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BURN WOUND SEPSIS IN ADULTS-A PROSPECTIVE STUDY

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CERTIFICATE

This is to certify that the work embodied in the dissertation entitled **'BURN WOUND SEPSIS IN ADULTS - A PROSPECTIVE STUDY'** has been carried out by **DR. S.MANICKAVASAGAM MS (ENT)**during the period between August 2005 & August 2008 in the Department of Burns, Plastic & Reconstructive surgery, Kilpauk Medical College, Chennai for the partial fulfillment of Mch BRANCH III PLASTIC SURGERY Degree Examination.

The work has been carried out with care and precision.

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INTRODUCTION

Although burn injuries are frequent in our society, many of these patients need hospitalization, and more than 40% die due to infections. Mortality is highest among the very young and the very old[1,17] Two-thirds of all burn accidents occur at home and most commonly involve young adult females and children. Young adults are most commonly burned by flammable liquids, while toddlers are most often scalded by hot liquids while playing in the kitchen. Structural fires[33] cause about 5% of burn accident. Inhalation injury has the biggest impact on both early and late mortality[2].

Advances in trauma and burn management has resulted in improved survival and reduced morbidity from major burns. 50 years ago, the mortality rate of a 50% body surface area (BSA) burn in a young adult was about 50% despite treatment[33,44]. Today, over 50% of these patients are surviving. Improved results are due to advancements in resuscitation, surgical techniques, infection control, nutritional and metabolic support.

THIS STUDY WAS CONDUCTED IN THE DEPARTMENT OF BURNS, PLASTIC& RECONSTRUCTIVE SURGERY, KILPAUK MEDICAL COLLEGE, CHENNAI-10.

AIMS AND OBJECTIVES

1. To find out the prevalence of infection in adult burn patients.

2. To find out the causative organisms and their antibiotic sensitivity.

3. To find out the most common type of infection.

4. Role of age, sex, buns percentage and general condition of the patient on the recovery.

5. To find out prevalence of drug resistant and changing pattern of microbes in burn patient.

HISTORY AND REVIEW OF LITERATURE

The Neanderthal man used extracts of plants to cover wounds. 1500 BC Egyptians used gum and goat's milk mixed with mother's milk to treat burns. The Chinese (600 to 500 BC) used extracts of tea leaves to treat burns. Hippocrates (460 BC) used swine's semen, resin, bitumen and Oak bark solution to cover burns. Celsus and Galen Roman physician used honey, bran and vinegar to bind burn wounds[44].

Rhases (9th century) advocated the use of cold water to wash wounds. PARE (1517 to 1596): advocated excision of dead tissue and application of ointment in burns.

David Cleghorn (1792) used vinegar and chalk poultice to cover burns. Edward Kentish (1797) revolutionized the use of pressure dressing in burns treatments. Syme (1827) used wool dressing and Lisfranc (1835) used calcium chloride dressing for burns. Passavant (1858) introduced the saline bath in burns treatment.

REVIEW OF LITERATURE

The skin is the largest organ in the body, comprising 15% of body weight and covering approximately 1.7 m² in an average adult. The function of the skin is complex: it warms, it senses, and it protects. Of its 2 layers, only the epidermis is capable of true regeneration. When the skin is seriously damaged, this external barrier is violated and the internal milieu is exposed and altered.[4]

BURN INJURY

A burn injury implies damage or destruction of skin and or its contents by thermal, chemical, electrical or radiation energies or combinations thereof. In our department thermal injuries are by far the most common type and frequently present with concomitant inhalation injuries.

A thermal injury involves, heating of tissues above the critical level (40^{o}C) at which damage occurs via protein denaturation[13,14] . Tissue injury depends on the heat content of the burning agent, the length of exposure and the thermal conductivity of the involved tissue.

The hydrophilic human skin possesses a high specific heat and a low thermal conductivity. Therefore, skin becomes overheated quite slowly, but also cools slowly. As a result, thermal damage continues after the burning agent is extinguished or removed.

BURN SYNDROME

Following a major burn injury, sequential physiologic changes occur that together comprise the clinical scenario of the burn patient. These derangements include:

ELECTROLYTE AND FLUID DERANGEMENT[7,12,29]

The burn wound becomes rapidly edematous due to micro vascular changes, induced by direct thermal injury and by release of chemical mediators of inflammation. This results in systemic intravascular losses of water, sodium, albumin and red blood cells. Unless intravascular volume is rapidly restored, shock will develop.

METABOLIC DERANGEMENT

This is evidenced by increased resting oxygen consumption (hyper metabolism), an excessive nitrogen loss (catabolism), leading to pronounced weight loss and malnutrition.

VITAL ORGAN DYSFUNCTION:

All major organ systems are affected by the burn injury. Renal insufficiency can result from hypoperfusion or Renal tubular obstruction by myoglobulin and hemoglobin. Pulmonary dysfunction may be caused by initial respiratory tract damage or progressive respiratory insufficiency due to pulmonary oedema or adult respiratory distress syndrome or bronchopneumonia[41].

Gastrointestinal complications include paralytic ileus, and gastrointestinal ulcerations. The small bowel ischemia and stasis promote bacterial translocation as a mechanism for endogenous infection. And multi-system organ failure is a common final pathway leading to late burn mortality.

BURN SHOCK [31,29].

The burn wound is a 3-dimensional mass of damaged tissue. At its margin is the zone of hyperemia and at its center is the zone of coagulation, surrounding the coagulation necrosis is the zone of stasis, so named because it starts with a circulation, which becomes static. Due to direct thermal effects, the blood vessels in this region dilate and its endothelial lining 'leaks' plasma and intravascular proteins.

Within minutes to hours, the circulation in this region ceases as the capillaries become packed with red blood cells and microthrombi, aggravating the inflammatory response. Although the cellular damage in this region is potentially reversible, injury to the microcirculation is progressive over 48 hours[12]. Extent of the injury is minimized by adequate fluid resuscitation. The inflammatory response in the zone of stasis is responsible for burns oedema. Regional edema occurs in the burned tissue due to increased microvascular permeability, vasodilatation, and increased extra vascular osmotic activity in damaged tissue, infiltration of tissues by leukocytes and release of vasoactive substances.

