Burn Wound Infections

An Essay

Submitted for partial fulfillment of M.Sc., degree in Medical Microbiology and Immunology

By

Nermin Samir Sayed Ahmed Rashwan
(M.B, B.Ch., Cairo University)

Supervised by

Prof. Dr. Amal Shafik Balbaa
Professor of Medical Microbiology and Immunology
Faculty of Medicine, Cairo University

Dr. Iman Ezzat Wali
Lecturer of Medical Microbiology and Immunology
Faculty of Medicine, Cairo University

Faculty of Medicine
Cairo University
2007

Acknowledgement

In the name of Allah, the most Great and the most Merciful. First of all, thanks to Allah who guided my way to the right path and with His Will I finished my essay.

I would like to express my sincere gratitude and heartfelt thanks to ***Prof. Dr. Amal Shafik Balbaa,*** *Professor of Microbiology, for her most valuable advice, kind supervision, unlimited guidance, patience and efforts which made this essay came to existence.*

I'm greatly indecbted to ***Dr. Iman Ezzat Wali,*** *lecturer of Microbiology, for her patience, kind supervision, precious suggestions, continuous cooperation and encouragement throughout this essay.*

Also, my special thanks to all the staff and my colleagues in Microbiology department.

Finally, I would like to express my deep thanks to ***all the members of my family****, especially my mother, for their contribution in the success of this essay and their endless support and care and to the soul of my father whom I wanted to be proud of me.*

Abstract

Burn wound infections is one of the most common causes of death in burn patients. It is caused by many organisms, the most common are: *Staphylococcus aureus* and *Pseudomonas aeroguinosa*. The types of burn wound infections occur are: impetigo, celluilitis, invasive infections and invasion into deep layers that may result in bacteraemia, sepsis and multiple-organ dysfunction syndrome. It is prevented by efficient infection control program, proper antimicrobials use and early wound excision and grafting.

Keywords: burn wound, infections, infection control.

Appendix

The Specific Infection Control Guidelines to be used in Burn Units:

- Use PPE (gowns, masks, gloves and footwear): HCWs must use fresh gloves every time they encounter a different patient, making sure to wash their hands before and after they don gloves. Clean gloves are sufficient for changing the dressings but sterile ones are needed when indwelling devices are inserted. Gowns, masks and footwear are used if there is risk of blood spills in addition to the usual contact precautions and airborne precautions taken in the burn unit.
- The burn unit must be built with exact specifications and should be access-restricted. It must have individual rooms, each with laminar airflow, positive pressure ventilation, equipped with HEPA filters and entry to the unit is through negative pressure anteroom where PPE are worn.
- Wash hands using an antimicrobial agent with continued killing action before and after each patient contact.
- Carefully manage potentially contaminated equipment and environmental surfaces and disinfect them with the proper disinfectant.
- Isolate the burn patients especially the colonized or infected.
- Chart patients who have egregious infections and chart the personnel who care for them.
- Educate personnel.
- Destroy and discard supplies in a contaminated environment after a patient is transferred or discharged.
- Hydrotherapy tanks must not be used. Showering hydrotherapy is preferred and the best to do is to do wound care at the bedside for each patient in his roo

Contents:

Abbreviations:

ACH	Air changes per hour
AIDS	Acquired immunodeficiency syndrome
Allo/BT	Allogeneic blood transfusion
BWI	Burn wound infection
C3a	Complement 3a
CD4+	Constant determinant 4
CD8+	Constant determinant 8
CDC	Center for disease prevention and control
CFU	Colony forming unit
CRP	C-Reactive protein
CVP	Central venous pressure
DC	Dendritic cells
EPS	Exopolysaccharide
Flk-2	Fetal liver kinase 2
Flt3L	Fms-like tyrosine kinase 3 ligand
HAIs	Healthcare associated infections
HBV	Hepatitis B virus
HCW	Healthcare workers
HEPA	High efficiency particle air
HIV	Human immunodeficiency virus
ICU	Intensive care unit
IgG	Immunoglobulin G
IL-1	Interleukin-1
INF-α	Interferon-alpha
INF-γ	Interferon-gamma

