

Protective role of selective nitric oxide synthase inhibitor for treatment of decompensated hemorrhagic shock in normotensive and hypertensive rats

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Background and Aims: Different vasoactive factors can modulate cardiovascular adaptation to hemorrhagic shock including Nitric Oxide (NO). In this study we investigated the effect of NO synthase inhibitor for treatment of decompensated hemorrhagic shock in normotensive and hypertensive rats.

Methods: twenty four male wistar rats were divided into two groups: normotensive and hypertensive groups. Hypertension was induced by DOCA-Salt method during 8 weeks. Then, the animals were experienced hemorrhagic shock by continuously withdrawing blood until the Mean Arterial Pressure (MAP) reached to 40 mmHg. The animals were maintained to shock state for 120 min. Then, they were randomly assigned to L-NAME-treated (10 mg/kg) and non-treated groups and monitored 60 min after L-NAME infusion. After recovering, the survival time was recorded on first 4h and every 12h up to 72h. Blood samples were taken before and after shock and 60 min after L-NAME administration.

Results: Infusion of L-NAME caused a significant increase of MAP in normotensive animals, however, slightly increased MAP in hypertensive animals. Heart rate did not significantly alter after L-NAME infusion in normotensive and hypertensive groups. Hemorrhage caused a marked increase in serum nitrite levels in both groups ($p < 0.05$). L-NAME treatment significantly reduced serum nitrite concentration in normotensive group ($p < 0.05$), without any change in hypertensive group ($p > 0.05$). All animals who received L-NAME treatment were survived at the end of experiment. 50% of hypertensive animals (three of six animals) were died 4 h after experiment. There were no significant differences in 72h survival rate between L-NAME treated groups.

Conclusions: L-NAME infusion during decompensated hemorrhagic shock has a protective role in improvement of hemodynamic responses and short-term survival rate in normotensive animals.

Keywords: Hypertension; Nitric oxide; Hemorrhagic shock