New Advances In Management of Septicemia In Burned patients

An Essay

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List of abbreviation

ADH	**Anti-diruetic hormone**
ALI	**Acute lung injury**
APRV	**Airway pressure release ventilation**
aPTT	**activated partial thrombo-plastin time**
ARDS	**Acute respiratory distress syndrome**
AT	**Anti-thrombin**
BSBA	**Body surface burned area**
BWI	**Burn wound infection**
DIC	**Disseminated intra-vascular coagulopathy**
ECs	**The endothelial cells**
GAGs	**Glycol amino-glycans**
HFOV	**High frequency oscillatory ventilation**
HSPGs	**Endothelial heparin sulphate polysaccharides**
IL-6	**Interleukin – 6**
LBP	**Lipopoly-saccharide binding protein**
MAP	**Mean arterial pressure**
MODS	**Multi organ dysfunction syndrome**
MOF	**multiple organ failure**
MPs	**Micro-particles**

MRSA	**Methicillin resistant staph – aureus**
NE	**Norepinephrine**
NO	**Nitric oxide**
PARs	**Protease activated receptors**
Pc	**Protein C**
PCT	**Pro-calcitonin**
PG	**Proteoglycans**
PT	**Pro-thrombin time**
SIRS	**Systemic inflammatory response syndrome**
TBSA	**Total body surface area**
TF	**Tissue factor**
TFPI	**Tissue factor pathway inhibitor**
TLR	**Toll-like receptors**
TNF	**Tumour necrosis factor**
TRICC	**Transfusion requirement in critical care**
TSST	**Toxic shock syndrome toxin**
VAP	**Ventilator associated pneumonia**
VILI	**Ventilator induced lung injury**
VP	**Vasopressin**

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Introduction

Burn injury is a major problem in many areas of the world and it has been estimated that ٧٥٪ of all deaths following burns are related to infection . It is therefore essential for every burn institution to determine its specific pattern of burn wound microbial colonization, time related changes in predominant flora and antimicrobial resistance profiles. This would allow early management of septic episodes with proper empirical systemic antibiotics before the results of microbiologic culture becomes available, thus improving the overall infection-related morbidity and mortality. ***(Sharma , ٢٠٠٥)***

Burn wound has a much higher incidence of sepsis as compared to other forms of trauma, because of extensive skin barrier disruption and an alteration in the cellular and humoral immune responses . ***(Winkelstein , ١٩٨٤)***

Burn injury is known to cause devitalization of tissues and produce extensive raw areas. The wound is moist due to the outflow of serous exudates at a temperature approaching ٣٧ C or above .This moist wound environment favor colonization and proliferation of a variety of microorganisms. ***(Lawrence , ١٩٩٢)***

Dysfunction of the immune system, a large cutaneous bacterial load , the possibility of gastro-intestinal bacterial translocation , prolonged hospitalization and invasive diagnostic and therapeutic procedures, all contribute to sepsis. ***(Jones et al., ١٩٩٠)***

The infecting organisms may gain access either through cross contamination or they may derive from the patient's own skin and gastrointestinal tract micro-flora ***(Weber and McManus , ٢٠٠٤)*** The survival rates for burn patients have improved substantially in the past few decades due to advances in modern medical care in specialized burn centers ***.(Lionelli et al., ٢٠٠٥)***

Improved outcomes for severly burned patients have been attributed to medical advances in fluid resuscitation , nutritional support , pulmonary care , burn wound care , and infection control practices. ***(Roth and Hughes , ٢٠٠٤)*** Seventy five percent of all deaths are currently related to sepsis from burn wound infection or other infection complication and or sepsis due to inhalational injury**.** ***(Atiyeh et al., ٢٠٠٥)***

Aim of the work

To discuss new causative agents , new strategies for diagnosis and treatment of septicemia in burned patients , aiming to improvement of outcome to reduce rate of morbidity and mortality .

Pathophysiology of sepsis in burned patients

Sepsis remains a critical and serious complication of a major burn injury with significant morbidity and mortality. Because burn causes a delay in epidermal maturation and leads to additional scar tissue formation ***(Edwards and Harding , ٢٠٠٤)*** . Invasion of microorganisms into the tissue layers below the dermis may result in bacteremia, sepsis, and multiple-organ dysfunction syndrome. Clinical diagnosis of burn wound infection relies on regular monitoring of vital signs and inspection of the entire burn wound surface .***(Pruitt et al., ١٩٩٨)*** Local signs of burn wound infection include conversion of a partial-thickness injury to a full-thickness wound , rapidly extending cellulitis of healthy tissue surrounding the injury, rapid eschar separation, and tissue necrosis. ***(Robson , ١٩٨٨)***

The burn wound typically has three characteristic areas of involvement

(i) the zone of coagulation, which comprises the dead tissues that form the burn eschar that is located at the center of the wound nearest to the heat source. (ii) the zone of stasis , which comprises tissues adjacent to the area of burn necrosis that is still viable but at risk for ongoing ischemic damage due to decreased perfusion .

