# Wound Sleuth

By Carol Ott, MD FRCPC and Alana Miller, MD FRCPC

## Why didn't this lower leg ulcer heal?

### History

RV is a 73-year-old female who presented to the wound clinic with a 10-year history of venous stasis, venous stasis dermatitis and leg ulcers. Her ulcers were painful and often became infected, requiring many courses of antibiotics.

Other past medical history included type 2 diabetes, atrial fibrillation, hypertension, osteoporosis and diastolic heart failure. Her medications were glicazide, metformin, sitagliptin, rivaroxaban, rosuvastatin and furosemide.

On examination, her dorsal pedal pulses were strong in both feet. Lipodermatosclerosis, with the inverted champagne bottle sign, was present bilaterally. Hyperkeratosis and venous stasis dermatitis were also present on both legs. An ulcer was located on the lateral side of her left leg.

**Q** How would you treat this patient?

**A** Treatment involved washing her left leg with a facecloth, soap and water to remove the hyperkeratosis. Betamethasone 0.1% cream was applied to the dermatitis, followed by an antiseptic primary dressing and an absorptive dressing. Tubular mild elastic compression of 8–12 mmHg was applied to reduce edema and treat the venous stasis.

This treatment continued for several months with improvement to the periwound area, but the central ulcer persisted (see Figure 1).

On a follow-up visit, 3 x 3 mm fragments were removed from the wound. We assumed the fragments were calcinosis that can occur in chronic wounds (see Figure 2).

The fragments were sent to pathology and identified as mature reactive bone, or hypertrophic ossification (HO) (see Figure 3).

