## **Case in Point**

# Adjunctive Treatment for a Nonhealing Pressure Ulcer in a Patient With Spinal Cord Injury

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A wound of 12 years' duration finally heals when negative pressure wound therapy and pulsed radio frequency energy supplement traditional wound bed preparation.

ressure ulcers are common problems in patients with spinal cord injuries, with many occurring soon after injury.<sup>1</sup> Those that become chronic, stage IV wounds are difficult to treat and slow to heal, especially in patients with such comorbid conditions as type 2 diabetes mellitus, coronary artery disease, and anemia of chronic disease. Good results often require complex treatment protocols, involving multiple advanced wound healing modalities. Even with proper wound care, however, many ulcers persist, further diminishing patients' quality of life-physically, psychologically, and socially. While traditional wound bed preparation (characterized by debridement, antimicrobial treatment, moisture control, and use of appropriate dressings) is an effective intervention against pressure ulcers,<sup>2</sup> chronic and refractory wounds often call for complex regimens that employ adjunctive therapies. Even

with their use, however, results are uncertain.

In this article, we report on the case of a 60-year-old man who presented to our spinal cord injury (SCI) clinic with quadriplegia, a stage IV pressure ulcer of 11 years' duration, and several comorbid conditions. Over the years, various treatments had induced intermittent improvement in the wound, but healing was not sustained. When the wound worsened following an elective surgery, we added negative pressure wound therapy (NPWT) and pulsed radio frequency energy (PRFE) to the standard treatment protocol to accelerate healing. This report describes the effects of these adjunctive modalities and highlights the importance of taking a multipronged approach to wound care.

### **INITIAL EXAM**

A 60-year-old black man presented to our SCI clinic in October 2007 with a stage IV, right proximal coccygeal ulcer that had been present for almost 11 years. From a fall he sustained in 1996, he had a cervical SCI at level C4 (American Spinal Injury Association class A, indicating no preserved motor or sensory function in sacral segments S4-S5). Painful muscle spasms arising with movement complicated his rehabilitation and contributed to immobility. Comorbid conditions included neurogenic bowel and bladder, atonic colon, diabetes mellitus, coronary artery disease, obesity, sleep apnea, and chronic pain. He developed the ulcer shortly after his SCI. Due to multiple issues around his complex care, he lived in nursing facilities for the next 11 years. One month prior to his visit to our clinic, circumstances allowed him to be able to be transferred home to the care of his family.

His first primary care visit at our SCI clinic revealed a  $1.8 \times 2.1 \times 1.4 \text{ cm} (5.3 \text{ cm}^3)$  stage IV pressure ulcer on the right proximal coccyx (Table). The family reported that the ulcer had worsened since his discharge from the nursing home 1 month prior.

#### **TREATMENT COURSE**

We changed the patient's wound care from daily wet-dry dressings to alginate dressings at his first visit to

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our clinic. It was difficult to communicate with the patient throughout treatment because he was angry and exhibited overt signs of depression. He voiced extreme displeasure at being essentially bed bound since his injury because his nonhealing ulcer prevented him from sitting in a wheelchair.

In November 2007, the patient underwent an elective subtotal colectomy with end ileostomy, following repeated medical intervention for functional bowel obstruction. The pressure ulcer worsened during his postsurgical hospitalization, though it was not severe enough to warrant a diverting colostomy. Inpatient examination revealed that the ulcer had increased in size to 2.5 x 2 x 2.5 cm (12.5 cm<sup>3</sup>) with 2.5 cm undermining from the 11 o'clock to the 1 o'clock position. The ulcer, located on the top of the coccygeal crease, was round and had a moist red wound base. There was no necrotic tissue, but a small amount of serosanguinous exudate, maceration around the ulcer margins, and a slightly foul odor were evident. We changed his wound care to include daily packing with alginate pads covered with gauze.

By December 2007, the wound had improved only minimally, so we initiated adjunctive NPWT, using a vacuum-assisted closure device (Kenetic Concepts Inc., San Antonio, Texas) at 125 mm/Hg, continuous. At the time, the ulcer measured  $4 \ge 1.2$ x 2 cm (9.6 cm<sup>3</sup>) with 1.8 to 2.5 cm undermining from the 9 o'clock to the 3 o'clock position. By January 2008, it had again enlarged, measuring 4.6 x 1.2 x 2.4 cm (13.2 cm<sup>3</sup>) with 1.3 to 3.8 cm undermining from the 9 o'clock to the 3 o'clock position; it had a foul odor, and a clean dark red crater with a moderate amount of serosanguinous exudate were



apparent (Figure 1). We added silver antimicrobial dressings to the treatment protocol to reduce bioburden.

