

Fat Graft Pulmonary Embolism: In favor of a Lymphatic Route.

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INTRODUCTION

Fat Pulmonary Embolism Syndrome (F-PES) is a dreadful complication associated to high-volume fat grafting. When pathologically examined, a minority of cases reveal vessel injury at injury site and gross adipose tissue is found in the right atrium. However, more frequently, post-mortem examination of post-fat grafting fat embolism yields a fat emboli histology described as “**lipid micelles**” lodged in pulmonary capillaries in absence of intravascular extracellular matrix or stromal cell elements (figure 1)

During harvest liposuction, adipocyte disruption releases free triglycerides (TGC) in liquid oil form, accounting for a raw lipoaspirate **lipocrit**. The present pilot study was aimed at identifying absorption and transport routes for free and micellar TGC during liposuction-lipografting procedures, which is immiscible with plasma (figure 2)

Interestingly, 1 buttock augmentation case suffered a F-PES. Accidental findings during her ICU management prompted reformulation of the following hypothesis.

HYPOTHESIS

Following fat grafting, liquid free oil within lipoaspirates is preferentially removed from the interstitium by lymphatics, concentrated in chyle, and finally emptied in the central venous system.

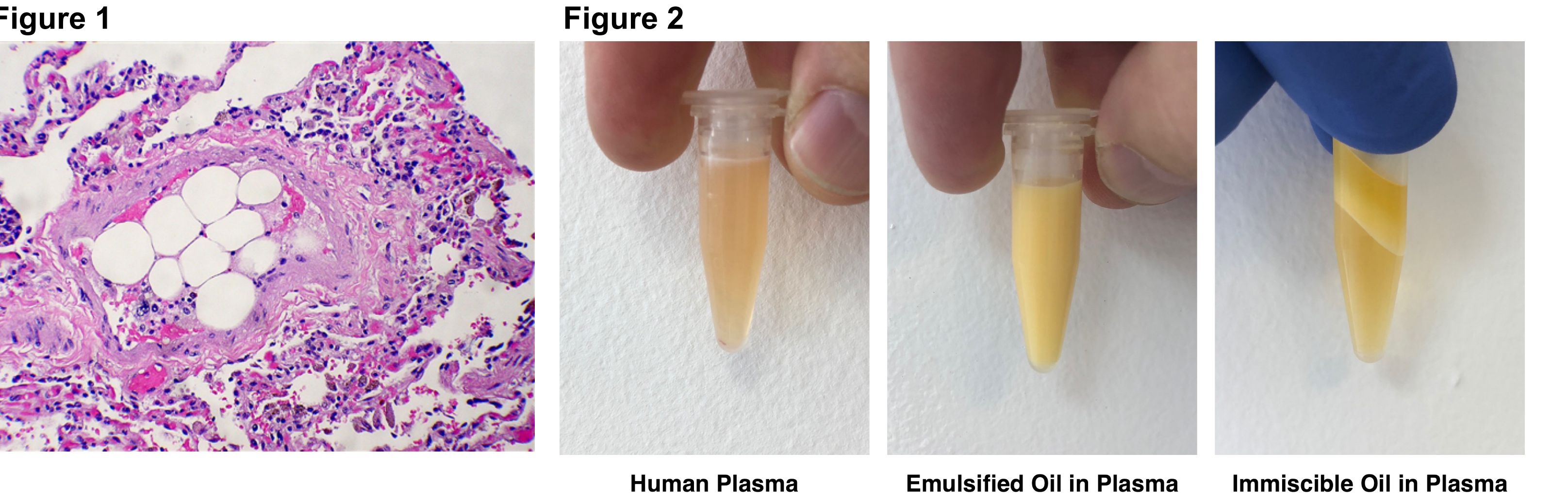
SAMPLE, MATERIALS and METHODS

In this prospective study, 27 consecutive cases underwent procurement liposuction and simultaneous fat grafting (20 breast, 7 buttocks), while:

- liquid oil fraction from fat graft volume (lipocrit) and
- TGC plasma concentration ([TGC]p) from peripheral blood samples preoperatively, immediately and 24h postoperatively.

In addition to the above and following ICU management of F-PES complication:

- Critical Micelle Concentration (CMC) of fat graft TGCs was analysed when lipoaspirated human TGCs were mixed in either lymphatic (chylous, CMCc) or plasmatic (CMCp) fluids, and
- Interstitial Pressures (IP) both intramuscular (im) and subcutaneous (sc) at recipient sites were also recorded (Figure 3)



