Management of the Pressure Injury Patient with Osteomyelitis: An Algorithm

Peter J Nicksic, BA[1], Sarah E Sasor, MD[1], Sunil STholpady, MD, PhD, FACS[1], William A Wooden, MD, FACS[1], Luke G Gutwein, MD[2]

[1]Division of Plastic & Reconstructive Surgery, Department of Surgery, Indiana University, School of Medicine, Indianapolis, IN

[2]Department of Anatomical Sciences, St. George's University School of Medicine, Grenada, West Indies

Disclosure Information: Nothing to disclose.

Correspondence address:
Peter J. Nicksic
Indiana University School of Medicine, Division of Plastic Surgery
535 Barnhill Drive, EH 232
Indianapolis, IN 46202
pnicksic@iupui.edu
(219) 263-8949

Running Head: Pressure Injury with Osteomyelitis

Keywords: pressure injury, pressure sore, decubitus ulcer, osteomyelitis, bone infection

Introduction

Pressure injury (PI) is a common complication of inpatient care, affecting an estimated 3 million patients annually in the United States (1). Risk factors include immobility, compromised sensation, malnutrition, urinary or fecal incontinence, and chronic medical illness (2). Compliance with established guidelines (pressure offloading, skin care, and frequent inspection) is imperative for the prevention of hospital-acquired pressure injury. Unavoidable PI does, at times, occur and is often related to advanced medical illness (3). Patients with physiologic, behavioral, or treatment-related risk factors may develop PI complicated by osteomyelitis (OM) despite the adherence to current standards of prevention (4).

The National Pressure Ulcer Advisory Panel's (NPUAP) defines pressure injury as localized damage to the skin and underlying soft tissue usually over a bony prominence or related to a medical device **(Table 1)** (5). The sacrum, trochanter, ischium, and heel are commonly affected (6). When bacteria within the wound adhere to bone, OM can develop. Propagation of bacteria and the formation of biofilms deep within bony structures cause an inflammatory response which leads to bone resorption - both by decreasing osteoblast function and increasing osteoclast activity (7). This process results in a necrotic hollowing of the bone called a sequestrum and is the distinguishing feature of chronic OM (8). Chronic OM is unresponsive to systemic antibiotics; surgical debridement is required for curative treatment (9). Nearly every case of OM secondary to a high grade PI is chronic (10).

Despite advancements in both surgical and medical therapies, the long-term recurrence of chronic OM remains 20-30 percent (11). Patients with personal, social, and economic risk factors for PI are at high risk for recurrence despite initial success at wound coverage. The goal of this paper is to review indications for surgical treatment of PI with associated OM and to introduce an algorithm for successful treatment.

Pressure Injury-Osteomyelitis Algorithm

We propose an accurate and simple algorithm for the diagnosis and treatment of OM secondary to PI for clinicians and surgeons to follow in patients with high-grade PI and suspected OM **(Figure 1)**. The algorithm delineates the steps necessary for an accurate diagnosis of OM secondary to a PI. The algorithm then walks through the treatment plan for patients with both curative and palliative treatment goals.

Diagnosis

Osteomyelitis secondary to PI should be suspected in a patient who presents with wounds with significant tissue necrosis or exposed bone. While non-specific, laboratory tests can be useful to rule out OM. C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) are nearly 100 percent sensitive (12). Unfortunately, absolute values do not correlate with short-term outcomes (13).

Imaging is important to support the diagnosis of OM and to localize and grade the severity of infection. Plain radiography is the first step - it is useful for detecting bony destruction and periosteal reaction (14). An involucrum, a thick sheath of new periosteal

bone surrounding a sequestrum, is sometimes visible (15). If OM is suspected on plain films, it is appropriate to proceed directly to bone biopsy and culture.