I. EARLY BURN MANAGEMENT[24]

Treatment of the burn injury begins at the scene of the accident. The first priority is to stop the burning. The patient must be separated from the burning source. For thermal burns, immediate application of water on burns can reduce tissue damage then the patient should be covered up to avoid shivering prior to shifting.

This application of water carefully done in large burns and in children, as prolonged cooling can precipitate a dangerous hypothermia.

For electrical burns the electricity should be switched off. The appliances or the source of contact should be removed from the patient. As with other forms of trauma, initial establishment of an adequate airway is vital. In chemical injuries, the agent should be washed off with copious water.

Endotracheal intubation[18,24.] is not an essential part of management of all

inhalation injuries. If the patient shows evidence of airway edema and impending obstruction in the form of hoarseness, wheezing, or stridor, then endotracheal intubation will be advised.

In all burn patients administer 100% oxygen by mask or tube to reduce the likelihood of problems from pulmonary dysfunction or carbon monoxide poisoning. In case of associated injury appropriate management like control of external hemorrhage and stabilization of fractures from concomitant trauma must be done. Burn wounds should be covered by a clean dressing.

Although, a 20-40% burn injury can initially appear normal burn shock can develop rapidly if fluid resuscitation is delayed. Burns of less then 15 % BSA in the conscious and cooperative patient can often be resuscitated by oral fluids.

The patient with more than 15% BSA burns, requires IV access. Cut downs or central lines are initially less desirable. Start infusion of Ringer's lactate solution of about 1000 ml/hr in adults, 400-500 ml/m²BSA/hr in children, until more accurate assessments of burn size and fluid requirements can be made. Foley's catheter should be placed to monitor urinary output. A nasogastric tube is inserted with the tip placed in the duodenam for gastric decompression and for early feeding.

Patient evaluation should include history of allergies, medications, pre-existing diseases, last meal, events of the injury including time, location and type of insult. Medicolegal aspect should be taken care of patient when needed.

History of loss of consciousness should be elicited, in patients who are

alcoholic, drug user, smokers or mentally unstable persons. Complete physical examinations should include careful neurological examinations to rule out cerebral anoxic injury. In all trauma patients, occult injuries must be ruled out.

Patients with facial burns should get an Ophthalmologists opinion regarding the status of cornea and eye ball.

Routine Laborotory investigations[18] should include CBC, electrolytes, Blood glucose, serum creatinine, serum protein, LFT and serum calcium. For proper Pulmonary assessment, arterial blood gases, chest x-ray, should be done. Despite a toxic level of a carbon monoxide (i.e. greater than 15%), pO_2 and saturation values may be normal.

An ECG is important in all cases of electrical injuries and in patients more than 40 years of age, and extremities should be examined for pulses, especially in circumferential burns. Evaluation of pulses can be assisted by use of a Doppler ultrasound flow meter. In case of circumferential burn, with absence of distal pulse inspite of adequate fluid resuscitation, should undergo urgent escharotomy for release of the constrictive eschar. In severe chest burns, escharotomy may also be indicated to relieve chest wall constriction and improve ventilation. Escharotomies[6,18.]are generally performed at the bedside under IV sedation.

Mid axial incisions are preferred and completed through the bleeding tissue, extending the full length of the eschar to achieve adequate release, limbs should be elevated above heart level. Distal limb viability and Pulses should be monitored for 48 hours. Occasionally escharotomy alone will fail to relieve intra-compartmental pressures in such cases fasciotomy under anesthesia is

indicated.

Distal numbness and tingling sensations are the earliest signs of ischemia. Absence of pulse is a late finding. If facilities are available patient central venous pressure should be monitered. Central venous pressure is normally 8-12 cm H2O and arterial pressure is typically 80-120 mmHg[31]. Venous compromise occurs much before the loss of arterial pulsations in an affected extremity. Intra-compartment pressures can be measured by using wick catheter or inserting a 25- 26 gauze needle connected to a pressure catheter, such as for arterial line, into all compartments of the affected extremity[44].Compartment pressures of more than 30 cmH2O require immediate attention.

Fasciotomies are always indicated in high voltage electrical injuries and in severe crush injuries. This should include Carpal Tunnel and Guyon (Ulnar) canal releases. An 'intrinsic-minus' hand deformity is indicates the need for deep muscle compartment decompressions. As the patient is resuscitated, new swelling and reperfusion injury can cause delayed onset of compartment syndromes, and this need continuous monitoring.

All patients with significant burns should receive 0.5 ml of tetanus toxoid. If prior immunization history is absent or unclear, or the last booster dose was more than 10 years ago, 250 units of tetanus immunoglobulin should be given.

II. FLUID RESUSCITATION [44,36].

The most crucial aspect of early management of burn patient is prompt initiation of volume replacement. Large quantities of salt-containing fluids should be started immediately sufficient to maintain adequate perfusion of vital

organs. Many formulas for burn resuscitation have proven clinically efficacious, and each differs in volume, sodium, and colloid content.

	Brooke	**Modified Brooke**	**Parkland**	**Monafo**	**Evans**
Day 1					
Colloid	0.5mL/kg/%burn	None	None	None	1mL/kg/%burn
Crystalloid	Lactated Ringer solution, 1.5mL/kg/% BURN	Lactated Ringer solution, 2mL/kg/% burn (adult), 3mL/kg/% burn (child)	Lactated Ringer solution, 4mL/kg/% burn	250mEq Na, 150mEq lactate, 100mEq CL, titrate to urine flow	Lactated Ringer solution, 1mL/ kg/% burn
5% D/W	2000mL/m^2	None	None	"Liberal" free water by mouth	2000mL/m^2
Urine	30-50mL/hr (adult)	30-50mL/hr (adult) 1mL/kg/hr) (child)	50-70mL/hr (adult)	30-50mL/hr (adult)	30-50mL/hr (adult)
Rate	½ total in first 8 hr, ¼ total in next 8 hr, ¼ total in next 8 hr	Same as Brooke	½ total in first 8 hr, ¼ total in next 8 hr, ¼ total in next 8 hr	Infuse Constantly	½ total in first 8 hr, ¼ total in next 8 hr, ¼ total in next 8 hr
Calculation of volume	Same as Evans	Same as Parkland	Use total burn area for all sizes of burn	Titrate to urine production, not burn	Use burn area up to a total of 50% TBSA; above 50% TBSA burn, calculate as 50% burn
Day 2					
Colloid	0.25mL/kg/% burn	0.3-0.5mL/kg/% burn	700-2000mL (adult) as required to maintain urine	-	0.5mL/kg/% burn
Crystalloid	Lactated Ringer solution, 0.5mL/kg/% burn	None	None	-	Lactated Ringer solution, 0.5mL/kg/% burn
5% D/W	1500-2000mL	Sufficient to maintain normal urine output	Sufficient to maintain normal urine output	-	15000-2000mL

In each case, half of this volume is administered in the first eight hours, the remaining half administered within 24 hours. The rate is adjusted hourly to maintain a urinary output of 30 ml/hr (o.5 ml/kg/hr) in adults and 1 ml/kg/hr in children and to keep the Serum albumin level more than 2.5 gm/dl.