LDI	Laser Doppler Imaging
LPC	Lipid protein complex
LTCFs	Long term care facilities
MBEC	Minimum biofilm eradication control
MODS	Multiple organ dysfunction syndrome
MRSA	Methicillin-resistant *Staphylococcus aureus.*
NK	Natural killer cells
NNIS	National Nosocomial Infections Surveillance System
NTM	Non-tuberculous mycobacteria
OD	Organ dysfunction
PBI	Primary bloodstream infection
PCR	Polymerase chain reaction
PDAs	Personal digital assistants
PE	Protective environment
PGE_2	Prostaglandins E_2
PPE	Personal protective equipment
SARS	Severe acute respiratory syndrome
SDD	Selective digestive decontamination
TBSA	Total burn surface area
Th-1	T-helper cell
TNF-α	Tumor necrosis factor alpha
TRIM	Transfusion-associated immunomodulation
TSST-1	Toxic shock syndrome toxin-1
VAP	Ventilator-associated pneumonia
VRE	Vancomycin-resistant enterococci

List of figures:

List of tables:

Introduction

The primary function of normal intact skin is to control microbial populations that live on the skin surface and to prevent the underlying tissues from being colonized and invaded by potential pathogens**.** Burns are one of the most common and devastating forms of trauma. Patients with serious thermal injury require immediate specialized care in order to minimize morbidity and mortality **(Church et al., 2006).**

Adult burn injury may result from domestic, industrial or work-related accident or may result from suicide attempts, assault and unintentional injury due to alcohol and/or drug use **(Horner et al., 2005)**. A significant proportion of adult burn patients also suffer from a high degree of mental illness **(Pruitt et al., 2002).**

The highest fatality rates occur among children 4 years of age or younger and adults over the age of 55 years **(Lionelli et al., 2005).** Burn-related deaths in these two age groups account for more than two-thirds of all fire deaths. Males are as twice as likely to die of burn-related injury as females in all age groups **(Wibbenmeyer et al., 2001).**

75% of all deaths are currently related to sepsis from burn wound infection or other infection complications namely urinary tract infections, ventilator-associated pneumonia and central venous catheter-associated bloodstream infections, sepsis syndrome and toxic shock, suppurative thrombophlebitis, myonecrosis and/or inhalation injury **(Atiyeh et al., 2005).**

However, improved outcomes for severely burned patients have been attributed to medical advances in fluid resuscitation, nutritional support, pulmonary care, burn wound care and infection control practices in specialized burn centers. As a result, burn-related deaths, depending on the extent of injury, have significantly decreased within the past 40 years **(Roth & Hughes, 2004, Lionelli et al., 2005).**

Aim of the work:

1. **T**o acquire a broad view of all the possible etiologies and risk factors that eventually lead to wound infections in burned patients.
2. **T**o plan infection control strategies to minimize such infections and review the possible measures to prevent and treat those infections, should they take place.

Burns

Burn injury may be thermal or chemical injury. Thermal injury may be caused by: direct contact with flame, a hot surface or hot liquid (scald), or a source of heat conduction, convection, or radiation which causes a degree of cellular damage to the skin that varies with the temperature and duration of exposure **(Roth & Hughes, 2004).**

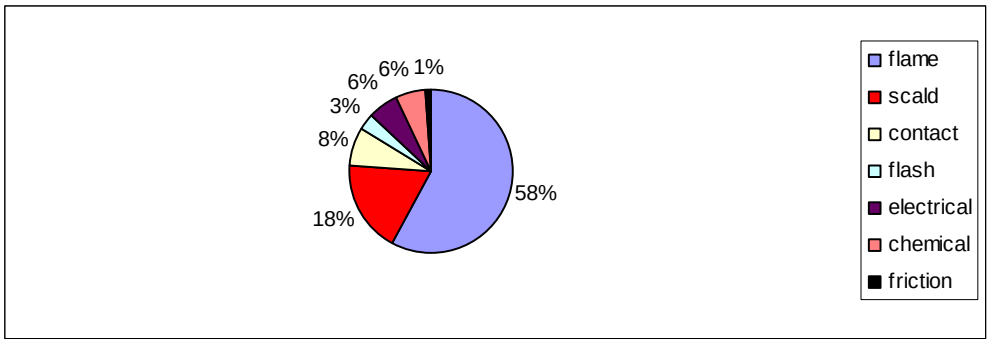

Figure (1): Percentages and types of burn injuries presented by patients. **(Wibbenmeyer et al., 2006).**

Chemical injury may also damage protein structures by chemical interaction. It may be caused by reducing agents, oxidizing agents,…etc **(Roth & Hughes, 2004).**

Table (1): Classification of chemicals that cause burn injury.