Plain XR has a high false negative rate in OM; it is positive only when half of the affected bone has necrosed which is typically occurs at least one month into disease progression (14). For this reason, more sensitive imaging techniques should follow a negative radiograph. Magnetic resonance imaging (MRI) is the imaging modality of choice for OM for its superior specificity and comparable sensitivity (16). In patients who are not candidates for MRI, three-phase technetium-99 bone scintigraphy and leukocyte scintigraphy are useful adjuncts (14).

While laboratory tests and imaging are useful in the work-up of OM, a bone biopsy and culture is needed to confirm the diagnosis (15). Three separate cultures and one histopathological analysis should be done per ulcer (17). Specimens should be obtained after debridement of overlying tissue to decrease the risk of contamination (9); cultures from superficial wounds or sinus tracts are not useful (18, 19). Wound cultures are considered positive if at least one of three shows a non-commensal organism concentration of >100,000 per gram tissue or all three biopsies are positive for commensal organisms (20). Histopathology typically shows a lymphoplasmacytic infiltrate and bone necrosis (9).

Medical Optimization

Many pressure injuries have the ability to heal without surgery once risk factors are corrected. When OM is present, infected or necrotic bone should be debrided, if possible (21). Patients should then be medically optimized before considering reconstructive surgery.

Patient repositioning, specialized mattresses, and foam coverings are the mainstay of pressure off-loading and are an integral in decreasing the size and severity of PI prior to intervention (22, 23). As many PI are malnourished or nutritionally deficient, it is also necessary for the patient to have adequate micronutrients, protein, and caloric intake to heal surgical wounds (24). Medical comorbidities should also be controlled prior to surgical intervention, as they may adversely affect wound healing. Lastly, social barriers affecting the patient's ability to care for their wounds should be assessed and corrected.

Treatment

Ideal candidates for reconstructive surgery do not use nicotine, have good social support, are nutritionally optimized, and have good surgical options for coverage. Patients who do not meet these standards are likely to benefit from a palliative approach to treatment with the goal of arresting progression of infection and reducing bioburden without reconstructive surgery.

Surgical principles for the treatment of pressure sores include complete excision of the wound (soft tissue, bursae, and affected bone), elimination of dead space, and wound resurfacing. Flaps should be large enough to provide adequate cushioning over any

remaining bony prominences. Suture lines should be placed away from areas with direct pressure. Flap design should allow for re-advancement, if possible, and should not violate adjacent flap territories.

Debridement

Surgical debridement is recommended for all patients with pressure injury and osteomyelitis who are deemed operative candidates (21). Aggressive, serial debridement back to healthy-appearing, vascularized tissue is required. The purpose of debridement is to convert a chronic wound into an acute wound; removing affected tissue and its inflammatory cytokines allows the remaining wound to heal as if it was acutely injured. Necrotic bone may harbor biofilms and should also be aggressively debrided **(Figure 2)**(10). Biofilms must be mechanically disrupted by either high-pressure washes or excision or else infection is likely to recur (25). The goal of debridement is a clean wound bed that has tissue of white, yellow, or red appearance (Figure 3).

Definitive Treatment

Antibiotic Therapy

For patients who are candidates for definitive wound closure, targeted parenteral antibiotic treatment should be initiated after debridement and continued for 4-6 weeks (10). Some sources recommend and additional 2-4 weeks of oral antibiotics beyond this (18). Choice of antibiotic agent should be guided by culture, susceptibility, and drug penetration into bone (19, 26, 27).

It is important to realize that antibiotic therapy alone cannot cure chronic OM, and a person with chronic OM should not be on systemic antibiotics indefinitely (28). The goal of targeted antibiotic therapy is to bridge the gap between debridement and wound closure.

Reconstruction

After debridement, the patient should have their wound dressed until reconstruction is possible. Negative pressure wound therapy (NPWT) is popular in this setting (10). After the local wound control and initiation of antibiotic therapy, CRP levels should trend down (13). When granulation tissue is present, it is safe to plan for permanent wound coverage (10). Soft tissue defect should be reconstructed with the least invasive procedure that will offer the most benefit to the patient. Myocutaneous and fasciocutaneous flaps offer similar resilience to compression (29). Common local flap options are V-Y advancement flaps, gluteal rotation or advancement flaps, hamstring flaps, and pedicled tensor fascia lata flaps (30). Free flaps are rarely indicated in this patient population.