In mid-April 2008, the periwound area appeared fragile. Although the wound had decreased in size to 2.2 x 2 x 0.8 cm ( $3.5 \text{ cm}^3$ ), the width had almost doubled with 2 cm undermining from the 9 o'clock to the 11 o'clock position. The patient and family were eager for resolution. Because published reports suggest that PRFE may accelerate ulcer healing,<sup>2,3</sup> we introduced this adjunctive therapy (Provant Therapy System, Regenesis Biomedical, Inc., Scottsdale, Arizona) into the treatment regimen, while continuing with the NPWT.

Twice daily, the patient received a 30-minute, 27.12 MHz PRFE treatment at home. Both dose and duration are preset, allowing caregivers to administer treatment without nursing supervision.<sup>4</sup> The applicator pad was placed on top of the wound dressing, so as to prevent contamination, cooling of the wound, or disruption of concurrent treatment.

After 2 months, the ulcer was dime-sized, without drainage or redness. We applied an adhesive foam dressing to the site to provide



Figure 1. Pressure ulcer before institution of pulsed radio frequency energy.



Figure 2. Healed pressure ulcer.

protection during transfers and seating trials. We continued with NPWT until early August and PRFE until the end of August to ensure a stable scar. By mid-August 2008, the right proximal coccygeal area had totally healed (Figure 2), and it remained healed on subsequent clinic visits. Furthermore, the patient demonstrated a remarkable, positive change in attitude from an angry demeanor, completely resistant to care and treatment, to a pleasant demeanor, largely adherent to his medical regime. Healing of the pressure ulcer had greatly improved his quality of life and, for the first time in years, he was able to sit upright in a wheelchair.

## **ABOUT THE CONDITION**

When pressure ulcers become chronic stage IV wounds, treatment is difficult and traditional wound care may need to be augmented by an advanced healing modality. NPWT is one of several that have been used with success in the treatment of complex nonhealing wounds to improve local wound conditions,<sup>5</sup> and Blume and colleagues<sup>6</sup> concluded that it was more effective than advanced moist wound therapy in treating diabetic foot ulcers.

In our patient, the ulcer size increased initially when NPWT was introduced into the treatment protocol. With the addition of silver antimicrobial dressings, ulcer size slowly decreased over a period of 3 months, but the periwound area appeared more fragile. At that point, we decided to introduce a trial of PRFE.

Porreca and colleagues<sup>2</sup> had reported on a patient with quadriplegia and a long-standing, stage IV sacral ulcer that healed with PRFE, and Larsen and Overstreet<sup>3</sup> had observed similar results in 2 patients with recalcitrant diabetic foot ulcers. In 2009, Frykberg and colleagues<sup>7</sup> reported encouraging results after using PRFE in 5 patients with chronic wounds.

Although its precise mechanisms of action are unknown, PRFE is thought to work at a cellular level, stimulating dermal cell proliferation in the wound bed and inducing

Table 1. Wound size from initial presentation, 12 years following SCI		
Date	Volume (cm <sup>3</sup> ) and wound appearance; intervention, if any	% change
Oct 4, 2007	5.3 cm <sup>3</sup> without undermining	N/A-initial assessment at SCI clinic
Nov 7, 2007	12.5 cm <sup>3</sup> with undermining of 2.5 cm, moderate serosanguinous exudate, odor, and maceration	235% increase, after patient hospitalized for surgery
Dec 20, 2007	9.6 cm <sup>3</sup> with undermining of 1.8 cm to 2.5 cm, moderate serosanguinous exudate, odor, and maceration; NPWT initiated	23% decrease from Nov
Jan 8, 2008	13.2 cm <sup>3</sup> with undermining of 1.3 cm to 3.5 cm, moderate serosanguinous exudate, odor, and maceration; NPWT continued	73% increase from Dec
Apr 15, 2008	3.5 cm <sup>3</sup> with undermining of 2 cm, fragile periwound area; PRFE therapy initiated and NPWT continued	72% decrease from Jan
Jun 17, 2008	Dime-sized ulcer	92% decrease
Aug 8, 2008	Ulcer healed; NPWT discontinued 8/8 PRFE discontinued at end of month	100% healed
SCI = spinal cord injury; NPWT = negative pressure wound therapy; PRFE = pulsed radio frequency energy.		

growth factors, cytokines, and extracellular matrix proteins to begin the inflammatory phase of wound healing.<sup>8</sup> An in vitro study designed to determine the effect of PRFE on dermal fibroblast and keratinocyte proliferation found a significant increase in cell mitosis after a 30-minute treatment.<sup>8</sup> Gilbert and colleagues<sup>9</sup> suggested that it may activate the p44/42 mitogen-activated protein, which induces proliferation of cells.