Additional fluids are commonly needed in inhalation injuries, electrical burns, associated trauma and delayed-resuscitated patients. The appropriate resuscitation regimen administers the minimal amount of fluid necessary for maintenance of vital organ perfusion as measured by adequate urine output.

Invasive haemodynamic monitoring with central venous catheters, arterial lines, and SwanGanz catheters are usually not needed.[4]

Central arterial lines especially carry an increased risk of sepsis, thrombophlebitis and endocarditis in thermally injured patients.

BURNS ASSESMENT

Once the cardiopulmonary assessment and resuscitations are over, more careful evaluation of the burn wound is performed. If the patient is adequately hydrated, appropriate doses of IV sedatives and narcotics may be safely administered. Excessive narcotics in patients with inadequate resuscitation, can precipitate burn shock.

The wounds are gently cleaned with normal saline, loose skin and blisters are debrided. Blister fluid contains high amount of inflammatory mediators, proteins and electrolytes, the loss of which increase burn wound ischemia. The blister fluid is also a rich media for bacterial growth[44]. Once the burn wound depth and percentage assessment is completed, the wounds are covered with silver sulfadiazine cream and an appropriate burn dressing applied. Estimation of burn size and depth assists in determinations of severity and prognosis of the patient. Burn size directs the amount of fluid resuscitation, nutritional support and surgical interventions. Estimation of burn

depth is a clinical judgment based on morphological appearance of the wound.

Degree	Depth	History	Etiology	Sensation	Appearance	Healing
1st degree	Superficial	momentary exposure	sunburn	sharp, uniform pain	blanches red, pink, edematous, soft, flaking, peeling	± 7 days
2nd degree	Superficial/deep	exposure of limited duration to lower temperature (40-55°C)	scalds, flash burn without contact, weak chemical	dull or hyperactive pain, sensitive to air/temp changes	Blisters thin walled,large,gelatinous,easily rupture,base bright red appearance/thick walled,small,difficult to rupture,base pale appearance	10-21 days
3rd degree	full thickness	long duration of exposure to high temperature	immersion, flame, electrical, chemical	painless to touch and pinprick, may hurt at deep pressure	no blanching, pale white, tan charred, hard, dry, leathery, hair absent	granulates, requires grafting

First degree burns are superficial and involves just epidermis. 1st degree burns are inconsequential in subsequent burn management. Partial-thickness injuries are 2nd degree burns that involve variable amounts of dermis. The hallmark of a partial-thickness burn is blisters and painful wound that will potentially heal within 2 to 3 weeks. Blister fluid contains rich protein, fluid and electrolytes. 3rd degree burns are full-thickness injuries, which require skin grafting following loss of the devitalized dermis (Eschar)[36].

Full-thickness burns are identified as dry or leathery wounds that are initially insensate to light touch or pinprick. In infants, 3rd degree burns may also appear cherry red. Determinations of burn depth can be misleading initially, as the tissue destruction is progressive over the first 48 hours.

Burn size is based on the percentage of 2nd and 3rd degree burns as compared to total body surface area. Burn injuries are quantifiable, and patho-physiological derangement is related to the size of burn injury. The surface area of a patient's palm is approximately 1% of their total body surface area and provides a quick estimation of burn size. Typically, burn size estimations are derived from the "Rule of Nines".

The body's surface is divided into areas of roughly 9% each, which includes the head and neck, the chest, the abdomen, the upper back, the lower back and buttocks, each thigh, each lower leg, and each upper extremity, the "Rule of Nines" overestimates burn size in children. The head and neck account for a larger proportion of the total body surface area (BSA) in children, and more than 21% BSA in toddlers and babies.

For accuracy and reproducibility, burn size should be determined by plotting the burn wound on Lund and Browder's burn diagrams (chart).

A major burn injury is defined as greater than 25% BSA involvement in adults (15% in children), or more than 10% BSA of full-thickness involvement. Major burns require aggressive resuscitation, hospitalization, and appropriate burn care. Additional criteria for major burns injury includes, deep burns of the hands, feet, eyes, ears, face, perineum, inhalation injuries and electrical burns.

Moderate thermal burns of 15-25% BSA, or 3-10% BSA full-thickness, often require hospitalization for optimal patient care.

Other criteria for admission include concomitant trauma, significant pre-existing disease, and suspicion of child abuse. Minor burns can generally be treated on outpatients basis.

Classification of Burn Injuries		
Major Burns	**Moderate Burns**	**Minor Burns**
Burn surface involvement of 25% body surface area. Full-thickness burns 10% body surface area. Deep burns of the head, hands, feet, and perineum. Inhalation injury. Chemical or high-voltage electrical burn.	Burn area of 15-25% body surface area. Superficial partial-thickness burns of the head, hands, feet or perineum. Suspected child abuse. Concomitant trauma. Significant pre-existing disease.	15% body surface area. Nothing involving the head, feet hands or perineum.

III. POST BURN INFECTION AND SEPSIS[33]

BACTERIAL INFECTION ON TISSUES:

The damaged integument creates a vast area for surface infection and invasion of microorganisms. Burned patients with a major thermal injury are unable to maintain an adequate immunologic defense, and this increases the risk of septic shock.

BACTEREMIA:

Is the transient presence of bacteria or other microorganisms in the blood (e.g. bacteria in the blood after brushing one's teeth).

