Fat Emboli in the Aorta in a Model of Fat Embolism Syndrome

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Introduction

Fat embolism (FE) induced by intravenous Triolein (T) injection in an experimental rat model has been shown to result in severe pulmonary damage, septal and arterial inflammation, and eventually diffused fibrosis (1,2). This response appears to show a peak 48 hours after the administration. Fat droplets were also observed in the brains and the retina of the same rats. The current study was carried out to determine if FE in our model was also associated with fat droplets in the aorta at 48 hours.

Methodology

26 Sprague-Dawley rats (250-330 grams) were divided into two groups (N=13 each) receiving either 0.2 mg intravenous T (0.2 mL) or the same volume of saline. All animals were euthanized 48 hours later. Aortas were isolated, snap frozen and processed for cryosectioning. Sections were stained for Oil-red O and imaged at 100x magnification and photographed by two pathologists unaware of their identity. Two photographs were taken for each aorta section. The fat droplets in each photograph were quantitatively by ImageJ (NIH program) evaluated by one independent observer and statistically compared utilizing a student T-test.

References/Support

1. Mc Iff T. E. et al. J. Orthoped. Res. 2010, 28 (2): 191-197
2. Mc Iff T. E. et al. J. of Trauma. 2011, 70 (5): 1186-1191

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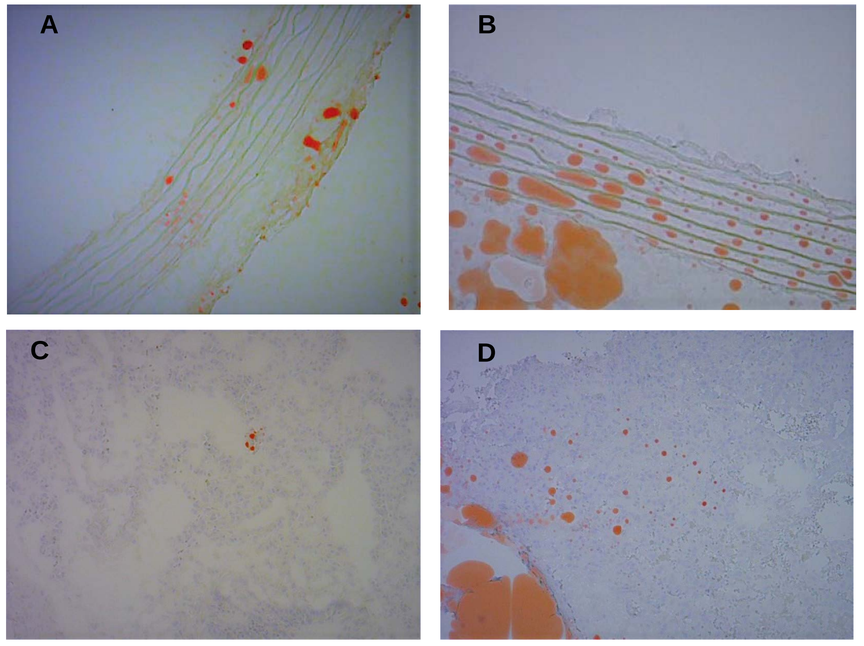

Figures A & B: Representative photographs of aorta of rats injected (A) with saline, and (B) with triolein. A larger number of fat droplets is evident in the media and the adventitia of triolein injected animals.

Figures C & D: Representative photographs of lung sections of rats injected (C) with saline, and (D) with triolein. Small number of fat droplets is evident in a lung section of a saline injected animal, (D) Triolein injection induced large numbers of fat droplets varying in their size and spread in the parenchyma.

Stain: Oil Red O, magnification 100x

Results

The aortas of the control group exhibited fat droplets in the media of 4 of the 8 vessels considered. These droplets were present in only limited sections of the vessels, were small in size, and close to the fat plaques of the nearby adventitia. The adventitia plaques were present in the aorta of all the rats including those without droplets in the media. Those fat droplets were mostly small and similar in their dimension. On the other hand, 7 of the 8 T treated rats exhibited a variable but usually large number of plaques, often involving the entire section of the aorta. Adventitia plaques were similar in number and shape to those of the controls and were present in all the sections. Few large droplets mostly close to the media were also observed. The difference of droplets presence in the two groups was quantitatively significant by ImageJ evaluation with an average of 10.625 +/- 1.369 SEM for the T injected rats versus 2.50 +/- 0.7906 SEM for the controls ($P<0.0009$). No statistically significant difference was seen for the adventitial plaques of the two groups.

Conclusion

While fat deposition is a nonspecific finding, the aorta sections of T injected rats showed an increased presence of fat in the media. Such an increase was also seen in lungs and brains. While FE appears to induce a marked inflammatory response within the lungs already at 48 hours, the aortas, like the brains do not seem to reciprocate such lung inflammatory development. Therefore, at 48 hours post injection, the aorta appears more like the brain sections and it is histopathologically different from pulmonary vessels showing no signs of inflammation. Further studies are necessary to ascertain whether the inflammatory changes may appear in the aortas over a longer period of time.

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