LETTER TO THE EDITOR

SODIUM NITROPRUSSIDE AS A NITRIC OXIDE DONOR IN A RAT INTESTINAL ISCHEMIA REPERFUSION MODEL: A NOVEL MOLECULAR MECHANISM

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I have read with great interest the article by Emre et al.[1] Their work showed that sodium nitroprusside could be elegantly used to reduce intestinal ischemia reperfusion. This is underscored by a decrease in inflammation as compared to using a placebo. I would like to further discuss this article by introducing a major route through which sodium nitroprusside could suppress inflammation.

The recent focus on ischemia-reperfusion injury has been on the interaction between neutrophils and endothelial cells. Transendothelial migration of neutrophils, with release of reactive oxygen species and cytokines, causes further damage to the injured tissue.[2,3] However, key components in the pathogenesis of reperfusion syndrome include the up-regulation of surface adhesion molecules on the vascular endothelium and their subsequent interaction with activated neutrophils.[4] The most important adhesion protein identified on neutrophils is the integrin lymphocyte function-associated antigen-1 (LFA-1; CD11a/CD18). This is the ligand for intercellular adhesion molecule-1 (ICAM-1), which is expressed on the endothelium. The LFA-1/ICAM-1 interaction is crucial for neutrofils to ingress into inflammatory sites.[5,6] Sodium nitroprusside down-regulates ICAM-1 and LFA-1 expression, and interferes with the ICAM-1–LFA-1 interaction by binding to LFA-1.[7,8] This important mechanism should be borne in mind as the major mechanism for sodium nitroprusside-induced inhibition of neutrophil activity.

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