SEPTICEMIA:

Is the invasion of the blood stream by pathological microbes from a focus of infection and an active proliferation of these microbes accompanied by hyperthermia or hypothermia and prostration. Frequently, it is diagnosed clinically by the presence of any three of these cardinal signs:

Obtundation, Hyperventilation, ileus, thrombocytopenia, hyperglycemia, leukocytosis or leukopenia.

COLONIZATION

Is the mere presence of bacteria, and establishment of a colony with Bacterial counts of 10^5 bacteria/gm[42] of tissue with no evidence of invasion into viable tissues.

Infection is the most common and most serious complication of a major burn injury related to burn size. Sepsis accounts for 50-60% of deaths[38] in burn patients despite improvements in antimicrobial therapies. Sepsis in burns is commonly due to bronchopneumonia, pyelonephritis, thrombophlebitis, or wound infection[44].

The burn wound is an ideal substrate for bacterial growth and provides a wide portal for microbial invasion. Microbial colonization of the open burn

wounds, primarily from an endogenous source, is usually established by the end of the first week[44].

Infection is promoted by loss of the epithelial barrier, malnutrition induced by the hypermetabolic response to burn injury, and by a generalized post-burn immunosuppression due to release of immunoreactive agents from the burn wound.

IV. CLINICAL DIAGNOSIS OF SEPSIS:

ATLEAST 3 OF THE FOLLOWING CRITERIA SHOULD BE MET[32] FOR DIAGNOSIS:

- Burn wound infection (>10^5 organisms/gm tissue) with histological or clinical evidence of invasion
- Thrombocytopenia (<50,000) or falling rapidly
- Leukocytosis or Leukopenia (>20,000 or <3,000)
- Unexplained hypoxia, acidosis or hyper/hypoglycemia
- Prolonged paralytic ileus
- Hyper/hypothermia (>39°C or <36.5°C),
- Positive blood cultures
- Documented catheter or pulmonary infection
- Altered mental status
- Progressive renal failure or pulmonary dysfunction

LOCAL EVIDENCE OF INVASIVE WOUND INFECTION[34,38,44].

- Black or brown patches of wound discoloration
- Rapid Eschar separation

- Conversion of partial thickness wounds to full-thickness
- Spreading peri-wound erythema.
- Punctuate hemorrhagic sub-Eschar lesions
- Violaceous or black lesions in unburned tissue

BLOOD CULTURE[36,37].

Blood cultures are essential in determining septic episodes. The best time to collect the specimen is before the temperature spikes. A temperature above 39.5 is the body's natural method of eliminating the bacteria from blood stream.

Most bacteria do not survive at temperature above 39.5^0C. Care must be taken not to contaminate the blood culture bottle. Site selection for collecting the specimen must be meticulously and aseptically cleansed with normal saline prior to specimen collection.

No spirit /antiseptic to be used. If the site is through a contaminated area, appropriate comments should be made on the request slip for culture.

URINARY TRACT INFECTION (U.T.I.)

If U.T.I. is suspected as the cause of sepsis, urine must be collected aseptically. Appropriate comments should be made on the request slip for culture.

UPPER RESPIRATORY TRACT INFECTION (U.R.I.)

If a U.R.I. is suspected or evident from clinical signs appropriate investigations like x-ray, sputum, and bronchial washings are essential in order to identify the etiologic agent or infection.

V. IMMUNOSUPPRESSION IN BURN INJURY[5,44.]

This is characterized by:

1. Decreased Serum levels of immunoglobulins,
2. Decreased Fibronectin
3. Decreased Complement levels
4. Diminished ability for opsonization.
5. Impaired chemotaxis, phagocytosis, and killing function of neutrophils, monocytes, and macrophages
6. Granulocytopenia.
7. Cellular immune response is impaired, as evidenced by delayed allograft rejection, anergy to common antigens.

8. Impaired lymphocyte functions, and altered mixed lymphocyte responsiveness.

9. Decreased Interleukin-2 (IL-2) production, T-cell and NK (natural killer) cell cytotoxicity, and helper to suppressor T-cell ratio (HSR).

INFECTION CONTROL:

The avascular burn eschar is rapidly colonized by 2nd post burn day, despite the use of antimicrobial agents[8]. If the bacterial density exceeds the immune defenses of the host, then invasive burn sepsis may develop.

When bacterial wound counts are >10^5 microorganisms per gram of tissue, risk of wound infection is great, skin graft survival is poor, and wound closure is delayed. The goals of local wound management are the prevention of dessications of viable tissue from the body surface and the control of bacterial infection. These are achieved by use of topical antimicrobial agents and/or biological dressings. It is very difficult to keep a burn wound sterile. Bacterial counts of less than 10^3 organisms/gm are not usually invasive and allow skin graft survival rates of >90%. Antimicrobial therapy is directed by bacterial surveillance through routine sputum, urine, and wound cultures[44].

Quantitative wound biopsy is a better determinant of significant pathogens. Organism identification and sensitivities to antibiotics are obtained within 48 hours. Agar diffusion assays can also be performed to test susceptibilities to topical antimicrobials.

If quantitative biopsies reveal microbial density of $>10^3$ organisms/gm, a change in topical therapy is indicated. If bacterial counts are 10^5, wound infection should be suspected[14] and rapid histological analysis and gram stain should be performed to confirm this. Wound infection is confirmed by histological evidence of tissue invasion or clinical sepsis. Systemic antibiotics must be instituted, and early care of the wound should be aimed at.

Diagnosis of sepsis in burn patients can be difficult to distinguish from the usual hyperdynamic, hyperthermic, hypermetabolic post-burn state.

Blood cultures are commonly negative. Fever spikes are not proportional to the degree of infection.[23.]

Appropriate systemic antibiotics are administered as indicated. The appropriate use of antibiotics should be based on the following definitions:

PROPHYLAXIS[35,44.]

It is a preventative or precautionary measure designed to preserve health and prevent the spread of disease.

PERIOPERATIVE:

This may also be considered prophylaxis. It is the administration of systemic antibiotics as a protective measure for any type of surgical intervention. The time frame for administration should be short-lived and is limited to 1 to 3 doses, depending on the operative procedure.

THERAPEUTIC:

This is the administration of antibiotics for the treatment of infection. Depending on the infection, therapy may be continued for several days. Systemic antibiotic treatment for burn wound sepsis is continued for at least 72 hours after evidence of sepsis has resolved[37]. If the wounds appear clean, other sources such as the lungs, the kidney, and peripheral veins should be suspected. In the absence of confirmed organisms antibiotic selection should be based on routine surveillance & cultures. Empiric antibiotic choice should also be based on culture sensitivities of the burns unit.

