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Wound Care

Contents

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Wound Care

A/Prof Goh Lee Gan

SFP2014; 40(3): 3

Wound care is a competency that should be in today's family doctor's tool kit. The tagline from one nursing expert in this area of work is - "Early detection and prevention are vital to reduce avoidable pressure ulcers" (Guy H, 2012)[1]. Disabled persons confined to bed or wheelchair run a high risk of developing wounds from pressure damage or combined pressure and shearing damage caused by sliding down an inclined bed, if these antecedent factors are allowed to continue to damage the integument. The care of pressure ulcer as a wound is a multidisciplinary responsibility, not just the tissue viability nurse alone. Many caregivers contribute to its success in prevention and treatment – attending doctors, nurses, the tissue viability nurse, as well as informal caregivers and family members. The common task is to prevent and relieve the skin of pressure damage or pressure and shear damage in all avoidable pressure ulcers.

The aim of this issue of The Singapore Family Physician is to provide the reader with an understanding of the pathophysiology of pressure damage, the healing process, the factors that perpetuate non healing and the action to take as first intent; the wound dressings that will be appropriate; and finally, how to approach a wound that is complex.

The College Council and the Institute of Family Medicine (IFM) wish to put on record their thanks to the writers and speakers in the forthcoming Family Practice Skills Course on Wound Care and their help in putting together this issue for the reader. It is a laudable collaborative effort between the family physicians and the nursing experts in wound care. There are 3 reading units in this family practice skills course.

Unit 1 on wound healing - by Dr Low Lian Leng and Dr Ng Joo Ming Matthew - covers the pathophysiology of wound healing; the risk and perpetuating factors preventing wound healing; the staging of pressure ulcers; the TIME acronym which provides a framework for systematic evaluation and intervention on the 4 factors that impede wound healing; and the role of adjuvants in speeding up healing.

Unit 2 on wound dressings - by Dr Lee Mei Gene Jasmine, Dr Pan Yow-Jeng Franny, Dr Ng Joo Ming Matthew and Yang Leng Cher - covers the common types of wound dressings in use and the choice related to stage of healing of the wound: hydrogels in the debridement stage; foams and low-adherence dressing in the granulation stage; and hydrocolloids and low adherence dressings for the epithelialization stage.

Unit 3 approach to complex wound management and adjunct therapy - by Tan Mui Lan and Goh Boon Ai Susie - covers what is a complex wound; the application of the TIME framework in dealing with a complex wound; the appropriate choice of wound care products based on wound type; and adjunct therapy.

The ten readings selected from current literature related to wound care adds to the foreground knowledge on the subject. The first is a must read (Mackintosh et al, 2014)[2]: the appearance and texture of fruits are used to help the caregiver visualise the stage of pressure ulcer: tomato for stage 1; partially peel potato for stage 2; apple with a bite off for stage 3; peach with a bite off for stage 4; a rotten part of a peach for unstageable pressure ulcer; and a purple brinjal for deep tissue injury. The rest are useful reading too. For example, prevalence of the prevalence of pressure ulcers is higher in Netherlands than Germany or Sweden by a lot. Why is this so? Find out from reading the last 2 readings in the 10 papers selected for you to read (Gunningberg et al 2013; Meesterberends et al, 2013)[3,4].

In addition, under the PRISM section is a case study by Dr Wang Mingchang and Dr Shum Oi Han, residents in the Family Medicine Residency Program. It is a case study on streptococcal pneumonia associated haemolytic uraemic syndrome (SP-HUS) in a 4-year old boy seen in a local hospital. This is a rare but serious condition. The child is actually immunised against strep pneumonia and why does he still come down with SP-HUS? Find out from the case report.

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2. Mackintosh R, Gwilliam A, Williams M. Teaching the fruits of pressure ulcer staging. J Wound Ostomy Continence Nurs. 2014 Jul-Aug;41(4):381-7. PubMed PMID: 24988517
3. Gunningberg L, Hommel A, Bååth C, Idvall E. The first national pressure ulcer prevalence survey in county council and municipality settings in Sweden. J Eval Clin Pract. 2013 Oct;19(5):862-7. PubMed PMID: 22640165.
4. Meesterberends E, Halfens RJ, Spreeuwenberg MD, Ambergen TA, Lohrmann C, Neyens JC, Schols JM. Do patients in Dutch nursing homes have more pressure ulcers than patients in German nursing homes? A prospective multicenter cohort study. J Am Med Dir Assoc. 2013 Aug;14(8):605-10. PubMed PMID: 23628407

"Wound Care" Family Practice Skills Course

- Overview Of "Wound Care" Family Practice Skills Course
- Unit 1 : Wound Healing
- Unit 2 : Wound Dressings: A Primer for the Family Physician
- Unit 3 : Approach to Complex Wound Management and Adjunct Therapy

OVERVIEW OF "WOUND CARE" FAMILY PRACTICE SKILLS COURSE

A/Prof Goh Lee Gan

SFP2014; 40(3): 5

INTRODUCTION

This is a one-afternoon Family Practice Skills Course. The aim of this skills course is to provide the reader with an understanding of the pathophysiology of pressure damage, the healing process, the factors that perpetuate non healing, and the action to take as first intent; the wound dressings that will be appropriate; and finally, how to approach a wound that is complex.

COURSE OUTLINE AND CME POINTS

This Family Practice Skills Course is made up of the following components. You can choose to participate in one or more parts of it. The CME points that will be awarded are also indicated below.

Components and CME Points

• Distance Learning Course – 3 units (3 Core FM CME points upon attaining a minimum pass grade of 60% in Distance Learning Online MCQ Assessment of 15 questions)
• 1 Seminar (2 Core FM CME points)
• 1 Workshop (1 Core FM CME point)
• 10 Readings – read 5 out of 10 recommended journals (maximum of 5 CME points for the whole CME year)

Distance Learning Course

Unit 1: Wound Healing
Dr Low Lian Leng, Dr Ng Joo Ming Matthew
Unit 2: Wound Dressings: A Primer for the Family Physician
Dr Lee Mei Gene Jesmine, Dr Pan Yow-Jeng Franny, Yang Leng Cher, Dr Ng Joo Ming Matthew
Unit 3: Approach to Complex Wound Management and Adjunct Therapy
Tan Mui Lan, Goh Boon Ai Susie

COURSE TOPIC DETAILS

Unit 1: Wound Healing
• Introduction
• Four phases of healing in acute wounds
• Progression to a chronic wound
• Risk factors affecting wound healing or causing non-healing in chronic wounds
• Local factors
• Systemic factors
• Approach to wound healing and strategies to enhance wound healing
• Role of adjuvants
• Conclusions

Unit 2: Wound Dressings: A Primer for the Family Physician
• Introduction
• Wound dressings and factors affecting selection
• Categories of wound dressings
• Advances in wound care technology
• Conclusions

Unit 3: Approach to Complex Wound Management and Adjunct Therapy
• Introduction
• Principles of wound healing
• Complex challenges and effective management strategies
  - TIME review

FACE-TO-FACE SESSIONS

Seminar: 27 September 2014, 2.00pm – 4.00pm
Unit 1: Wound Healing
Dr Low Lian Leng

Unit 2: Wound Dressings: A Primer for the Family Physician
Yang Leng Cher

Unit 3: Approach to Complex Wound Management and Adjunct Therapy
Goh Boon Ai Susie

Workshop: 27 September 2014, 4.30pm – 5.30pm
Assessment of Wounds
Goh Boon Ai Susie

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UNIT NO. 1

WOUND HEALING

Dr Low Lian Leng, Dr Ng Joo Ming Matthew

ABSTRACT

Wound healing is achieved through four coordinated and overlapping phases, 1) haemostasis, 2) inflammatory, 3) proliferative and 4) remodelling. This complex process can be disrupted by local or systemic risk factors, resulting in delayed healing and progression to a chronic wound. Chronic wounds interact closely with a patient's comorbid illnesses, social circumstances and functional status. The Family Physician plays an important role to optimise patient and wound risk factors that impair wound healing. Strategies to enhance wound healing include optimising local wound care based on TIME principles, identification and optimising the underlying causes for poor wound healing and education to the patients and their caregivers in wound care, dressing changes and avoidance of risk factors to prevent recurrence. Complex chronic wound care may need a multi-disciplinary approach involving allied health members to provide additional nutritional, nursing and psychosocial support. There is a role for adjuvants such as hyperbaric oxygen therapy and platelet derived growth factor gels to enhance healing in certain wounds but stronger evidence is required to support its routine use.

Keywords:
Wound healing, Family Physician

SFP2014; 40(3): 6-16

INTRODUCTION

A wound is a disruption of the normal structure and function of the skin and skin architecture and chronic wounds occur when healing does not occur in an orderly and timely manner to restore anatomic and functional integrity.[1] Chronic wound care is complex and may need a multi-disciplinary approach involving allied health members to provide additional nutritional, nursing and psychosocial support. Family Physicians (FPs) are well positioned in the community to coordinate such care and can better serve their patients with knowledge and proficiency in chronic wound management.

The costs of prolonged treatment of chronic wounds in an acute hospital setting are unsustainable and many patients can be managed in the community or in step-down care facilities. The current mindset of delegating wound management to nurses also needs to change. Despite its growing importance, current undergraduate medical and post-graduate family medicine training do not place much emphasis on chronic wound care education and training.[2]

The objectives of this module is to provide FPs with a basic understanding of the pathophysiology in the different phases of healing in acute wounds, classification of the risk factors and their mechanisms in causing non-healing in chronic wounds, and a systematic approach and strategies to enhance wound healing appropriate at the family physician level.

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NG JOO MING MATTHEW, Head Medical Services, Bright Vision Hospital; Consultant, Department of Family Medicine and Continuing Care, Singapore General Hospital

FOUR PHASES OF HEALING IN ACUTE WOUNDS

Wound healing is achieved through four coordinated and overlapping phases (Figure 1)[3], 1) haemostasis, 2) inflammatory, 3) proliferative and 4) remodeling. These complex interactions ensure successful wound healing. Any disruption to this sequence and time frame can result in suboptimal or delayed healing, resulting in progression of an acute wound to a chronic wound. A basic understanding of the pathophysiology in various phases of wound healing (Table 1)[4] will allow the FP to appreciate the factors affecting wound healing and the strategies to enhance wound healing. However an in-depth discussion on the cellular and molecular mechanisms of wound healing is beyond the scope of this article.

1. Haemostasis (immediate)

The immediate response to skin injury is vasoconstriction and platelet-mediated activation of the intrinsic clotting cascade to achieve haemostasis within the first 5 to 15 minutes. Platelets release essential growth factors and cytokines (e.g., platelet-derived growth factor, transforming growth factor-β) that are important for the initiation and progression of wound healing. The resulting fibrin matrix stabilises the wound, provides a provisional scaffold for the arriving neutrophils, monocytes, fibroblasts, and endothelial cells and concentrates the cytokines and growth factors.

2. Inflammatory Phase (first 24-48 hours to two weeks)

Neutrophils and macrophages phagocytize debris and microorganisms and secrete a variety of chemotactic and growth factors such as fibroblast growth factors to direct the next stage of wound healing.

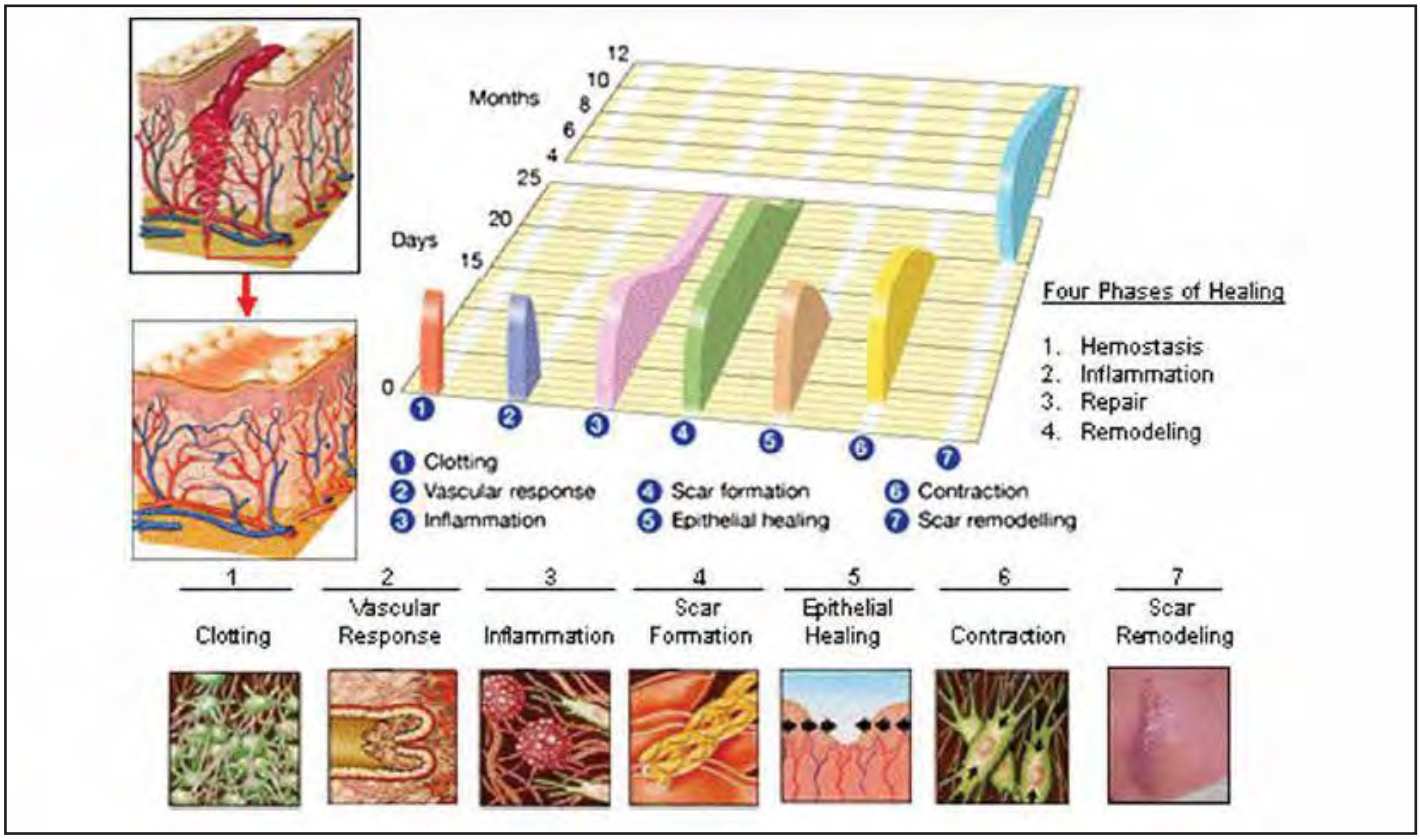
3. Proliferative Phase (Day 4 to 21)

The proliferative phase overlaps the initial phases of haemostasis and inflammation, and includes fibroplasia, epithelization, angiogenesis and provisional matrix formation. Epithelial cells migrate from the basement membrane and wound edges to fill the wound defect. Angiogenesis marked by endothelial cell migration and capillary formation is stimulated by tumour necrosis factor alpha. The final part of the proliferative phase is granulation tissue formation. Fibroblasts migrate into the wound site from the surrounding tissue, become activated, and begin proliferating and synthesising collagen. Some fibroblasts will transform into myofibroblasts for wound contraction to reduce the exposed area requiring repair by scar formation.

4. Remodelling Phase (few days after injury to 2 years)

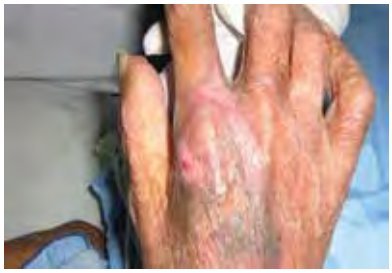
The main feature of this phase is the deposition of collagen in an organised and well-mannered network. Key elements of

FIGURE 1. SEQUENCE OF CELLULAR AND MOLECULAR EVENTS



Source: Stojadinovic A et al. Topical advances in wound care

TABLE 1. FOUR PHASES OF WOUND HEALING

Phase	Clinical Progress	Cellular and Bio-physiologic Events
Hemostasis	 *(Day 1 of injury)*	1. Vascular constriction 2. Platelet aggregation, degranulation, fibrin formation (thrombus)
Inflammation		1. Neutrophil infiltration 2. Monocyte infiltration and differentiation to macrophage 3. Lymphocyte infiltration
Proliferation	 *(Day 14 of injury showing epithelization)*	1. Re-epithelization 2. Angiogenesis 3. Collagen synthesis 4. Extracellular matrix formation
Remodeling	 *(Day 26 of injury)*	1. Collagen remodeling 2. Vascular maturation and regression

maturation include degradation of disorganised collagen, collagen cross-linking to enhance tensile strength, remodeling, wound contraction and repigmentation. The tensile strength of the wound is directly proportional to the amount of collagen. Maximum strength depends upon the interconnection of collagen subunits. However the collagen in the scar (even after a year of maturing) will never become as organised as the collagen found in uninjured skin and maximum final strength is approximately 80 percent.

PROGRESSION TO A CHRONIC WOUND

Chronic wounds develop when a wound fails to progress through the normal phases of healing and cannot be repaired in a timely, orderly manner to produce anatomic and functional integrity.[1,5] The 4 most common types of chronic wounds are arterial ulcers, venous ulcers, diabetic ulcers and pressure ulcers. Rarer causes include vasculitis, haematological conditions and malignancy.

