Wound Infections in Critical Care

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KEYWORDS

• Wound healing • Evidence-based nursing • Wound management • Wound infection • Critical care nursing

KEY POINTS

- Antimicrobial resistance rates in the critical care environment are higher because of the widespread use of broad-spectrum antibiotics, multiple invasive procedures, and transmission of multiple drug–resistant bacteria between patients.
- Clinical signs and symptoms of wound colonization and infection share similarities and can be difficult to differentiate.
- Nurses must be adept at distinguishing normal wound drainage from the initial inflammatory response from an overt infectious process.
- Biofilms form rapidly in wounds given the right environment, delay wound healing by competing for metabolic resources, and prolong the inflammatory phase.
- Numerous commercially available antimicrobial/antiseptic agents have been developed and should be considered as part of a comprehensive wound management strategy.

Patients admitted to critical care units are at high risk for increase morbidity and mortality from skin and deep wound infections. Antimicrobial resistance rates are higher because of the widespread use of broad-spectrum antibiotics, multiple invasive procedures, and transmission of multiple drug–resistant bacteria between patients.[1,2] *Staphylococcus aureus* is part of the normal skin flora found on 20% to 50% of healthy adults. In hospitals, *S aureus* is the major cause of acute bacterial skin and skin structure infections, especially surgical site infections.[3] *S aureus* isolates increased from 35,553 isolates in 1996 to 190,654 isolates in 2008 in United States hospitals, a ~5.4-fold increase.[4] Methicillin-resistant *S aureus* (MRSA) isolates accounted for

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53% of all *S aureus* isolates in US hospitals from 2006 to 2008 with more than half cultured in the critical care environment.[4] Prevention, early recognition, and management of wound infections are critical to successful outcomes in this patient population. This article addresses the physiologic basis for wound healing and the complexities of the wound microenvironment, distinguishing inflammation from infection, recognition of biofilms, and evidence-based wound management guidelines to promote wound healing.

PHYSIOLOGIC BASIS FOR WOUND HEALING

Skin Anatomy and Physiology

The skin is the human body's largest organ and the first line of defense from environmental insults (**Box 1**). The skin and its appendages (eccrine glands, apocrine glands, and hair follicles) make up this complex organ system. Although the distribution of appendages and skin thickness vary depending on the location, the basic structural components remain constant.[5] Human skin is divided into 2 layers: a superficial epidermal layer generally about 0.04 mm thick, and a deeper connective tissue layer called the dermis. The epidermis and dermis overlay the hypodermis, a layer of subcutaneous fat tissue containing large blood vessels that supply the skin and play a crucial role in thermoregulation and pressure redistribution.[5–7] The epidermis of human skin is a living ecosystem colonized by more than 10^3 microorganisms (per gram of tissue) including bacteria, fungi, and viruses.[8] These symbiotic microorganisms provide protection against invasion and overgrowth of more pathogenic organisms.

The epidermis is a stratified epithelium composed of 4 layers over most of the body and a fifth layer (stratum lucidum) of translucent cells found on the palms of the hands and soles of the feet. Keratinocytes are the primary cells in the epidermis. The stratum basale or stratum germinativum is the deepest layer of the epidermis and attaches to the dermis by an adhesive basement membrane. The basement membrane is the layer involved in blister formation.[9] A single layer of mitotically active keratinocytes forms the stratum basale and is responsible for all epidermal regeneration. When the epidermis is injured, cell division in the basal layer greatly accelerates. As new cells form, older cells migrate toward the skin surface through the stratum spinosum and stratum granulosum, undergoing terminal differentiation by the time they reach the outermost layer, the stratum corneum.[9]

The stratum corneum is made up of 20 to 30 layers of dead keratinocytes, which provides a protective, durable overcoat that is tough, waterproof, and fairly insensitive to biological, chemical, and physical assault. Normal skin is slightly acidic (5.5 pH) and the layered keratinocytes and lipids of the epidermis form the acid mantle that inhibits microbial growth. Underneath the surface, immune cells, including polymorphonuclear neutrophils, Langerhans cells, and macrophages, destroy pathogenic

Box 1
Skin functions

- Protection
- Prevention of fluid loss
- Thermoregulation
- Sensation
- Synthesis of vitamin D