Vasculitis, a process caused by inflammation of the blood vessel walls, is categorized by the size of the vessels involved (i.e., large-vessel, medium-sized-vessel, or small vessel); it may be self-limiting, with short-term courses, or may involve multiple organ systems, with life-threatening complications. Cutaneous small-vessel vasculitis (CSVV) involves arterioles, venules, and capillaries, with occasional involvement of small or medium arteries. Skin lesions may result from the inflammatory process and can present as petechiae, “palpable” purpura, blisters, deep nodules, ulcers, or livedo on the extremities, which may lead to ulceration and gangrene. Necrotic tissue delays healing, increases inflammation, and serves as a nidus for infection. Excess inflammation within a wound prevents healing and causes further tissue damage.

Multiple etiologies for CSVV have been identified, including infections, medications, chemical exposure, malignancies, and food allergies; the underlying cause is unknown in up to 60% of patients. A paucity of easily accessible accurate information, combined with a low level of suspicion for vasculitis, make the diagnosis difficult to consider, identify, and confirm. Treatment options are limited by patient discomfort, wound chronicity, and multiple comorbidities that require continuing medications that might be a “triggering” source. For disease that is limited to the skin, conservative treatment is preferable because the long-term effects of systemic treatment and its sequelae are unknown.

Active *Leptospermum* honey (ALH) promotes autolytic debridement and has anti-inflammatory, immunomodulatory, and antioxidant properties. ALH has been shown to modulate the activity of monocyte cells in the wound; biopsies of tissue treated with honey have shown decreased numbers of inflammatory cells. This is not solely attributed to honey’s antimicrobial or debridement effect; rather, ALH-specific mechanisms and components stimulate cytokine induction and modulate the inflammatory response. The anti-inflammatory effect reduces pain and increases opening of the blood vessels; thereby, reducing edema and exudates. ALH scavenges free radicals after they have been formed and may prevent their formation in the first place, resulting in less inflammation.

The antioxidant activity of ALH may be partly responsible for the anti-inflammatory effect.

**Case Study**

A 61-year-old Caucasian woman with a history of a questionable chemical exposure at work developed a malar rash followed by erythematous nodules on her lower extremities. She presented to the wound clinic on August 27, 2008 with a 3-month-old, 0.5 cm x 0.5 cm x 0.1 cm necrotic, nonhealing ulcer on the right lateral gaitor aspect of the leg, surrounded by edema and erythema (see Figure 1).

A thorough history and physical exam, including family history, current and past medications, comorbidities, and nutritional and occupational status, was performed. Prior wound treatments included over-the-counter topical antibiotics and clobetasol propionate cream. Prior medical history included superficial phlebitis, hypothyroidism, mixed hyperlipidemia, hypertension, peptic ulcer disease, osteoarthritis, temporomandibular joint (TMJ) syndrome, and a blood transfusion. Past surgical history included a tubal ligation. The patient was taking the following medications: methylprednisolone dose...
pack, cetirizine hydrochloride, lovastatin, sulindac, atenolol, levophedoxine, tramadol hydrochloride, albuterol sulfate, budesonide and formoterol fumarate, clorazepate dipotassium, cyclobenzaprine, clotetasol propionate cream, acetaminophen, ciclopirox olamine, and omeprazole. Allergies included latex, sulfamethoxazole and trimethoprim, and aspirin (gastrointestinal distress).

A wound assessment and biopsy were performed; necrotic tissue was debrided. The size of the wound increased slightly to 0.8 cm x 0.5 cm x 0.1 cm. An antimicrobial cellulose dressing was prescribed until biopsy results were obtained. One week later, periwound edema, erythema, increased exudate and periwound maceration were noted. Meanwhile, biopsy results confirmed suspected CSVV (see Figure 2).

An ALH hydrocolloid dressing (MEDIHONEY® Honeycolloid™, Derma Sciences, Inc., Princeton, NJ) was initiated on September 3, 2008, covered with an absorbent dressing, and secured with one layer of tubular elastic bandage (the patient was intolerant to higher compression and declined increasing the compression due to discomfort). The dressing was changed every 3 days. Rapid reduction in necrotic slough tissue, edema, exudate, and maceration were noted (see Figure 3). Complete healing was achieved by December 3, 2008 (see Figure 4).

Chronic wounds, specifically those caused by CSVV, cause a great deal of morbidity for patients. ALH helps improve quality of life by decreasing pain at the wound site, cost and complexity of wound care, and the need to spend unnecessary time away from work for wound management. In addition to this case, other chronic wounds where inflammation is believed to cause nonhealing have been treated. The outcomes of six additional cases have been positive; as a result, ALH has become this clinician’s product of choice when inflammation is at the root of wound chronicity. Further studies are indicated to delineate additional benefits of ALH.

References