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State of the Art Management of Burns

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INTRODUCTION

It is a great opportunity and pride for me to deliver this prestigious oration today before such an enlightened audience. Col. Lal was the Director of Medical Services in the Madras Presidency and he chose to be Surgeon in-charge of a unit at the Medical College. I had the unique privilege of being his student in Surgery, and my husband late Dr M.S. Ramakrishnan was his Surgical Registrar. His daughter Puspha was my father's student at Presidency College, Chennai. One becomes almost nostalgic, while thinking of Col. Lal and those years.

I chose the topic of burn management today, because as a Plastic Surgeon I have devoted well over 25 years in teaching and treating Burns at the Kilpauk Medical College, Chennai, where a well equipped Intensive Care Burn Unit attached to the Reconstructive Unit with 50 beds is present. This unit was established before 25 years. Though the speciality of burns is not by any means glamorous like our other counterpart, the cosmetic surgery, I chose to remain as the Professor and Head of the Department in this unit for twenty years till I superannuated.

Over the last two decades the mortality due to burns have steadily decreased all over the world, but we cannot boast of such an event in India, mainly because of lack of accurate statistical data, epidemiological surveillance and lack of implementation of good preventive programmes. The actual decline in burn mortality is essentially due to a better understanding of pathophysiology of inflammatory response in burn trauma and the availability of superior antibiotics in infection control enabling early surgery for burns. The advent of skin replacements, synthetic skin and cultured human skin has revolutionised the management of burn wounds.

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EPIDEMIOLOGY

Burns being a preventable condition, an effective preventive programme can be implemented, only after we identify the epidemiological and aetiological factors and the statistics on occurrence, mortality and survival of burns. The following charts would establish the gravity of the problem. As burn injury is not a notifiable disease, and due to the lack of National Burn Registry, our country can never talk of the actual incidence of the condition. However our statistics reveal that majority of burn injury occur in women, due to flame and there is a large proportion of paediatric burns in India. There is also a significantly high incidence of suicides (11%), mainly in women in the age group of 20 to 40 years.

IMMUNOLOGICAL RESPONSE TO BURN INJURY

With every decade of history new insights based on technological advances change our understanding of the world around us. No where has there been more prolific change than in the field of medical research where new ways of looking at the disease have change its treatment. In the recent past relevant immunological changes were noticed to have taken plac}e over since allografts had been used to cover the burn wound. During the 1970s, our understanding of cellular and humoral immunity increased markedly. We noticed that several T-cell functions failed following burns. Burn injury also caused particular reduction in the number of helper T-cells. In 1980's the immunological alterations produced by burns were more clearly understood and every known assayable parameters were identified. It is in this era that recombinant technologies gave to the researcher unprecedented tools such as interleukins [IL], ways of assessing their function and their receptors. Consequently the examination of T-cell failure in Burn injury could be made at the molecular level. The characterization of IL-1, and IL-2 and their receptors gave us the understanding of the cell to cell interaction and broader time-course of immune events. These achievements have all been focussed on the molecular details of the immune response in burns. However as each cytokine was discovered it became evident that in burns most cytokine activities are chaotic and abnormal. Other products of immune cells like prostaglandins, oxygen free radicals and consequently lipid peroxides were also found to be involved. As this picture emerged over 1980, and more details were provided in the literature, more compound and diffuse the picture became. The newest description then, of burn immune failure has changed from simply categorising what parameters fail, to describing the condition with a unifying term 'systemic inflammatory response' (SIR), which is responsible for the eventual outcome. The concept became established because today 50% patients who die due to severe burns, do not die due to infection, as was thought previously. Today the chaotic cytokine array is deemed responsible for what ultimately kills the patient. But one has to bear in mind that assessment of cytokines is of little use as predictors of critical events because they themselves are effects rather than causes. What is needed today is an interpretation which encom-

passes most, if not all, phenomena in one hypothesis.

The first action that takes place after burn injury is macrophage activation. Then three cytokines IL-1, IL-6 and TNF-α are released. Macrophages are also responsible for producing oxidation products of lipids like prostaglandins, leukotrienes, and indirectly peroxides which are all toxic to the patient. Hence it is imperative to find out how the thermal injury activates the macrophages or neutrophils, and this would be extremely important due to a simpler treatment intending to prevent activation, rather than to arrest the consequent activity of several run away phagocyte products.