**Q** What would your next steps be?

**A** To determine whether the HO was a local or diffuse process, X-rays of both legs and

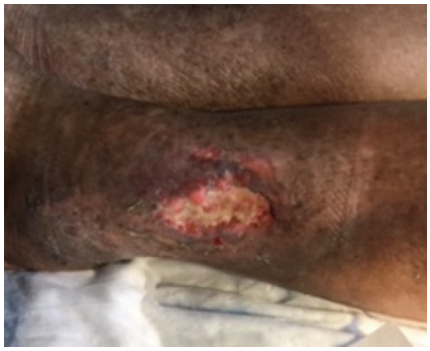

**Figure 1:** Non-healing ulcer on left leg

**Figure 2:** Fragments removed from wound

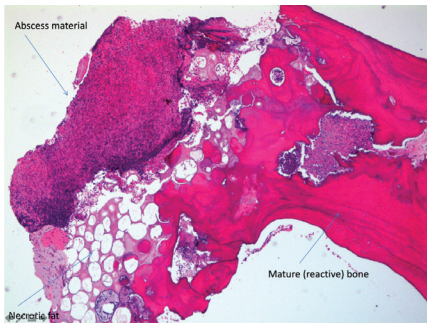


**Figure 3:** Microscopic view of the fragments

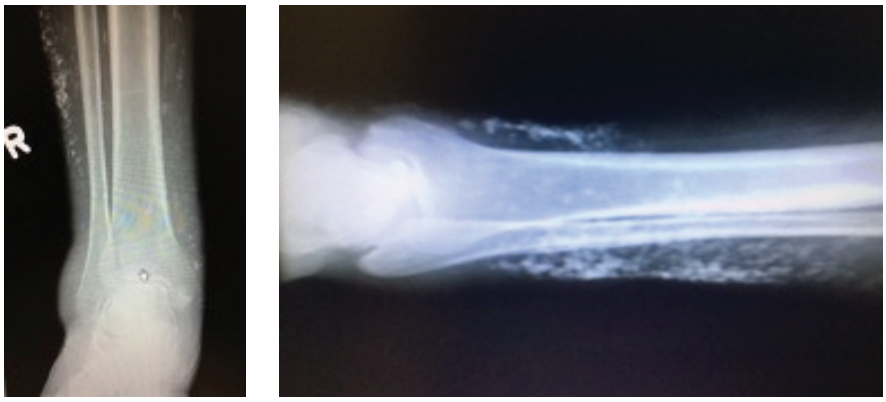


**Figure 4:** Right and left ankle radiographs showing hypertrophic ossification

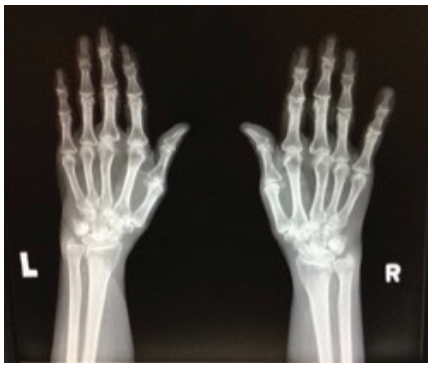


**Figure 5:** Right and left radiographs NOT showing hypertrophic ossification

hands were performed. If the process was diffuse, a work-up for systemic disease-causing hypertrophic ossification is necessary, whereas a localized HO is likely related to the chronic wounds and recurrent infections. The imaging revealed peripheral radiodensities consistent with subcutaneous hypertrophic ossification in the region of chronic venous insufficiency (see Figures 4 and 5).

## Discussion

Heterotrophic ossification is extra-articular bone in soft tissue adjacent to bone.[1] It was first recognized in 1692.[2] This condition is distinguished from metastatic calcifications, which occur mainly in hypercalcemia, and dystrophic calcification in tumours.[2]

HO is a relatively common complication following central nervous system disorders such as brain injuries, tumours, spinal cord injuries and encephalitis.[2] HO can occur almost anywhere in the body, including laparotomy scars, the kidney, skeletal muscle and the oral cavity.[3] Hereditary causes include fibrodysplasia ossificans progressive, progressive osseous heteroplasia and Albright's hereditary osteodystrophy.

The pathophysiology of HO is not completely understood. It's believed that three conditions must be met: First is the presence of osteoinductive factors such as inflammation. The second is the presence of osteoprogenitor cells, and finally, an environment conducive to osteogenesis, which leads to unwanted bone formation.[1]

Two theories exist regarding how these three conditions occur in chronic edema and cause HO. The first is that periosteal or cartilaginous cells are traumatically displaced from skeletal structures and are activated by the traumatic stimulus (and possibly other factors) to form bone.[3]

The second theory is that a metaplastic change occurs in ubiquitous pluripotent mesenchymal cells, which transforms the undifferentiated cells into osteoblasts. The trigger may be chronic edema and recurrent cellulitis leading to nutritional injury to the skin and subcutaneous tissues of the leg.[3]

Subcutaneous ossification is a frequent late complication of chronic venous insufficiency.[4] A series of 600 cases of venous insufficiency were studied in the 1960s, and heterotopic bone formation was identified in 10% of patients.[4]

HO due to chronic venous insufficiency is most frequently reported in post-menopausal women.[5] On examination, the typical presentation of HO involves a recurrent or non-healing ulcer.[5] Other features of chronic insufficiency are often present, such as telangiectasia, leg edema and lipodermatosclerosis. On blood work, the serum calcium and phosphate are normal.

The differential diagnosis of HO includes collagen vascular disorders, morphea, dermatomyositis, hyperparathyroidism or malignancy such as sarcoma or metastatic carcinoma.[5] X-ray