VI. TOPICAL ANTIMICROBIALS[29,44.]

Currently, number of topical agents are available to assist in microbial control of the burn wound, including Silver sulfadiazine, Mefenideacetate, 0.5% Silver nitrate, Bacitracin/polymyxinB, Mupirocin, and Mycostatin.

No single agent is totally effective and each has advantages and disadvantages. Almost all agents will affect wound healing.

SILVER SULFADIAZINE[19]

Silver sulfadiazine is the most commonly used topical antimicrobial agent in burns. Its antimicrobial properties are derived from dual mechanisms of its silver and sulfa moieties and has a broad spectrum of antimicrobial coverage including gram positive bacteria, most gram negative bacteria, and some yeast forms.

Unlike mafenide or silver nitrate, silver sulfadiazine does not hinder

epithelialization, although it does hamper contraction of fibroblasts.

Furthermore, silver sulfadiazine is painless on application and reduces pain significantly once applied. It has high patient acceptance, it is also easy to use with or without dressing. Although true allergic sensitivities are rare, some of the patients will develop a transient leucopenia secondary to margination of circulating white blood cells. This leucopenia is generally harmless needs observation, but not cessation of treatment. However, if the white blood cell count drops below 3000/cumm, the medication is sometimes withheld until the WBC count raises to >4000-5000/cumm.

MAFENIDE ACETATE 11.2% CREAM[30]

It is one of the oldest and effective topical antimicrobial agents. Mafenide has a broad spectrum of antimicrobial activity, including silver sulfadiazine-resistant pseudomonas and enterococci, but reduced antifungal properties. Its exact mechanism of action is not clear, but thought to be related to its water-soluble sulfa moiety.

Mafenide cream is toxic to epithelial cells and fibroblasts. Unlike other topical agents, mafenide has good penetration through the eschar. For this reason mafenide is commonly used on dirty or infected burn wounds, or electrical burns, and on burned ears to prevent chondritis.

Following the application of mafenide produces painful sensation for several minutes. Mafenide can cause an allergic skin rash. Through carbonic anhydrase inhibition, mafenide can also cause bicarbonate wasting in the kidneys, hyperchloremia, systemic metabolic acidosis and compensatory

hyperventilation. To protect against such metabolic abnormalities one can monitor serum electrolyte levels and treat abnormal values with appropriate intravenous replacement therapy.

Alternatively, applications of mafenide cream can be limited to no more than 20% of the BSA at any one time[44]. The sites of mafenide applications can then be rotated every 2 hours until the entire burn has been treated. Alternate mafenide and silver sulfadiazine creams on seriously contaminated wounds is called 'pulse therapy'.

SILVER NITRATE 0.5% SOLUTION[20,21.]

It is a broad spectrum, non-penetrating, painless antimicrobial agent. It requires multiple daily applications the disadvantage is messy and staining of dressings. The solution is hypotonic, so electrolyte wasting like hyponatremia, and hypokalemia are common. A rare complication of silver nitrate use is methemoglobinemia.

Silver nitrate dressings can also be used in the treatment of toxic epidermal necrolysis syndrome and patients allergic to silver sulfadiazine or mafenide.

Petroleum-based antimicrobial ointments such as bacitracin and/or polymyxin B are painless on application, and allow for easy wound observation. These agents are commonly used for treatment of facial burns, graft sites, healing donor sites, and small partial-thickness burns.

Povidone iodine topical ointment has a broad spectrum of antimicrobial

activity, against bacteria, fungi, and some viral forms.

Mupirocin has improved activity against gram positive bacteria, especially methicillin resistant staph aureus(MRSA)[22]and selected enteric bacteria.

Gentamicin ointment can be used for selective and resistant organisms, but it diminish effectiveness of parenteral gentamicin.

MYCOSTATIN:

In severely burned patients (>40% BSA), the combination of Mycostatin ointment with other topical agents reduces the incidence of fungal super-infections and improves antimicrobial action. Mycostatin should not be combined with mafenide, because both become inactivated. In addition, Mycostatin 5-15 ml given orally 3 times daily reduces alimentary fungal overgrowth.

Topical antimicrobial creams are usually used with closed dressings. This provides greater patient comfort and less dessications than the open technique. The creams are spread on fine mesh gauze, applied on the wounds then covered with bulky protective gauze dressing. Dressing changes are performed every 12 to 24 hours. At each dressing change, wounds are gently cleaned prior to re-application

VII. BIOLOGICAL DRESSINGS[25].

Biological dressings usually have no antimicrobial properties. However, they create a wound environment that prevents dessications, diminishes

bacterial proliferation, reduces loss of heat, water, protein and red blood cells, and promotes more rapid wound healing. Biological dressings also reduce burn wound pain.

Biological dressings should be applied as early as possible. Before application all dead tissue have to be removed thoroughly and early application will be ideal.

Fresh skin allograft can be used as temporary coverage of the clean open burn wound. Allograft achieves an environmental 'seal' of the burn wound.

Allogenous human skin graft[26,27] can be obtained from fresh cadavers within 18 hours of death or from living relatives to assure that skin cells within the graft are viable. The graft can re-vascularize once applied to the wound. Allograft provides the best temporary cover for the excised burn wound. However, the Langerhans cells in the transplanted epidermis retain their antigenicity, and the skin allograft will undergo rejection within 7 to 14 days in normal patients.

Fresh skin allograft has a high price tag, a limited supply, a short shelf life (2-3 weeks), and a need for refrigerated storage. Although shelf life may be improved by freezing or lyophilization, these processes diminish cell viability, graft adherence, and protective functions of the allograft. Fresh skin allograft should only be used on clean wounds.

AMNION:

Amnion is readily available from labour ward and is inexpensive.

However, it adheres poorly to the wound. Usually amnion following LSCS used for this purpose. Although amnionic coverage can promote angiogenesis and increased wound capillary density. Due to the increased prevalence of HIV infection amnions are no longer advised

PORCINE SKIN[25].

