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Lidocaine-induced apoptosis and necrosis in U937 cells depending on its dosage

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ABSTRACT

Local anesthetics are known to affect a variety of cellular responses other than the action of anesthetics through the Na^+ channel blockade. In this study, we examined the effect of a common local anesthetic lidocaine on the cellular activity and viability of human histiocytic lymphoma U937 cells. The cellular activity and viability were assessed by WST-1 reduction activity and trypan blue exclusion test, respectively. Induction of apoptosis was monitored by DNA ladder formation, reduction of mitochondrial transmembrane potential ($\Delta\Psi m$), caspase-3 activity and nuclear morphology. Lidocaine at concentrations below 12 mM induced apoptosis characterized by DNA fragmentation and chromatin condensation dose- and time-dependently. A pan-caspase inhibitor and a caspase-3 inhibitor blocked DNA ladder formation followed by the reduction of cell death. However, the caspase inhibitors did not affect the $\Delta\Psi m$, but cyclosporin A inhibited the collapse of $\Delta\Psi m$ followed by a reduction of cell death. Lidocaine-induced apoptosis was mitochondria- and caspase-dependent, but the collapse of $\Delta\Psi m$ was independent of caspase activation. At concentrations above 15 mM, lidocaine induced necrosis with early disruption of membrane integrity. These results indicate that lidocaine induced apoptosis and necrosis in U937 cells depending on its dosage.

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