Remifentanil and hydrogen peroxide-induced oxidative stress on human keratinocytes

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Background

Many patients suffer from wound healing problems after surgery. During wound healing process, reactive oxygen species (ROS) are produced and overfull ROS are detrimental to wound repair because of their high reactivity. Remifentanil can decrease the production of ROS and inflammatory response. Therefore, we investigated the effects of remifentanil on human keratinocytes (HaCaT cell) during H2O2-induced oxidative stress and whether this effect has connection with autophagy.

Materials and Method

Human keratinocytes (HaCaT cell line) were randomly assigned to 4 groups: control group, cells incubated in normoxia without remifentanil; H2O2 group, cells exposed to H2O2 (300 μM) for 2 h; remifentanil pretreatment (RPC) + H2O2 group, cells pretreated with remifentanil (1 ng/mL) for 2 h and exposed to H2O2; and 3-methyladenine (MA) + RPC + H2O2 group, cells pretreated with 3-MA (1 mM) for 1 h and exposed to remifentanil, H2O2. MTT assay was conducted to analyze cell viability. Apoptosis was measured via Hoechst staining. A wound healing assay was used to measure cell migration. The role of autophagy was ascertained using autophagosome staining.

Results

Cell viability significantly decreased in H2O2 group and improved by RPC. RPC effectively decreased H2O2-induced apoptosis in HaCaT cell. Remifentanil restore cell proliferation and migration injured by H2O2. However, 3-MA inhibited the protective effect of remifentanil at cell apoptosis.

Conclusions

Cell viability significantly decreased in H2O2 group and improved by RPC. RPC effectively decreased H2O2-induced apoptosis in HaCaT cell. Remifentanil restore cell proliferation and migration injured by H2O2. However, 3-MA inhibited the protective effect of remifentanil at cell apoptosis.