In burns, the area of burnt skin quantitatively is correlated with the mortality. Hence skin today is recognised as an immune organ and the effect of heat on the skin could well figure in the origin of the pathophysiology encountered. Area of skin burned also correlated with the degree of T-cell functional failure.

In experimental animals, a toxic material has been isolated from the burnt skin, which when infected into normal animals exhibits all the symptoms of toxic shock seen post-burn. The toxic material is a polymerized aggregate and is a lipoprotein complex [LPC]. Heat input had induced toxicity simply by polymerization. This experimental work indicates that a heat toxic polymer, from burnt skin is responsible for the death of the patient, and this mortality was linked to immune depression. The LPC acts in the following ways:

- It suppresses cell mediated immune response.
- It inhibits lymphocytes immuno globulin synthesis.
- It depresses granulocyte colony formation. LPC circulates in the serum after burns and antibodies develop. The type of dysregulation of immune function which develops post-burn seems to begin with the inability of cells to respond positively with regard to the immune response. The toxic LPC reduced the resistance of burnt mice to pseudomonas infection.

INITIAL MANAGEMENT OF SHOCK AND WHAT IS NEW IN FLUID RESUSCITATION

An accurate clinical assessment of the extent and depth of burn injury is the first step in effective resuscitation. With regard to fluid resuscitation in the early phase of burn injury, clinicians are guided by different standard management protocols. Each department over a period of years develops its own preference to formulae. In the initial examination, presence or absence of smoke inhalation and concomitant injuries must be identified, particularly because a patient with smoke inhalation may require 30% more fluid than the one without it.

Resuscitation fluids are mainly crystalloids in the form of Ringer lactate and colloids in the form of fresh frozen plasma [FFP], albumin and whole blood.

In this part of the world, Parkland formula of 4 ml/kg/% burns in the form of Ringers lactate is the most popular. Whole blood and FFP are also used. But strict vigil on viral disease transmission must be kept in mind, for human immunodeficiency virus [HIV] and hepatitis B virus. In Europe

today FFP is sterilised with a detergent so as to be completely free from virus. The goal of fluid resuscitation is to support the patient through the initial 24 to 48 hours of hypovolaemia due to sequestration of fluid into the burnt tissues. Adequate resuscitation is monitored by an urinary output of 0.5 ml to 1 ml/kg/hr. Weight based formulae overload children, hence formula based on body surface area is used in paediatric patients. However for patients with severe burns, the use of urinary output and vital signs may lead to sub-optimal resuscitation. For these patients invasive cardio-respiratory monitoring may be indicated to optimise fluid therapy.

METABOLIC RESPONSE

The degree of metabolic changes experienced by patients are directly related to the extent of injury. During the initial phase called "ebb" phase, there is lowering of cardiac output and metabolic rate. Once resuscitation takes place, cardiac output and metabolic rate may return to normal and may become elevated. This hypermetabolic phase may show a high core temperature of about 38.5 °C and remains high for 5-15 days post-burn. This is due to the direct stimulation of the hypothalamus by inflammatory mediators like cytokines that increase the thermoregulatory set point and alter the endocrine function also. Secretion of cortisol, glycogen and catecholamines are all increased. These being strongly catabolic, produces negative nitrogen balance, loss of tissue protein and bone minerals. Growth hormone [GH] and insulin like growth factor IGF-1 levels also decrease following severe burns.

If untreated, the hypermetabolic phase literally wastes the patient and he succumbs. For therapy today recombinant GH 2 mg/kg per day is given to massively burnt children. The effects of IGF-1 and GH are complementary. While GH increases the protein synthesis the IGF-1 decreases protein degradation. These have been proved by several randomized trials. But the cost of the treatment is prohibitive. Since the cardiac rate is high in burns patients, beta-blocking agents such as propranolol can reduce the rate and also the ventricular work index. Since the catecholamine levels are very high in severe burns, one can safely give propranolol.