RISK FACTORS AFFECTING WOUND HEALING OR CAUSING NON-HEALING IN CHRONIC WOUNDS

The complexity of wound healing makes it vulnerable to interruption at many levels. Multiple factors can affect physiologic responses and cellular function and disrupt wound healing by prolonging one or more phases of haemostasis, inflammation, proliferation or remodeling. A continuous state of inflammation in the wound creates a cascade of tissue responses that together perpetuate a non-healing state.

These factors can be categorised into local or systemic factors (Table 2)[4] for easy classification. Local factors directly affect the characteristics of the wound itself while systemic factors are the overall health or disease states of the patient that affects his or her wound healing ability through local mechanisms acting on the wound.

LOCAL FACTORS

Oxygenation

Healing is an energy dependent process and an adequate oxygen level is crucial for cell metabolism and optimal wound healing. Initial hypoxia stimulates wound healing through the release of growth factors and angiogenesis. Thereafter adequate oxygen sustains the healing process by inducing angiogenesis, increasing keratinocyte differentiation, migration, and re-epithelialisation, enhancing fibroblast proliferation and collagen synthesis, and promoting wound contraction.[5,6] In addition, the level of superoxide production (a key factor for oxidative killing pathogens) by polymorphonuclear leukocytes is critically dependent on oxygen levels.

Wound infection and foreign bodies

All wounds contain bacteria at levels ranging from contamination through colonisation to critical colonisation and finally to infection.[7] Contamination is the presence of non-replicating organisms on a wound, while colonisation is defined as the presence of replicating microorganisms on the wound without tissue damage. Critically colonised wounds with bacterial concentrations exceeding 10^5–10^6 bacteria colony-forming units per gram of tissue, or the presence of β-haemolytic streptococci exceeds the ability of host defenses to clear the bacterial biofilm and result in impaired healing. The bioburden precipitates an overproduction of serine proteases, leading to the degradation of the extracellular matrix. Several factors such as the bioburden, virulence and host resistance determine transition from colonisation to infection.[7,8] The presence of foreign bodies also prevent an effective immune response. The transition to infection occurs when bacterial proliferation overcomes the host's immune response and host injury occurs.[9] Infection interferes with epithelialisation, wound contraction, collagen deposition, prolonging the inflammatory phase and inhibiting normal progression to the proliferative phase of wound healing.

Biofilm

A biofilm is a complex community of aggregated bacteria embedded in a self-secreted extracellular polysaccharide matrix that acts as a physical barrier to the permeation and the action of antimicrobial agents.[10,11] The biofilm environment provides physical protection to the bacteria from a potentially hostile external environment and is also a habitat where bacteria can communicate with each other (quorum sensing), which may lead to an increase in virulence and propensity to cause infection.[12] Chronic wounds offer ideal conditions for biofilm production

TABLE 2. FACTORS AFFECTING WOUND HEALING

Local	Systemic
Ischaemia	Age and Gender
Infection	Stress
Foreign bodies	Sex hormones
Elevated tissue pressure/oedema	Alcoholism and smoking
	Diseases: diabetes, cardiovascular, respiratory diseases
	Obesity
	Medications: glucocorticoid steroids, non-steroidal anti-inflammatory drugs, chemotherapy drugs
	Immunocompromised conditions:Cancer, radiation therapy, Acquired immunodeficiency syndrome
	Nutrition

because proteins (collagen, fibronectin) and damaged tissues are present, which can allow attachment. The biofilm, in turn, becomes a primary impediment to the healing of chronic wounds.[13] Most of the chronic wound pathogens, such as methacillin resistant Staphylococcus aureus (MRSA) and Pseudomonas, are typical biofilm producers. Bacteria that reside within mature biofilms are highly resistant to many traditional therapies. Bacteria within biofilms have been reported to be up to 500 times more resistant to antibiotics than planktonic (unattached, freely living) cells.[14,15].

Oedema
Elevated tissue pressure from pressure or compartment syndrome induces capillary closure through its effect on critical closing pressure, causing prolonged, severe hypoxia.

SYSTEMIC FACTORS

Systemic factors act through the local effects that impact wound healing. These include age, gender, stress, sex hormones, diseases (e.g. diabetes, cardiovascular, respiratory diseases), obesity, medications (e.g. glucocorticoid steroids, non-steroidal anti-inflammatory drugs, chemotherapy drugs), alcoholism, smoking, Immunocompromised conditions (e.g. cancer, radiation therapy, acquired immunodeficiency syndrome) and nutrition. The mechanisms by which some of these factors affect wound healing are described below.

Diabetes mellitus (DM)
DM can affect wound healing via metabolic, vascular, and neuropathic pathways. Sorbitol accumulation and increased dermal vascular permeability results in pericapillary albumin deposition, which impairs the diffusion of oxygen and nutrients.[16] Hyperglycemia-associated nonenzymatic glycosylation inhibits the function of structural and enzymatic proteins essential for healing.

Age
Age-related changes are evident in all phases of healing, including enhanced platelet aggregation, increased secretion of inflammatory mediators, delayed infiltration of macrophages and lymphocytes, impaired macrophage function, decreased secretion of growth factors, delayed re-epithelialisation, delayed angiogenesis and collagen deposition, reduced collagen turnover and remodeling, and decreased wound strength.[17,18]

Nutrition
Protein malnutrition and particularly deficiencies in the amino acids arginine and methionine are associated with compromised wound healing because of prolonged inflammation and disruption of matrix deposition, cellular proliferation, and angiogenesis.[19,20] Glucose is the main fuel for wound repair and malnutrition is associated with decreased deposition of collagen in skin wounds. Micronutrients such as vitamins and minerals are critically important in immune function and wound healing.

Smoking
The harmful effects of smoking are related to toxic substances in the cigarette. Nicotine is a vasoconstrictive substance that decreases proliferation of erythrocytes, macrophages, and fibroblasts. Hydrogen cyanide is inhibitory to oxidative metabolism enzymes. Carbon monoxide decreases the oxygen-carrying capacity of hemoglobin by competitively inhibiting oxygen binding.[21] Smoking increases the individual's risk for atherosclerosis and chronic obstructive pulmonary disease, two conditions that might also lower tissue oxygen tension.

Steroids
The anti-inflammatory effects of steroids and suppression of cellular wound responses inhibit healing by reducing the effectiveness of phagocytosis by neutrophils and macrophages. Steroids also have a direct inhibitory effect on fibroblasts, and interfere with fibrogenesis, angiogenesis and wound contraction. In contrast, topical low-dosage corticosteroid treatment of chronic wounds has been found to accelerate wound healing, reduce pain and exudate, and suppress hyper granulation tissue formation in 79% of cases. While these positive results are promising, prolonged use should be carefully monitored as there is potential increased risk of infection.[22]

APPROACH TO WOUND HEALING AND STRATEGIES TO ENHANCE WOUND HEALING

A holistic approach to wound healing is essential as chronic wounds often complicate a patient's health and interact closely with the other co-morbid illnesses, social circumstances and functional status. Therefore the FP needs to address both patient and wound factors that impair wound healing.

A comprehensive patient history is necessary to identify the etiology, risk factors and disease states that impair wound healing. The patient's social circumstances, finances, function and care environment also impact on healing. Requirement for intensive wound care and a lack of caregiver to do or bring the patient for daily dressings may necessitate admission to a community hospital for wound management. Locally, home care nurses provide wound care support to patients who have difficulty in wound dressing and travelling to an outpatient clinic for dressing changes. Finally the psychosocial impact of a chronic wound on the patient and their caregiver's quality of life should not be neglected. The patient with a non-healing wound suffers from a reduced quality of life and may become socially isolated and depressed as a result of pain and discomfort, foul odor, discharge from the wound, reduction of his functional level and damage to his body image.[23]

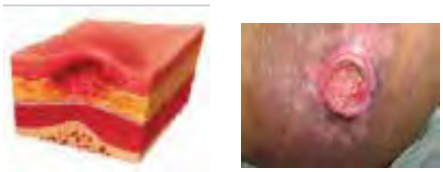
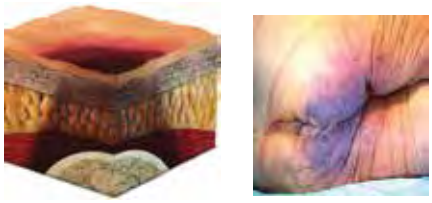
A detailed wound assessment should include the site, number, type, size and depth of wound, identifications of barriers to healing in the wound bed and stage of pressure ulcer if applicable[24] (Table 3). Measurement of the percentage reduction of wound area over time should be calculated to monitor the wound progress and identify need for treatment changes (Annex A). Serial photography may be helpful for documentation and can be an important part of ongoing wound assessment. The use of objective wound photography decreases inter-observer variability and allows for consistent and accurate assessment of

changes in wound area over time. A careful and accurate assessment of the neurovascular status is essential when a patient presents with a chronic wound of the extremity. A thorough neurovascular exam should include sensation testing, palpation of the pulses and capillary refill.

Three broad strategies to enhance wound healing are:

1. Optimise local wound care.
2. Identify and optimise the underlying causes for poor wound healing.
3. Education to the patients and their caregivers in wound care, frequency of dressing changes and the need for compliance to dressing changes and avoidance of risk factors to prevent recurrence.

TABLE 3 NPUAP STAGING SYSTEM FOR PRESSURE ULCERS

Stage	Illustration	Characteristics
Stage 1		Non-blanchable erythema of intact skin, localised usually over bony prominence. Coloration is pink, red or mottled after pressure is relieved. For patients with darker skin tone, blanche may not be visible. Colour may differ from the surrounding skin.
Stage 2		Partial-thickness skin loss of dermis; shallow open ulcer; red or pink wound bed without slough or bruising. May be intact or serum filled blister. Shiny or shallow ulcer; if bruised; suspect deep tissue injury; not skin tear, tape burn, perineal dermatitis maceration or excoriation.
Stage 3		Full thickness skin loss. Subcutaneous tissue may be visible. No tendon, muscle or bone is visible or palpable. Slough does not obscure depth of tissue loss; undermined or tunnelled depth varies with location: Shallow on bridge of nose, ear, occiput or malleolus or deep where fat layer is thick.
Stage 4		Full-thickness skin; exposed bone, tendon or muscle visible or palpable.Slough or eschar may be present on parts of ulcer. Often includes tunnelling or undermining. Depth varies with location: Shallow on bridge of nose, ear, occiput or malleolus; may extend into muscle and/ or supporting structure; osteomyelitis possible.
Unstageable		Full thickness tissue loss. Base of ulcer covered by: slough (yellow, tan grey, green) or eschar (tan, brown or black). Until enough slough or eschar is removed to expose wound base, staging cannot be determined. Do not remove stale dry black heel eschar.
Deep Tissue Injury		Purple or maroon discoloured skin or blood-filled blister, may be painful, warm or cool, boggy or firm. Difficult to detect if skin tone is dark. Evolution may include thin blister over dark wound bed; may progress to thin eschar cover. May evolve rapidly exposing additional layers of tissue, even with optimal treatment.

Source: Adapted from the National Pressure Ulcer Advisory Panel. Pressure Ulcer stages

ANNEX A. WOUND ASSESSMENT FLOW CHART

SNO	Date / Time						
1	**Wound Serial No**						
2	**Site of Wound**						
3	**Type of Wound**						
	Blisters						
	Skin tears (Category 1, 2 or 3)						
	Post-op wound						
	Diabetic Ulcer						
	Pressure Ulcer						
	Others: (Specify)						
4	**Size (in cm)** Length x Width x Depth						
5	**Pressure Ulcer Cannot Be Staged**						
6	**Pressure Ulcer Stages (1, 2, 3, or 4)**						
7	**Suspected Deep Tissue injury (sDTI)**	Y / N	Y / N	Y / N	Y / N	Y / N	Y / N
8	**Cavity with Undermining**	Y / N	Y / N	Y / N	Y / N	Y / N	Y / N
9	**Cavity with Tunneling**	Y / N	Y / N	Y / N	Y / N	Y / N	Y / N
10	**Wound Description**						
	Epithelial tissue (Pink)						
	Healthy Granulating tissue (Red)						
	Unhealthy Granulating tissue (Dull/Dusky)						
	Hyper Granulation (Red Raised)						
	Slough (Soft or Adherent Yellow)						
	Infected tissue (Green)						
	Necrotic tissue (Moist or Dry Black)						
11	**Exudates: Type/Color**						
	Serous						
	Haemo-serous						
	Sanguineous (Frank Blood)						
	Greenish/Yellow (may indicate infection)						
	Purulent / Haemo-Purulent (P / HP)						
12	**Exudates: Amount** – None (N) Slight (+) Moderate (++) Large (+++)						
13	**Wound Odor**	A / P	A / P	A / P	A / P	A / P	A / P
14	**Wound Related Pain**	A / P	A / P	A / P	A / P	A / P	A / P
15	**Peri-Wound Skin**						
	Erythema (skin redness)	A / P	A / P	A / P	A / P	A / P	A / P
	Induration (abnormal hardness of tissue)	A / P	A / P	A / P	A / P	A / P	A / P
	Skin Maceration(wound margins white)	A / P	A / P	A / P	A / P	A / P	A / P
	Edema (swelling)	A / P	A / P	A / P	A / P	A / P	A / P
	Others: (Specify)						
16	**Conservative Sharp Wound Debridement (CSWD) done**	Y / N	Y / N	Y / N	Y / N	Y / N	Y / N
	RN Signature						

- Tick√ or Circle(O) as appropriate
- A / P means Absent or Present
- Y / N means Yes or No
- Time: The time wound is assessed

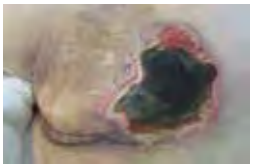
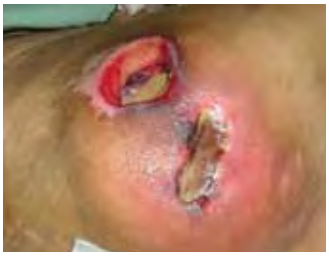
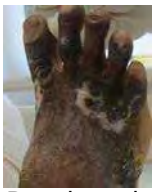
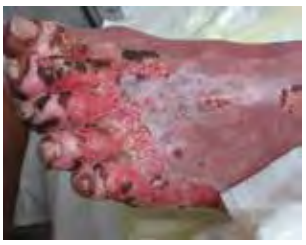
1. Optimising local wound care using wound bed preparation

Appropriate wound bed preparation removes local barriers to healing and accelerates endogenous healing or to facilitate the effectiveness of other therapeutic measures. The "TIME" acronym was first developed in 2003 by an international group of wound healing experts to provide a systematic and practical assessment and management of all the critical components of a non-healing chronic wound.[23] The clinical components of wound bed preparation according to "TIME" [Tissue, non-viable or deficient; Infection or Inflammation; Moisture imbalance; Non-advancing or undermined epidermal margin or Edge] defined the underlying pathophysiology of impaired healing, proposed wound bed preparation-based clinical interventions, outlined the effects of these interventions at a cellular level, and described anticipated clinical outcomes (Table 4)[25]. Wound bed preparation is the first step in the treatment of any chronic wound. The Advisory Board emphasised three important elements of wound bed preparation in chronic wounds: judicious debridement, management of exudate, and resolution of bacterial imbalance.[25]

T (tissue – non viable/ deficient) – T is for identifying the presence of non-viable tissue that manifests as necrotic tissue,

eschar and slough. Debridement is required to remove non-viable tissue and numerous techniques such as sharp surgical, autolytic (hydrocolloid, occlusive dressings), mechanical (curettage, waterjet), enzymatic, biological (maggot debridement therapy) are available. Selection of the appropriate method of debridement requires the consideration of the clinical setting and capability of the physician, patient's overall condition, nature and extent of the wound, exudate and presence of infection as well as the goal of therapy (removal of necrotic tissue versus preservation of granulation tissue). Autolytic debridement using hydrocolloid or occlusive dressings is most commonly used if a slow, more conservative option is preferred.