(iii) the zone of hyperemia, which comprises normal skin with minimal cellular injury that has predominant vasodilation and increased blood flow as a response to injury. ***(Roth and Hughes , ٢٠٠٤)***

The burn wound surface (in deep partial-thickness and in all full-thickness burns) is a protein-rich environment consisting of a vascular necrotic tissue (eschar) that provides a favorable niche for microbial colonization and proliferation . The a vascularity of the eschar results in impaired migration of host immune cells and restricts delivery of systemically administered antimicrobial agents to the area , while toxic substances released by eschar tissue impair local host immune responses. ***(Erol et al., ٢٠٠٤)***

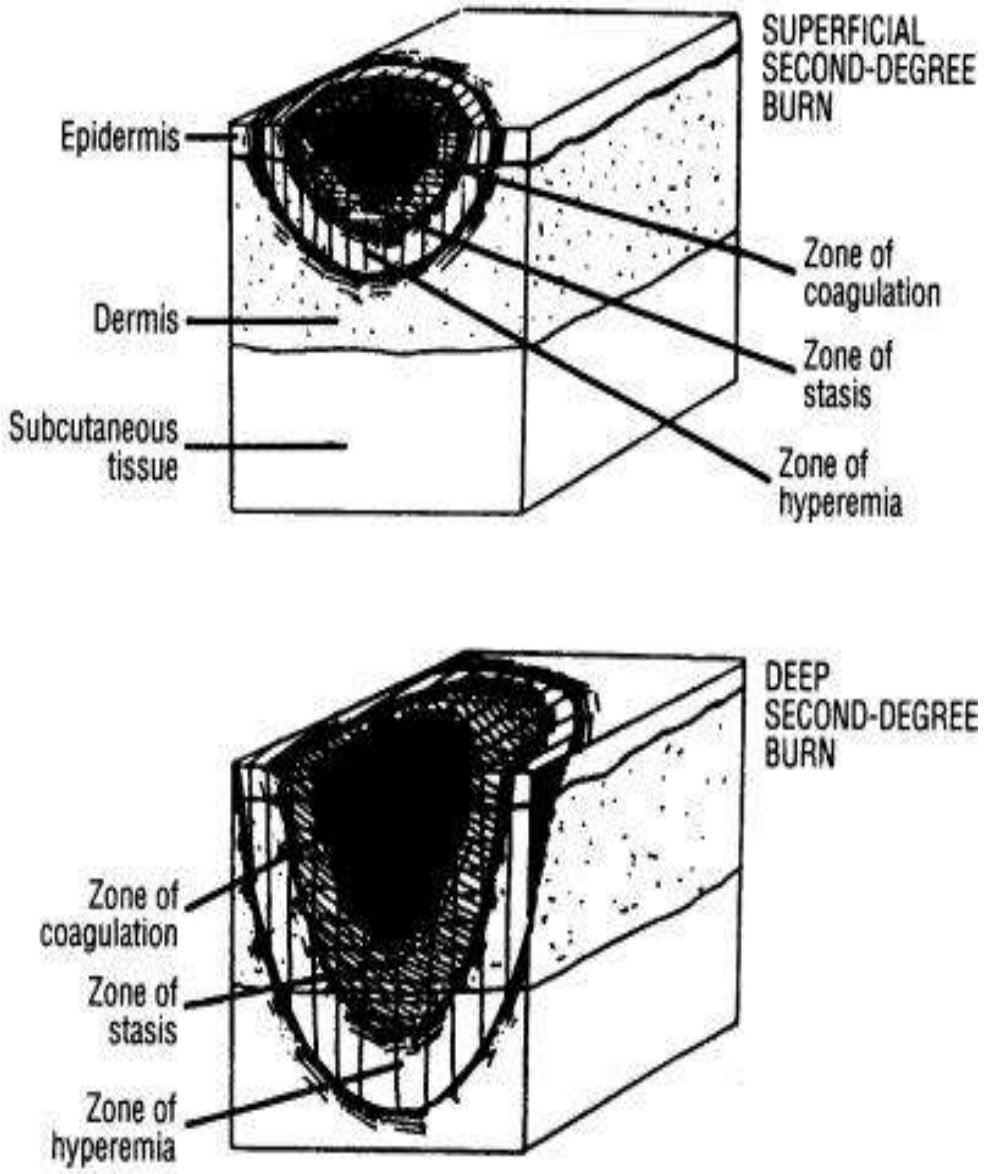

Figure (١) : wound areas in superficial and deep burn

(Roth and Hughes, ٢٠٠٤)

Although burn wound surfaces are sterile immediately following thermal injury, Gram-positive bacteria that survive the thermal insult, such as staphylococci located deep within sweat glands and hair follicles, heavily colonize the wound surface within the first ٤٨ h unless topical antimicrobial agents are used . ***(Altoparlak et al., ٢٠٠٤)***

Eventually (after an average of ٥ to ٧ days), these wounds are subsequently colonized with other microbes, including gram-positive bacteria , gram-negative bacteria, and yeasts derived from the host's normal gastrointestinal and upper respiratory flora and / or from the hospital environment or that are transferred via a health care worker's hands . ***(Erol et al., ٢٠٠٤)***

Sepsis is widely recognized as a clinical syndrome, resulting from an overwhelming, systemic host response to infection. ***(Annane et al., ٢٠٠٥)*** The key clinical manifestations of sepsis are not caused directly by the invading pathogens; rather, the hypotension, coagulopathy, and multisystem organ dysfunction that characterize severe sepsis are predominantly a result of dys-regulation of host-derived mediators of inflammation. Sepsis is the most common cause of death among hospitalized patients in non-cardiac intensive care units . In the last years, tremendous progress has been made in understanding the complex triad of infection, inflammation, and coagulation during sepsis . ***(Angus et al., ٢٠٠١)***