

Radiological and Histological Assessment in Perforator Flap Microvasculature Following Pretreatment with Topical Negative Pressure Therapy: An Experimental Rat Model

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Background: Surgical delay and ischemic preconditioning have been traditionally used to precondition flaps to render flaps more vascular and resistant to changes in their microcirculation. Negative pressure wound therapy (NPWT) has been extensively in clinical practice and associated with angiogenesis and accelerated wound healing. This experimental model evaluated topical negative pressure as a mechanism for non-invasive preconditioning for perforator flap microvasculature and perfusion.

Methods: Two gluteal perforator flaps (matched control and intervention) were designed on sixteen 400g Sprague-Dawley male rats. NPWT was applied to the flap area directly continuously at -125mmHg for 7 days, after which the rats were divided into two principal groups. Group A (n=8) underwent 4D computed tomographic and angiography (CTA) with a body volume perfusion protocol after NPWT and euthanized. Group B (n=8), control and intervention flaps were raised, isolated on a single pedicle and laid back down and monitored for a further 7 days. Group B flaps were assessed using laser-assisted indocyanine fluorescence angiography before surgery, following flap harvest and at 7 days prior to euthanasia. Half of all rats in each group were analyzed with Micro-CT to assess the microvasculature. All paired specimens were assessed histologically with H&E and immunohistochemistry (IHC).

Results: There was a 17% increase in CT tissue perfusion in skin treated with NPWT versus matched controls ($P=0.001$). LA-ICGFA demonstrated higher perfusion following NPWT treatment ($P=0.006$), however no significant difference immediate post flap harvest ($P=0.19$) but a difference was seen 7 days postoperatively ($P=0.03$). Micro-CT evaluation showed an increase in average vessel volume (%) from 0.005 in control to 0.009 in the NPWT flaps ($P=0.04$). H&E analysis showed significant difference in the epidermal thickness ($P<0.001$), but comparable dermal thickness ($P=0.34$). Quantitative analysis of CD31 IHC demonstrated a mean area fraction percentage of 4.30 and 2.68 in the NPWT and control flaps respectively ($P=0.002$). There was partial necrosis in the control (n=3) and NPWT flaps (N=1), however this was <5% in the NPWT flap.

Conclusion: We present novel multimodal approaches using static and dynamic imaging and histological assessment to provide a proof of concept on the use of NPWT for non-invasive conditioning of flaps. The study provides the basis for further investigation and clinical studies with potential for direct translation into clinical practice.

