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The effects of dexmedetomidine on human neutrophil apoptosis

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ABSTRACT

The purpose of the present study was to evaluate the effect of dexmedetomidine, used as a sedative in the intensive care unit, on human neutrophil apoptosis and superoxide production in vitro. Neutrophils from healthy volunteers were incubated in different concentrations of dexmedetomidine (1, 10 and 100 ng/mL). Apoptosis was assessed by Hoechst 33342 staining, caspase activities and loss of mitochondrial transmembrane potential (MTP). Superoxide production was determined by the WST-1 assay. After 24 h of incubation, dexmedetomidine accelerated neutrophil apoptosis in a dose-dependent manner and 100 μM yohimbine did not inhibit the apoptosis. Treatment with 100 ng/mL of dexmedetomidine significantly enhanced the activation of caspases-3/7, -8 and -9, and also markedly increased the number of neutrophils with decreased MTP. At 24 h, the suppression of superoxide production was dependent on dexmedetomidine concentrations. However, a clinically relevant concentration (1 ng/mL) of dexmedetomidine did not affect neutrophil apoptosis and superoxide production. These results suggest that high doses of dexmedetomidine induce apoptosis without α_2-adrenoceptors stimulus and inhibit superoxide production after long-term incubation. The mechanisms of dexmedetomidine-induced apoptosis are associated with the caspase cascade and loss of MTP.

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