Interleukin-10 controls the protective effects of circulating microparticles from patients with septic shock on tissue-engineered vascular media

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Résumé en anglais	During sepsis, inflammation can be orchestrated by the interaction between circulating and vascular cells that, under activation, release MPs (microparticles). Previously, we reported that increased circulating MPs in patients with sepsis play a pivotal role in ex vivo vascular function suggesting that they are protective against vascular hyporeactivity. The present study was designed to investigate the effects of MPs from patients with sepsis on the contractile response of TEVM (tissue-engineered vascular media). TEVM that were composed only of a media layer were produced by tissue engineering from human arterial SMCs (smooth muscle cells) isolated from umbilical cords. TEVM was incubated with MPs isolated from whole blood of 16 patients with sepsis. TEVM were incubated for 24 h with MPs and used for the study of vascular contraction, direct measurements of NO and O2- (superoxide anion) production by EPR and quantification of mRNA cytokine expression. MPs from patients with sepsis increased contraction induced by histamine in TEVM. This effect was not associated with inflammation, neither linked to the activation of NF-kappaB (nuclear factor kappaB) pathway nor to the increase in iNOS (inducible NO synthase) and COX (cyclo-oxygenase)-2 expression. In contrast, mRNA expression of IL (interleukin)-10 was enhanced. Then, we investigated the effect of IL-10 on vascular hyporeactivity induced by LPS (lipopolysaccharide). Although IL-10 treatment did not modify the contractile response in TEVM by itself, this interleukin restored contraction in LPS-treated TEVM. In addition, IL-10 treatment both prevented vascular hyporeactivity induced by LPS injection in mice and improved survival of LPS-injected mice. These findings show an association between the capacity of MPs from patients with sepsis to restore vascular hyporeactivity induced by LPS and their ability to increase IL-10 in the tissue-engineered blood vessel model.
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