Class	Example(s)	Mode of action
Reducing agents	Hydrochloric acid	Bind free electrons in tissue proteins
Oxidizing agents	Sodium hypoclorite	Oxidized on contacting proteins producing toxic by-products
Corrosive agents	Phenol	Denatures tissue proteins
Protoplasmic poisons	Hydrofluoric acid Acetic acid	Bind calcium or other ions essential to cell function
Vesicants	Dimethyl sulforide Cantharides Mustard gas	Ischemia with anoxic necrosis
Desiccants	Sulfuric acid Muriatic acid	Dehydration Exothermic reaction

(Church et al., 2006).

The extent of burn injury can be estimated using either the age-specific Lund-Browder chart (table 2) or by using the patient's palm, which represents approximately 1% of his body surface area **(Palmieri & Greenhalgh, 2002)** or by using the rule of nines **(Roth & Hughes, 2004)** (Figures 3 &4)**.**

Table (2): Lund-Browder chart of body surface area size by age group.

%Total body surface area												
	Head	Neck	Anterior trunk	Posterior trunk	Buttock[a]	Genitalia	Upper arm[a]	Lower arm[a]	Hand[a]	Thigh[a]	Leg[a]	foot[a]
0-1 year	19	2	13	13	2.5	1	4	3	2.5	5.5	5	3.5
1-4 years	17	2	13	13	2.5	1	4	3	2.5	6.5	5	3.5
5-9 years	13	2	13	13	2.5	1	4	3	2.5	8	5.5	3.5
10-14 years	11	2	13	13	2.5	1	4	3	2.5	8.5	6	3.5
15 years	9	2	13	13	2.5	1	4	3	2.5	9	6.5	3.5
adult	7	2	13	13	2.5	1	4	3	2.5	9.5	7	3.5
a represents percentage of body surface area per side, e.g. right upper arm is 4% and left upper arm is 4% of the total body surface area												

(Palmieri & Greenhalgh, 2002).

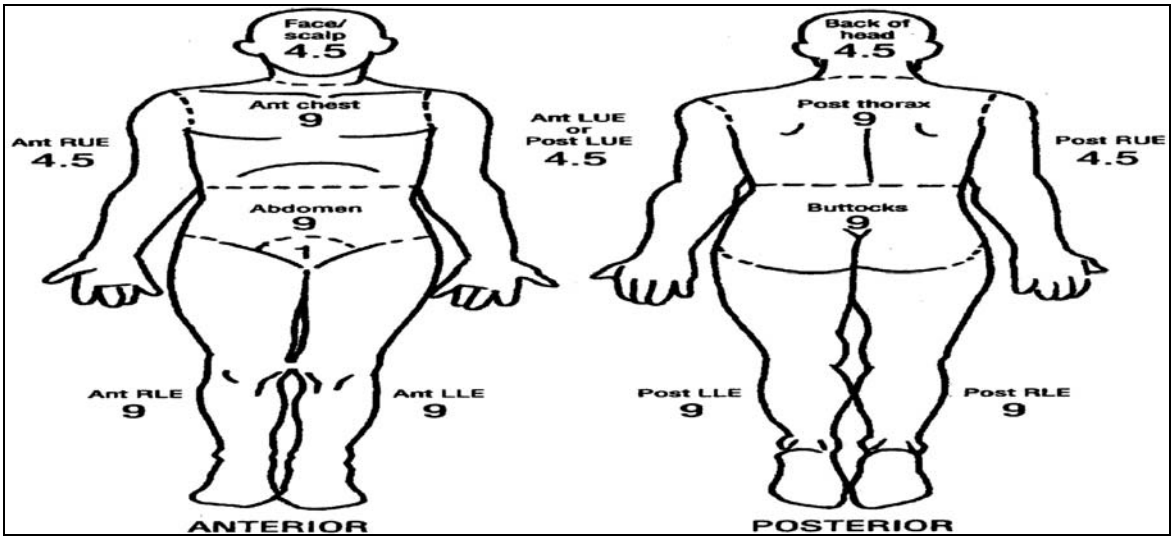

Figure (2): Body diagram for estimation of total burned surface area (%TBSA) in adults, using the rule of nines (numbers are for anterior only and posterior only) **(Roth & Hughes, 2004).**