Fat Emboli in the Rat Brain in a Model of Fat Embolism Syndrome

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Introduction

Fat emboli released into the microcirculation after a number of clinical procedures including orthopedic surgery, liposuction, bone marrow transplant, caesarian section, etc. can lead to fat embolism (FE). Both mechanical damage from obstructed vasculature and biochemical damage from inflammatory pathways have been proposed to contribute to the clinical manifestations of fat embolism syndrome (FES). We have investigated the cellular and biochemical consequences of fat emboli in a rat model. We have previously shown that fat embolism resulting from intravenous triolein (T) injection in an experimental rat model has been shown to induce severe pulmonary damage, septal and arterial inflammation, and eventually fibrosis (1). This tissue response appears to feature a peak at 48 hours(2). Fat droplets have previously been reported in the brains of patients suffering severe FES (3,4). The current study was carried out to determine if FE in our model was also associated with fat droplets in the brain 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 or saline. All animals were euthanized under isoflurane anesthesia 48 hours later. Lungs and brains were isolated, snap-frozen, and processed for cryosectioning. Sections were stained for Oil Red O and hematoxylin and eosin. Sections were imaged at 400x magnification and counted by two pathologists blind to section identify. These counts were decoded by an independent observer and statistically compared utilizing students' T-test.

Credits/Disclosure/Support

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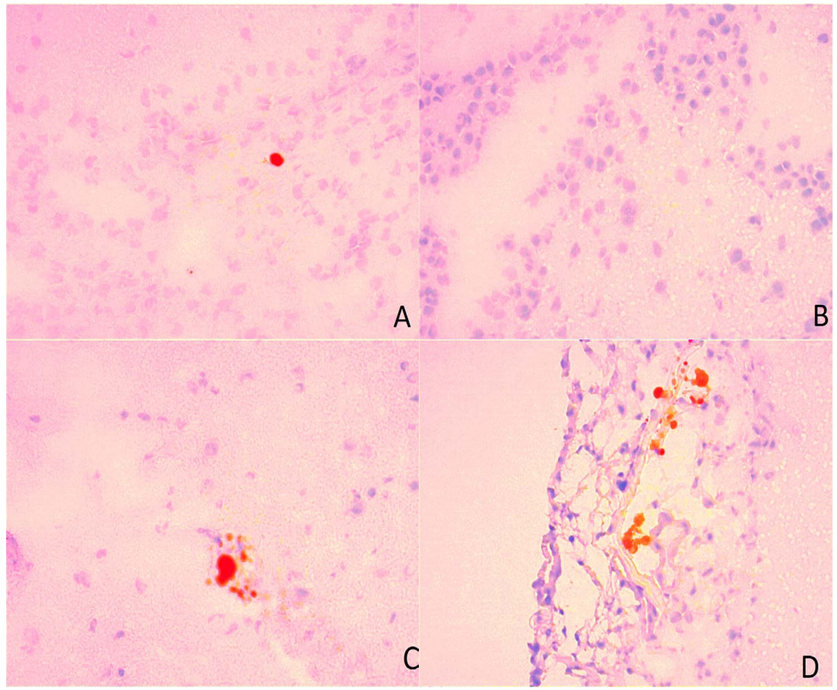
Supported by the Catherine T Geldmacher Foundation, St. Louis, MO.



- **Figures A & B:** Representative images of control rat brain, injected with saline, showing minimal Oil Red O staining.
- **Figures C & D:** Representative images of experimental rat brain, injected with triolein. Fat droplets were observed most frequently with minimal inflammatory reaction within experimental group. The size and shape of these droplets were heterogeneous.
- Stain: Oil Red O, magnification at 20x.

Results

The brains of the control group exhibited fat droplets in 3 of the 12 brains considered. These droplets were small in size and primarily localized to the cerebral cortex with minimal associated parenchymal reaction. 10 of the 12 brains observed in the experimental group demonstrated fat droplets which were localized to the cerebral cortex, cortical arteries, and especially the meningeal arteries. These droplets were much larger and were associated with an appreciable meningeal vascular thickening within affected vessels. Lungs of the control group minimal amounts of small scattered fat droplets in the same 3 subjects while the lungs of the experimental group all featured large fat droplets with the expected septal and associated arterial damage pattern previously published.

Conclusion

While fat deposition is a non-specific finding, the difference in tissue response demonstrated in rat lungs and brains is evident. While FE appears to provoke a marked inflammatory response within the lungs even at 48 hours, the brain does not appear to reciprocate this change. While the droplets noted within the experimental group are larger and more associated with a mild vessel response, the parenchyma of the brain appears much less affected compared to the lungs within the same animals. As the tissue reaction within the lungs is biphasic and the observed changes within the brain differ significantly by histology, further studies are necessary to ascertain if said changes might not evolve over a longer period of time.