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Hyperthermic preconditioning

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Keywords

Heat shock proteins, hyperthermia, peritonitis, preconditioning

Comments

An interesting experimental study examining the protective effects of hyperthermia preconditioning in a peritonitis animal model. The mechanism of this phenomenon is still far from understood and this study provides some evidence that the immune system functions better following preconditioning. However preconditioning at present remains a difficult concept to achieve in clinical practice since we are unable to preempt the noxious insult in the majority of situations.

Introduction

Heat shock proteins (HSPs) are endogenous factors which are induced by a variety of noxious stimuli and have important cytoprotective effects. HSP70 has been shown to be responsible for ischaemic preconditioning of the heart, and other HSPs protect cells from cytotoxic mediators released in septic shock. However, the mechanism responsible for this protection awaits elucidation. The effects and mechanisms involved in hyperthermic (HT) preconditioning for sepsis are explored in this paper.

Aims

To investigate whether HT preconditioning protects against peritonitis by actions on the immune system.

Methods

Male Sprague-Dawley rats were divided into five groups; control group, HT group (subjected to 42^0C, 15 mins and then peritonitis induced 8 h later), Normothermic (NT) group (subjected to NT

followed by induction of peritonitis 8 h later), and two corresponding sham laparotomy groups. Severity of peritonitis, measurement of free oxygen radicals in peritoneal fluid, serum TNF-α and percentages of $CD4^+$, $CD8^+$, $CD4^+CD56^+$, $CD8^+$, $CD11b^+$, $NK^+$and B cells were measured.

Results

Severity of peritonitis was graded as moderate and severe in the HT and NT group respectively, and although seven day survival was improved in the HT rats, it did not reach statistical significance. Although free oxygen radicals increased in the peritoneal fluid of the HT group with peritonitis when compared to the sham HT group, this was not statistically significant when compared to the NT groups. HT prevented the increase in $CD11b^+$ and decrease in $CD4^+$ and B cells, found in the NT rats with peritonitis. No significant effects were found for other lymphocyte phenotypes or TNF-α.

Discussion

Peritonitis suppresses cell mediated and humoral immunity. However, in this study the increase in $CD11b^+$ (an adhesion molecule induced by inflammation) and decrease in $CD4^+$ (plays a protective role in inflammation) and B cells, were prevented by hyperthermic preconditioning. Thus the protective effect of HT preconditioning may be in part due to direct effects on the immune system. HT preconditioning does not appear does not appear to influence cytokine release.

References

1. Ozveri ES, Bekraki A, Cingi A, Yuksel M, Demiralp EE, Yegen BC, Aktan AO: The effect of hyperthermic preconditioning on the immune system in rat peritonitis. Intensive Care Med. 1999, 25: 1155-1159.