Palliative treatment

Some patients are not candidates for definitive wound coverage for various medical and non-medical reasons. In these instances, it is best to pursue palliative treatment with the goals of arresting disease progression, decreasing bacterial bioburden, and reducing wound size. If the patient is an operative candidate, it is best to adequately debride infectious or necrotic material as described above. Bacteria in high concentrations alter

the microenvironment and cellular metabolism within the wound, favoring progression of chronic wounds (31). With adequate debridement there is still potential to restore a balance in the cellular microenvironment (**Figure 2**) (32). If a patient's condition does not allow for complete debridement, additional solutions include partial debridement, bone trephination, and drainage tube placement (10).

Systemic antibiotic therapy is inappropriate in a palliative care patient if the patient is stabilized after debridement (33). Antibiotics will not penetrate the largely avascular bone abscess or biofilms if they have not been completely cleared from the wound; they also carry the risk of an opportunistic infection.

An important aspect of palliative management is wound care. It is critical to keep the wound clean. This can be difficult, especially in patients that are incontinent. Temporary or permanent urinary and/or fecal diversion may benefit some patients. There are innumerable wound care products available for the treatment of pressure injury. A meta-analysis of various dressings and topical agents found no significant difference in outcomes (34). Options include mechanical debridement with wet-to-dry dressings or Dakin's-soaked gauze, enzymatic debridement with collagenase or Manuka honey, and various antibiotic impregnated foams and sponges, among others. NPWT is of special utility in this population as it aids in both wound contraction and optimization of the microenvironment of the wound for epithelialization while shielding the wound from further inoculation (35). NPWT should not be applied to wounds with grossly infected or necrotic tissue present (35).

If palliative care is the known to be the goal at the onset of PI diagnosis, imaging studies, bone biopsies, and cultures should not be performed since they will not guide treatment.

Conclusion

Pressure injury complicated by chronic osteomyelitis is a common problem in hospitalized patients. An algorithmic approach to diagnosis and treatment is useful to help determine if a patient may benefit from definitive wound coverage. Many patients may be better served by a palliative approach to care. With a 49 percent 12-month recurrence rate, curative measures for PI should only be pursued in a medically and nutritionally optimized patient with a robust social support system (36).

Our algorithmic approach to pressure ulcer injuries has aided understanding and communication of the complex care in this patient population across disciplines in medicine. For successful wound closure, multiple clinical variables must be addressed such as debridement, incontinence, antibiotic therapy, nutritional state, optimal wound care, off-loading, and the social support of the patient. With a plan to address each of these variables, care of the pressure injury patient is optimized.

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Table 1. Pressure Injury Staging

Stage	Description
I	Intact skin with nonblanchable erythema. Pigmented skin may not differ from surrounding skin.
II	Loss of epidermis, partial thickness loss of dermis. Open or closed blister.
III	Full-thickness loss of skin. Exposure of subcutaneous tissue. No exposure of muscle, tendon, or bone.
IV	Full-thickness loss of skin. Exposure of muscle, tendon, or bone.
US	Unstageable injury. Eschar exposed with unknown depth of necrotic tissue.

