Abnormal Vessel Architecture Persists in the Microvasculature of the Massive Weight-Loss Patient

Authors: Evan B. Katzel, MD; Sameer Shakir, MD; Nataliya Kostereva, PhD; Bernd Lannau, BA; Michael Gimbel, MD; Vu T. Nguyen, MD; Carolyn De La Cruz, MD; Kacey Marra, PhD; Jeffrey A. Gusenoff, MD

Disclosures: None of the authors has a financial interest in any of the products, devices, or drugs mentioned in this manuscript.

Introduction: Research demonstrates a link between obesity and increased circulating inflammatory cytokines, leading to changes in the microvasculature. Massive weight loss (MWL) patients often experience delayed wound healing following body contouring procedures, however no studies exist to explore the inflammatory response of MWL on microvasculature. This study hypothesized MWL patients undergoing body-contouring procedures maintain persistently elevated inflammatory markers in the microvasculature that delay wound healing.

Methods: Descriptive data were queried from normal weight and MWL patient charts to assess baseline demographics, medical histories, medications, and smoking status. Superficial inferior epigastric artery (SIEA) vessels were harvested during abdominally-based free flap surgery and abdominal contouring surgery for normal weight and MWL patients, respectively. Vessels were histologically assessed using immunohistochemistry to quantify anti-interleukin-1 (IL-1), IL-6, and TNFα expression. Trichrome staining was performed to assess and compare vessel architecture. Statistical analyses included independent samples two-tailed t-tests and Fisher’s exact test.

Results: All patients (n=23) were female. There were no significant differences in patient demographics including preoperative BMI. Quantitative analysis of IL-1, IL-6, and TNFα expression revealed no difference between normal weight and MWL patients (figure 1). Trichrome staining demonstrated abnormal vessel architecture in the MWL group with decreased collagen composition of the tunica adventitia and disorganized smooth muscle in the tunica media (figure 2).

Conclusions: Despite the return to normal levels of inflammatory markers following MWL, trichrome staining demonstrated irregular composition in the tunica adventitia and tunica media. This suggests vessel pathology that could cause friability and poor delivery of nutrients which could ultimately explain delayed wound healing in the MWL population.

Figure Legends:
Figure 1. Quantification of inflammatory cytokine staining between groups. Results are expressed as percentage of stained versus unstained area per artery cross-section. Normal weight patients are displayed in white while the black bars represent MWL. Bars represent mean with error bars representing standard deviation.
Figure 2. Histologic arterial cross-section using Masson Trichrome from normal weight patients (A) and MWL (B) at 10x magnification. Note aberrant vessel architecture in MWL group (B) with minimal tunica adventitia (blue), altered tunica intima, and tunica media organization (dark red) Keratin/Muscle (red); Collagen (blue); Cytoplasm (light red/pink); Nuclei (black).