

Involvement of the AT1 Receptor in the Pathophysiology of the Respiratory Failure Induced by Scorpion Venom

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Abstract

Background: Renin-Angiotensin System (RAS) is related to cardiovascular diseases. It is also involved in the pathogenesis of pulmonary dysfunction, through the induction of proinflammatory mediators in lungs.

Respiratory failure is a life-threatening complication in scorpion envenomation, a public health problem in Algeria.

Our aim was to explore the role of angiotensin type I receptors (AT1R) in the respiratory dysfunction induced in an envenomed model with *Androctonus australis Hector (Aah)* venom, in the presence or absence of an AT1R antagonist.

Methods: Respiratory failure is assessed by the arterial blood gas analysis (pO₂ and pCO₂, sO₂, hemoglobin and HCO₃ concentration and pH), by neutrophil infiltration (myeloperoxidase activity) and oxidative stress markers (Nitrites, MDA, H₂O₂, catalase and GSH), in lungs and the pulmonary artery.

Results: A severe respiratory dysfunction, including a marked perturbation of the oxygenation parameters and the acid base balance, reflecting a state of hypoxia and acidosis were induced by the venom. An important infiltration of neutrophils into the tissues and the generation of ROS with the alteration of the antioxidant system were found.

Inhibition of the AT1R before envenomation revealed the recovery of the respiratory parameters in sera. The reduction of inflammatory cells infiltration and the prevention of the redox status imbalance were also recorded.

Conclusion: These results indicate the involvement of Ang II through the AT1R in the respiratory failure induced by *Aah*'s venom. However, further studies are still required to understand the RAS role in the pulmonary injury developed in the envenoming syndrome, a serious public health surgery that must be immediately treated.

Key words: envenomation, respiratory failure, pulmonary artery, angiotensin II.

1. Conflict of interest statement

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2. Authors' biography

No Biography

3. References

No references