TABLE 4. THE PRINCIPLES OF WOUND BED PREPARATION (WBP) BASED ON "TIME"

Clinical observations	Treatment Objectives	Effect of WBP actions	Clinical outcomes
T (Tissue non-viable)  The letter 'T' indicates tissue debridement	Identify and debride the presence of non-viable tissue - Autolytic, sharp surgical, enzymatic, mechanical or biological	Restoration of wound base and functional extracellular matrix proteins	Viable wound base
I (Inflammation/Infection)  The letter 'I' characterises inflammation or infection within and surrounding the wound site	Determine the underlying cause of infection Identify the severity of infection in the wound Remove or reduce bacterial load using antimicrobials and debridement of non-viable tissue	Low bacterial counts or controlled inflammation: ↓ inflammatory cytokines ↓ protease activity ↑ growth factor activity	Bacterial balance and reduced inflammation
M (Moisture Imbalance)  Macerated  Dessicated The letter 'M' signifies Moisture Imbalance	Determine the reasons why the wound is out of moisture balance Identify the moisture status of the wound-dry (None), minimal (+), moderate (++) or heavy (+++) Select a suitable dressing that will either remove exudate if there is too much present, or add fluid to the wound if it is too dry	Restored epithelial cell migration, desiccation avoided Oedema, excessive fluid controlled, maceration avoided	Optimal moisture balance
E (Edge advancement)  The letter 'E' describes the quality of the wound edge, not advancing	Revisit TIM issues (Tissue, Inflammation /Infection and Moisture)	Migrating keratinocytes and responsive wound cells Restoration of appropriate protease profile	Advancing epidermal margin If edge of wound not advancing after two to four weeks, reassess interventions for improvement

Sharp surgical debridement and newer modalities such as low-frequency ultrasound and hydro-surgical debridement require advanced surgical knowledge. Advances in debridement technology such as low-frequency ultrasound, hydrosurgery and add-on use of negative pressure wound therapy or vacuum assisted devices have led to better outcomes, as have advances in traditional non-surgical debridement methods such as larval and enzymatic debridement.[26]

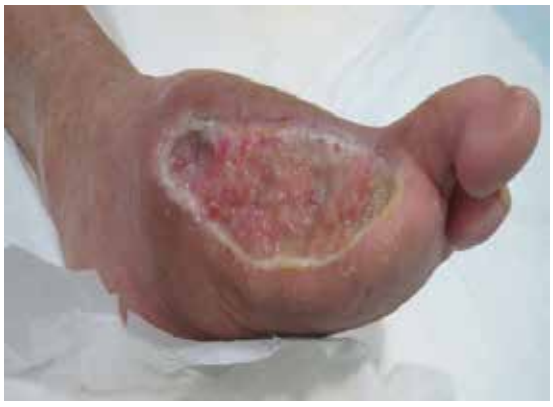
I (Infection/ Inflammation) – I is for the presence of inflammation or infection, or both. Inflammation is a physiological response to wounding but excessive or inappropriate inflammation, often in the presence of infection, may impair wound healing. While low levels of bacteria can facilitate wound healing by producing enzymes such as hyaluronidase that stimulate neutrophils, excessive bacteria burden leads to a continued inflammatory response which eventually leads to overt wound infection, and/or a systemic toxicity. Signs of infection in chronic wounds include delayed healing, increased exudate, bright red discoloration of granulation tissue, friable and exuberant tissue, new areas of slough, undermining, malodour and wound breakdown.[27] Deep infections can cause erythema and warmth beyond wound margins. Redness, heat, pain, swelling, and exudate may be minimal or absent as a result of the presence of factors that commonly contribute to the formation of chronic wounds. Comprehensive wound care must include cleansing, debridement, and exudate management.

Most chronic wounds are invariably colonised, and therefore, superficial swabs cultures should be avoided. Ideally, quantitative or semi quantitative tissue cultures should be obtained to guide antibiotic therapy. A properly obtained swab culture may be helpful in routine clinical practice. Anti-pseudomonal coverage is important for non-healing wounds more than 4 weeks old with deep tissue infection (e.g., cellulitis extending N1 cm beyond the wound margin) and systemic response (fever, chills, night sweats, rigors). Systemic antibiotics with appropriate Staphylococcal, Streptococcal, coliform, and anaerobic coverage[28-30] should only be used in the treatment of sepsis, osteomyelitis, cellulitis, lymphangitis, abscess formation, and other signs of invasive tissue infection. Continued topical antimicrobial therapy is advised as systemic antibiotics do not reach therapeutic levels in the relatively avascular infected wound tissue.[31-32] Biofilms should be considered if wounds fail to improve or degenerate despite a healthy appearance (Figure 2). The best way to disrupt biofilm is by debridement. Sharp debridement physically disrupts and removes biofilm and regular debridement to reduce the biofilm potential for regrowth. Once disrupted, the biofilm is more vulnerable to antimicrobials and use of a topical broad-spectrum antimicrobial such as silver or iodine or topical antiseptic solutions such as Prontosan® can also prevent biofilm reconstitution.

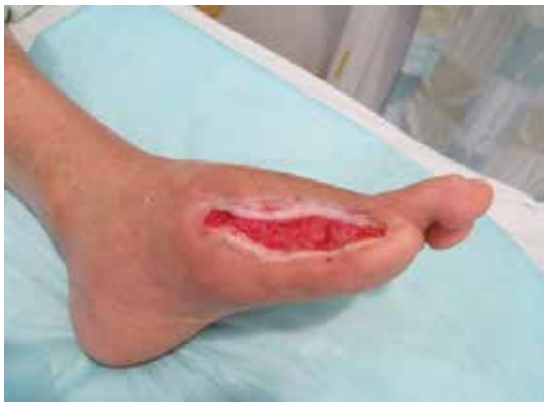
M (moisture imbalance) – M describes the state of moisture balance, ranging from dessication to maceration. Appropriate wound moisture is required for the action of growth factors, cytokines and cell migration. Too much exudate can cause damage to the surrounding skin, while too little can inhibit cellular activities and lead to eschar formation, which inhibits wound healing. Chronic wound fluid has high levels of proteases and pro-inflammatory cytokines and elevated levels of MMPs. This increased proteolytic activity damages the wound bed, degrade the extracellular matrix and aggravate the integrity of the peri-wound skin[33], while the high levels of cytokines promote and prolong the chronic inflammatory response seen in these wounds.[34]

Dressings should maintain an appropriate moisture balance and avoid maceration or dessication of the wound bed. Choosing a topical dressing to restore moisture balance in a wound depends on the amount of exudate, the anatomic location of the wound, the presence of dead space, the condition of surrounding skin, the caregiver ability, whether or not healing is expected, and product cost. The ideal dressing for patient comfort and convenience is one that is not bulky, not painful to change and reduces the number of dressing changes needed. The status of the wound bed determines the type of therapeutic intervention required to restore moisture balance in the wound. If the wound is dry or desiccated, moisture should be added. Modern dressings

FIGURE 2. TREATMENT OF BIOFILM WITH PRONTOSAN SOLUTION



Picture on left shows Right Ray's amputation wound with presence of biofilm.



Picture on right shows improvement in wound bed after cleansing with Prontosan solution.

fulfill the dual role of removing exudates and maintaining a moist wound environment that support wound healing. Cavities should be filled but not over packed to allow for granulation and epidermal migration.[35] Specific examples of appropriate type of dressings for different wound types will be further elaborated in the module on "Types of Dressings".

Negative pressure wound therapy involves the application of a controlled sub atmospheric pressure to a wound covered with a foam dressing and is proving to be an increasingly valuable tool to enhance wound healing. It reduces oedema surrounding the wound, stimulates circulation, and increases the rate of granulation tissue formation. Negative pressure wound therapy is useful to manage large defects until closure can be performed. It has also been used with modest success in the treatment of pressure ulcers[36,37], and diabetic wounds.[38, 39]

E (edge of the wound, epithelium) – E refers to the wound edge, whether it is non-advancing or undermined, or the extent of re-epithelialization. The final stage of wound healing is epithelization, which is the active division, migration and maturation of epidermal cells from the wound margin across the open wound.[40] Epithelial edge advancement and an improved state of the surrounding skin is the clearest sign of healing. A 20-40% reduction in wound area after 2 and 4 weeks of treatment is seen as a reliable predictive indicator of healing and confirm either the effectiveness of the wound treatment being used or the need for re-evaluation. New therapies to improve wound edge epithelialization include electromagnetic therapy; laser therapy, ultrasound therapy and negative pressure wound therapy.

2. Identify and optimise the underlying causes for poor wound healing

The underlying cause of the chronic wound/ulcer should be addressed whenever possible. Patients with critical ischemia from severe peripheral vascular disease should be considered for revascularisation with angioplasty or bypass surgery to improve the vascular supply and oxygenation to the wound. Definitive surgery such as ligation of the saphenopopliteal junction, stripping of the long saphenous vein with multiple stab avulsions should be considered for patients with chronic venous insufficiency and venous ulcers. For pressure ulcers, strategies to relieve pressure, shear and moisture include frequent repositioning every 3-hourly using the 30 degree tilt, pressure relieving mattresses, regular changing of diapers and insertion of a urinary catheter.[41]

Although there is insufficient clinical evidence to support tight short-term glycaemic control or routine nutritional supplementation to enhance wound healing outcomes[41-43], most guidelines recommend a minimum calorie intake of 30–35 kcal per kg per day, protein intake of 1.25-1.5g per kg per day, fluid intake of 30 ml per kg per day and optimal glycaemic control when treating wounds and infections. Patients with modifiable risk factors such as smoking and steroids should be encouraged to stop if possible and for the additional health benefits.

3. Patient and caregiver education

Patient education should start from day of admission to hospital or during presentation in the doctor's clinic. Patient and caregiver should be taught fundamentals such as:

1. Hygiene: Caregiver should maintain hand hygiene during the dressing procedure. They should be taught proper hand washing technique.
2. Wound care: Caregiver should be taught step by step methods of caring for the wound and the proper selection of dressings.
   a. Make sure all supplies are available.
   b. Remove the old dressing and discarding it properly in a bag.
   c. Inspect the wound for depth, size and odour.
   d. Look out for systemic signs of infection such as fever, confusion and increasing redness.
   e. Dressings should only be use once only.
3. Managed expectation. Patients should be made aware that wounds take time to heal. Superficial wounds involving the epidermis and dermis usually healed faster that deep wounds involving the deeper fat and muscle layers.
4. Proper nutrition, eat a balance diet with increase fluids and proteins. Supplementation with vitamins maybe necessary to promote healing.

ROLE OF ADJUVANTS

Many adjuvants are available to help with the treatment of chronic wounds, but good quality randomised trials and strong evidence on their effectiveness to support routine use are still lacking. Becaplermin is a platelet-derived growth factor (PDGF) gel preparation that promotes cellular proliferation and angiogenesis, and thereby improves wound healing.[44] It is approved for use in the United States as an adjuvant therapy for the treatment of non-infected diabetic foot ulcers with an adequate vascular supply and is the only pharmacological agent approved for treatment of chronic wounds. Hyperbaric oxygen therapy (HBOT) has been used as an adjunct to wound care in the therapy of acute and chronic ulcers due to venous, arterial and diabetic disease. Although hyperbaric oxygen may benefit some types of wounds (e.g., diabetic ulcers), systematic reviews have concluded that there is insufficient evidence to support its routine use.[45-47]

There is some evidence of improved outcomes with the use of ABOUND® or IMPACT®, which is a targeted therapeutic nutrition drink mix containing Revigor, arginine and glutamine that has been clinically shown to support tissue repair,[48] and to help build and maintain lean body mass (LBM).[49] Cilostazol and pentoxifylline have been used with some success in the treatment of arterial ulcers and venous ulcers respectively.[50,51] However these treatments are not routinely practiced in Singapore and definitive treatment such as revascularisation and chronic venous insufficiency surgery such as ligation and stripping should be performed if indicated.

CONCLUSIONS

Many local and systemic factors can affect the physiologic

responses and cellular function to disrupt the wound healing process. The Family Physician should take a holistic approach to wound healing as chronic wounds are part of a patient's health problems and interact closely with his other co-morbid illnesses, social circumstances and functional status. Strategies to enhance wound healing include wound bed preparation using the TIME acronym, optimising and removing underlying risk factors for poor wound healing and patient education on dressing changes and avoidance of risk factors. Many adjuvants are available but their routine use is not supported by current evidence.

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LEARNING POINTS

- **Chronic wounds complicate a patient's health and interact closely with the other co-morbid illnesses, social circumstances and functional status. Therefore the Family Physician needs to address both patient and wound factors that impair wound healing.**
- **There are three broad strategies to enhance wound healing:**
  i) Optimise local wound care.
  ii) Identify and optimise the underlying causes for poor wound healing.
  iii) Education to the patients and their caregivers in wound care, frequency of dressing changes and the need for compliance to dressing changes and avoidance of risk factors to prevent recurrence.
- **The TIME principles should be used for local wound bed preparation.**
- **Adjuvants such as Hyperbaric oxygen therapy (HBOT) and Vacuum assisted closure (VAC) devices are promising to enhance wound healing but stronger evidence are required to define its roles for specific wounds and support routine use in clinical practice.**

UNIT NO. 2

WOUND DRESSINGS: A PRIMER FOR THE FAMILY PHYSICIAN

Dr Lee Mei Gene Jesmine, Dr Pan Yow-Jeng Franny, Yang Leng Cher, Dr Ng Joo Ming Matthew

ABSTRACT

Given the myriad of choices available on the market, selecting the appropriate wound dressing remains a challenge for most healthcare workers. It is important to exercise discretion and adopt a systematic approach in dressing selection following wound assessment, as this will directly impact on rates of wound healing, which in turns affects the patient's quality of life and overall healthcare costs. This paper provides an overview of the common types of wound dressings in use currently and gives a brief synopsis of some of the latest advances in wound care technology and their applications in management of complex wounds. The consensus to date is for the use of hydrogels in the debridement stage, foams and low-adherence dressings in the granulation stage and hydrocolloids and low-adherence dressings for the epithelialization stage. Additional studies and research need to be undertaken to further evaluate the application of advanced wound technology in clinical practice.

Keywords:
Wound dressings, Wound care

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INTRODUCTION

It is of emerging importance that doctors are equipped with skills in proper wound management; since it is not only a common problem outside of the acute-care setting, but is of increasing prevalence in our rapidly ageing population in the community[1,2]. The estimated cost associated with healing of an ulcer can be as high as $45,000 and this does not account for the decreased quality of life, restricted mobility, psycho-social impact and/or intractable pain associated with the wound[1,3].

As physicians, we should familiarise ourselves with the different types of dressings available and know how to choose the appropriate dressings for different types of wounds. With a better understanding of the wound healing process at the cellular level, as well as interactions of the cellular components found within the chronic wound environment, better products are now being created to change the wound milieu to aid the healing process. This article aims to help the family physician navigate through the jungle of wound products; and shed some light on the latest advances in wound care technology.

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WOUND DRESSINGS AND FACTORS AFFECTING SELECTION

Wound dressings are described as primary where materials are placed into wound beds and interact with the actual wound surface, while those described as secondary refer to dressings that are used to cover and secure the primary dressings in place.

The key to understanding the various types of wound dressings is to learn the basic properties of each category of wound dressing. The dressings within each category are not identical, but they do possess many of the same properties.

Wound dressings can also be described as passive (inert) or interactive. **Passive dressings** simply serve a protective function and do not actively interact with wound properties to facilitate wound healing. An example is gauze. Although they remove excess exudates, the fibrous nature of the dressing increases its potential for leftover lint and particulate materials in the wound. This introduces foreign bodies into the wound environment and increases the risk of infection. Furthermore, it adheres to the wound surface causing trauma and pain during change. The damage to the neodermis delays wound healing. On the other hand, **interactive dressings** not only create a moist wound environment, but actively interact with local wound properties such as exudates and growth factors to accelerate wound healing. They promote healing through reduction of bacterial colonisation and level of exudates, retention of moisture, strengthening wound collagen matrix, removal of cellular products and protection of the epithelializing bed[4,5].

It must be stressed that an ideal dressing for all wound types does not exist (see Table 1: Characteristics of an ideal dressing). There is no single dressing that will be able to manage all the nuances within the wound environment. Adequate wound assessment is vital; this is the cornerstone of dressing selection. A wound is an evolving entity; the same dressing cannot be used from the beginning to the end. Dressings are selected according to wound characteristics; therefore when the wound changes, so should the dressing. At each dressing change, it is advisable to review the condition of the wound, as this allows for monitoring of the effectiveness of the previous dressing used. This includes measurement of the wound, as well as taking photographs. Review the treatment objectives and select the appropriate dressings (See Figure 1).

An invaluable consensus list of recommendations published in 2007 by a panel of wound experts advocated the use of hydrogels in the debridement stage, foams and low-adherence dressings in the granulation stage and hydrocolloids and low-adherence dressings for the epithelialization stage[6]. The panel also made specific suggestions regarding the use of low adherence dressing on fragile skin, alginates on bleeding wounds and activated

FIGURE 1. REVIEW TREATMENT OBJECTIVES AND SELECT APPROPRAITE DRESSINGS

Patient A	Treatment objective	Types of dressing	Outcome
	1. Determine the underlying cause of infection 2. Identify the severity of infection in the wound 3. Remove or reduce bacterial load using antimicrobials and debridement of non-viable tissue	• Silver dressing • Cadexomer Iodine • Metronidazole gel • Systemic antibiotics	Reduce inflammation and infection

charcoal dressings on malodorous wounds[6]. Besides the recommendations, the following points should also be assessed when choosing the appropriate dressings[7,8,9]:

- Etiology of the wound
- Wound site, size and position
- Current state of the wound and surrounding skin
- Amount of wound exudate
- Presence of infection
- Characteristics of wound dressings (Table 1)
- Contraindications to dressing use e.g. allergies
- Ease of application, change and removal
- Need for secondary dressing

Hand in hand with dressing selection comes the question of frequency of dressing change. This is a decision made based on clinical judgment. If the dressing is soiled, loose, slipping or curling at the edges, it is obvious that it should be changed. If there is accumulation of fluid and/ or debris and the dressing is saturated, it needs change. If infection is present, increased frequencies of change need to be considered. Most dressings come with manufacturer recommendations on the frequency of change or how long each dressing can maintain its efficacy; however these should only be used as guidelines, clinical judgment still rules.

The ideal wound dressing should provide the optimum environment to meet treatment objectives and protect the wound from further injury. See Table 1.

CATEGORIES OF WOUND DRESSINGS

Traditionally, dressings are classified into seven different categories. These are gauze, films, alginates, foams, hydrogels,

TABLE 1. CHARACTERISTICS OF WOUND DRESSINGS

Characteristic of dressings:	Rationale:
Promotes or retains moisture	Dry wound bed inhibits wound healing
Manages excess exudates	Prevents maceration and further wound breakdown
Provides thermal insulation	Reducing temperature at wound bed reduces fibroblast activity
Impermeable to bacteria	Prevent exit and entry of bacteria
Causes minimal trauma on removal	Prevents damage and reduces pain
Cost effective	Makes best use of available resources
Available in hospital and community	Accessible to all carers

hydrocolloids, and composite dressings. However, with better understanding of wound healing and improvement in technology such classification no longer suffices (Refer to Table 2 for types of common wound dressings and their indications).

