Investigation of the Protective Effect on the Rat Lung Tissue of Enoxaparin and Ticagrelor Pretreatment Against Ischemia-Reperfusion Injury

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Abstract
This study was conducted to reveal possible protective effects of ticagrelor and enoxaparin pretreatment on the lung tissue to ischemia-reperfusion (IR)-induced injury at light microscopic level. Wistar albino rats (n=36) were randomized into four groups as follows: group-1 (sham-control), group-2 (control-saline+IR), group-3 (ticagrelor+IR), group-4 (enoxaparin+IR). Normal saline, ticagrelor and enoxaparin were administrated, respectively in group-2, group-3 and group-4 before the ischemic period. In the 2th-4th groups, IR injury was induced by clamping the aorta infrarenally for 2 hs, followed by 4 h of reperfusion except group-1. After sacrifice of animals, lungs were removed and fixed with %10 neutral buffered formalin for histological examinations in all rats. Lung tissues were evaluated in paraffin sections with stained H&E. Apoptosis was evaluated by caspase-3 immunoreactivity. Semiquantitative analyze for caspase-3 reaction performed as positive staining cells distribution in each section of the lung. Data were statistically analyzed by SPSS programme. H&E staining slides showed that lung tissues were normal histological structure in group-1. Group-2 showed disorganised epithelial cells, hemorrhage, inflammatory cell infiltration in alveolar wall. Lung sections of treatment groups were more better than group-2. In group-2, apoptotic cells were significantly higher than sham-control and treatment groups (p<0.001). Apoptotic cells were noticeable in the sections of group-2, and these cells were lower in the treatment groups. In enoxaparin+IR group, caspase-3 immunostaining was lower than ticagrelor+IR group. The histological appearance was similar to that of normal lung architecture in group-3 and group-4. Evaluation with caspase-3 IHC showed that pre-treatment with enoxaparin gave better healthy by light microscopy.

Keywords: ischemia-reperfusion, rat lung tissue, caspase-3, apoptosis.