Porcine skin is nonviable, adheres less than allograft, and does not undergo re-vascularization by the recipient bed. It undergoes progressive degenerative necrosis rather than classic rejection. Also, Xenograft does not provide the same level of protection from infection as allograft, so porcine skin is often embedded with salts of antimicrobial agents to increase its bacteriostatic potential.

Febrile responses can be caused by reaction to the treated porcine skin or to hidden wound infections, and fever spike requires temporary xenograft removal. However porcine skin is well-suited for temporary coverage of full and partial-thickness burn wounds.

Porcine skin is cheaper and more available than allograft. Its recommended uses include protective coverage of partial-thickness wounds to allow spontaneous healing,[25] temporary coverage of clean granulating wound beds between autografting procedures,[39] and to serve as a 'test graft' to decide suitability for autograft closure. Porcine skin should not be used on densely contaminated or non-viable wound surfaces.

On deep dermal defects, porcine skin should be changed every 3-4 days to prevent infection.

Synthetic biological dressings also provide wound protection from dessications, contamination, increase the rate of wound healing, and reduce patient discomfort. When used to cover clean partial-thickness wounds, the dressing detaches as re-epithelialization and keratinization occurs underneath.

BIOBRANE[38,40].

It is a synthetic, bilaminar membrane with an outer semi-permeable silicone layer bonded to an inner collagen nylon matrix. In-growth of granulation tissue into the inner Biobrane layer increases its adherence. Its elasticity and transparency allows easy drape ability, full range of movement and easy wound inspection.

Biobrane is suited for donor sites, superficial partial-thickness burns, and clean, excised wounds prior to grafting. Biobrane gloves on partial-thickness hand burns reduce discomfort and increase motion, allowing earlier aggressive physiotherapy. Biobrane can also be used to cover meshed skin grafts[38] to prevent slipping and dessications. The problem with Biobrane are its expense and its lack of inherent antimicrobial properties.

COLLAGEN:

It is a semi-occlusive, self adhering and sterilized Type 1 collagen.

SALIENT FEATURES

- Single application and effective barrier against infection.
- Promote epithelialization and protects regenarating epithelium.
- Preserve local heat and effective wound healing.
- Non toxic, non immunogenic and biocompatible.

APPLICATION:

Before application denuded areas thoroughly cleaned with normal saline. Once the pack was opened, collagen sheets were washed with normal saline and applied immediately.

USES

- 2nd degree non-infected superficial and deep dermal burns.
- As a temporary cover for 3rd degree Burns, after Escharectomy and tangential excision.
- Skin donor sites.
- Traumatic loss of skin cover.
- Protective cover over widely meshed autografts.

CONTRAINDICATIONS:

- Patients allergic to Collagen.
- Patients with known sensitivity to bovine material.
- Grossly infected wounds.

MATERIALS AND METHODS

INTRODUCTION

This was a prospective study which was conducted in the DEPARTEMENT OF BURNS, PLASTIC& RECONSTRUCTIVE SURGERY, KILPAUK MEDICAL COLLEGE, CHENNAI. Two hundred and eight (208) patients with acute burns were included in this study which was conducted from January 2006 to December 2007.

INCLUSION CRITERIA

1. All patients with acute burns admitted within 12 hours of the incident.
2. Chemical , Electrical , Flame Burns
3. Patients aged between 12 to 70 years
4. Patients with 20-60% burns

EXCLUSION CRITERIA

1. Patients admitted after 12 hours from the incident.
2. Admitted and treated at other hospitals and subsequently referred patient.
3. Patients aged less than 13 years and over 70 years.
4. Patients with associated systemic diseases like diabetes, Systemic hypertension, renal, hepatic disorders etc.
5. Burns more than 60%

METHOD

All patients having burns admitted via casualty to the burns department were meticulously followed up from admission to till discharge. The clinical course during the treatment was studied.

EXAMINATION

All the admitted patients were thoroughly examined clinically. The percentage of burn accurately calculated by using LUND-BROWDER chart, and the degree of burns assessed by morphological appearance of burn wound.

LABORATORY INVESTIGATION

- Heamoglobin, heamatocrit, blood urea, blood sugar, serum creatinine, serum alkaline phosphatase, serum proteins, serum Electrolytes, SGOT, SGPT, and serum bilirubin.
- ECG (In case of Electrical injury, and for patients aged more than 40years)
- Wound swab for culture and sensitivity was taken on admission, after 48hours, 4th and 7th post burn day in the study group.
- In the control group, burn wound swab was taken on 7th post burn day only
- Blood culture was done to find out the causative organism, in case of wounds swab negative for culture& sensitivity (after 48hrs) and patients condition was detoriating.

MANAGEMENT

In all patients the degree and depth of burns were assessed clinically, after removing all clothes. Depending on the degree of burns intravenous ringer lactate solution calculated by modified Parkland formula (3ml/kg/%of burns) was administered.

For early feeding and monitoring urine output, Ryle's tube and Foley's catheter were inserted for most of our patients.

104 patients were included in the study group and equal number in the control group using similar inclusion & exclusion criteria. Swab culture and sensitivity was taken in all patients, five antibiotics were selected (Ampicillin,Ceftriaxone, Cloxacillin, Ciprofloxacin, Cefotaxime, as they were the easily available IV antibiotics supplied in the hospital). They were randomly allocated to patients in both groups. The antibiotics were continued for 7 cotinuous days in the control group and then swab culture and sensitivity was repeated to check the organism and its sensitivity pattern.

In the study group, swab C&S was done 1) on admission, 2) at 48 hours, 3) 4th PBD, 4) 7thPBD. As per the report, the antibiotics were changed as & when the C&S reports became available.

The pattern of change of flora, their sensitivity, resistance and associated mortality after 7 days were noted.

Using the agar diffusion method, culture for aerobic organism was done and sensitivity was checked using disc method.

Oral feeds were encouraged as early as possible. Most of our patients have no problem in taking oral feeds, so they are encouraged.

Supplemental diet like KANJEE (sathumavu kanjee) with added sugar and milk with extra rations of fresh fruits, eggs are given. Patients are encouraged to take plain yogurt, which maintains the normal intestinal flora and reduce the risk of diarrhea.

Wound swabs repeated after 48hrs, 4th, and 7th post burn day. Antibiotics

were altered depending on the culture & sensitivity reports. In the control group the same prophylactic antibiotic was continued for 7 days and the end of 7th day wound swab for culture& sensitivity was taken.