For practical purposes, the dressings in this paper are broadly divided into five categories: Moisture-retentive dressings, absorbent dressings, anti-microbial dressings, composite dressing and protective dressings. The applications and limitations of each will be discussed in further detail in each section.

1. Moisture Retentive Dressings

Moisture in the wound environment is needed to increase epidermal cell movement, retain growth factors, increase angiogenesis and decrease fibrosis[10]. These dressings not only serve as an effective barrier to trauma and microbes but allow for less frequent dressing change and reduce pain and scar formation[10].

Hydrocolloids - Made from gelatin, sodium carboxymethylcellulose or pectin with a polyurethrane waterproof outer layer, these are adhesive, occlusive and conformable dressings[11]. By trapping protein and cytokine-containing exudate, hydrocolloids promote autolytic debridement, increase cellular proliferation, and encourage granulation tissue formation and epithelialisation of low to moderately exudative wounds[4,5,10,11]. The advantage of this dressing is that it can be left in place for 2-4 days provided that the wound is not infected[10]. Users must be aware of the possible maceration to surrounding skin and its tendency to produce a brown and malodorous exudate often mistaken for infective exudates[10,11].

Hydrogels - They are composed of a matrix of insoluble modified carboxymethycellulose polymers with propylene glycol humectant[4]. Hydrogels contain 60-70% water and are available in sheets or liquid gel dressings embedded in gauze[12]. These soothing and absorbent dressings are most ideal for wound rehydration facilitating natural autolysis of necrotic tissue[4,5]. It is non-adhesive, easy to use (requires change every 2-3 days), cause minimal pain on removal and is cost effective[11]. A secondary dressing is usually needed to hold hydrogels close to the wound bed.

Films - Films are made from thin and semi-permeable sheets of polyurethane[5,12]. They are most useful in holding primary dressings in place especially over the joint areas and uneven wound surfaces as they are highly adherent and flexible[5,12]. They are frequently used to protect the skin from friction and shear forces but extra caution must be practiced when removing these highly adhesive dressings[7,10]. Being transparent and permeable to air and water vapour, the wound bed and moisture level is easily visualised[5,10].

2. Absorbent Dressings

Absorbent dressings play an important role in the management of moderate- heavily exudative wounds. Their main function lies in absorbing exudates whilst minimally adhering to the wound bed[11]. The amount of fluids that can be handled varies with each product. These dressings are more costly compared to the traditional gauze but they have been found to reduce overall cost and treatment time[11].

Alginates - Alginates are composed of calcium or sodium salts of alginic acid derived from brown seaweed (Phaeophyceae)[4,13]. They are available in sheets, ribbons, beads or pads[10]. Alginates partially dissolve on contact with wound fluid to form a gel that is able to absorb up to 20 times its own weight hence it is recommended to be used on wounds with moderate to heavy level of exudate[5,11]. They promote healing and granulation by maintaining a physiologically moist environment ideal for healing. An important advantage of alginates lies in its haemostatic property allowing it for use in minor bleeds[4,11]. Some have added silver for antimicrobial effects. Alginate dressings can be used to fill a cavity but should always be covered with a secondary dressing. Issues limiting the use of alginates include peri-wound maceration and residual fibres in the wound after removal[11].

Hydrofiber - These are white fibrous dressings compose of 100% Hydrofiber (sodium carboxymethylycellulose)[4,10]. Hydrofibers are best used for moderately exudative wounds because of its capacity to absorb large amounts of wound exudate and bacteria to create a soft, cohesive gel that conforms to the wound surface[4,10]. This helps with autolysis and removal of necrotic material from the wound surface. Some have added silver for its antimicrobial properties[4]. It can be easily removed in one piece without causing trauma to the underlying wound[4,10].

Foam dressings - These are semi occlusive dressings manufactured as polyurethrane or silicone foams. They are non-adhesive and much thicker than most other dressings. Being soft and conformable, they can provide padding over bony prominences such as heel, ankle, sacrum and hip[10]. Foams are also absorbent and can be used over mildly and moderately exudative wounds[10]. They have an additional benefit of providing thermal insulation and moisture vapour and oxygen to the wound, allowing for enhanced rates of wound healing[5]. Some have added silver for antimicrobial effects and they can last up to seven days.

3. Antimicrobial Dressings

It has been found that the presence of any trace of β-hemolytic streptococci or bacterial concentration over 10^5 or 10^6 bacteria colony-forming units per gram of tissue in wound is associated with impaired healing[14]. The recommendation to date is to reduce or eliminate the bioburden through a combination of frequent debridement, vigorous physical cleansing, and use of appropriate dressing material, extensive high-dose systemic antibiotics or topic biocides to disrupt its reconstitution[15]. The following section describes some of the readily available types of antimicrobial dressings.

Cademoxer Iodine - Cademoxer iodine is released from a starch lattice when it comes in contact with the wound exudate to exert its broad spectrum bacteriostatic activity against organisms including Staphylococcus aureus and Pseudomonas aeruginosa[15]. 1 g of Cademoxer iodine is able to absorb up to

7ml of fluid, making it a useful dressing for infected wounds[5]. Because iodine may be absorbed systematically, it should be avoided in patients with thyroid disorders[5].

Silver – Silver comes in many different forms including elemental, Inorganic and organic silver available in various formulations[10]. It combines properties of broad spectrum antimicrobial action, toxin and odour control. Upon exposure to moisture, the inert metallic silver (Ag^0) is converted to the reactive silver ion, Ag^+, which is the active antimicrobial agent[15]. Once it comes in contact with wound exudate, there is exchange of Ag^+ (dressing) with negatively charged particles such as DNA, RNA and chloride ions[16]. Its broad spectrum bactericidal action covers gram-positive, gram-negative bacteria, yeast and fungi. Silver is not only of low toxicity to skin but rates of bacteria resistance to Ag^+ have been found to be extremely low[16]. Silver preparations are available in the form of silver nitrate and silver sulfadiazine and nanocrystalline silver technology[16]. Whilst in the past, silver nitrate preparations had to be applied up to twelve times a day to maintain its effectiveness, the newer preparations can exert effects that last up to 7 days[16]. A major disadvantage of silver product is its potential to cause discolouration or irritation to surrounding skin (argyria)[11].

Honey - A recent Cochrane review showed that honey may improve healing times in mild to moderate superficial and partial thickness burns though it has limited benefits for other types of ulcers[10,17]. Honey dressings have gained popularity in treatment of other wounds in recent years due to its anti-inflammatory, antimicrobial and debriding properties[18]. The nectar from the Leptospermum plants is harvested by the honey bee (Apis Mellifera) and it is formulated into a gel or impregnated dressing[18,19]. The high sugar content results in a highly osmolar wound environment which makes it non- conducive for bacterial growth[18,19]. In addition, it has been shown to stimulate granulation and epithelialization and reduce pain and edema[18].

4. Composite Dressings

Composite dressings are multi-layered dressings that can be used as primary or secondary dressings. They usually comprise of three layers, an inner non-adherent layer, a middle area that absorbs and wicks away moisture, and an outer semipermeable film. The inner non-adherent layer prevents trauma to the wound bed during dressing change, the middle layer can consist of a hydrogel, hydrocolloid or alginate which provides a moist wound healing environment and the outer layer serves as a barrier to bacteria. These dressings are pre-packaged, have less flexibility in terms of indications of use and can be costly. Their water proof nature makes them a popular choice for areas prone to moisture assault from incontinence.

5. Protective dressings

Gauze- plain gauze, made of cotton, is inexpensive, readily available, and most useful as secondary dressings in most wounds. It is available in square dressings or rolled forms[10]. Gauze may promote wound dessication[16] in wounds with minimal exudates unless they are impregnated with zinc, iodine or petrolatum or used in combination with another type of dressing.

Non adherents - Composed of porous silicone or tulles, they are often used as a primary dressing for lightly exuding or granulating wounds[4,5,12]. Some have limited capacity for absorption and strikethrough can occur; while others are more absorbent and can be used for moderately exudative wounds. Being non adherent, these dressings are most useful when pain during dressing application and change is the main concern or in patients with sensitive or fragile skin[5].

ADVANCES IN WOUND CARE TECHNOLOGY

The art of wound care has evolved throughout the ages. A papyrus dating back to 3000 BC was discovered by American Egyptologist Edwin Smith in 1862. When it was finally translated in 1930, it was found that the ancient Egyptians used a paste out of honey, grease and lint to remove necrotic tissues and promote healing in open wounds[10]. Strips of linen and sticky gum were described to have been used to close wounds and green copper pigment and chyrsoedla used as antiseptics in open wounds. During the war time in the 19th century, various remedies from boiling oil to concoctions of turpentine, egg yolks and rose oil were used to treat firearm wounds[10]. Today, the wound care scene is going through another wave of revolution with the invention and application of novel techniques and modalities. Although most are resource intensive and lack the high level evidence to validate their integration into regular clinical practice, their contribution to wound care should not be undermined as their potential impact on the total cost of care in the long term may justify their higher cost per treatment[20]. This section provides a brief summary of some of the advances in wound care.

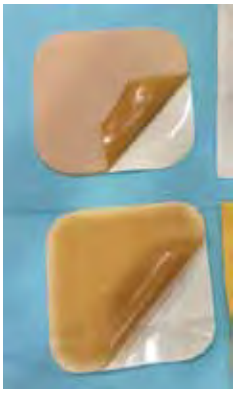
Maggot debridement therapy (MDT)

The first postulated mechanism of action of MDT is from the wriggling and the probing of the hook and the mandibles of the maggots on the wound bed[23]. It was later found that the proteolytic action from the saliva of the green bottle fly larvae (Lucilia Phaenicia) served as a form of biologic debridement through liquefaction of necrotic tissue, providing antimicrobial and wound healing effects[10]. The larvae used need to be medical grade sterile and left in the wound bed for 48-72 hours and changed[10]. To optimise effects of MDT, the maggots require optimal body temperature with adequate oxygen and moisture. Indications for maggot therapy include disinfection of chronic sloughy necrotic wounds[23]. In the past few years restructured hospitals like Tan Tock Seng Hospital; Singapore General Hospital and National University Hospital have been offering maggot therapy for wound debridement. Once the wound is deemed suitable for maggot debridement, the maggots are placed on a gauze or in a bag and applied onto the wound bed. After 2 days the dressings are removed and the maggots are flushed away by saline. This treatment typically takes up to 2 to 3 applications over the course of a week.

Growth factors - Recombinant human platelet derived growth factor (PDGF)

Growth factors (GFs) promote angiogenesis, stimulate fibroblasts and granulation tissue formation[20]. Beneficial effects

TABLE 2. TYPES OF COMMON WOUND DRESSINGS [4, 5, 7, 10, 12, 16]

Types of Dressing	Indications	Special Considerations	Examples
Hydrocolloids			
	• Dry and desiccated wounds • Abrasions • Necrotic eschars • Wounds with minimal exudates • Superficial or healing wounds	• Not recommended for highly exudative or infected wounds, diabetic foot ulcers and other wounds requiring frequent wound inspection • Beware fragile skin due to potential for maceration of surrounding skin • During application, foams size must be extended beyond wound edges to ensure good adherence • Silver can be applied under the hydrocolloid dressing centrally for antimicrobial effects	• Duoderm • Comfeel
Hydrogels			
	• Very dry and minimally exuding wounds • Necrotic wounds • Arterial ulcers • Dry venous ulcers • Warfarin induced necrotic wound • Rheumatologic ulcers	• Gels may be squeezed directly into cavity and covered with a secondary dressing • Periwound skin may need protection from maceration (PP)	• Purilon • Duoderm Hydroactive Gel
Alginates			
	• Recommended for highly exudative and deep wounds e.g. chronic pressure ulcers • Can also be used for split skin graft donor site and • diabetic foot wounds, • heavily exudative venous leg ulcers	• Users may experience foul odour but may be from seaweed rather than wound itself • Not recommended for use on dry wounds • Due to low tensile strength, avoid packing into deep sinuses • Can be used as part of a multilayer compression wrap on lower limbs	• Algisite • Algisorb • Seasorb • Kaltostat • Biatain Alginates
Hydrofiber			
	• Moderately exudative wounds	• Not recommended for use in bleeding wounds, dry or necrotic wounds • Due to low tensile strength, avoid packing into narrow deep sinuses • Cost effective	• Aquacel • Aquacel-Ag • Aquacel-Ag rope

Types of Dressing	Indications	Special Considerations	Examples
Foams (polyurethranes or silicone)			
 	• Wide range of moderate to highly exudative wounds • Wounds subjected to sustained or unrelieved pressure	• Occlusive foams without silver should not be used on infected wounds • Not suitable for dry or eschar covered wounds • May require secondary dressing to keep in place	• Allevyn, • Allevyn Gentle • Mepilex Ag • Mepilex Lite • Biatain Ag • Hydrasorb
Cademoxer iodine			
	• Chronic exuding infected wounds • Infected diabetic ulcers • Pressure ulcers	• Beware hypersensitivity to iodine • May need systemic antibiotics if evidence of deeper tissue infection	• Iodosorb powder, ointment and paste
Silver barrier dressing			
	• Infected wounds especially when antibiotic resistance is a concern	• May need systemic antibiotics if evidence of deeper tissue infection • Silver absorbed into the skin may cause argyria, which is a permanent depigmentation of skin	• Silver Nitrate • Silver sulphadiazine • Ionic silver available in acticoat
Non adherent synthetic			
	• Mainly used as a primary dressing on lightly exuding or granulating wounds • Painful or friable wounds • Wounds requiring application of topical medications	• May require secondary dressing • Strikethrough may occur with heavier level of exudates	• Primapore • Mepitel • Meloline

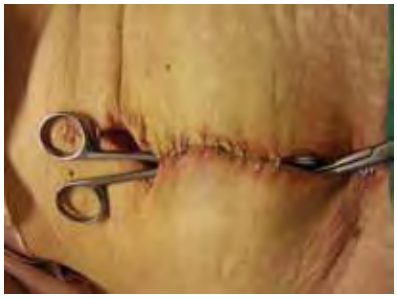
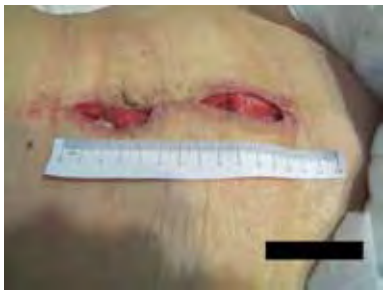
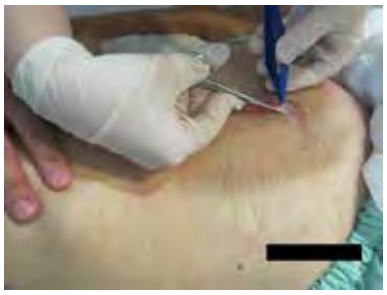
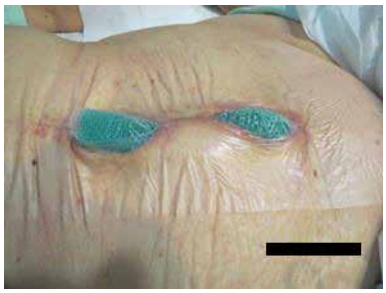
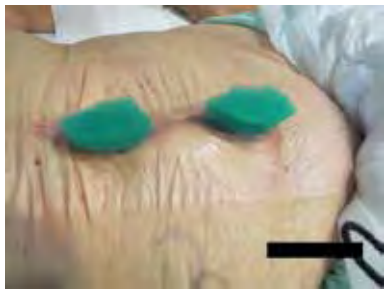
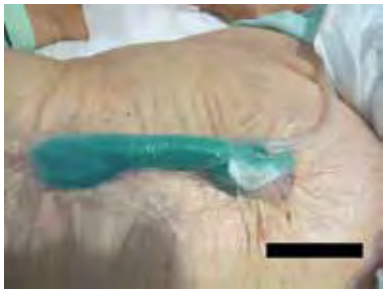
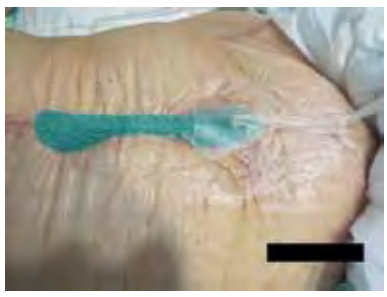
Types of Dressing	Indications	Special Considerations	Examples
Films/ membranes			
 	• Primary or secondary dressings for minimally exudative or dry wounds • Superficial lacerations • "difficult" anatomical sites for e.g. over joints • Minimally exudative wounds including thin burn wounds, venous catheter sites, donor sites for split skin grafts or partial thickness wounds	• Skin around wound must be intact for a good seal • can be used as a secondary dressing in combination with alginates or hydrofibers • Avoid in draining or infected wounds	• Tegaderm • Opsite
Gauze			
	• Highly exudative wounds • Useful as a secondary dressing	• May adhere to viable areas of wound bed and cause pain during removal	• Gauze • Vaseline gauze • Xeroform and • Telfa
Composite Dressing			
	• Use as Primary or secondary dressing	• Combine physically distinct components to a single product to provide multiple functions • Serve as bacterial barrier, absorbent and a adhesion	• Primapore • Versiva

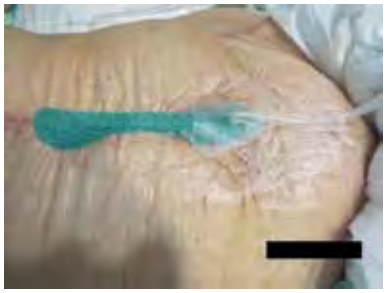
of GFs such as platelet derived Growth Factor (PDGF) and Fibroblast Growth Factor (FGF) in wound healing have been demonstrated in clinical trials[20]. Research is currently ongoing with trials on hepatocyte growth factor and other cell therapy products that contain lymphocytes, monocytes and neutrophils[20]. Becaplermin is a FDA approved PDGF- derived gel that has shown efficacy in diabetic ulcer healing; however, it is also associated with increased rates of malignancy[10].

