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Data in Brief

Data article

Data on necrotic and apoptotic cell death in acute myocardial ischemia/reperfusion injury: the effects of CaMKII and angiotensin AT_1 receptor inhibition

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ARTICLE INFO

ABSTRACT

Content of particular proteins indicating cellular injury due to apoptosis and necrosis has been investigated in ischemic/reperfused (IR) hearts and ischemic/reperfused hearts treated with CaMKII inhibitor and/or AT_1 receptor inhibitor. This data article provides information in support of the original research article "Oxidative activation of CaMKIIδ in acute myocardial ischemia/reperfusion injury: a role of angiotensin AT_1 receptor-NOX2 signaling axis" [1].

AT$_1$ receptor blockade
Apoptosis
Necrosis

Specifications Table

Subject area	*Biology*
More specific subject area	*Myocardial ischemia/reperfusion injury; Cellular injury; Protein kinase*
Type of data	*Figures, Text*
How data was acquired	*Immunoblotting, Chemiluminescence*
Data format	*Raw, Analyzed*
Experimental factors	*SDS-PAGE, Western blotting in samples of left ventricles of the hearts subjected to ischemia/reperfusion injury with and without angiotensin AT$_1$ receptor inhibitor and CaMKII inhibitor*
Experimental features	*Analysis of certain proteins indicating cellular injury due to apoptosis and necrosis*
Data source location	*Bratislava, Slovak Republic*
Data accessibility	*Data is supplied in this article*

Value of the data

- The data can be useful for other researchers investigating signaling pathways associated with CaMKII and AT$_1$ receptor activation under setting of ischemia/reperfusion in the heart.
- These data also provide significant contribution in understanding of pharmacological properties of AT$_1$ receptor inhibitors, drugs used in patients at high cardiovascular risk.

1. Data

In ischemic/reperfused (IR) group treated with CaMKII inhibitor, but not with AT$_1$ receptor inhibitor, Bcl-2/Bax ratio was significantly increased (Fig. 1) and the expression of caspase-3 and 89 kDa apoptotic fragment of PARP1 was changed by neither CaMKII inhibition nor angiotensin AT$_1$ receptor inhibition (Fig. 2). ~40 kDa necrotic fragment of PARP1 was upregulated in the IR group and no intervention was able to normalize these levels (Fig. 3). In the double-treated IR hearts, the levels of individual apoptotic and necrotic did not differ from those observed in either of mono-treated IR; however, the p~40PARP1/total PARP1 ratio was increased when compared to the IR group treated with CaMKII inhibitor alone.

2. Experimental design, materials and methods

Samples from the untreated and losartan and/or KN-93-treated hearts subjected to ischemia/reperfusion injury were used to examine protein expression by immunoblotting.