RESULTS

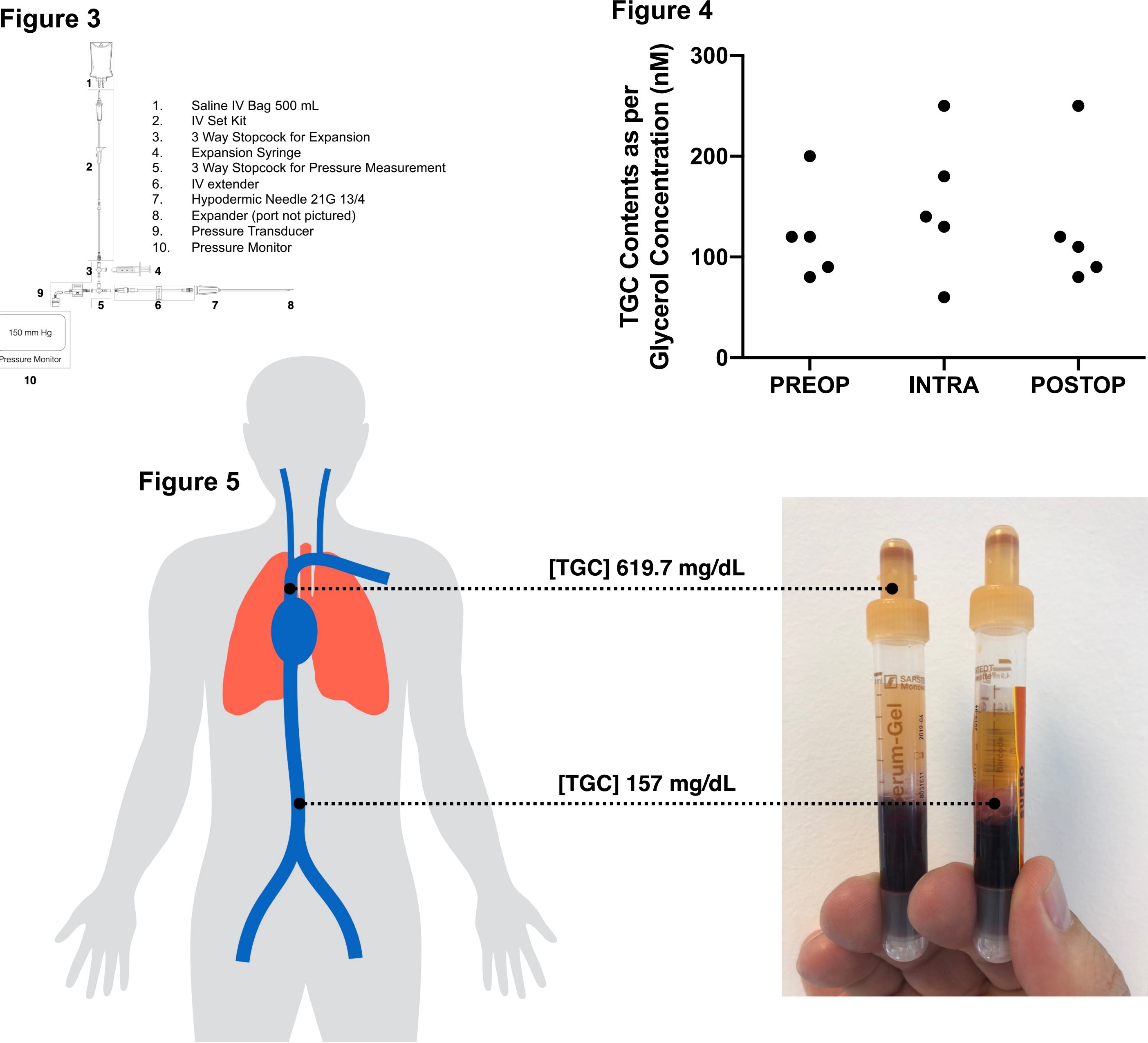
Lipocrit (200-600 cc total graft volume) ranged between 19-27%.

[TGC]p pre-, post- and at 24h displayed no significant differences among them in all cases (paired t-student). All measurements within normal range (x=199 mg/dL) (Figure 4)

However, central right subclavian vein sample taken at ICU admission, demonstrated a lipemic plasma with [TGC]p 7-fold higher than that of the simultaneously drawn peripheral sample, returning to equivalent peripheral levels 24 h later (Figure 4)

CMCc is consistently higher than CMCp (CMCp=110 mg/dL, CMCc=320 mg/dL, n=3, P<0.001)

IPsc progressively raised to pressures lower than 50 mmHg (Starling's venous drainage threshold) and returned to baseline (x=7 mmHg) at 24 h post grafting. However, IPim rapidly raised above 50 mmHg and persisted past 24 h post grafting (Figure 6 and 7 respectively))



CONCLUSIONS (Figure 8)

There F-PES appears associated to increased [TGC]p detectable at the central venous system only, yet seemingly undetectable in peripheral samples.

TGC micelle formation was shown to be more difficult in plasma rather than in chyle, suggesting a preferential interstitial uptake of grafted oil at the recipient site and a fast greater micelle formation when chyle drains in plasma.

This uptake could be facilitated when graft oil is implanted intramuscularly, where IP-im reach pressures that prevent venous drainage, possibly favoring lymphatic absorption instead (Starling independent drainage).

Fluid TGC contained in fat grafts may be preferentially absorbed by tissue lymph ducts, concentrated in chyle where they form larger micelles as they ascend via pelvic nodal network, cisterna magna and thoracic duct, and finally drip in the central venous system as large oil droplets to be showered in the pulmonary capillary network.