Bioengineered skin substitutes

Both synthetic and cultured autologous engineered skin can be used as a source of non- senescent fibroblasts in promoting wound healing[16]. The two major types currently available are living and non-living cell/tissue[17]. Problems of rejection and possible transmission of disease are potential setbacks in the development of allografts and xenografts. Skin substitutes have established its place mainly in the realm of burns and large wounds[16].

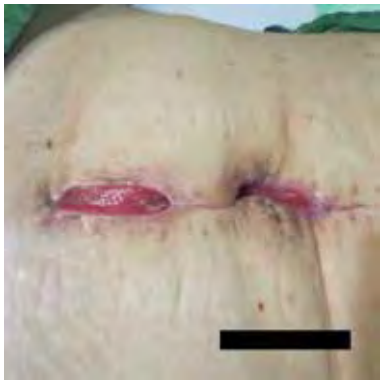
FIGURE 2. NEGATIVE WOUND PRESSURE WOUND THERAPY

Patient B	NPWT/VAC dressing
 Abdominal wound with 2 gaping wound after alternate removal of stitches	
 Prepare required foam size and silicone dressing according to wound size for insertion to wound bed	 Place sterile film on abdominal wound first and cut film open to expose the two wound cavity.
 Insert foam with silicone wrap to wound bed. Seal wound with sterile film again	 On the surface of the wound, place 2 pieces of foams over the two exposed wound areas.
 Connect the two pieces of foams with a piece of bridging foam. Seal with sterile film again. Create a small slit at the distal wound site for placement of the transfer pad device.	 Transfer pad device sealed with sterile film and connect to the canister and pump



Maintain on negative pressure 125mmHg on continuous mode. Change VAC dressing every three days

Vacuum device which maintain a continuous negative pressure



Wound after discontinuation of NPWT

Foam dressing

Negative pressure wound therapy

NPWT has been in use since 1995 for the following: chronic and acute wounds, dehisced incisions, chronic diabetic wounds, pressure ulcers, grafts and flaps[22]. It is non-invasive and acts by delivering negative pressure at the wound bed[22]. The exact mechanism of action is not known although it has been postulated to work via promoting changes at the cellular level to enhance formation of granulation tissue, adhesion of wound edges and reducing exudates[21,22]. The controlled subatmospheric pressure improves local oxygenation and peripheral blood flow[19]. NPWT has also been found to reduce the overall volume and dimensions of the wound, reducing the need for complex plastic reconstruction needed for wound closure[16].Contraindications for NPWT include fistulas to organs and body cavities, eschars, non-debrided necrotic tissue, untreated osteomyelitis, malignant wounds, bleeding wounds, patients on anticoagulants[22]. See Figure 2.

Oxygen therapy

Hyperbaric oxygen therapy (HBOT) is usually used as an adjunct in wound management. It consists of a course of multiple treatments in a pressurised sealed chamber containing 100% oxygen[16]. A synergistic response between oxygen and growth factors have been demonstrated in addition to supplying oxygen to the wound site[16]. Oxygen is needed for neutrophils and macrophages mediated bacterial killing as well as for tissue repair processes[16]. In addition, pressurised oxygen has been shown to stimulate stem cell and endothelial progenitor cell release from bone marrow, promoting wound healing[16]. HBOT is indicated for use in crush injuries, compartment syndrome, acute traumatic ischemia and ischemic reperfusion injuries, radiation injuries, compromised skin grafts and refractor osteomyelitis and anaerobes infected wounds[16]. It has been found to be most useful in reducing the rates of major amputation in diabetic foot ulcers[19]. There are few contraindications for hyperbaric oxygen therapy and these include reactive airway disease, untreated pneumothorax and concurrent chemotherapy[16]. Other side effects which can occur with use of HBOT include otic or sinus discomfort, claustrophobia and oxygen toxicity at high pressures[16].

Ultrasound therapy

By using different frequencies of ultrasound (Low frequency- Hertz in thousands range and high frequency- Hertz in millions range), it has been discovered that non-healing or stagnated wounds can be stimulated to progress on in the cycle of wound repair[20]. It works via penetration of deep tissue to stimulate cells beneath the wound bed and promotes debridement of necrotic tissue[20]. Ultrasound therapy has been tried and tested in the treatment of a variety of wounds including diabetic foot ulcers, chronic venous ulcers, pressure sores, and burns and for bone debridement[20]. Currently, there is limited evidence supporting its routine use[19].

Low energy light treatment or low- power laser therapy

Laser therapy makes use of low energy band lasers to promote fibroblast activity, collagen metabolism and epithelialization via increasing reactive oxygen species, stimulating gene expression, promoting angiogenesis and reducing inflammation[20]. It is used in venous leg ulcers, diabetic ulcers and burns[19]. Again, there is

limited evidence supporting its routine use in clinical practice.

CONCLUSIONS

With an ageing population and the rising incidence of chronic diseases such as diabetes and peripheral vascular disease, the cost of wound care will inevitably become a cause for concern in our local healthcare system. Choosing the right wound dressing remains one of the most critical considerations to enhance rates of wound healing. There is no one dressing that fits all wounds and current selection of dressings is based on wound assessment and treatment objectives. The experiences and knowledge of the wound care practitioner and availability of dressings on the market also plays an important role in wound management. Wound management should be based on a systematic, patient-centred and multidisciplinary approach as this has been repeatedly demonstrated to significantly increase healing rates, reduce wound associated pain and the frequency of treatments needed[1,24]. Of equal importance is the proper education of patients and care givers which has been shown to improve compliance to treatment and overall outcome[14]. Today's rapid technological advances in wound care should serve as an impetus for us as medical professionals to positively impact medical education and the management of wounds.

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LEARNING POINTS

- **There is no single dressing that will be able to manage all the nuances within the wound environment currently. Adequate wound assessment together with adequate knowledge of basic properties of each dressing category is vital and this is the cornerstone of dressing selection.**
- **A wound is an evolving entity; the same dressing cannot be used from the beginning to the end. Dressings are selected according to wound characteristics; therefore when the wound changes, so should the dressing.**
- **At each dressing change, it is advisable to review the condition of the wound, as this allows for monitoring of the effectiveness of the previous dressing used. This includes measurement of the wound, as well as taking photographs.**
- **The frequency of dressing change made based on clinical judgment. If the dressing is soiled, loose, slipping or curling at the edges, it is obvious that it should be changed. If there is accumulation of fluid and/ or debris and the dressing is saturated, it needs change. If infection is present, increased frequencies of change need to be considered.**

UNIT NO. 3

APPROACH TO COMPLEX WOUND MANAGEMENT AND ADJUNCT THERAPY

Tan Mui Lan, Goh Boon Ai Susie

ABSTRACT

The ageing of our population and rise in chronic diseases has resulted in the complex profile of the patients in the community. Complex wounds such as diabetic foot ulcers, infected pressure ulcers and other complications of non-healing wounds are common encounters in the primary health settings. The challenges of these complex wounds lie in its multi-factorial nature of the person, the wound and the environment. This requires a team approach to care within the limited resources boundary.

As part of the care continuum, it is essential for primary care physicians to be familiarized with the approach to care of complex wounds and the adjunct therapy. This article seeks to provide a broad framework using the systematic assessment framework via T.I.M.E (Tissue, Inflammation/Infection, Moisture imbalance, Epithelial edge of wound) for wound bed preparation to guide primary care physicians/clinicians in their approach to complex wounds. It also highlighted the complexities of chronic wound management pertaining to the person, the wound and the environment as well as the recent advances adjunct therapy in chronic wound care. In addition, it seeks to enable primary care physician and wound clinicians to translate wound-healing principles into effective management strategies to provide better clinical care to our patients.

Keywords:
Complex, Chronic, Non healing wounds management, Biofilms, Wound bed preparation, Debridement, Topical negative pressure therapy, Transforming wound healing

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INTRODUCTION

The healing of wounds is a series of complex process consisting of hemostatsis, inflammation, proliferation/repair and maturation or remodeling. The molecular and cellular activities in tissue repair is a continuous process which cells undergo a number of complicated biological changes to enable hemostasis, combat infection, migrate into wound space, deposit a matrix, form new blood vessels and contract to close the defect. However, complex or chronic wounds undergo a complicated prolonged disorganised manner of healing process[2, 3].

Chronic wounds such as diabetic foot ulcers, pressure ulcers and infected ulcers usually heal by secondary intention due to the imbalance of the molecular and cellular environment.[1-4] Chronic wounds are those that have failed to progress through the normal stages of healing and therefore enter a state of pathologic inflammation. The presence of high level of pro-inflammatory cytokines, high protease, reactive oxygen species, low mitogenic activity and senescent cells are found in chronic wound exudates.[2-4]. As a result, the healing process is delayed, incomplete and does not proceed in a coordinated manner, subsequently resulting in poor anatomical and functional outcome.[4] A myriad of factors in complex wounds such as chronic diseases like diabetes, devitalised tissues, prolonged inflammation, excessive protease in exudates, infection and also psychosocial factors can impair or delay wound healing.[2-5] These factors increased our challenges in managing complex wounds.

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PRINCIPLES OF WOUND HEALING

The principles of wound centered on the 3 key components: the person, the wound and the environment. The approach to chronic wound management utilised the principles of wound healing by optimising factors that aids healing.

Accurate assessment and identifying the etiologies of the wound is key toward treatment and wound healing.[6, 7, 8].It includes relevant medical, surgical, psychological and drug history together with appropriate physical assessment to identify etiologies and barriers to healing. Delay in wound healing can occur in persons' with co-morbidities such as renal or peripheral vascular disease. Refer to Table 1 on differential diagnosis of chronic wounds. Baseline laboratory such as complete blood cell count, creatinine level, erythrocyte sedimentation rate or C-reactive protein level, and HbA1c level are useful to identify etiologies of delay in wound healing.[8]

Assessment of the wound. Comprehensive assessment, recognition of wound characteristics that will promote or impede the healing process and preparing the wound bed is to allow the healing cascade to occur.[6, 8] The evidence of wound infection includes erythema, increased exudate/pus, swelling, warmth, pain and pyrexia. The goal will be to restore the bacterial balance. The intervention includes medical review, wound swab, wound cleansing, exudate control, use of topical antimicrobials and systemic antibiotic as warranted. When the roadblocks or barriers to healing are eliminated will achieve a stable microenvironment for repair, granulation and contraction of the wound to take place.[10]

Wound Bed Preparation (WBP). This is a concept to enable clinicians to evaluate on the critical components of a non healing wound to identify the cause and treat it. A structured approach using the TIME framework[6] is a useful tool in the evaluation of chronic wounds. The key components in TIME are: T = tissues that non-viable or devitalised; I= inflammatory/Infection; M= moisture too or too little; and E= Epithelial edge of wound. Identifying clinical presentations in the wound bed with debris such as necrotic/non-viable tissues, moisture imbalance,

TABLE 1. DIFFERENTIAL DIAGNOSIS OF A NON HEALING WOUND

Vascular	
Lymphatic	Lymphedema
Mixed venous -arterial	
Vasculitis	Systemic lupus erythematosis, rheumatoid arthritis, scleroderma, polyarteritis nodosa, Wegener's granulomatosis
Venous	Venous stasis
Pressure	Spinal cord injury, bedbound, elderly
Neuropathic	Diabetes, peripheral neuropathy
Hematologic	Polycythemia rubra vera, sickle cell disease
Traumatic	Burns, cold injury, radiation, factitious
Neoplastic	Basal carcinoma, squamous cell carcinoma, melanoma, Marjolin's ulcer, Bowen's disease
Others	Sarcoidosis, obesity, tropical ulcer, pyoderma gangrenosum, necrobiosis lipoidica diabetecorum

excessive colonisation of microorganism and lack of epithelial edge advancement is to enable targeted treatment to 'jump start' the process of healing[4, 5, 6, 7, 10] The goals of WBP are: maintain moisture balance; optimise pH and wound temperature, promote granulation, contraction and eptithelisation. Refer to Table 2 on clinical evaluation and strategies based on TIME framework.

Tissue. Repetitive and maintenance debridement and wound cleansing had been recognised its value in removal of devitalised tissues and control of bioburden, infection and biofilm that impede granulation and healing[4, 6, 9]. Wound cleansing using saline or distilled water irrigation is to remove cellular debris such as bacteria, exudate, purulent material and residual topical agents from the previous dressings.[4, 9] Debridement is the act of removing necrotic material, eschar, devitalised tissues, infected tissue, slough, foreign body, debris to promote healing. Necrotic tissue serves as a medium culture for bacteria and deters wound healing. The removal of these dead tissues is to enable granulation and consequently epithelialisation to occur.[8, 11]

Infection/ Inflammation

As stated earlier, chronic wounds exhibit a prolonged inflammatory response, thus providing an ideal environment for bacterial infiltration and proliferation.[17-18] Maintenance debridement is a proactive way to "jump-start" the wound and keep it in a healing mode. In addition, clinical recognition of infection, persistent inflammation and biofilm is critical. The appropriate use of topical antiseptics to control bioburden and inflammation is a useful measure in managing chronic wounds.[16-18] Physical disruption of the biofilm using irrigation or ultrasound or surfactants with antimicrobials such as polyhexamethyl biguanide (PHMB) and octenidine for cleansing as well as sharp debridement are effective means of removing and preventing reconstitution of the biofilm.[9-13]

Presence of infection impedes healing and requires timely recognition and treatment to restore bacterial balance. The presence of friable hyper-granulation, tissue bridging, pocketing, rolled wound edges, increased exudate and static healing are the evidences of critical colonisation.[13-16] The goal of care in this context will be to eradicate the biochemical and cellular burden. The interventions will entails wound cleansing, review frequency of dressing change, exudate management and topical antimicrobials. The eradication of biofilm and strategies to normalise proteases levels in modulating bacteria load is a crucial aspect in the management of chronic wound such as diabetic foot ulcers.[9, 13, 18]

Moisture

Regulation of moisture balance in wound bed preparation is vital for adequate moist wound healing and wound edge contraction. Chronic wounds contains high levels of proteases, pro-inflammatory cytokines and elevated levels of matrix metalloproteinases (MMPs) damages the wound bed, destroys the extracullar matrix and affects the integrity of the peri-wound edge that further hinders wound edge epithelisation.[6-10] Studies had linked biofilm formation to poor exudate control.[11, 14,15,20] Recent evidences have recognised the role of negative pressure wound therapy (NPWT) in exudate management and promote granulation.[23, 28]

Edge of wound

Considerable developments and improvement in NPWT has evolved in the last few years. It proved to be a valuable tool for exudate control, biofilm reduction, edge of wound contraction and is effective on hard-to-heal wounds. These adjunct therapies include electromagnetic therapy, laser, ultrasound and systemic oxygen therapy.[6, 22, 23, 24]

Selecting wound care products

The main categories of dressings include films, hydrogels, acrylics, hydrocolloids, calcium alginates, hydrofibers, and foams. In general, absorptive dressing (calcium alginates/ hydrofillers/ foam) is needed for high exudate wounds and moisture balance dressing (hdrogel/ hydrocolloids) is needed to give moisture to the dry wounds. Antimicrobial impregated silver or idodine based dressings (idosorb powder/ paste, silver dressing or calcium alginate with silver) are used to reduce the bioburden in chronic wounds.[13, 16] Refer to Table 3 on selection of wound care products.

Adjunct therapy. Negative pressure wound therapy (NPWT) has revolutionised the approach to complex wounds, enabling a

TABLE 2. TIME FRAMEWORK[6]

Clinical Observation	Wound Bed Preparation	Developments/ strategies
Tissue	Necrotic, non-viable tissues to debride. Episodic or continuous debridement to remove defective matrix and cell debris Wound cleansing	New methods: Low frequency ultrasound; Hydrosurgery Existing methods: Autolytic (honey/hydrogel) NPWT add on to existing debridement method Antimicrobial irrigation solution
Infection	Bacterial balance Persistent inflammation Excessive colonisation of microorganism; inappropriate/persistent inflammation; biofilm (increases activities of proteases); wound breakdown, friable granulation; increase in wound size and pain	Eradicate Biofilm. Use debridement and antiseptic agents to disrupt and prevent reconstitution of biofilm. Role of proteases and pro - inflammatory markers in chronic wound Remove infected foci through use of topical or systemic antimicrobials to remove bioburden and contain inflammation
Moisture	Moisture imbalance The increased proteolytic activity of chronic wound exudates inhibits healing by damaging the wound bed. Lead to formation of biofilm. Too little exudate. Lack of growth factors to promote wound healing	Control excessive fluid and avoid wound maceration. Use moisture balancing dressing, compression or negative pressure to remove the fluids Adjunct therapy: NPWT for both acute surgical and chronic wounds
Edge of wound	Epithelial edge advancement and contraction determines wound closure Improve peri-wound edge. Dry or macerated wound edge affects the ability of wound to contract	Adjunct therapy NPWT to encourage contraction. Laser, Ultrasound, systemic oxygen therapy

breakthrough in wound management in both acute and chronic wounds. Indications for NPWT include diabetic foot ulcers, stage III & IV pressure ulcers, or post operative dehisced surgical wounds.[4, 9, 22, 26] It is generally used for deep wounds that require assistance with contraction and granulation tissue formation. The primary treatment goal of NPWT in most chronic wounds is to achieve wound closure (either by secondary intention or preparing the wound for surgical closure). NPWT is useful in moisture imbalance (high exudate) management to facilitate moist wound healing. The secondary goals are to reduce wound dimensions, and to improve the quality of the wound bed. There are strong evidences for use of NPWT in non-ischaemic diabetic foot ulcer, chronic recalcitrant diabetic foot ulcers and also wounds in the diabetic limb following surgical debridement or partial amputation.[4, 9, 23, 27, 28] It is generally well tolerated and appears to stimulate a robust granulation tissue response compared with other wound healing modalities.