INFECTION CONTROL

After the shock phase, prevention of infection is most important in burn patients. In developing countries the maximum mortality in burns is due to septicaemia. In infection control, prevention is accomplished by strict vigil on nursing, air sterilization, and providing isolated rooms for every patient. Prophylactic antibiotics need not be given, and a definite strategy on the administration of antibiotics is followed. Routine surface and biopsy would cultures are done and then appropriate antibiotics in the necessary dosage is given. In invasive sepsis, aggressive antibiotics and antifungal treatment are given.

Local wound is dressed every day, and each surgeon has his or her own way of taking care of the wound. In our set up:

- Superficial and superficial partial thickness wounds are treated as outpatient

cases with amniotic membrane or collagen as biological dressings. In deep partial thickness burns also, biological dressings are used to prevent infection and progress of depth of burn. However if it extends, it is treated as full thickness burn.

- Topical antimicrobials are liberally used in certain cases of circumferential burn and silver sulphadiazine invented by Fox has stood the test of time. To cover the wound with autoskin is always the primary aim of wound care.

NUTRITION

Patient with severe burns (>40%) have metabolic rates that are 100% to 150% above the basal metabolic rate. These patients need energy and proteins in order to prevent impaired wound healing, cellular dysfunction and decreased resistance to infection. Diet is provided orally, enterally or parenterally. Several formulae are available. But each physician plans a diet that would suit the patient individually. One can aim at a calorie intake anywhere between 2400 kcal to 3600 kcal and may even go up to 5000 kcal in rare occasion. Regardless of the mode administration 20% of calories must be from proteins, 30% must be fat and 50% must be carbohydrate. The role of dietary additions such as glutamine, arginine, vitamin C, vitamin E, fish oil are all under investigation. Total parenteral nutrition [TPN] in burn patients has been associated with metabolic and immunological complications and its use is limited today to supporting patients with severe gastrointestinal dysfunction.

WOUND HEÀLING

As an aggressive approach to burns wound excision is the standard of care today. All burns over 30% must be aggressively treated. However all surgical excisions must be tailored to the individual circumstance. In developing countries due to constraint on the available skin replacements and blood the management varies. The treatment of superficial and superficial partial thickness burns have already been mentioned.

- Deep burns if they are circumferential immediate escharotomy and decompression must be done to save the vascularity of the limb.
- Deep burns over 30% are tangentially excised with adequate blood support and the whole are is autografted. In the absence of sufficient autograft, homografting from the parents of the child can be contemplated. One has to be very cautious about the area to be excised, and intense planning has to be done so as to judiciously use the skin replacements. Repeated graftings are done by sequential harvesting of limited donor sites.
- In electric burns, if the area is limited, we can do primary full thickness excision and immediate flap cover. Micro surgical free flaps also can be used.
- Cadaver skin is not available in India (legislation does not permit us to use it) and what is available today to be used are [i] Biobrane which is a synthetic cover and [ii] Dermagraft JC which is essentially biobrane populated with neonatal firboblasts which secrete

structural support proteins and cytokines prior to being inactivated. Next is the cultured autologous epidermal keratinocytes. This is very expensive, and due to lack of structural integrity on a long term basis, it is becoming less popular even in the western world.

- Recently we have skin substitutes which are very useful. One is Integra which is a bilaminate of collagen with chondroitin sulphate with a silastic cover, and Alloderm which is a deepith-elialized pathogen free cadaver skin.

Ultimately the area has to be closed with patient's own skin. When none of the above is available, the patients survival gets priority and the patient is allowed to get into contractures which are later released and grafted. This extreme measure is only very rarely adopted.

RECONSTRUCTION AND REHABILITATION

Several plastic surgical procedures including flap covers, and microsurgical free flaps are used in burn reconstruction. Every reconstruction has to be followed with physical therapy and rehabilitation. Pressure bandages, garments and splints are most useful.

PSYCOTHERAPY AND OCCUPATIONAL THERAPY

Burnt victims go through a phase of depression, and disfigurement can be very traumatic. Treatment by a psychiatrist is often needed. During the phases when a person waits for staged reconstructive procedures placing them in suitable occupations will help them to rebuild the confidence.

To conclude burn management needs a multidisciplinary research and multi-professional approach. Tertiary care treatment alone will not solve this devastating problem.

RESEARCH

In burn management, research is an ongoing process. Study of the process of inflammation/immune response, its failure and the production of newer synthetic skin replacements are being conducted in many centres of the world. Every burn surgeon must be interested in research.

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