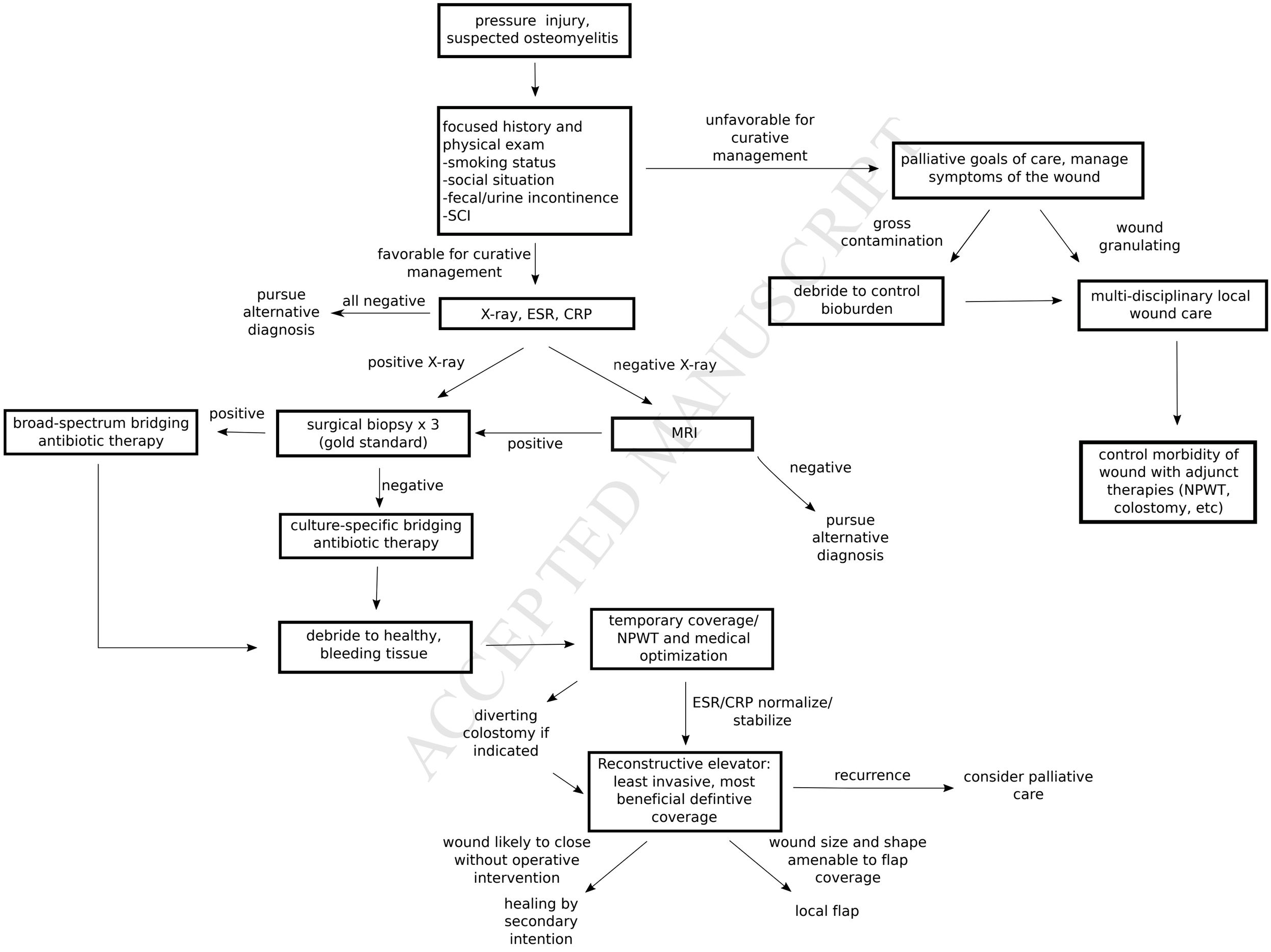
NPUAP staging guidelines for pressure injury.(5)