COMPLEX CHALLENGES AND EFFECTIVE MANAGEMENT STRATEGIES – TIME REVIEW

The approach to the management of complex wounds using the

TABLE 3. TYPES OF WOUND CARE PRODUCTS BASED ON WOUND TYPE

Tissue	Type of dressing	Goal
Thick dry slough/necrotic	Hydrogel Hydrocolloid	To hydrate; soften slough and debris. Soothing and cooling properties
Moderate exudate	Calcium alginate	Absorbent non-adherent, turn to gel-like upon contact with exudate
High exudate	Calcium alginate with silver/foam/NPWT	Exudate control; reduce bioburden
Inflamed/infected	Antimicrobial dressings	Control of bioburden

TIME framework is effective and relevant in clinical settings given the complexity of healing in chronic wounds. Part of routine assessment is to have continuous review and monitoring of the wound bed is absolutely in dealing with chronic wounds. This will optimise wound bed preparation in term of recognition, disruption and eradication of biofilm and control of bioburden.[4-6, 9, 10] The value of physical disrupting the biofilm and maintenance/episodic debridement is facilitate transformation from non-healing into healing wounds by preventing reconstitution of biofilm.[10-13] Selection and use of topical antiseptics, wound cleansing agents, sharps or autolytic debridement and wound products need to be evaluated frequently as the wound bed progress from non-healing to healing phase.[8, 13] Review of treatment aims during dressing change is important to re-ascertain if the treatment goals are met and wound is progressing at a expected rate. Effective management will yield reduction in bioburden, decrease in wound bed size, increase in granulation and epithelial contraction of the wound edge.[6, 9, 16, 20]

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LEARNING POINTS

- **T.I.M.E is a useful clinical framework for assessing and managing chronic wounds in the primary care settings.**
- **Effective chronic wound management includes holistic assessment, accurate diagnosis, treatment of underlying cause and partnership with patient & other health care professionals.**
- **On-going assessment of complex wounds using TIME framework for wound bed preparation aid toward diagnosis and treatment progress.**
- **Episodic or continuous biofilm disruption via vigorous cleansing, debridement and use of antimicrobial agent is to prevent the reformation of the biofilm and transform the chronic non-healing wound into healing phase.**
- **Adjunct therapy such as NPWT has proved useful in the treatment of diabetic foot ulcers especially in exudate control, expediting wound closure and limb preservation.**

ASSESSMENT OF 15 MCQs

FPSC NO: 59
WOUND CARE
Submission DEADLINE: 11 NOVEMBER 2014, 12 NOON

INSTRUCTIONS

- **To submit answers to the following multiple choice questions, you are required to log on to the College Online Portal (www.cfps2online.org)**
- **Attempt ALL the following multiple choice questions.**
- **There is only ONE correct answer for each question.**
- **The answers should be submitted to the College of Family Physicians Singapore via the College Online Portal before the submission deadline stated above.**
- **There will be NO further extension of the submission deadline.**

1. The healing of an acute wound is described as a process of four phases. Which of the following is an INCORRECT description?

A. Inflammatory phase.
B. Epithelial cell migration phase.
C. Remodelling phase.
D. Proliferative or repair phase.
E. Haemostasis phase.

2 One of the tasks in wound assessment is to determine factors that will hinder healing and remove them as far as possible. Also such factors can be grouped as local or systemic factors. Which of the following is a local factor?

A. Non-steroidal anti-inflammatory drugs.
B. Nutrition.
C. Alchoholism.
D. Ischaemia.
E. Gender.

3. A sacral pressure ulcer is noted to have the following features: subcutaneous tissue is visible; no tendon, muscle or bone is visible; minimal slough is present; and no undermining is noted. Which stage of pressure ulcer will this be classified as?

A. Stage 1.
B. Stage 2.
C. Stage 3.
D. Stage 4.
E. Deep tissue injury.

4. About a guideline to support the healing of a pressure ulcer, which of the following guideline statement is INCORRECT?

A. A minimum calorie intake of 30-35 kcal per kg per day.
B. Protein intake of 1.25 – 1.5 kg per day.
C. Fluid intake of 30 ml per kg per day.
D. Steroids can be given to promote tissue growth.
E. Smoking should be stopped.

5. Many adjuvants are available to help with the healing of wounds. Which of the following is NOT an adjuvant therapy or procedure?

A. Use of ABOUND OR IMPACT which is a therapeutic nutrition drink mix.
B. Venous insufficiency surgery.
C. Use of pentoxyfylline.
D. Hyperbaric oxygen therapy.
E. Use of a platelet-derived growth factor get preparation.

6. A pressure ulcer is noted to be in the granulation stage. There is some exudate present. Which of the following would be a suitable wound dressing to use?

A. Alginate dressing.
B. Cademoxer iodine dressing.
C. Polyurethrane foam dressing.
D. Silver barrier dressing.
E. Hydrofiber dressing.

7. An infected pressure ulcer is being dressed. There is some exudate but not heavy. There is also some concern that there is antibiotic resistance. Which of the following dressing will be appropriate?

A. Polyurethrane foam dressing.
B. Hydrogel dressing.
C. Hydrocolloid dressing.
D. Hydrofiber dressing.
E. Silver barrier dressing.

8 A 67-year-old diabetic patient is noted to have a highly exudative and deep foot ulcer. It does not look infected. Which of the following would be a wound dressing of choice?

A. Hydrocolloid dressing.
B. Hydrofiber dressing.
C. Alginate dressing.
D. Hydrogel dressing.
E. Silver barrier dressing.

9. Maggot debridement therapy (MDT) is used as a way of therapy for pressure ulcers. Which of the following pressure ulcer would be the most likely to benefit?

A. Stage 1 pressure ulcer.
B. Stage 2 pressure ulcer.
C. Stage 3 pressure ulcer.
D. Unstageable pressure ulcer.
E. Deep tissue injury.

10. Negative pressure wound therapy is a newer way of wound care. The controlled subatmospheric pressure improves local oxygenation and peripheral blood flow. Which of the following is a contraindication to this way of treatment?

A. Fistulas to organs and cavities.
B. Chronic wounds.
C. Acute wounds.
D. Chronic diabetic wounds.
E. Grafts and flaps.

11. A healing pressure ulcer is being dressed. It is noted to be excessively dry. Which of the following will be a suitable dressing to use?

A. Silcone foam dressing.
B. Hydrogel dressing.
C. Silver barrier dressing.
D. Alginate dressing.
E. Hydrofiber dressing.

12. A 55-year-old man has a diabetic ulcer that is non healing. Necrotic, non-viable tissue is present. Which would be the treatment of choice?

A. Low frequency ultrasound.
B. Silicone foam dressing.
C. Alginate dressing.
D. Antimicrobial dressing.
E. Hydrofiber dressing.

13. A biofilm prevents a chronic ulcer from healing. Which of the following is NOT a characteristic of biofilm?

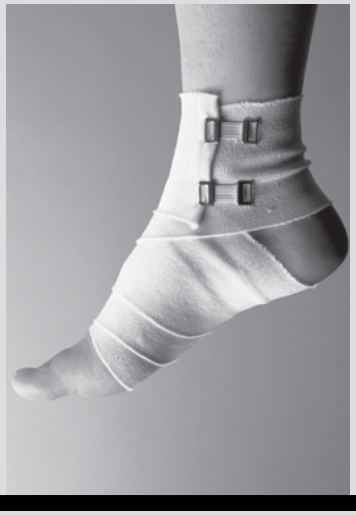
A. Granulation with exudate on the ulcer surface.
B. Non healing ulcer.
C. Shiny ulcer surface.
D. Malodour.
E. Unresponsive to antimicrobial intervention.

14. Negative pressure wound therapy has emerged to be a useful modality to use in chronic pressure ulcer care. In which of the following is negative pressure wound therapy the MOST useful?

A. A fistula wound.
B. Untreated osteomyelitis.
C. Eschars.
D. Bleeding wound.
E. Edge of wound contraction.

15. A chronic pressure ulcer is noted to have a thickened biofilm. What would be the most useful application to use for this ulcer?

A. Alginate dressing.
B. Hydrocolloid dressing.
C. Aquacel dressing.
D. Hydrogel dressing.
E. Prontosan lotion.



READINGS

A SELECTION OF TEN READINGS ON TOPICS RELATED TO WOUND CARE

A SELECTION OF TEN READINGS ON TOPICS RELATED TO WOUND CARE

some available as free full-text and some requiring payment

Selection of readings made by A/Prof Goh Lee Gan

READING 1 – USING FRUITS TO STAGE PRESSURE ULCERS

Mackintosh R, Gwilliam A, Williams M. Teaching the fruits of pressure ulcer staging. J Wound Ostomy Continence Nurs. 2014 Jul-Aug;41(4):381-7. PubMed PMID: 24988517

URL: http://ovidsp.tx.ovid.com./ doi:10.1097/WON.0000000000000049. – Free full text

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ABSTRACT

BACKGROUND: Accurate pressure ulcer staging is an important skill for nurses, physicians, physical therapists, and certified nursing assistants. Current education is based on the National Pressure Ulcer Advisory Panel's staging system. A review of the literature indicates variability in staging abilities of numerous healthcare providers. With this problem in mind, a new method of teaching pressure ulcer staging by visual analogy was developed.

METHODS: We used the current National Pressure Ulcer Advisory Panel definitions to create a training tool based on a visual analogy between the different pressure ulcer stages and common fruits and vegetables.

RESULTS: Initial feedback from a western states wound care conference indicates successful integration of teaching into nursing practice. A poster was also presented at the annual 2011 Wound, Ostomy and Continence Nurse's National Conference. Positive feedback was received from numerous Wound, Ostomy and Continence Nurse's members who requested an electronic copy of the poster.

CONCLUSIONS: Visual analogies can provide a method of teaching pressure ulcer staging across different disciplines with different levels of training involved in patient care.

READING 2 – NUTRITIONAL STRATEGIES TO REDUCE PRESSURE ULCERS

Posthauer ME, Collins N, Dorner B, Sloan C. Nutritional strategies for frail older adults. Adv Skin Wound Care. 2013 Mar;26(3):128-40; quiz 141-2. doi: 10.1097/01.ASW.0000427920.74379.8c. PubMed PMID: 23426414.

URL: http://www-ncbi-nlm-nih-gov /pubmed?term=23426414&report=abstract&format=text – free full text

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Comment in
Adv Skin Wound Care. 2013 Mar;26(3):102.

The objectives of this continuing education article are to analyze the aging process and its effect on the nutritional status of frail older adults; determine how sarcopenia, anorexia, malnutrition, and Alzheimer disease increase the risk for pressure ulcer development and impact the healing process; and to apply evidence-based nutrition guidelines and implement practical solutions for wound healing.

READING 3 – PRESSURE RISK MANAGEMENT GUIDELINES WORK

Kapp S. Successful implementation of clinical practice guidelines for pressure risk management in a home nursing setting. J Eval Clin Pract. 2013 Oct;19(5):895-901. doi: 10.1111/j.1365-2753.2012.01870.x. Epub 2012 Jun 5. PubMed PMID: 22672390.

URL: http://onlinelibrary.wiley.com./doi/10.1111/j.1365-2753.2012.01870.x/pdf - Payment required

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ABSTRACT
RATIONALE: This paper reports an initiative which promoted evidence-based practice in pressure risk assessment and management among home nursing clients in Melbourne, Australia.

AIM AND OBJECTIVES: The aim of this study was to evaluate the introduction and uptake of the Australian Wound Management Association Guidelines for the Prediction and Prevention of Pressure Ulcers.

METHOD: In 2007 a pilot study was conducted. Nurse perspectives (n=21) were obtained via survey and a client profile (n=218) was generated. Audit of the uptake and continued use of the pressure risk screening tool, during the pilot study and later once implemented as standard practice organizational wide, was conducted.

RESULTS: Nurses at the pilot site successfully implemented the practice guidelines, pressure risk screening was adopted and supporting resources were well received. Most clients were at low risk of pressure ulcer development. The pilot site maintained and extended their pilot study success, ensuring more than 90% of clients were screened for pressure risk over the 18 months which followed. All other sites performed less well initially, however subsequently improved, meeting the pilot sites success after 18 months. Two years later, the organization continues to screen more than 90% of all clients for pressure risk.

CONCLUSION: Implementation of clinical practice guidelines was successful in the pilot project and pressure risk screening became a well-adopted practice. Success continued following organizational wide implementation. Pilot study findings suggest it may be prudent to monitor the pressure ulcer risk status of low risk clients so as to prevent increasing risk and pressure ulcer development among this group.

READING 4 – REPOSITIONING FOR PRESSURE ULCER PREVENTION

Gillespie BM, Chaboyer WP, McInnes E, Kent B, Whitty JA, Thalib L. Repositioning for pressure ulcer prevention in adults. Cochrane Database Syst Rev. 2014 Apr 3;4:CD009958. doi: 10.1002/14651858.CD009958.pub2. PubMed PMID: 24700291.

URL: http://onlinelibrary.wiley.com./doi/10.1002/14651858.CD009958.pub2/pdf -- Payment required

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ABSTRACT
BACKGROUND: A pressure ulcer (PU), also referred to as a 'pressure injury', 'pressure sore', or 'bedsore' is defined as an area of localised tissue damage that is caused by unrelieved pressure, friction or shearing forces on any part of the body. PUs commonly occur in patients who are elderly and less mobile, and carry significant human and economic impacts. Immobility and physical inactivity are considered to be major risk factors for PU development and the manual repositioning of patients in hospital or long-term care is a common pressure ulcer prevention strategy.

OBJECTIVES: The objectives of this review were to:1) assess the effects of repositioning on the prevention of PUs in adults, regardless of risk or in-patient setting;2) ascertain the most effective repositioning schedules for preventing PUs in adults; and3) ascertain the incremental resource consequences and costs associated with implementing different repositioning regimens

compared with alternate schedules or standard practice.

SEARCH METHODS: We searched the following electronic databases to identify reports of the relevant randomised controlled trials: the Cochrane Wounds Group Specialised Register (searched 06 September 2013), the Cochrane Central Register of Controlled Trials (CENTRAL) (2013, Issue 8); Ovid MEDLINE (1948 to August, Week 4, 2013); Ovid EMBASE (1974 to 2013, Week 35); EBESCO CINAHL (1982 to 30 August 2013); and the reference sections of studies that were included in the review.

SELECTION CRITERIA: Randomised controlled trials (RCTs), published or unpublished, that assessed the effects of any repositioning schedule or different patient positions and measured PU incidence in adults in any setting.

DATA COLLECTION AND ANALYSIS: Two review authors independently performed study selection, risk of bias assessment and data extraction. MAIN RESULTS: We included three RCTs and one economic study representing a total of 502 randomised participants from acute and long-term care settings. Two trials compared the 30º and 90º tilt positions using similar repositioning frequencies (there was a small difference in frequency of overnight repositioning in the 90º tilt groups between the trials). The third RCT compared alternative repositioning frequencies. All three studies reported the proportion of patients developing PU of any grade, stage or category. None of the trials reported on pain, or quality of life, and only one reported on cost. All three trials were at high risk of bias. The two trials of 30º tilt vs. 90º were pooled using a random effects model (I^2 = 69%) (252 participants). The risk ratio for developing a PU in the 30º tilt and the standard 90º position was very imprecise (pooled RR 0.62, 95% CI 0.10 to 3.97, P=0.62, very low quality evidence). This comparison is underpowered and at risk of a Type 2 error (only 21 events).In the third study, a cluster randomised trial, participants were randomised between 2-hourly and 3-hourly repositioning on standard hospital mattresses and 4 hourly and 6 hourly repositioning on viscoelastic foam mattresses. This study was also underpowered and at high risk of bias. The risk ratio for pressure ulcers (any category) with 2-hourly repositioning compared with 3-hourly repositioning on a standard mattress was imprecise (RR 0.90, 95% CI 0.69 to 1.16, very low quality evidence). The risk ratio for pressure ulcers (any category) was compatible with a large reduction and no difference between 4-hourly repositioning and 6-hourly repositioning on viscoelastic foam (RR 0.73, 95% CI 0.53 to 1.02, very low quality evidence).A cost-effectiveness analysis based on data derived from one of the included parallel RCTs compared 3-hourly repositioning using the 30º tilt overnight with standard care consisting of 6-hourly repositioning using the 90º lateral rotation overnight. In this evaluation the only included cost was nursing time. The intervention was reported to be cost saving compared with standard care (nurse time cost per patient €206.6 vs €253.1, incremental difference €-46.5; 95%CI: €-1.25 to €-74.60).

