Initial Experience using an Autologous Blood Clot to Promote Closure in Chronic Pressure Ulcerations

Wahab N., MD; Lapucha M., MD; Chauhan, C., RN; Abellera, J., RN
Wahab Consulting & Research, Las Vegas, NV

Introduction

Over the past several years, there has been an outpouring of novel products for the treatment of hard-to-heal wounds. Pressure ulcerations are one example of hard-to-heal wounds. Pressure ulcers are chronic wounds in which the microenvironment is ischemic due to microvascular compromise and nutrient depletion. Improved healing outcomes have been shown for chronic pressure ulcerations by recreating a wound microenvironment in which the acute phase of wound healing has been restarted by directing acute phase reactants to the wound. One such product showing potential promise for wound healing is an autologous blood clot formulated to serve as a topical dressing. The proposed mechanism of action postulates that wound healing is stimulated by cell-to-cell signaling within the local wound milieu (Guo et al. 2010). This includes release of growth factors, particularly VEGF, TGF, & PDGF, from the macrophages and secondary platelet plug complex within the whole-blood autograft. These factors mobilize monocyte migration to the wound, stimulate differentiation into macrophage subtypes, and promote angiogenesis (Eming et al. 2014). In effect, the whole blood autograft is thought to recreate the acute phase of healing (see Figure 1). We present a case series elucidating progress in wound area and volume reduction and closure of pressure wounds in a state of persistent non-healing despite conventional standard-of-care treatments.

Materials & Methods

Patients were selected using inclusion criteria in affiliated wound care centers in Las Vegas, NV. Wound care of acceptable study patients was transitioned from these centers to one research clinic. The whole blood-autograft was individually prepared for each patient per manufacturer recommendations and titrations using the product kit. After providing standard-of-care to the wound bed, the autograft would be applied. Patients would return to clinic for weekly applications of the whole-blood autograft as tolerated. Primary endpoint was complete wound healing as assessed by two practitioners. Secondary endpoints were greater than 40% area and volume reduction at 4 weeks.

Results & Conclusion

❖ 6 patients were enrolled with 7 wounds in total
❖ All tolerated phlebotomy and coagulated adequate autograft
❖ An average of 5 applications were performed over 8 weeks
❖ 6 wounds achieved >40% wound healing area at 4 weeks, ranging from 55% to 100% when excluding Patient 6 (see Graph 1)
❖ 6 wounds achieved >40% wound healing volume at 4 weeks, ranging from 65% to 100% when excluding Patient 6
❖ 1 wound (Patient 6) deteriorated with whole-blood autograft treatment. This patient had a large refractory pressure ulcer that had failed treatment with other advanced modalities.
❖ 3 of the 7 wounds achieved complete resolution
❖ A selection of patient pictures is presented below.

This case series demonstrates that a whole blood autograft is a useful addition to the armamentarium of the wound care physician for the treatment of hard-to-heal pressure ulcers, particularly those with greater surface areas. The whole-blood autograft is a cost-conscious and effective product that warrants further study.