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Short Communication

Hearts, Macrophages and Mast Cells in a Rat Model of Fat Embolism

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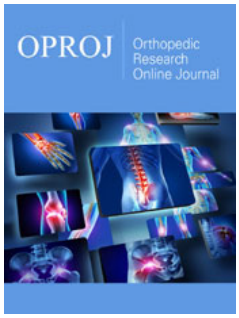

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Introduction

Fat embolism induced in rats lungs, by intravenous injections of Triolein (T), leads to severe pulmonary inflammation, fibrosis, and vasculitis as early as 48 hours that persists at 10 weeks post injections [1,2]. Similar histopathological changes were not observed in the hearts of these rodents at the same interval times [3]. Mast cells and macrophages make a significant contribution to inflammation. To determine whether pulmonary emboli lead to alteration and different expression of the macrophages and mast cells, these markers were examined in the lungs and the hearts of triolein injected rodents.

Methodology

24 Sprague Dawley Rats were divided into four groups and intravenously injected in the tail with 0.2ml of T or 0.2ml of saline. Six animals of each group were collected 48 hours or ten weeks after injections, fixed in formalin and stained with hematoxylin eosin (H&E), trichrome and immunostained with antibodies to CD117 and CD68 to identify macrophages and mast cells. Stained slides were evaluated by two pathologists blind to the slide's identity at 400x magnification. Cells were counted, histopathologically described, photographed and the statistical significance evaluated using graph pad software.

Results

As previously reported the H and E staining of the lungs in the T injected rats induced severe septal infiltration of neutrophils and lymphocytes, alveolar emphysema, and vasculitis and arterial thickening [1,2]. The quantitation of the severity of the damage was statistically significant when compared to that of the lungs of the rats injected with saline. Trichrome staining also induced a very severe expression of collagen, statistically significant in the lungs of the T injected animals when compared to the lungs of those ones injected with saline.

No significant increases of the inflammatory response was evidenced in the hearts of T injected animals; and collagen expression was not altered compared to saline counterparts [3]. Staining with CD117 and CD68 to identify mast cells and macrophages, showed these cells were significantly increased in the lungs of the T treated rats. In these lungs both cell types were located close to the septa and two distinct types of macrophages were identified, a normal size population and a second cell type with larger dimensions. Saline treated rats only showed small size macrophages and smaller number of both inflammatory cells.

The hearts of the T treated animals showed only macrophages of small dimension. Some animals had few macrophages between myocardial fibers or close to their pericardial

structure, while others showed only very few cells and four of them did not show any cells. The percentage of stained macrophages was very similar in the slides of both T treated and saline treated groups with no statistically significant differences. Hearts stained by CD117 did not show presence of mast cells in any group of animals killed at both interval times.

Conclusion

These findings confirm previous studies exhibiting an inflammatory response with macrophages and mast cells in lungs, but not in the heart in response to T administration. Macrophages and mast cells secrete components of the renin angiotensin system (RAS) and those components play a relevant role in the development of pulmonary vasculitis, hypertension and fibrosis [3,4]. Administration of drugs such as captopril, losartan and aliskiren which interfere with different components of the RA system prevent the development of the pulmonary damage [5,6]. We suggest staining of renin in hearts of T treated rodents and compare its intensity and amounts to those found in the lungs of the same animals.

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