Noncontact ultrasound therapy also has shown benefit in helping to heal wounds that are refractory to conventional treatment.<sup>10–12</sup> With the use of adjunctive ultrasound therapy, Bell and Cavorsi<sup>10</sup> observed a 79% reduction in ulcer size, and Kavros and colleagues<sup>11</sup> reported wound healing rates of more than 50%. Ultrasound is believed to speed wound healing by stimulating signal-transduction pathways, which (like PRFE) affect cellular activity.<sup>11</sup> In an experimental study, ultrasound was also shown to reduce bacterial count.<sup>13</sup> Adjunctive treatments, including topical growth factor and electrical stimulation, have been used with variable results in treating wounds.<sup>14</sup>

The cost of treating pressure ulcers can be staggering. Over the 10 months in which we treated this patient, we calculated our gross costs to be more than \$60,000, which doesn't include the cost of caring for the wound over the prior 11 years. Our costs included medications: numerous courses of oral antibiotics, topical antibiotics, multivitamins and vitamin C, iron, and protein supplements, numerous basic and advanced dressing supplies, the NPWT and PRFE devices, VA-sponsored transportation, as well as clinic and home health visits. Often with pressure ulcers of this complexity in the quadriplegic patient, inpatient care becomes necessary which would have increased the cost by \$2,400 to \$3,000 per month in a skilled nursing home or \$1,500 per day in a regional SCI facility.

## **IN SUMMARY**

We used NPWT and PRFE in addition to traditional wound care treatment in a patient with a stage IV ulcer that had persisted for 11 years and had reached a wound volume of 13.2 cm<sup>3</sup>. With these adjunctive modalities, the wound completely healed within 7 months. The patient tolerated the therapy well and had no complications. The response of this patient suggests that NPWT in conjunction with PRFE may benefit other patients with SCI and nonhealing wounds. Further studies of larger patient populations, segmented by different diagnoses (such as pressure vs ischemic or diabetic ulcers), and further studies of the combined use of these 2 modalities with complex pressure ulcers would help define the degree to which the adjunctive use of NPWT and PRFE is beneficial.

#### Author disclosures

The authors report no actual or potential conflicts of interest with regard to this article.

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#### REFERENCES

- Mawson AR, Biundo JJ, Neville P, et al. Risk factors for early occurring pressure ulcers following spinal cord injury. *Am J Phys Med Rehabil*. 1988;67(3):123–127.
- Porreca EG, Giordano-Jablon GM. Treatment of severe (Stage III and IV) chronic pressure ulcers using pulsed radio frequency energy in a quadriplegic patient. *Eplasty.* 2008;8:e49.
- Larsen JA, Overstreet J. Pulsed radio frequency energy in the treatment of complex diabetic foot wounds: Two cases. J Wound Ostomy Continence Nurs. 2008;35(5):523–527.
- Regenesis Biomedical. Operating Manual: Provant Wound Therapy System, 2501-01L. Scottsdale, AZ: Regenesis Biomedical, Inc., 2007.
- Wada A, Ferreira MC, Tuma Junior P, Arrunategui G. Experience with local negative pressure (vacuum method) in the treatment of complex wounds. *Sao Paulo Med J.* 2006;124(3):150–153.
- Blume PA, Walters J, Payne W, Ayala J, Lantis J. Comparison of negative pressure wound therapy using vacuum-assisted closure with advanced moist wound therapy in the treatment of diabetic foot ulcers: A multicenter randomized controlled trial. *Diabetes Care.* 2008;31(4):631–636.
- Frykberg R, Tierney E, Tallis A, Klotzbach T. Cell proliferation induction: Healing chronic wounds through low-energy pulsed radiofrequency. Int J

Low Extrem Wounds. 2009;8(1):45-51.

- George FR, Lukas RJ, Moffett J, Ritz MC. In vitro mechanisms of cell proliferation induction: A novel bioactive treatment for accelerating wound healing. *Wounds.* 2002;14(3):107–115.
- Gilbert TL, Griffin N, Moffett J, Ritz MC, George FR. The Provant Wound Closure System induces activation of p44/42 MAP kinase in normal cultured human fibroblasts. *Ann NY Acad Sci.* 2002;961:168–171.
- Bell AL, Cavorsi J. Noncontact ultrasound therapy for adjunctive treatment of nonhealing wounds: Retrospective analysis. *Phys Ther.* 2008;88(12):1517–1524.
- Kavros SJ, Miller JL, Hanna SW. Treatment of ischemic wounds with noncontact low-frequency ultrasound: The Mayo Clinic experience, 2004-2008. Adv Skin Wound Care. 2007;20(4):221–226.
- Schmuckler J. Acoustic pressure wound therapy to facilitate granulation tissue in sacral pressure ulcers in patients with compromised mobility: A case series. Ostomy Wound Manage. 2008;54(8):50–53.
- Serena T, Lee SK, Lam K, Attar P, Meneses P, Ennis W. The impact of noncontact, nonthermal, lowfrequency ultrasound on bacterial counts in experimental and chronic wounds. Ostomy Wound Manage. 2009;55(1):22–30.
- Whitney J, Phillips L, Aslam R, et al. Guidelines for the treatment of pressure ulcers. Wound Repair Regen. 2006;14(6):663–679.