Treatment of Chronic Non-Healing Ulceration Utilizing A Transposition Flap With Vascular Delay Technique: A Case Report

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Statement of Purpose

Chronic non-healing ulcerations are regularly encountered in the foot and ankle in patients with peripheral vascular disease and areas of decreased tissue perfusion. Vascular delay or ischemic preconditioning, describes a phenomenon that when a tissue is rendered partially ischemic, it will undergo neovascularization and enhance its vascularity. Vascular delay technique has been shown to promote flap survival, increase the length-to-breadth ratio in random pattern flaps and facilitate in transfer of a larger volume axial pattern flap. This technique has been widely used in field of reconstructive surgery, however, seldomly implemented and reported in foot and ankle reconstructive surgery during management of chronic non-healing ulcerations. Herein, we present a case report in surgical management of chronic non-healing ulceration with exposed Achilles tendon utilizing a transposition flap with a vascular delay technique.

Literature Review

Gaspar Tagliacozzi was first to popularize vascular delay technique in upper arm flaps in 1597. In 1967, Myers used rabbit model to show optimal time for tissue transfer to be 8-10 days. Anatomical studies have demonstrated increase in number, size, and a change in direction of vessels. Jonsson et al in 1988 measured tissue oxygen tension in delayed flaps in 7 dogs, and anatomic exploration in 10 rabbits. They observed that oxygen tension fell after initial elevation but rose back to normal by day 14, and upon final elevation minimal changes from normal was noted. They also found that dominant vessels in rabbit flap model demonstrated reorientation up to 10 days after initial incision, and upon re-incision at 14 days parallel vessels dilated significantly. These results were later confirmed by two new studies. Callegari and Taylor in 1992 used dog flap model and demonstrated dilation and hypertrophy occurred maximally in zone of choke vessels. Morris and Taylor in 1995 demonstrated that maximal choke vessel dilation was seen in first 48-72 hours in rabbit flap model.

In 1985, Murphy et al compared delayed flaps with acute flaps in rat skin flap model. Initially, elevated levels of vasoconstricting metabolites prostaglandin F2alpha and thromboxane and decreased vasodilator PGE2 was seen. In models with 14-day delayed flaps, vasoconstricting metabolite levels decreased toward normal with subsequent increase in PGE2, thus increasing flap survival. In acute flaps, this phenomenon was not observed, leading to progressive ischemia and decreased flap survival. Lineaweaver el at performed a study on 20 rat model flaps They reported 81.9% whole flap viability in models with 7-day delay technique compared to 40.9% flap viability in models without delay. In another study, Tepper et al used mouse flap model with 7 day delay. They found elevated levels of VEGF which subsequently mobilized endothelial progenitor cells (EPC) from bone marrow. By 14 days, EPC clusters coalesced into vascular cords and becoming functional vessels by day 21.

Case Study

HPI: A 65 year old, diabetic, male patient presented to the office with a long-standing, right posterior lower extremity ulceration. The wound had been present for nearly six months at presentation. He reported having a basal cell carcinoma removed by a dermatologist that led to an eventual dehiscence and non-healing wound complicated by an exposed Achilles tendon, his diabetes, and daily tobacco use.

Physical Exam: At presentation, the wound measured 6.0cmX3.0cmX0.4cm with visible Achilles tendon in the wound bed, but no other signs of infection or neoplasm. Protective sensation was diminished but vascular status was intact.

Procedure: After confirmation that clean borders had been obtained with the carcinoma excision and no infection present the decision was made to move forward with surgical intervention to aid in wound closure. Plans were made to proceed with a staged procedure involving external fixation to immobilize the ankle and thus the Achilles tendon, delayed technique for rotational flap and graft closure.

Stage one involved patient presenting to the operating room where he underwent a thorough wound debridement with preparation of the planned rotational flap, application of an amniotic graft to help prep the wound bed and an application of an external fixator. The patient tolerated the procedure well with no complications or infections (Figure 1).

He presented to the operating room two weeks later where the previously prepared flap was lifted and rotated to cover the wound with the exposed Achilles tendon. Full coverage of the wound was obtained with no tension on the flap. The region where the flap was lifted was then covered with an amniotic graft but no tendinous tissue was visible
(Figure 2).