Proteins were separated using the SDS-PAGE and transferred to PVDF membrane (EMD Millipore, USA). The primary antibodies used were Bcl-2 (Sigma-Aldrich, USA), Bax (Cell Signaling, USA), PARP1 (Cell Signaling, USA), Caspase-3 (csp-3) p17 (Santa Cruz Biotechnology, USA) and β-actin (Sigma-Aldrich, USA). A HRP-conjugated donkey anti-rabbit antibody (GE Healthcare Life Sciences, UK) was
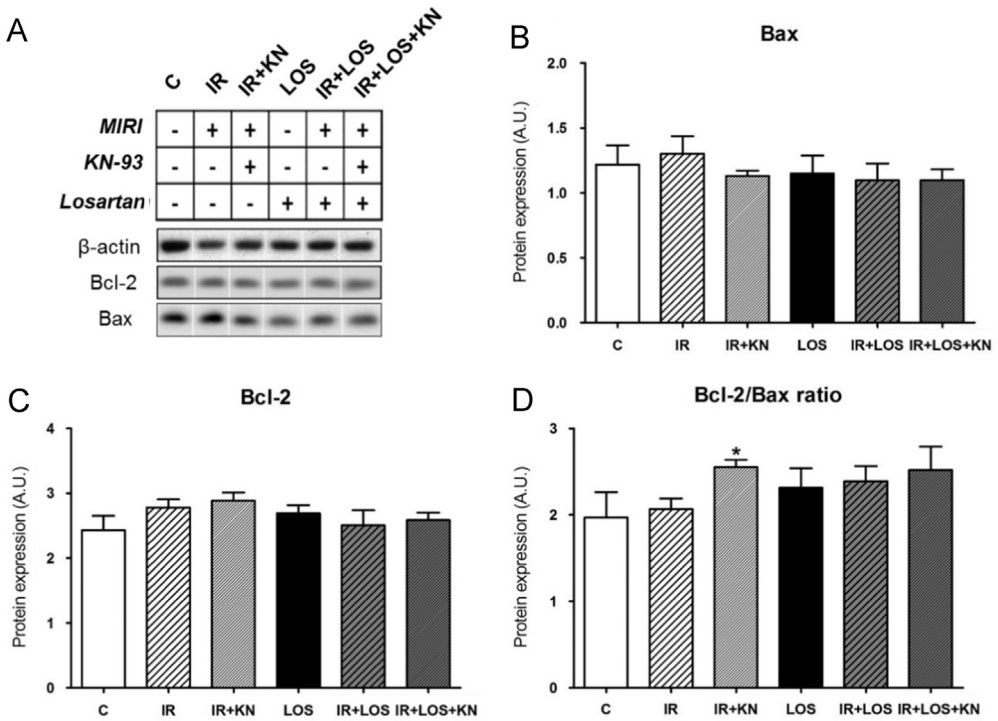
Fig. 1. Expression of Bcl-2 and Bax. (a) Representative western blots (b) Bcl-2 and (c) Bax expression (d) Bcl-2/Bax ratio. The values are expressed as the means $\pm$ S.E.M. ($n=5$–7 hearts per group). *$P < 0.05$ vs. IR group.
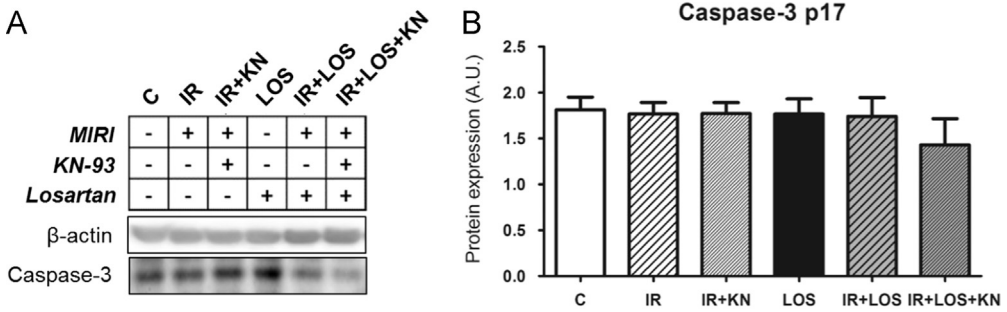