Escharectomy was done after 7 days, once the general condition of the patient stabilized. Early excision (within 3 days) with grafting is not routinely done in our department.

TABLE I

AGE DISTRIBUTION

AGE	NUMBER	PERCENTAGE
< 20 YRS	20	19.23%
20-40 YRS	69	66.34%
> 40YRS	15	14.42%

TABLE II

SEX DISTRIBUTION

SEX	NUMBER	PERCENTAGE
MALE	37	35.57%
FEMALE	67	64.42%

TABLE- III

ANTI-BIOTIC SENSITIVITY VS RESISTENCE

Post Burn Day	Positive culture	Antibiotic sensitivity	Antibiotic Resistance
AFTER 48 HOURS	45	35	10
4^{TH} PBD	100	68	32
7^{TH} PBD	94	70	24

TABLE-IV

AMPICILLIN GROUP

Organism	48 Hours culture	4^{TH} Day culture	7^{TH} Day culture
KLEBSIELLA	01	08	09
PSEUDOMONAS	02	06	02
STAPH AUREUS	02	04	02
E.COLI	01	00	02
PROTEUS	00	00	02
MRSA	01	02	02
NO GROWTH	13	00	01
TOTAL	20	20	20

TABLE-V

CEFTRIAXONE GROUP

Organism	48 Hours culture	4TH Day culture	7TH Day culture
KLEBSIELLA	03	01	07
PSEUDOMONAS	00	04	01
STAPH AUREUS	01	03	00
E.COLI	01	01	01
PROTEUS	00	02	00
MRSA	00	01	01
NO GROWTH	07	00	02
TOTAL	12	12	12

TABLE-VI

CLOXACILLIN GROUP

Organism	48 Hours culture	4TH Day culture	7TH Day culture
KLEBSIELLA	00	02	05
PSEUDOMONAS	02	05	04
STAPH AUREUS	00	02	01
E.COLI	02	00	01
PROTEUS	00	01	01

MRSA	00	01	00
NO GROWTH	09	02	01
TOTAL	13	13	13

TABLE-VII

CIPROFLOXACIN GROUP

Organism	48 Hours culture	4TH Day culture	7TH Day culture
KLEBSIELLA	01	10	07
PSEUDOMONAS	04	03	01
STAPH AUREUS	03	04	05
E.COLI	00	00	00
PROTEUS	00	01	02
MRSA	00	01	02
NO GROWTH	11	00	02
TOTAL	19	19	19

TABLE-VIII

CEFOTAXIME GROUP

Organism	48 Hours culture	4TH Day culture	7TH Day culture
KLEBSIELLA	11	13	15
PSEUDOMONAS	05	14	05
STAPH AUREUS	03	08	04

E.COLI	00	02	06
PROTEUS	00	01	04
MRSA	03	01	02
NO GROWTH	18	01	04
TOTAL	40	40	40

TABLE-IX

MICROBES DISTRIBUTION IN CONTROL GROUP

Organism	**7^{TH} Day culture**
KLEBSIELLA	32
PSEUDOMONAS	06
STAPH AUREUS	22
E.COLI	08
PROTEUS	05
MRSA	04
NO GROWTH	27
TOTAL	104

TABLE-IX(A)

COMPARISION BETWEEN ANTIBIOTIC GROUP AND CONTROL GROUP ON 7TH DAY CULTURE

ORGANISM	ANTIBIOTIC GROUP		CONTROL GROUP	
	NO	%	NO	%
KLEBSIELLA	43	41.34%	32	30.76%
PSEUDOMONAS	14	13.46%	10	09.61%
STAPH AUREUS	20	19.23%	22	21.15%
E.COLI	09	08.65%	08	07.69%
PROTEUS	08	07.69%	05	04.80%
NO GROWTH	10	09.61%	27	25.96%
TOTAL	104	100%	104	100%

TABLE –X

BACTERIAL DISTRIBUTION on 7th post burn day

BACTERIA	NUMBER	PERCENTAGE
KLEBSEILLA	43	41.34%
STAPH AUREUS	20	19.23%
PSEUDOMONAS	14	13.46%
E.COLI	9	9.61%
PROTEUS	8	7.69%
NO GROWTH	10	9.61%

TABLE -XI

ANTI BACTERIAL SENSITIVITY DISTRIBUTION (7th PBD)

ANTIBIOTIC	NO OF PATIENT	PERCENTAGE
AMPICILLIN	30	31.9%
AMOXYCILLIN	27	28.7%
CEPHALEXIN	32	34.2%
CEFOTAXIM	29	30.9%
CEFTRIAXONE	26	27.8%
AMIKACIN	52	55.4%
DOXYCYCLIN	23	24.6%
CIPROFLOXACIN	31	32.9%
CLOXACILIN	24	25.7%
GENTAMICIN	40	42.6%
CO-TRIMAXOZOLE	22	23.5%

TABLE-XII

ANTIBIOTICS RESISTANCE DISTRIBUTIONS

Prophylactic antibiotic	48 Hours culture	4TH PBD culture	7TH PBD culture
AMPICILLIN	01	07	05
CEFTRIAXONE	01	00	03
CLOXACILLIN	01	03	04
CIPROFLOXACIN	01	08	06
CEFOTAXIM	04	14	11
CONTROL GROUP			30

TABLE XIII

BURNS MORTALITY

Total no of patients	No of death in antibiotic group	%	No of death in control group	%
104	43	41.34%	54	51.92%

DISCUSSION

This study was conducted in the DEPARTEMENT OF BURNS, PLASTIC & RECONSTRUCTIVE SURGERY, KILPAUK MEDICAL COLLEGE, CHENNAI-10, between January 2006 & December2007.

PREVALENCE OF INFECTION:

The prevalence of burn wound infection was found to be 59.61%. Incidence of burn wound infection on admission was 8.65%,after 48 hours it became to 43.26%,4th day it increased to 96.15% and 7th day it stabilized at 90.38%.Though burn wounds were initially sterile ,they rapidly became colonized by pathological microbes(TABLE-III).

AGE AND SEX DISTRIBUTION:

In our study females in the age group of 20yrs to 40 yrs were more (66.34%) when compared to males(TABLE-I) (TABLE-II).