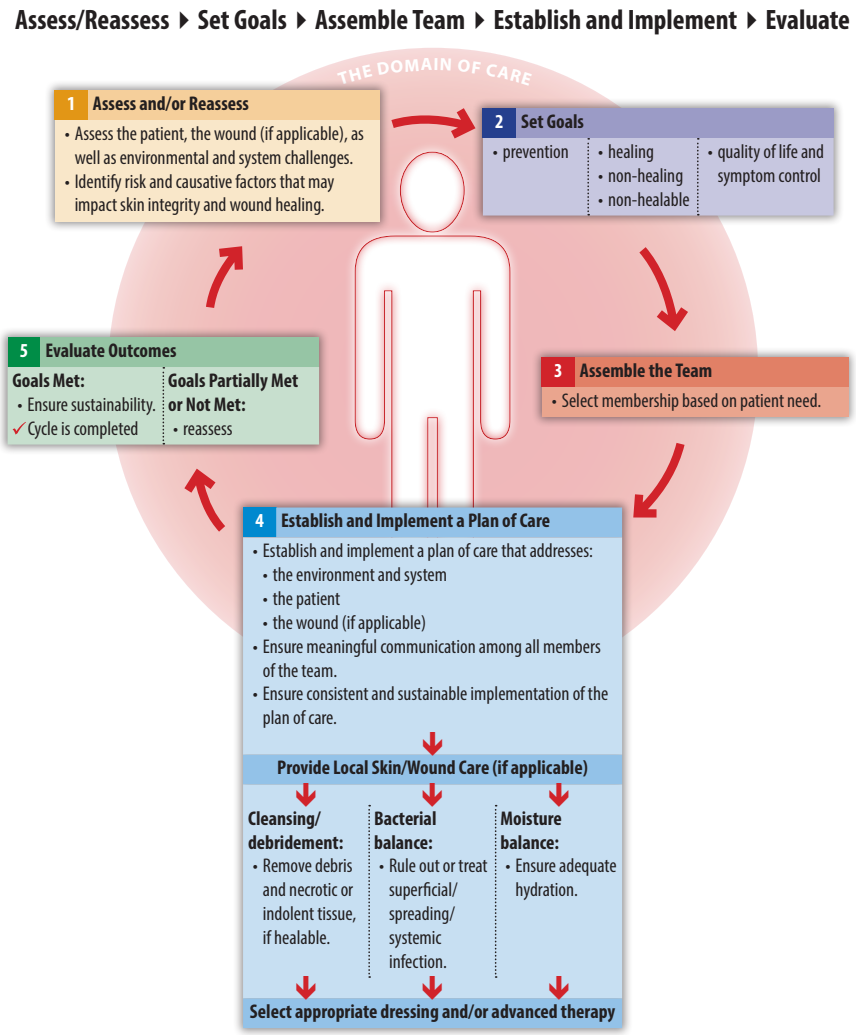


**Figure 6:** Wound Prevention and Management Cycle

or CT imaging will reveal extensive calcification in soft tissues. Pathology reveals mature cancellous bone with hemosiderin-laden macrophages.[5]

## Management

Management is primarily preventative. The chronic venous insufficiency should be treated with compression stockings and extremity elevation to prevent ossification. Once ossified tissue is present, the ossified fragments must be removed, otherwise the ulcer(s) will not heal.[5] Once resected, aggressive wound care for promotion of wound healing and infection prevention is needed. If severe, wound coverage with autograft and biosynthetic skin substitute can be considered.[5]

## Conclusion

Our team, a wound care physician and the home care nursing staff, followed Wound Canada's Wound Prevention and Management Cycle (see Figure 6) to heal RV's wound (see Figure 7). We identified and treated the etiology: venous stasis. When initially the ulcer did not heal, we reassessed, identified the ossified fragments and diagnosed HO with the assistance of pathology. Once the HO was discovered, the ossified fragments in the wound were removed and the ulcer healed. Over the next few years, the wound recurred, and the management plan was repeated with success.

