**A potential role of endothelins in rheumatic mitral stenotic valves**


Background: The genesis of rheumatic mitral valve stenosis has been correlated with the action of endothelin subtype 1 (ET-1) and its receptors (ETRA and ETRB). We aim to analyze, through real time PCR (polymerase chain reaction), the gene expression of ET-1 in rheumatic mitral valves. Methods: This is a randomized and experimental study. We collected ten mitral valves in two hospitals of Aracaju, Brazil. Each valve had suffered fragmentation, originating three segments that were subjected to extraction of total RNA. Then, each sample of total RNA was quantified by spectrophotometry. Through reverse transcription reaction, the total cDNA was obtained from each sample and then, the technique of amplification of target fragment by real time PCR with the quantification of each sample was performed. Data were tabulated and analyzed by CFX96 Real Time System (BIORAD), and the calculations of quantitation of each sample was performed. Data were tabulated and performed every 15 min from the beginning of the protocol.

Endothelin-1 (ET-1) is a mediator of vascular inflammation, cell proliferation, and fibrosis, and is, in addition, a potent vasoconstrictor. Previously, treatment with ET-1 antagonists was shown to reduce pulmonary vascular leak and inflammation in several models of lung injuries as well as in experimental acute respiratory distress syndrome (ARDS). The current study used an experimental model of lavage-induced surfactant depleted ARDS, to investigate the circulatory and pulmonary levels of ET-1. In addition, we also tested the effects of open endotracheal suctioning (OES) [a known inducer of alveolar de-recruitment] and the post-OES hyperinflation (HI) (performed to recover the alveolar de-recruitment using bagging) on ET-1 levels. Briefly, 18 Japanese White Rabbits were anesthetized and intubated. Normal saline was instilled into the lung and washed mildly. After instillation, rabbits were ventilated at definite settings; total OES and HI duration was for 3 h and performed every 15 min from the beginning of the protocol. Circulatory levels of ET-1 were found to have decreased from baseline (3.26 ± 1.01) to after lavage (1.82 ± 1.59, p = 0.003), without any significant change in mean blood pressure (baseline 112 ± 13.8; after lavage 113 ± 12.5, p = 0.848). In contrast, pulmonary ET-1 levels were almost unchanged irrespective of the induction of lavage-induced lung injury from baseline. It must be noted that, in lung injury state, PaO₂ was significantly decreased, having a parallel relation with ET-1. Either OES or HI failed to recover the down-regulated circulatory ET-1 level. For now, we cannot rule out the mechanism of differential pattern of circulatory ET-1 levels observed in the current model compared to other ARDS models.

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**Blood pressure independent downregulation of plasma endothelin-1 levels in a lavage-induced surfactant depleted rabbit ARDS model:**

Effects of various respiratory maneuvers on endothelin-1 levels

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Methods: This is a randomized and experimental study. We collected ten mitral valves in two hospitals of Aracaju, Brazil. Each valve had suffered fragmentation, originating three segments that were subjected to extraction of total RNA. Then, each sample of total RNA was quantified by spectrophotometry. Through reverse transcription reaction, the total cDNA was obtained from each sample and then, the technique of amplification of target fragment by real time PCR with the quantification of each sample was performed. Data were tabulated and analyzed by CFX96 Real Time System (BIORAD), and the calculations of quantitation of each sample was performed. Data were tabulated and performed every 15 min from the beginning of the protocol.

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