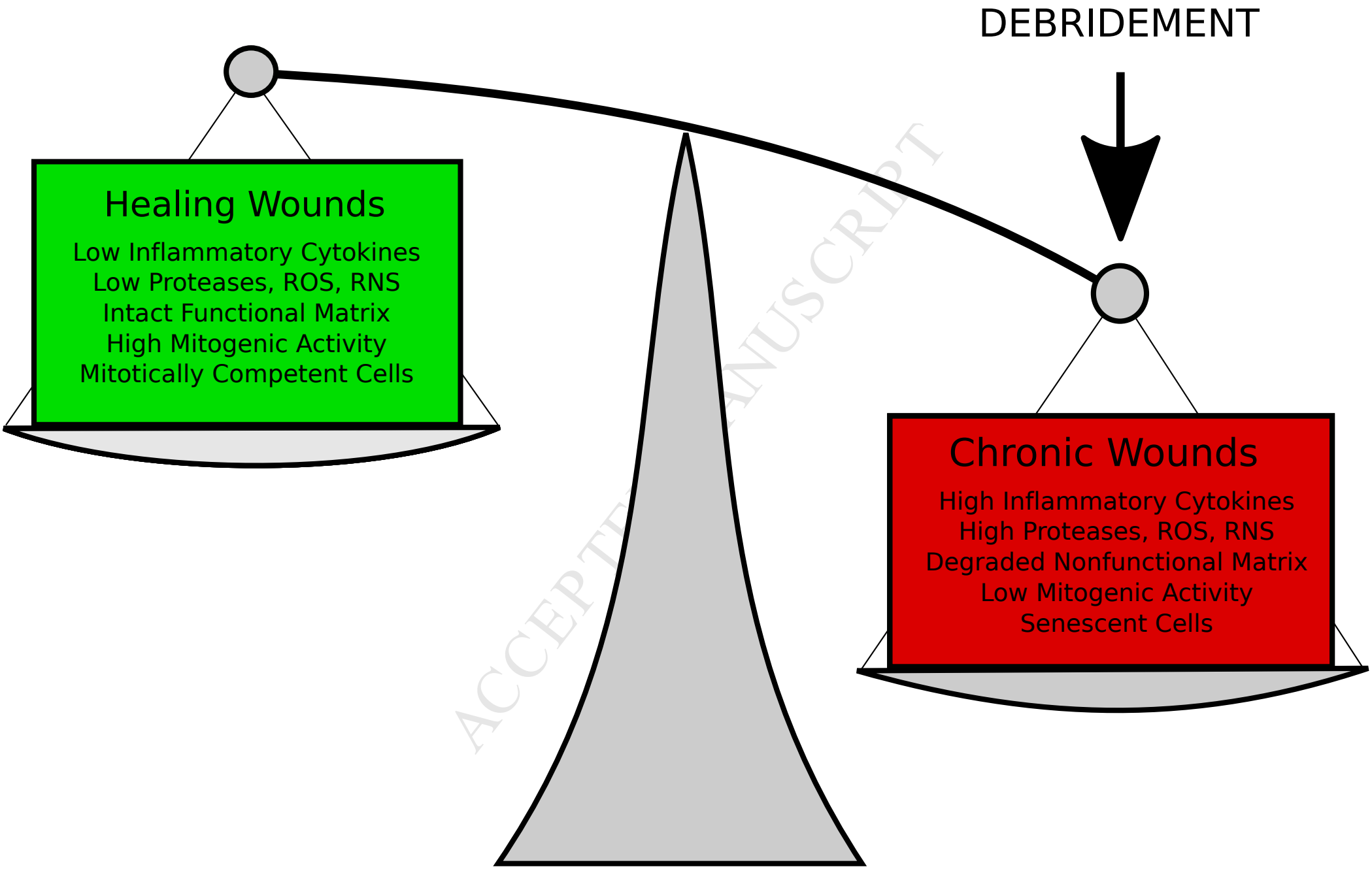
Figure Legends

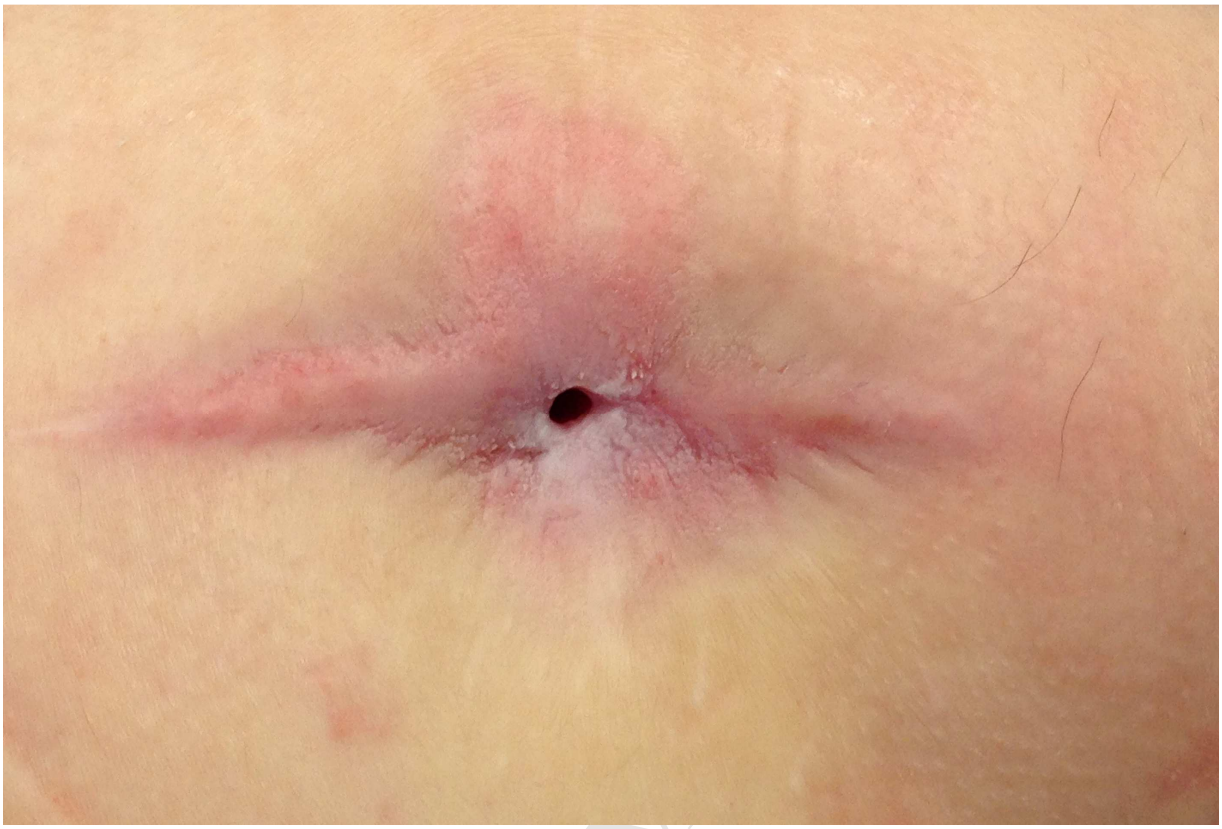
Figure 1. Diagnosis and treatment algorithm for the pressure injury patient with suspected osteomyelitis. The algorithm includes both curative and palliative treatment goals. NPWT, negative pressure wound therapy; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; SCI, spinal cord injury.

