Lack of Effect of the Direct Renin Inhibitor Aliskiren on Cardiac Mast Cells and Macrophages in an Acute Model of Fat Embolism in Rats

VanDillen M¹, Arif D¹, Siddiqi A¹, Ponnapreddy R Fotouhi A¹, Tappeta K, Khan S¹, Hamidpour S¹, Poisner A², Molteni A¹
¹University of Missouri-Kansas City School of Medicine, Kansas City, MO
²University of Kansas Medical Center, Kansas City, KS

INTRODUCTION

Fat embolism (FE) induced by Triolein injection causes both acute and chronic lung injury in rats, and the histopathological damage is not limited to the lungs but also involves the heart (1). The renin-angiotensin system (RAS) plays a role in the pathogenesis of such damage. Captopril and losartan treatment prevented such damage both in lungs and heart at 10 weeks after Triolein injection (2). More recently we reported that the renin inhibitor aliskiren induced the same protective effect in the lungs (3) and in the heart of the same animals either alone or Triolein + aliskiren treatments. In contrast to the lungs, where we have observed two types of macrophages, only one type was present in the heart. The lack of change in mast cells and macrophages in the heart and the occurrence of no fibrosis with only a modest change in SMA at this early time suggests that the occurrence of no fibrosis with only a modest change in SMA at 48 hours after Triolein, there were no alterations in mast cells or macrophages in the heart of the same animals either after Triolein alone or Triolein + aliskiren treatments. In contrast to the lungs, where we have observed two types of macrophages, only one type was present in the heart. The lack of change in mast cells and macrophages in the heart and the occurrence of no fibrosis with only a modest change in SMA at this early time suggests that such cellular response for the heart is happening only in a late phase following fat embolism.

METHODS

Twenty-two rats were divided into controls (n=4, Group A) and three FE groups (n=6, groups B, C & D). FE induction was done with Triolein 0.2 ml i.v at 0 hours, and an hour later animals received 0.2 ml i.p saline (Groups A&B), aliskiren at 50 mg/kg (group C) or at 100 mg/kg (Group D). Rats were euthanized at 48 hours and hearts were fixed and stained with H&E for general histopathology evaluation, Masson’s Trichrome (MT) for collagen presence and distribution, and SMA for myofibroblasts incidence. Mast cells were stained with CD 117 and macrophages with CD 68.

RESULTS

The only damage observed in the experiment was a statistical significant increase (P<0.05) in SMA staining in the adventia of the coronary arteries, in the myocardium of the rats injected with Triolein + saline vs the controls. No other significant difference was observed for all the other parameters. Mast cells and macrophages did not show any statistical difference among the four groups. The macrophages did not show the two types of cells that we have observed in the lungs, one with large cytoplasm with small vacuoles and the second with small compact cytoplasm (6).

SUMMARY

Despite the significant cellular response in the lungs at 48 hours after Triolein, there were no alterations in mast cells or macrophages in the heart of the same animals either after Triolein alone or Triolein + aliskiren treatments. In contrast to the lungs, where we have observed two types of macrophages, only one type was present in the heart. The lack of change in mast cells and macrophages in the heart and the occurrence of no fibrosis with only a modest change in SMA at this early time suggests that such cellular response for the heart is happening only in a late phase following fat embolism.

CREDITS/DISCLOSURE/REFERENCES


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