Remifentanyl and hydrogen peroxide-induced oxidative stress on human keratinocytes

Eun-Jung Kim¹, Ji-Young Yoon¹, Cheul-Hong Kim¹, Sang-Hoon Shin², Yong-Deok Kim², Ji-Uk Yoon³

¹Pusan National University Dental Hospital, Dept of Anaesthesiology, Yangsan, Korea, Republic of,

²Pusan National University Dental Hospital, Dept of Surgery, Yangsan, Korea, Republic of

³Pusan National University Yangsan Hospital, Dept of Anaesthesiology & Pain Medicine, Yangsan, Korea, Republic of,

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. Remifentanyl can decrease the production of ROS and inflammatory response. Therefore, we investigated the effects of remifentanyl 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 remifentanyl; H₂O₂ group, cells exposed to H₂O₂ (300 μ M) for 2 h; remifentanyl pretreatment (RPC) + H₂O₂ group, cells pretreated with remifentanyl (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 remifentanyl, 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

Fig. 3. Cell apoptosis was evaluated by apoptotic nuclei staining with Hoechst 33342

Fig. 4. Acridine orange staining of autophagosomes

Fig. 6. Monodansylcadaverine staining for autophagic vacuoles

Conclusions

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