CHANGING PATTERN OF MICROBIAL ORGANISM

For all patients in the study group, after taking swabs, prophylactic antibiotic was started. Because many of the patients have dirty clothing, poor personal hygiene, they may roll on floor, and applied mud, dosa batter, ink etc.. So we decided to take culture& sensitivity on admission which is supposed to be sterile[44] and we started prophylactic antibiotic. Wanted to check how each organism behaved & how early wound infection occurs so we take culture& sensitivity after 48 hours, the results of which were ready on 4th PBD, so for 4 days, the day one antibiotic had been continued. The pattern of change of flora

& their sensitivity were noted. If the flora changed, on 4th PBD (after 48 hours culture& sensitivity results) the antibiotic also was changed. We wanted to check if such early changes in antibiotic helped in eradication of infection or only resulted in further change in flora. Hence on 4th day, we again checked for the same. The results being received on 6th PBD, since the changed antibiotic had been on only for 3 days, we waited for one more day to have uniformity of them. On 7th PBD again swab culture and sensitivity was taken and the (4th PBD culture and sensitivity report) antibiotic change required was instituted. The 7th PBD culture and sensitivity was received and all the four batches of reports were correlated as below:

In the control group after starting prophylactic antibiotic, swab was taken only on the 7th post burn day. Up to 7 days the same antibiotic was continued.

Swab C&S on admission found to be, sterile or showed only commensals in both groups.

AMPICILLIN GROUP

In 48 hours culture, out of the 20 patients only 7 cases showed growth, staph aureus and pseudomonas being predominant (TABLE-IV).

In 4th day culture, positive in all 20 patients, klebsiella was the predominant organism (TABLE-IV), but antibiotic resistance seen in 7 patients (TABLE-X).

In 7th day culture Klebsiella was the predominant organism (TABLE-IV), and antibiotic resistance was noted in 5 patients (TABLE-XII).

CEFTRIAXONE GROUP

Out of 12 patients in this group, 48 hours culture was positive only in 5 patients, and klebsiella was the predominant organism (TABLE-V).

On 4th day culture, all patients had positive culture, and pseudomonas was the predominant organism (TABLE-XII). No antibiotic resistance was noted.

On 7th day culture, 10 patients showed positive growth, 3 cases showed antibiotic resistance (TABLE-XII).

CLOXACILLIN GROUP

Out of 13 patients, 4 became swab positive, and one case showed resistance after 48 hours culture (TABLE-XII).

On 4th day culture all patients had positive growth, where pseudomonas and klebsiella were predominant organisms (TABLE-VI), and in 3 cases antibiotic resistance was seen (TABLE-XII).

On 7th day culture, klebsiella was isolated as predominant organism (TABLE-VI), 4 patients showed antibiotic resistance (TABLE-XII).

CIPROFLOXACIN GROUP

After 48 hours culture, out of 19 patients 8 patients had positive growth, and one patient developed antibiotic resistance (TABLE-XII).

All patients showed positive growth on 4th day where klebsiella was the predominant organism (TABLE-VII). 8 patients had drug resistant organism (TABLE-XII).

On 7th day culture 17 patients had positive growth, out of which 6

patients had drug resistance (TABLE-XII).

CEFOTAXIM GROUP

After 48 hours culture, out of 40 patients 22 becames culture positive. Pseudomonas was the predominant organism (TABLE-VIII), 4 patients had antibiotic resistance (TABLE-XII).

On 4th day culture 39 patients become swab positive in which pseudomonas and klebsiella were predominant organism (TABLE-VIII). 14 patients developed drug resistance (TABLE-XII).

7th day culture was positive in 36 patients, where klebsiella was the predominant organism (TABLE-VIII). 11 patients developed drug resistance (TABLE-XII).

CONTROL GROUP

On 7th post burn day culture positive in 77 patients, where klebsiella and staph aureus were predominant organism (TABLE-IX). 30 patients developed antibiotic resistance.

Generally after 48 hours culture pseudomonas and staph aureus were the predominant organism isolated.

On 4th post burn day, klebsiella and pseudomonas were predominant organism isolated.

At the end of first week the most common organisms isolated in burn wound, were klebsiella, followed by staph aureus and pseudomonas.

ANTIBIOTIC SENSITIVITY

Over all, antibiotic sensitivity for all organisms was highest for

amikacin on 4th & 7th post burn day (TABLE-XI).

ANTIBIOTIC RESISTANCE

Antibiotic resistance was noted in 30.76%isolates on 4th post burn day and in 23.07% isolates on 7th post burn day. In control group 28.84% isolates showed antibiotic resistance (TABLE-XII).

BURNS MORTALITY

During that study period of 7 days, there were no mortality but, in the 7to 15 days period 43 cases in the study group and 54 cases in the control group expired due to burn wound sepsis(TABLE-XIII).

CONCLUSION

According to this study

- More number of female patients in 20-40 years age group were admitted with burns than compared to male.
- Initially all burn wounds are sterile or only has commensals
- Frequent change of antibiotics results in changing pattern of infective organism.
- Klebsiella is the most common infecting organism seen in our department.
- MRSA infection is insignificant in our unit
- Amikacin was the effective antibiotic in comparison to ampicilln,ceftriaxone,cloxacillin,ciprofloxacin, cefotaxim. But it is preferred as second line antibiotic, since the raw area was still be there (potentially infected). If amikacin is started as first line of defense, chances of developing resistant is high.
- Personal hygiene of both patients & care givers, aseptic precaution during dressing, separate dressing trolley for each patients, periodical bacterial survivallence and fumigation of burns ward can significantly reduce the mortality among adult burn patients.

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ABBREVIATIONS

BSA : Body Surface Area.

CBC : Complete Blood Count.

ECG : Electro Cardio Gram.

HSR : Helper Suppressor T Cell Ratio.

IL : Inter Leukin.

IV : Intra Venous.

LFT : Liver Function Test.

MRSA : Methicillin Resistent Staphylococcus Aureus.

NK cells : Natural Killer Cells.

PBD : Post Burn Day.

SGOT : Serum Glutamic-Oxaloacetate Transaminase.

SGPT : Serum Glutamic-Pyruvic Transaminase.

UTI :Urinary Tract Infection.

URI :Upper Respiratory Tract Infection.

WBC : White Blood Cells