Results

Patient returned a month later to have the external fixator removed and the remaining wound dressed with an Integra bilayer graft. Over the next two months the patient simply performed regular dressing changes and went on to heal uneventfully. At latest follow up, nearly two years from the original procedure, the patient was still healed with no recurrent break down.

Figure 1

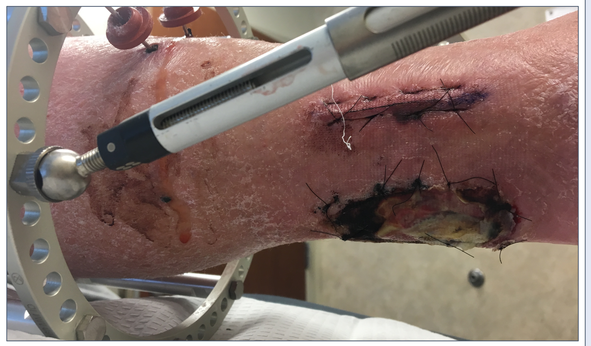

Figure 1: underwent a thorough wound debridement with preparation of the planned rotational flap, application of an amniotic graft to help prep the wound bed and an application of an external fixator

Figure 2

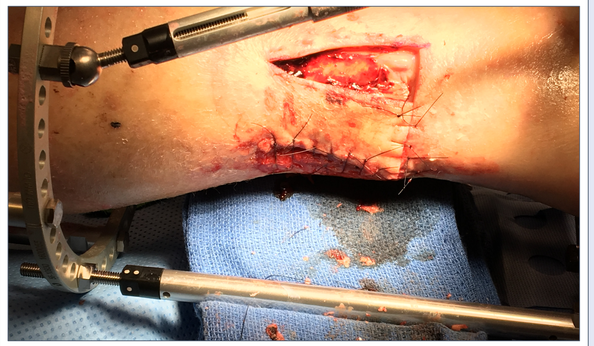

Figure 2: After two weeks later, the previously prepared flap was lifted and rotated to cover the wound with the exposed Achilles tendon with no tension on the flap.

Discussion

Vascular delay affects the target tissue in two phases – early and late. Early effects derive predominantly from transection of sympathetic fibers leading to dilation and reorientation of choke vessels. The late phase effects lead to enhanced flap vascularity due to new vessel growth. The early phase is characterized by three findings: changes in sympathetic tone, dilatation and reorientation of choke vessels, and alterations of metabolic pathways. During the initial flap dissection, the transected sympathetic nerves release norepinephrine into the tissue, leading to a hyperadrenergic state. This sudden catecholamine infusion can constrict the blood vessels up to 30 hours, amplifying the early ischemic state. When the severed nerve endings are depleted of these neurotransmitters, the hyperadrenergic state resolves leading to a reactive vasodilation and increased. Concurrently, the choke vessels reorient along the long axis of the flap, thereby enhancing blood supply to regions of the flap most prone for necrosis.

Vascular delay creates an imbalance between vasodilating (prostaglandin E2) and vasoconstricting (prostaglandin F2, thromboxane) metabolites. Murry found that subsequent elevation of a delayed flap leads to a blunted vasoconstrictor response, and an elevated vasodilator response, when compared with an un-delayed flap. This is thought to minimize ischemic injury. Most of the long-term benefits of vascular delay, however, derive from neovascularization of the flap. Vascular delay technique induces neovascularization by angiogenesis via increased production of angiogenic growth factors and vasculogenesis via recruitment of bone-marrow derived endothelial stem cells.

Delayed flaps experience a decrease in fat necrosis and partial flap loss compared with flaps which were not delayed. Vascular delay can further decrease these complications and also increase the number of candidates with vascular co-morbidities which would otherwise be restricted from surgery or be higher-risk patients. Patients with varying degrees of vascular compromise include those with radiated tissue, crush injuries, burns or otherwise traumatized tissues. Similarly, vascular delay benefits smokers, obese patients, and diabetics. Patients who had previously undergone surgery to the proposed flap region with scarred tissue and disrupted vessels are also candidates for vascular delay to increase the vascularity of the tissue.

By delaying and expanding free flaps prior to transfer, the delay phenomenon increases the amount of well-perfused, viable tissue available as well as to improve the tissue's condition at the donor site to facilitate primary closure.

References

Available upon request