AUTHORS' CONCLUSIONS: Repositioning is an integral component of pressure ulcer prevention and treatment; it has a sound theoretical rationale, and is widely recommended and used in practice. The lack of robust evaluations of repositioning frequency and position for pressure ulcer prevention mean that great uncertainty remains but it does not mean these interventions are ineffective since all comparisons are grossly underpowered. Current evidence is small in volume and at risk of bias and there is currently no strong evidence of a reduction in pressure ulcers with the 30º tilt compared with the standard 90º position or good evidence of an effect of repositioning frequency. There is a clear need for high-quality, adequately-powered trials to assess the effects of position and optimal frequency of repositioning on pressure ulcer incidence. The limited data derived from one economic evaluation means it remains unclear whether repositioning every 3 hours using the 30º tilt is less costly in terms of nursing time and more effective than standard care involving repositioning every 6 hours using a 90º tilt.

READING 5 – COMPARING PRESSURE ULCER TREATMENT STRATEGIES

Smith ME, Totten A, Hickam DH, Fu R, Wasson N, Rahman B, Motu'apuaka M, Saha S. Pressure ulcer treatment strategies: a systematic comparative effectiveness review. Ann Intern Med. 2013 Jul 2;159(1):39-50. doi: 10.7326/0003-4819-159-1-201307020-00007. Review. PubMed PMID: 23817703.

URL: http://www-ncbi-nlm-nih-gov /pubmedhealth/PMH0057472 – Free full text

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ABSTRACT

BACKGROUND: Pressure ulcers affect as many as 3 million Americans and are major sources of morbidity, mortality, and health care costs.

PURPOSE: To summarize evidence comparing the effectiveness and safety of treatment strategies for adults with pressure ulcers.

DATA SOURCES: MEDLINE, EMBASE, CINAHL, Evidence-Based Medicine Reviews, Cochrane Central Register of Controlled Trials, Cochrane Database of Systematic Reviews, Database of Abstracts of Reviews of Effects, and Health Technology Assessment Database for English- or foreign-language studies; reference lists; gray literature; and individual product packets from manufacturers (January 1985 to October 2012).

STUDY SELECTION: Randomized trials and comparative observational studies of treatments for pressure ulcers in adults and noncomparative intervention series (n > 50) for surgical interventions and evaluation of harms.

DATA EXTRACTION: Data were extracted and evaluated for accuracy of the extraction, quality of included studies, and strength of evidence.

DATA SYNTHESIS: 174 studies met inclusion criteria and 92 evaluated complete wound healing. In comparison with standard care, placebo, or sham interventions, moderate-strength evidence showed that air-fluidized beds (5 studies [n = 908]; high consistency), protein-containing nutritional supplements (12 studies [n = 562]; high consistency), radiant heat dressings (4 studies [n = 160]; moderate consistency), and electrical stimulation (9 studies [n = 397]; moderate consistency) improved healing of pressure ulcers. Low-strength evidence showed that alternating-pressure surfaces, hydrocolloid dressings, platelet-derived growth factor, and light therapy improved healing of pressure ulcers. The evidence about harms was limited.

LIMITATION: Applicability of results is limited by study quality, heterogeneity in methods and outcomes, and inadequate duration to assess complete wound healing.

CONCLUSION: Moderate-strength evidence shows that healing of pressure ulcers in adults is improved with the use of air-fluidized beds, protein supplementation, radiant heat dressings, and electrical stimulation.

READING 6 – ENZYMATIC SUPERIOR TO AUTOLYTIC DEBRIDEMENT

Waycaster C, Milne CT. Clinical and economic benefit of enzymatic debridement of pressure ulcers compared to autolytic debridement with a hydrogel dressing. J Med Econ. 2013 Jul;16(7):976-86. doi: 10.3111/13696998.2013.807268. Epub 2013 Jun 7. PubMed PMID: 23701261.

URL: http://informahealthcare.com. /doi/pdf/10.3111/13696998.2013.807268 – Free full text

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ABSTRACT

OBJECTIVE: The purpose of this study was to determine the cost-effectiveness of enzymatic debridement using collagenase relative to autolytic debridement with a hydrogel dressing for the treatment of pressure ulcers.

METHODS: A 3-stage Markov model was used to determine the expected costs and outcomes of wound care for collagenase and hydrogel dressings. Outcome data used in the analysis were taken from a randomized clinical trial that directly compared collagenase and hydrogel dressings. The primary outcome in the clinical trial was the proportion of patients achieving a closed epithelialized wound. Transition probabilities for the Markov states were estimated from the clinical trial. A 1-year time horizon was used to determine the expected number of closed wound days and the expected costs for the two alternative debridement therapies. Resource utilization was based on the wound care treatment regimen used in the clinical trial. Resource costs were derived from standard cost references and medical supply wholesalers. The economic perspective taken was that of the long-term care facility. No cost discounting was performed due to the short time horizon of the analysis. A deterministic sensitivity analysis was conducted to analyze economic uncertainty.

RESULTS: The number of expected wound days for the collagenase and hydrogel cohorts are estimated at 48 and 147, respectively. The expected direct cost per patient for pressure ulcer care was $2003 for collagenase and $5480 for hydrogel debridement. The number of closed wound days was 1.5-times higher for collagenase (317 vs 218 days) than with the hydrogel. The estimated cost/closed wound day was 4-times higher for the hydrogel ($25) vs collagenase ($6).

CONCLUSIONS: In this Markov model based on a randomized trial of pressure ulcer care in a long-term care setting collagenase debridement was economically dominant over autolytic debridement, yielding better outcomes at a lower total cost. Since it was a single institution study with a small sample size, the results should be interpreted with caution. Specifically, the findings may not necessarily be generalized to other hydrogel dressings, healthcare settings, age groups, or to wounds of other etiologies.

READING 7 – DIABETIC FOOT INFECTIONS

Gemechu FW, Seemant F, Curley CA. Diabetic foot infections. Am Fam Physician. 2013 Aug 1;88(3):177-84. PubMed PMID: 23939696.

URL: http://www.aafp.org/afp/2013/0801/p177.pdf -- Free full text

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ABSTRACT

Diabetic foot infection, defined as soft tissue or bone infection below the malleoli, is the most common complication of diabetes mellitus leading to hospitalization and the most frequent cause of nontraumatic lower extremity amputation. Diabetic foot infections are diagnosed clinically based on the presence of at least two classic findings of inflammation or purulence. Infections are classified as mild, moderate, or severe. Most diabetic foot infections are polymicrobial. The most common pathogens are aerobic gram-positive cocci, mainly Staphylococcus species. Osteomyelitis is a serious complication of diabetic foot infection that increases the likelihood of surgical intervention. Treatment is based on the extent and severity of the infection and comorbid conditions. Mild infections are treated with oral antibiotics, wound care, and pressure off-loading in the outpatient setting. Selected patients with moderate infections and all patients with severe infections should be hospitalized, given intravenous antibiotics, and evaluated for possible surgical intervention. Peripheral arterial disease is present in up to 40% of patients with diabetic foot infections, making evaluation of the vascular supply critical. All patients with diabetes should undergo a systematic foot examination at least once a year, and more frequently if risk factors for diabetic foot ulcers exist. Preventive measures include patient education on proper foot care, glycemic and blood pressure control, smoking cessation, use of prescription footwear, intensive care from a podiatrist, and evaluation for surgical interventions as indicated.

READING 8 – DIABETIC FOOT ULCER ORGANISMS

Gardner SE, Hillis SL, Heilmann K, Segre JA, Grice EA. The neuropathic diabetic foot ulcer microbiome is associated with clinical factors. Diabetes. 2013 Mar;62(3):923-30. doi: 10.2337/db12-0771. Epub 2012 Nov 8. PubMed PMID: 23139351; PubMed Central PMCID: PMC3581190.

URL: http://diabetes.diabetesjournals.org. /content/62/3/923.full.pdf+html – Full free text

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Comment in Diabetes. 2013 Mar;62(3):679-81.

ABSTRACT

Nonhealing diabetic foot ulcers (DFUs) are a common and costly complication of diabetes. Microbial burden, or "bioburden," is believed to underlie delayed healing, although little is known of those clinical factors that may influence microbial load, diversity, and/or pathogenicity. We profiled the microbiomes of neuropathic nonischemic DFUs without clinical evidence of infection in 52 individuals using high-throughput sequencing of the bacterial 16S ribosomal RNA gene. Comparatively, wound cultures, the standard diagnostic in the clinic, vastly underrepresent microbial load, microbial diversity, and the presence of potential pathogens. DFU microbiomes were heterogeneous, even in our tightly restricted study population, but partitioned into three clusters distinguished primarily by dominant bacteria and diversity. Ulcer depth was associated with ulcer cluster,

positively correlated with abundance of anaerobic bacteria, and negatively correlated with abundance of Staphylococcus. Ulcer duration was positively correlated with bacterial diversity, species richness, and relative abundance of Proteobacteria, but was negatively correlated with relative abundance of Staphylococcus. Finally, poor glycemic control was associated with ulcer cluster, with poorest median glycemic control concentrating to Staphylococcus-rich and Streptococcus-rich ulcer clusters. Analyses of microbial community membership and structure may provide the most useful metrics in prospective studies to delineate problematic bioburden from benign colonization that can then be used to drive clinical treatment.

READING 9 – PRESSURE ULCERS PREVALENCE IN SWEDEN

Gunningberg L, Hommel A, Bååth C, Idvall E. The first national pressure ulcer prevalence survey in county council and municipality settings in Sweden. J Eval Clin Pract. 2013 Oct;19(5):862-7. doi: 10.1111/j.1365-2753.2012.01865.x. Epub 2012 May 29. PubMed PMID: 22640165.

URL: http://onlinelibrary.wiley.com /doi/10.1111/j.1365-2753.2012.01865.x/pdf – Payment required

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ABSTRACT

AIM: To report data from the first national pressure ulcer prevalence survey in Sweden on prevalence, pressure ulcer categories, locations and preventive interventions for persons at risk for developing pressure ulcers.

METHODS: A cross-sectional research design was used in a total sample of 35,058 persons in hospitals and nursing homes. The methodology used was that recommended by the European Pressure Ulcer Advisory Panel.

RESULTS: The prevalence of pressure ulcers was 16.6% in hospitals and 14.5% in nursing homes. Many persons at risk for developing pressure ulcers did not receive a pressure-reducing mattress (23.3-27.9%) or planned repositioning in bed (50.2-57.5%).

CONCLUSIONS: Despite great effort on the national level to encourage the prevention of pressure ulcers, the prevalence is high. Public reporting and benchmarking are now available, evidence-based guidelines have been disseminated and national goals have been set. Strategies for implementing practices outlined in the guidelines, meeting goals and changing attitudes must be further developed.

READING 10 – PRESSURE ULCERS IN GERMANY & NETHERLANDS

Meesterberends E, Halfens RJ, Spreeuwenberg MD, Ambergen TA, Lohrmann C, Neyens JC, Schols JM. Do patients in Dutch nursing homes have more pressure ulcers than patients in German nursing homes? A prospective multicenter cohort study. J Am Med Dir Assoc. 2013 Aug;14(8):605-10. doi: 10.1016/j.jamda.2013.03.005. Epub 2013 Apr 28. PubMed PMID: 23628407

URL: http://ac.els-cdn.com/ doi: 10.1016/j.jamda.2013.03.005. – Payment required

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ABSTRACT

OBJECTIVES: To investigate whether the incidence of pressure ulcers in nursing homes in the Netherlands and Germany differs and, if so, to identify resident-related risk factors, nursing-related interventions, and structural factors associated with pressure ulcer development in nursing home residents.

DESIGN: A prospective multicenter cohort study. SETTING: Ten nursing homes in the Netherlands and 11 nursing homes in Germany (around Berlin and Brandenburg).
PARTICIPANTS: A total of 547 newly admitted nursing home residents, of which 240 were Dutch and 307 were German. Residents had an expected length of stay of 12 weeks or longer.

MEASUREMENTS: Data were collected for each resident over a 12-week period and included resident characteristics (eg, demographics, medical history, Braden scale scores, nutritional factors), pressure ulcer prevention and treatment characteristics, staffing ratios and other structural nursing home characteristics, and outcome (pressure ulcer development during the study). Data were obtained by trained research assistants.

RESULTS: A significantly higher pressure ulcer incidence rate was found for the Dutch nursing homes (33.3%) compared with the German nursing homes (14.3%). Six factors that explain the difference in pressure ulcer incidence rates were identified: dementia, analgesics use, the use of transfer aids, repositioning the residents, the availability of a tissue viability nurse on the ward, and regular internal quality controls in the nursing home.

CONCLUSION: The pressure ulcer incidence was significantly higher in Dutch nursing homes than in German nursing homes. Factors related to residents, nursing care and structure explain this difference in incidence rates. Continuous attention to pressure ulcer care is important for all health care settings and countries, but Dutch nursing homes especially should pay more attention to repositioning residents, the necessity and correct use of transfer aids, the necessity of analgesics use, the tasks of the tissue viability nurse, and the performance of regular internal quality controls.

PRISM SECTION

(Patients' Revelations as Insightful Studies of their Management)

- Streptococcal Pneumonia Associated Haemolytic Uraemic Syndrome (Sp-hus) in A 4-year-old Boy - A Rare But Serious Condition. What Should Primary Care Physicians Know?

STREPTOCOCCAL PNEUMONIA ASSOCIATED HAEMOLYTIC URAEMIC SYNDROME (SP-HUS) IN A 4-YEAR-OLD BOY - A RARE BUT SERIOUS CONDITION. WHAT SHOULD PRIMARY CARE PHYSICIANS KNOW?

Dr Wang Mingchang, Dr Shum Oi Han

ABSTRACT

We report a case of streptococcal pneumonia associated haemolytic uraemic syndrome (SP-HUS) in a 4-year old child. It is a rare complication of invasive streptococcus pneumoniae infection. This article touches on the how the patient's mother was unusually calm after hearing the bad news. Factors that could account for her reaction are explored. Other issues triggered by this case were questions on the pathophysiology, clinical features, treatment and prognosis of this complication. We also discussed "catch-up" vaccination for children immunised with the old 7-valent pneumococcal vaccine, early diagnosis of community acquired pneumonia, recognising antibiotic failure and SP-HUS.

Keywords:
Streptococcus pneumonia, Haemolytic uraemic syndrome, Acute renal failure, Anaemia, Thrombocytopenia, Complication

SFP2014; 40(3): 43-47

INTRODUCTION

This 4-year-old child presented when I was doing my paediatric rotation as a first year Family Medicine Resident towards the end of 2011. Streptococcal pneumonia associated haemolytic uraemic syndrome (SP-HUS) is a rare but serious complication of pneumococcal infection. Primary care physicians need to have a high index of suspicion in a paediatric patient with community acquired pneumonia that is not resolving.

PATIENT'S REVELATION: WHAT HAPPENED?

A 4-year-old Chinese boy first presented to a tertiary hospital's children's emergency with a 4-day fever of temperature 39°C, cough and rhinorrhea. His parents were also recently ill with upper respiratory tract symptoms. There was no travel history. His vaccinations were up to date, and he had received a single dose of the 7-valent pneumococcal protein conjugate vaccine 2 years earlier. This boy has a history of H1N1 influenza virus-induced febrile status epilepticus 2 years earlier and is on sodium valproate. He has been asymptomatic since and is neuro-developmentally appropriate for his age.

On clinical examination, he was non-toxic and not in respiratory distress. Temperature was 39.1°C, heart rate 135/min, respiratory rate 20/min and spO2 100% on room air. He had crepitations in both lungs. A full blood count showed a white cell count of 5.14 x 10^9/L. Chest X-ray (Figure 1) showed bilateral hilar infiltrates. He was diagnosed with atypical pneumonia and sent home with oral clarithromycin. Two days later, he re-presented to the emergency department again with persistent fever and body aches. Temperature was 38.7°C and vital signs were comparable to his previous visit. His parents were advised to continue clarithromycin. He returned three days later with worsening cough and body aches. He was lethargic and dehydrated. There were bilateral crepitations, decreased breath sounds, and dullness to percussion over the right lung base. Repeat chest X-ray (Figure 2) showed bilateral infiltrates with a right pleural effusion. Laboratory investigations showed leukopenia (TW3.81 x 10^9/L) and CRP of 515 mg/L. Haemoglobin, urea and creatinine levels were normal.

He was admitted. Blood cultures were sent off. Intravenous ceftriaxone was started. An ultrasound thorax showed a right-sided loculated pleural effusion with thin internal septations and fibrous material. There was consolidation of the whole right lower lobe.

On day 11 of illness, he had persistent lower abdominal discomfort with episodes of watery stool. He was anuric for 10 hours, and was tachycardic (180beats/min) and tachypneic (64 breaths/minute). Generalised oedema, a gallop rhythm, hepatomegaly and ascites were present.

