**INTRODUCTION**

Fat embolism (FE) by IV injection of triolein (T) causes vasculitis, septal inflammation, and fibrosis in rat lungs at 48 hours that is mitigated by two drugs acting on the renin-angiotensin system (RAS): captopril and losartan (1). The histopathologic changes found at this time are also affected by administration of the direct renin inhibitor aliskiren; however an increase in mast cells is noted (2). This study examines the impact of captopril and losartan on the observed number of mast cells in the rat lung exposed to T.

**METHODS**

Sprague-Dawley rats (280-300g) were treated with 0.2 ml IV T (N=31) or saline (N=7). One hour later 0.2 ml of saline (N=7), aliskiren (50 mg/kg N=6, 100 mg/kg N=6), captopril (150 mg/kg N=6), or losartan (10 mg/kg N=6) were administered IV in subgroups. 48 hours later necropsy examination was performed on all subjects following isofluorane anesthesia. Lung tissue was collected with 10% formalin fixation, H&E, Masson trichrome, and CD117 immunostains following. Two blinded pathologists then took ten random photographs of all slides at 400x for mast cell counts.

**RESULTS**

As previously reported, T administration resulted in severe histopathological damage partially ameliorated by all RAS drugs (1-3) with associated increase in mast cells (3). However while both aliskiren and losartan decreased observed mast cell numbers, captopril did not.

**FIGURES**

![Saline Control](Saline Control)

![Saline + T](Saline + T)

![T + Alisk 50](T + Alisk 50)

![T + Alisk 100](T + Alisk 100)

![T + Captopril](T + Captopril)

![T + Losartan](T + Losartan)

**CONCLUSION**

Mast cells may be involved in the pathological damage found after FE as suggested by their increased numbers observed in this rat model. The observed change with RAS modifying agents implicates the RAS system in the change noted. However, with the heterogeneity of tissue response provoked by captopril the suggestion of other pathways/peptides involved in the pathologic response to FE is evident.

**Credits/Disclosures/References**


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