Blood Coagul Fibrinolysis. 1993 Dec;4(6):999-1005. Related Articles, Links

The role of endotoxin immunity, neutrophil degranulation and contact activation in the pathogenesis of post-operative organ dysfunction.

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Gut mucosal hypoperfusion is associated with a poor outcome following major surgery but the pathogenetic mechanisms remain poorly understood. We have examined the relationship between gut mucosal hypoperfusion, endotoxin core antibodies (EndoCAb), neutrophil elastase alpha-1 antitrypsin complexes (NE) and components of the contact system during elective major surgery. Of the 26 patients studied 16 developed gut mucosal hypoperfusion (pHi < 7.32) by the end of surgery; of these four developed multiple organ failure (MOF) and three subsequently died. In this group there was a significant rise in NE ($P < 0.005$) and significant reductions in components of the contact system (factor XII, antithrombin III, prekallikrein and C1-inhibitor; $P < 0.001$) from immediately before surgery to 24 h later. Ten patients maintained gut mucosal perfusion (pHi > or = 7.32); none of these developed life threatening complications. In this group there was no significant increase in NE and, although there were significant reductions in some components of the contact system ($P < 0.01$), levels of C1-INH were not reduced. All patients demonstrated a significant reduction in both IgG and IgM EndoCAbs ($P <$ or $= 0.005$) indicating exposure to endotoxin. However, the group that maintained gut mucosal perfusion had significantly higher IgG EndoCAb levels at baseline and 24 h ($P <$ or $= 0.005$). These data suggest that all patients were exposed to endotoxin and that high levels of anti-endotoxin antibodies may contribute to the prevention of endotoxin-induced contact activation, neutrophil degranulation and gut mucosal hypoperfusion occurring during major surgery and thus reduce the likelihood of the development of post-operative MOF.