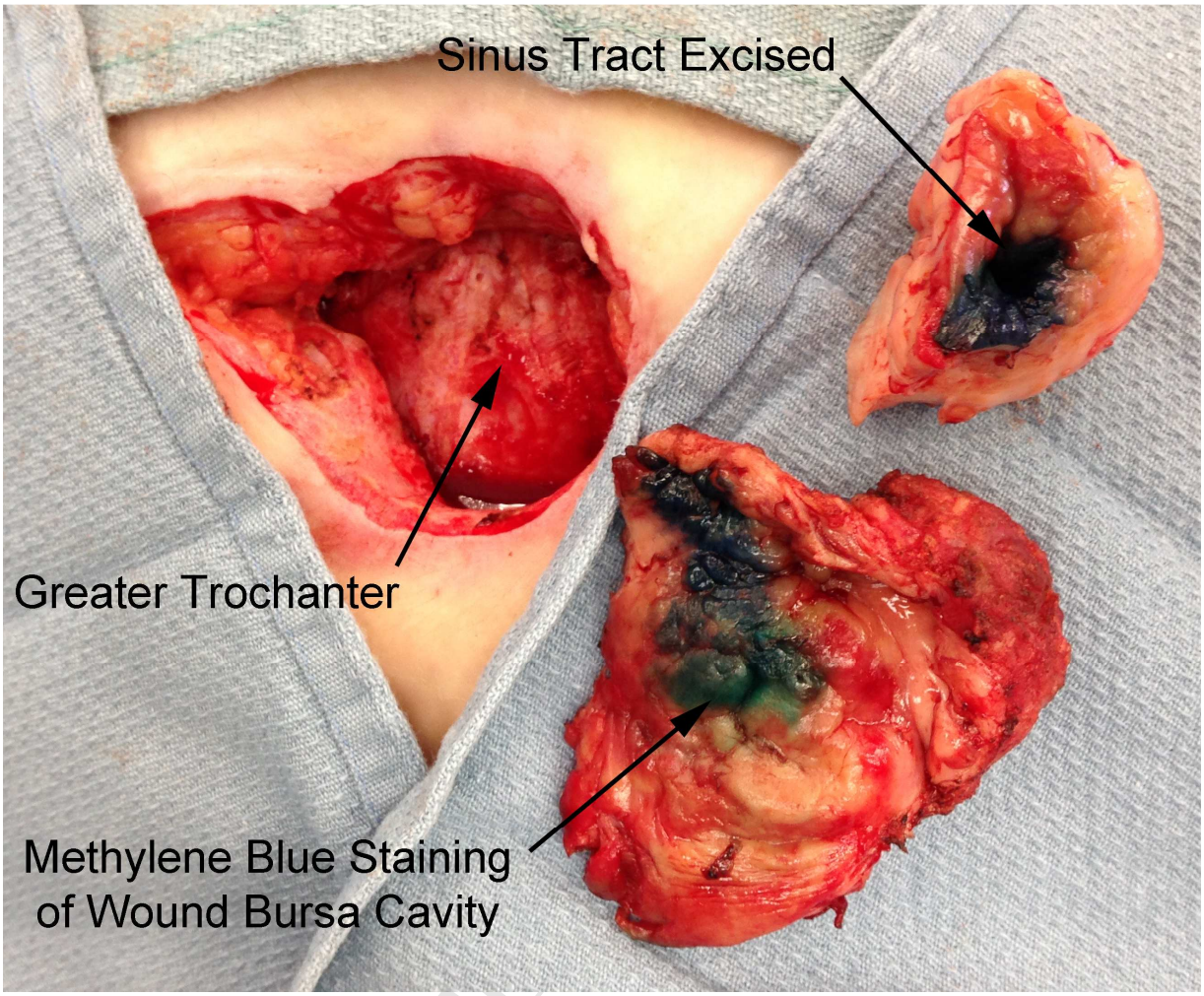
Figure 2. The role of debridement in converting cellular microenvironment of chronic wounds to healing wounds. There is an imbalanced cellular microenvironment between healing wounds and chronic wounds. This imbalance can be rectified through adequate debridement of devitalized tissue, which perpetuates the chronic immunological response inhibiting normal wound healing. ROS, reactive oxygen species; RNS, reactive nitrogen species.

Figure 3. (A) An unstageable trochanteric pressure ulcer harbors chronic inflammatory tissue that inhibits the normal phases of wound healing. (B) The sinus tract is stained with methylene blue to visually guide complete excisional debridement of the chronic inflammatory tissue and wound bursa. After excision and pulse irrigation washout, tissue is sent for culture and if osteomyelitis is suspected bone biopsy and cultures are obtained. (C) The wound is packed for 12 hours with moistened gauze to ensure hemostasis. Transition to negative pressure wound therapy is performed on postoperative day 1.








Sinus Tract Excised
Greater Trochanter
Methylene Blue Staining
of Wound Bursa Cavity