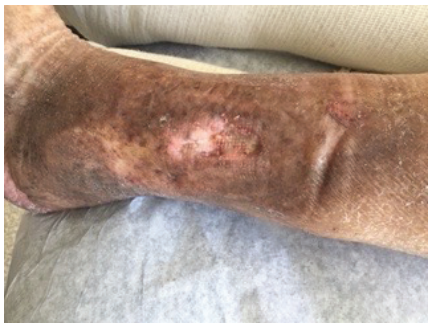

**Figure 7:** Lateral wound now healed

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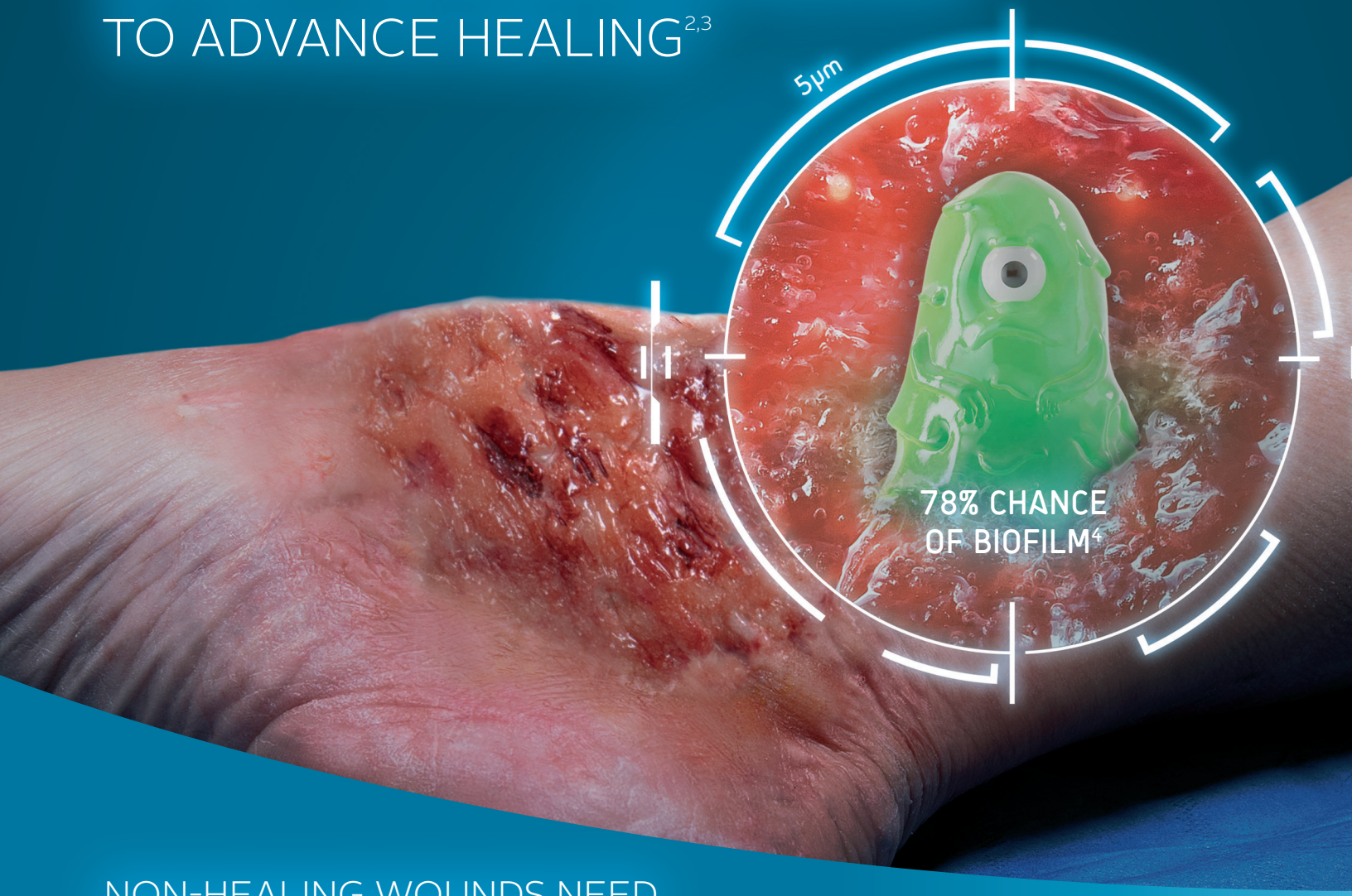


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