FIGURE 1. CHEST X-RAY OF PATIENT ON DAY 4 OF ILLNESS

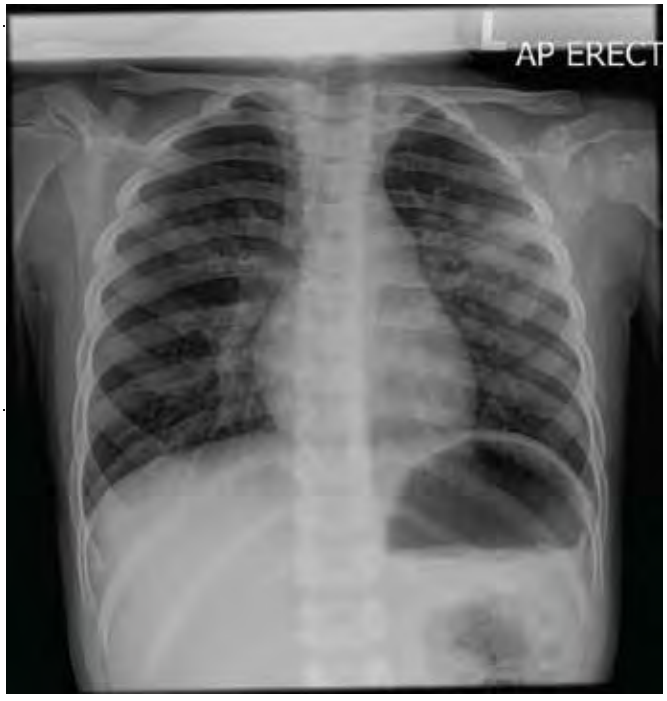

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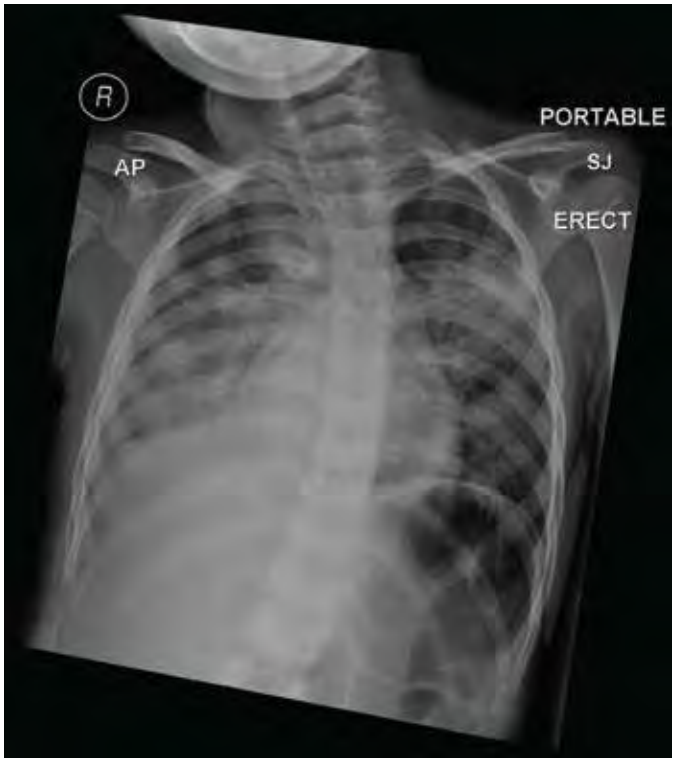
FIGURE 2. CHEST X-RAY OF PATIENT ON DAY 9 OF ILLNESS



He was transferred to the paediatrics intensive care unit (PICU) for monitoring and ventilatory support. Investigations supported a diagnosis of SP-HUS - anaemia, thrombocytopenia and acute renal failure. Haemoglobin dropped from 11.5 to 6.5 g/dL. Platelets decreased from 152 to 6 x 10^9/L. Peripheral blood film showed microspherocytes and marked red blood cell fragmentation, suggestive of microangiopathic haemolysis. His creatinine rose from 39 to 90 umol/L, and urea from 6.9 to 15.7mmol/L.

Blood cultures grew streptococcus pneumoniae, serotype 19A, sensitive to ceftriaxone and vancomycin. This serotype was not covered by the 7-valent vaccine he received 2 years earlier.

A family conference was held with the parents. When informed that the patient will require blood product transfusions and haemodialysis in addition to intravenous antibiotics, the mother accepted placidly. She even commented: "You are all more worried than me!"

Transfusions
The patient was transfused with "washed" red blood cell concentrate and plasma-reduced platelets for his anaemia and thrombocytopenia respectively.

Dialysis
He underwent continuous veno-venous haemodialysis for 10 days in view of his acute renal impairment and fluid overload state. A Tenckhoff catheter was inserted thereafter and peritoneal dialysis was commenced in anticipation of prolonged renal recovery.

Respiratory support
His fluid overload state also resulted in pulmonary oedema, which, coupled with ongoing pneumonia, led to significant respiratory distress. He was intubated and also underwent thoracoscopic decortication of his right lung empyema. A chest tube was inserted for continued drainage of pus post-operatively.

Antibiotics
He had a persistent fever despite being on intravenous ceftriaxone. He was subsequently placed on intravenous vancomycin followed by piperacillin-tazocin and then meropenem. Endotracheal tube culture grew sternotrophomonas sensitive to sulfamethoxazole and trimethoprim (Bactrim), which was then added on to the antibiotic regimen. Pleural fluid, empyema culture and repeat blood cultures did not yield any bacterial growth.

Progress
The patient improved and was extubated on day 17 of illness. He was transferred to a general ward after 19 days in the PICU.

His antibiotics were converted to oral cefuroxime after completing 11 days of intravenous meropenem and 2 weeks of oral Bactrim. He completed a total of 33 days of antibiotics.

His fever was on a downward trend. Inflammatory markers were also downtrending and chest X-ray showed resolving consolidation and effusion. His chest tube was removed.

The Tenckhoff catheter was leaking on day 10 of peritoneal dialysis and had to be removed. However, his renal function showed continuing improvement. He produced good urine output with intravenous frusemide. Serum creatinine improved from 334 to 55umol/L. Enalapril was started for renal protection.

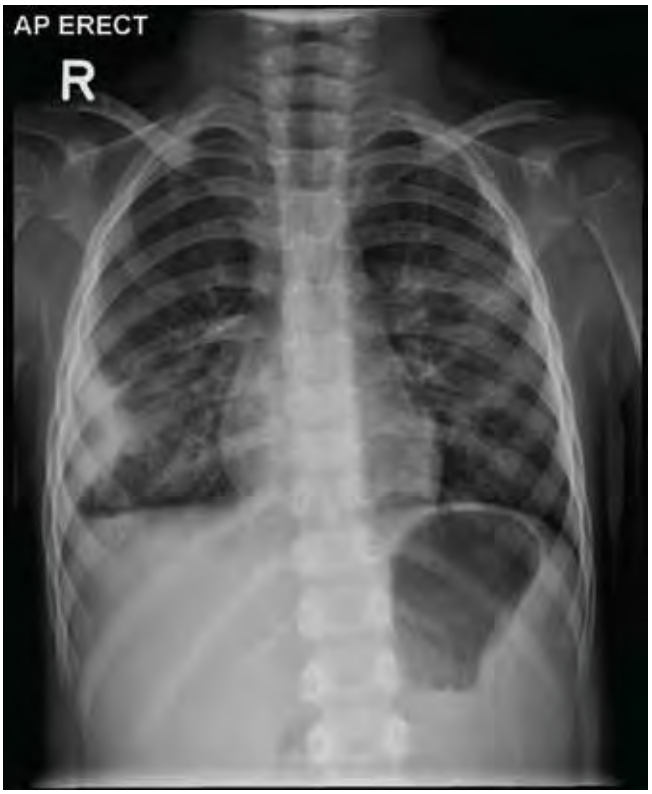
Discharge and follow-up
The patient was discharged well after 37 days in hospital. Reviewed in the outpatient clinic 1 week later, he was chatty, active and ambulant. Urine output was normal. A repeat chest X-ray (Figure 3) showed an almost resolved consolidation and no effusion was seen.

One month later, his haemoglobin was 9.5 g/L and his serum creatinine was stable at 65umol/L.

GAINING INSIGHT: WHAT ARE THE ISSUES?

This case triggered several issues:
i) What are the factors that could account for the mother reacting so calmly in the face of a grave disease that required prolonged and intensive treatment?
ii) What are the pathophysiology, clinical features, treatment and prognosis of SP-HUS?
iii) How do we prevent this condition and what is the choice of first-line antibiotics to treat community acquired pneumonia in children?

FIGURE 3. CHEST X-RAY OF PATIENT ONE WEEK AFTER DISCHARGE FROM HOSPITAL



STUDY THE MANAGEMENT: HOW DO WE APPLY THE INSIGHTS IN OUR CLINICAL PRACTICE?

Patient's mother's reaction

The patient's mother was unusually calm even after being told that her son needed intensive care. This response is even more significant considering that the patient is her only son. During the family conference, the team paused at frequent intervals to allow for questions. She mainly sought confirmation of what she understood. She did not at any point ask how the complication could have been prevented, and seemed to accept her son's condition as it was.

There were several reasons that could account for the mother's placidness. Seeing how she readily accepted management decisions, she probably had confidence in the team of doctors looking after her child. Another explanation would be that she could not comprehend the gravity of the situation, or that she was in a state of denial. My management of this patient was limited to two days in the general ward as he was transferred to intensive care thereafter. In my limited interactions with the parents, I found the mother well spoken, articulated and highly educated. It was unlikely she could not grasp the severity of the situation. Her husband was the silent type but was constantly by her side. He probably was her source of emotional support, simply by just being there. It may have helped her handle the bad news better.

In a cohort study by Jee RA et al,[1] coping strategies of parents in a paediatric intensive care unit in the UK were evaluated. It was found that the main coping strategies employed by parents were related to trust, assurance, and believing in positive outcomes. In this case, it could be her trust in the medical team and her belief in positive outcomes which led to her calm response.

A qualitative study was done on American physicians' experiences in communicating with families of children who suffer from acute life-threatening conditions[2]. This study revealed some helpful points which we can practise. Most families wanted timely and accurate information about the child's condition, and a private room where they can express their feelings or grieve. Start off by finding out the parents' level of understanding, and ask if they have any questions first. Bring them up to date using terminology appropriate to their level. Stage the delivery of bad news, giving it in increments depending on how much the family can take. It may have to take place over multiple meetings. Do not give false hope. The important role of a nurse or social worker to provide psychosocial, emotional and spiritual support was also emphasised.

Haemolytic uraemic syndrome

This is a clinical syndrome characterised by microangiopathic haemolytic anemia, thrombocytopenia and progressive renal failure. It predominantly affects children and has a peak incidence between six months and four years of age[3]. The most frequent cause is Shiga toxin-producing Escherichia Coli O157:H7[4] which is associated with bloody diarrhoea.

HUS can also be caused by invasive streptococcus pneumoniae infection, as demonstrated by this case. This variant is uncommon and involves 5% of all cases of HUS in children. The incidence of HUS following invasive pneumococcal infections is estimated to be 0.4 to 0.6%[5,6].

Pathophysiology

SP-HUS is due to an antigen-antibody reaction. The Thomsen-Friedenreich (TF) cryptantigen is a component of the surface structure of erythrocytes, platelets and glomerular endothelial cells. This antigen is normally hidden by neuraminic acid.

Pneumococci produce neuraminidase which cleaves neuraminic acid, exposing the TF antigen. Preformed host IgM antibodies then bind the TF antigen and initiate a cascade of events leading to autoimmune complement-mediated destruction of the affected cells, resulting in anaemia, thrombocytopenia and glomerular endothelial cell damage which characterises HUS.[4]

All serotypes of streptococcus pneumoniae have neuraminidase activity. Different serotypes may produce varying amounts of neuraminidase, thereby influencing the likelihood of a patient's developing HUS.[4]

Clinical Features

Features of pneumococcal HUS usually develop 7 to 9 days after initial infection.[7] Patients develop oligo-anuria and are found to have elevated plasma creatinine secondary to renal

failure. They have generalised oedema from the resultant volume overload. The patient may progress to end-stage renal failure in the long term.

Other key features in the acute stage are generalised pallor due to anaemia, and petechiae due to thrombocytopenia. Anaemia and volume overload will stress the heart. The patient may develop cardiac failure which can manifest as pulmonary oedema, hepatomegaly from liver congestion as well as ascites from third-spacing.

Treatment Overview

Management revolves around controlling the infection with antibiotics, as well as instituting supportive measures for anaemia and acute renal failure. Vancomycin and an extended spectrum cephalosporin should be started for treatment of invasive pneumococcal infection.[8] A specific antibiotic can be used once sensitivities are available. Dialysis is usually instituted if patients have prolonged periods of anuria or electrolyte abnormalities. Any type of dialysis - peritoneal, haemodialysis, continuous renal replacement therapy - may be used. Red blood cell transfusions are commonly administered to patients with symptomatic anaemia. These patients are usually tachycardic and in cardiac failure. Platelets may also be transfused if the patient shows signs of petechial haemorrhage, gum bleeding or epistaxis. Blood products have to be "washed" prior to transfusion to eliminate the pre-formed IgM antibodies which mediate TF antigen destruction. Other supportive measures include maintaining fluid and electrolyte balance and providing nutritional support. Feeding can be enteral or parenteral.

Prognosis

A PubMed literature search done using the key words "streptococcus pneumoniae", "haemolytic-uraemic syndrome" and "paediatric" produced 5 case series which were pooled, totalling 106 cases with known outcomes. The 106 cases were from a series from New Zealand (11)[9], Taiwan (20)[10], Hong Kong (5)[11], US (37)[12] and UK (43)[13], excluding 10 cases which did not have outcomes recorded. The pooled results showed full recovery in 29% of patients and mortality of 8.5%. 62.5% had renal, pulmonary, and neurological complications.

We note that renal dysfunction occurred in both Asian and Western patient groups but a smaller percentage of the Asian patients required dialysis. Months after the acute illness abated, Asian patients had a better renal prognosis than their western counterparts in terms of chronic renal impairment and dialysis requirement. However, as the follow-up period and data on types of complications were different in each of the studies, no clear unifying conclusion can be drawn with regards to long-term prognosis of SP-HUS.

DISCUSSION

This patient received the 7-valent pneumococcal conjugate vaccine (PCV7) 2 years earlier. However, invasive pneumococcal disease produced by non-PCV7 serotypes, particularly 19A, have been increasing in prevalence and antibiotic resistance in the past decade.[14] A newer vaccine containing 13 conjugate components (PCV13) was developed to expand serotype coverage and to address the disease burden caused by emerging serotypes. PCV 13 covers 6 additional strains of serotypes 1, 3, 5, 6A, 7F and 19A. It has been incorporated into our national immunisation program.

In the event that a child <24 months of age received ≥1 dose of PCV7, the immunisation series should be completed with PCV13. Children aged 14 to 59 months who are fully vaccinated with PCV7 should receive a single "catch up" dose of PCV13.[15]

Early diagnosis of community acquired pneumonia is not easy. A high index of suspicion is needed. Suspect pneumonia when URTI symptoms, such as cough, rhinitis or vomiting, which develop over one to several days are later followed by high fever. However, clinical presentations can be misleading. In a study by Toikka et al, it was noted that as many as one-fourth of the patients do not have any respiratory symptoms.[16] 38% of patients presented with gastrointestinal symptoms. To help in diagnosis, a chest radiograph should be obtained, especially from children with high fever (≥39deg C) and ill appearance.

Based on guidelines by the Pediatric Infectious Diseases Society and the Infectious Diseases Society of America[17], amoxicillin should be used as first-line therapy for previously healthy, appropriately immunised infants and preschool children with mild to moderate community-acquired pneumonia suspected to be of bacterial origin. Alternatives would be a second or third-generation cephalosporin, or levofloxacin. For patients allergic to penicillins or cephalosporins, alternatives are levofloxacin and clindamycin. Levofloxacin is the preferred antibiotic for streptococcus pneumoniae resistant to penicillin.

We also need to suspect antibiotic failure early and SP-HUS if the patient does not improve. Based on Toikka et al's study, most pneumonia patients become afebrile within 24hours after starting antibiotics. Should the patient fail to respond to therapy after a day or two, we need to consider antibiotic resistance or invasive pneumococcal disease, which can lead to SP-HUS. We must keep in mind that SP-HUS can develop 3 to 13 days after a pneumococcal infection,[4] hence the importance of close follow-up. Have a high index of suspicion in a child with pneumonia who develops signs of anaemia, fluid overload and/or thrombocytopenia, which are hallmarks of SP-HUS. It was fortuitous that the mother of this child did not do doctor hopping but brought the child back for repeat consultation at the same point of care 3 times.

CONCLUSION

A disease less often encountered and with a threat to life generates great worry amongst the medical team. Such challenging episodes leave an indelible impact and physicians who managed these cases will be better primed to diagnose and

manage medically challenging situations in the future.

The points of note in this case study are:
1) Haemolytic uraemic syndrome is a rare but potentially fatal complication of streptococcal pneumonia infection. A high index of suspicion for such an occurrence is needed.
2) Symptoms and signs suggestive of SP-HUS in a patient with pneumonia are: remaining unwell despite use of antibiotics, decreased urine output, tachycardia, a gallop rhythm, pallor, petechiae, peripheral oedema. Laboratory investigations done at this stage will show anaemia, thrombocytopenia and elevated creatinine.
3) Communication with parents of a paediatric patient is important. Parents want timely and accurate information. Bad news has to be given in increments or over multiple meetings, depending on how much the family can take. Trust in the team, reassurance and believing in positive outcomes are major coping mechanisms of parents.
4) Children aged 14 to 59 months who are fully vaccinated with PCV7 should receive a single "catch up" dose of PCV13.
5) Amoxicillin should be used as first-line therapy for appropriately immunised infants and preschool children with mild to moderate community-acquired pneumonia suspected to be of bacterial origin. Levofloxacin is the preferred alternative for those with penicillin allergy.

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The patient's mother has given permission for the authors to publish the X-ray images in this article.

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