Fig. 2. Expression of cleaved caspase-3. (a) Representative blots (b) content of cleaved caspase-3. The values are expressed as the means $\pm$ S.E.M. ($n=5$–7 hearts per group).
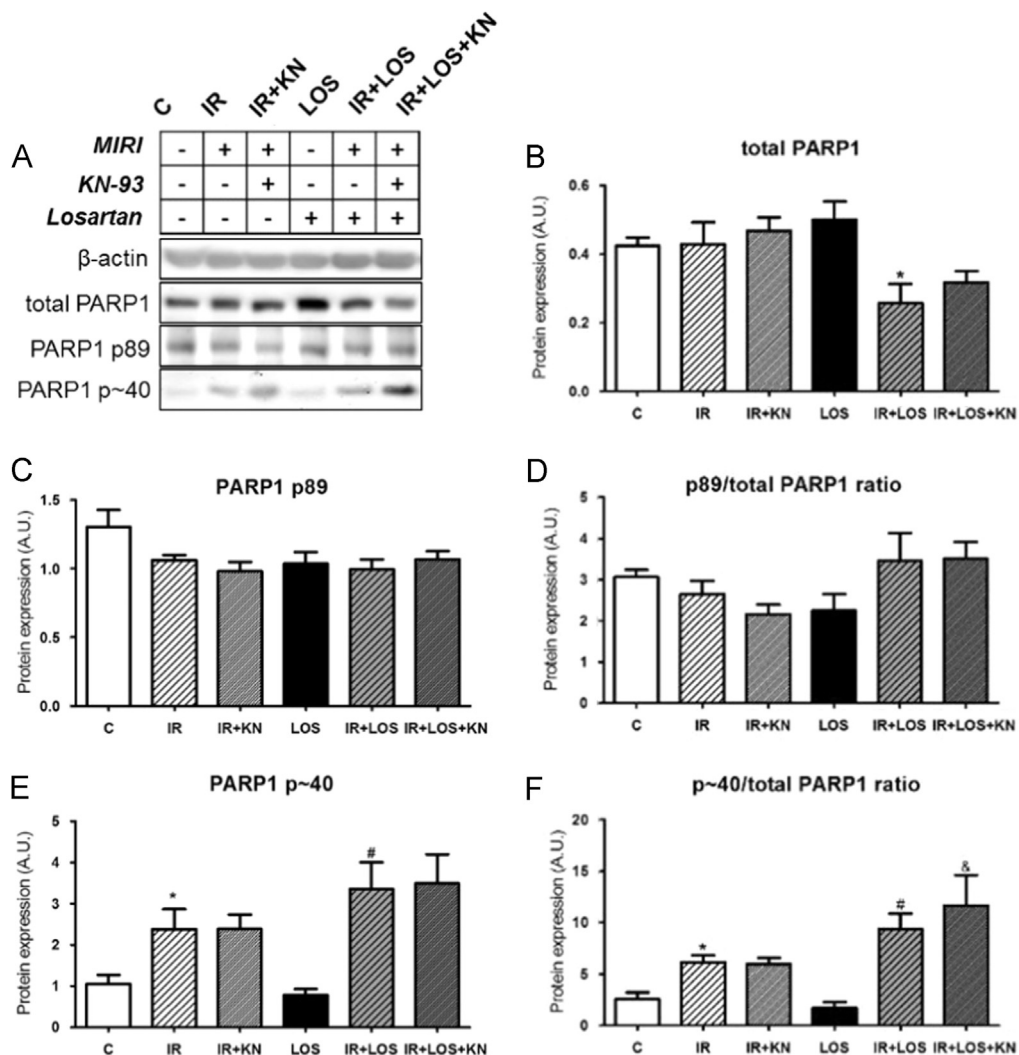
Fig. 3. Expression of total and cleavage fragments of PARP1. (a) Representative blots, (b) expression of total protein, (c) content of apoptotic PARP1 p89 fragment, (d) apoptotic p89 fragment to total PARP1 ratio, (e) necrotic PARP1 $p\sim40$ fragment and (f) ratio of necrotic PARP1 fragment to total PARP1. The values are expressed as the means $\pm$ S.E.M. ($n=5$–7 hearts per group). *$P < 0.05$ vs. C; #$P < 0.05$ vs. LOS group; &$P < 0.05$ vs. IR+KN.

used as the secondary antibody. The signals of proteins were detected using enhanced chemiluminiscence (Luminata, EMD Millipore, USA) and the blots were acquired using the MyECL Imager (Thermo Scientific, USA).

Data are expressed as the means $\pm$ S.E.M. Differences between variables with normal distribution were statistically analyzed by ANOVA with Newman–Keuls post-hoc test. Differences were considered as significant at $P \leq 0.05$.

Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.dib.2016.03.017.

Reference

[1] T. Rajtik, S. Carnicka, A. Szobi, Z. Giricz, J. O-Uchi, V. Hassova, P. Svec, P. Ferdinandy, T. Ravingerova, A. Adameova, Oxidative activation of CaMKIIδ in acute myocardial ischemia/reperfusion injury: a role of angiotensin AT_1 receptor-NOX2 signaling axis, Eur. J. Pharmacol. 771 (2016) 114–122. http://dx.doi.org/10.1016/j.ejphar.2015.12.024.