department of Pathology, Faculty of Medical Sciences, University of Kragujevac, Serbia
[c] Department of Physiology, Institute for Biological Research, University of Belgrade, Serbia

HIGHLIGHTS

- Chronic cisplatin treatment harmed cardiodynamics and coronary flow in rat heart.
- Chronic cisplatin treatment increased oxidative stress in rat heart.
- Chronic cisplatin treatment induced damage of myocardium and coronary vessels.
- NAC supplementation abolished cisplatin-induced cardiotoxicity.

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ABSTRACT

The aim of this study was to evaluate the effects of chronic NAC administration along with cisplatin on cisplatin-induced cardiotoxicity by means of coronary flow (CF), cardiodynamic parameters, oxidative stress markers and morphological changes in isolated rat heart.

Isolated hearts of Wistar albino rats (divided into four groups: control, cisplatin, NAC and cisplatin + NAC group) were perfused according to Langendorff technique at constant coronary perfusion pressure starting at 50 and gradually increased to 65, 80, 95 and 110 cm H_2O to evaluate cardiodynamic parameters within autoregulation range. Samples of coronary venous effluent (CVE) were collected for determination of CF and biochemical assays, and heart tissue samples for biochemical assays and histopathological examination.

Cisplatin treatment decreased CF and heart rate, and increased left ventricular systolic pressure and maximum left ventricular pressure development rate. Cisplatin increased H_2O_2 and TBARS, but decreased NO_2^- levels in CVE. In tissue samples, cisplatin reduced pathological alterations in myocardium and coronary vessels, with no changes in the amount of total glutathione, as well as in activity of glutathione peroxidase and glutathione reductase.

NAC coadministration, by reducing oxidative damage, attenuated cisplatin-induced changes of cardiodynamic and oxidative stress parameters, as well as morphological changes in myocardium and coronary vasculature.

Abbreviation: NAC, *N*-acetylcysteine.

* Corresponding author at: Department of Physiology, Faculty of Medical Sciences, University of Kragujevac, Serbia Svetozara Markovica 69, 34000 Kragujevac, Serbia.
E-mail addresses: grosic@medf.kg.ac.rs (G. Rosic), dragica984@gmail.com (D. Selakovic), jovana_joksimovic@yahoo.com (J. Joksimovic), ivan_srejovic@hotmail.com (I. Srejovic), vladimirziv@gmail.com (V. Zivkovic), nikola.tatalovic@ibiss.bg.ac.rs (N. Tatalović), zoranaor@ibiss.bg.ac.rs (Z. Orescanin-Dusic), smitrovic@medf.kg.ac.rs (S. Mitrovic), lena.ilic@gmail.com (M. Ilic), drvladakgbg@yahoo.com (V. Jakovljevic).

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