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Burns are notorious in the sense that they break the continuity of skin and produce raw area, which is prone for invasion by micro-organisms and abnormal loss of body constituents. The water retention ability of skin depends on its effective vapour pressure and diffusion barrier offered by keratin layers and lipid contents in the stratum corneum. This lipid is thermolabile, when this barrier is removed after thermal injuries, the effective vapour pressure gradient is increased by 15-20 times. This results in a large amount of evaporative water loss amounting to an increase of 3-10 times the normal rate in sensible water loss (40ml/hr), the amount and duration for which the loss persists depends on the depth and percentage of burn.

In the treatment of burn injury the, the focal points are control of shock and infection and skin grafting (Peter Zellner). The improvements in infusion therapy in burn patients have lead to clear reduction in mortality (Zellner & Metzger 1976). There is consensus that infection is the primary source of morbidity and mortality from extensive burn injury in the present era. Bacterial infection has always been considered one of the most serious complication of burn injury.
Burn sites are potential areas for entry of heterogeneous microflora with which human co-exist. The problem is further compounded by the severe depression of host immunological response to a degree directly proportional to the severity of burn.

The avascular nature of burn tissue as a result of thrombosis of vessels, limits the delivery of endogenous phagocytic cells and systemically administered antibiotics may not always achieve an optimal burn wounds' concentration and, thus, may not be able to limit the injection of that site. Early closure of burn wound. Logically would limit the site of entry of infection but this is not always achievable. In addition to the infection, wound maceration and pressure necrosis also favour microbial proliferation and impairs circulation. At deep burn sites due to prolonged ischemia in the sub-escharal plane, systemically administered antibiotics reach only by diffusion gradient from the wound periphery (Koch DM 1985, William WM, Bruce F 1987, Yurf RW, McManus AT et al 1984). The large raw area produced by the burn wound with it's exudate or serum, works like a huge culture plate on which organisms can multiply uninhibitedly. The superficial burn can convert to a deep burn in the presence of infection. Thus the burn wound despite
the use of topical antibacterial agents remains a constant, potential source of systemic sepsis until eschar separation is complete, (Charles R. Baxter et al 1973). Once generalised burn wound sepsis has developed, the chance of survival is 10% or less (Mc Marus WF et al 1981).

Although it is still thought that burn wound can be virtually sterile at the time of injury, this concept is purely academic. Adequate sampling of burn surface will reveal bacteria in every instance, although during the first few hours the concentration may be very low (Robert B Lindberg).

In large burn areas, dense colonisation of pathogens can occur within 24 hours (William WM, Bruce F 1987 and Krupps et al 1985). In untreated patients, immediately after injury few bacteria can be recovered and these are predominately gram positive. The type and density of organisms present in the untreated burn wound change with time. By the fifth post burn day, pseudomonas can be recovered (William WM, Bruce F 1987). By the middle of the second post burn week the burn wound organisms are predominately gram negative, rods and fungi especially candida (Artz Cp et al 1979, Order & Moncrief 1965, William W Monafo, Bruice F 1987). The organism penetrates the eschar by migration and extends down to the viable-nonviable tissue
interface. At this site further microbial proliferation commonly occurs and promotes lysis of the denatured collagen and spontaneous sloughing of the eschar. (Order & Moncrief 1968). In patients with inadequate host defence capacity or those in whom the topical therapy is ineffective, the subescharal organisms invade the underlying unburnt tissue and may spread systemically (Moncrief AJ, Teplitz C 1964, William WM, Bruce F 1987).

Evaluation and treatment of the microflora in the burn wound to prevent septic complication are a challenging clinical problem. Bacterial colonisation of the burn wound may reach a concentration of $10^6$ to $10^7$ per gram of burn tissue before changes are evident either in the appearance of the burn wound or in the detection of clinical signs or symptoms of systemic sepsis (Charles R Baxter et al 1973). Surface culture techniques fail to predict accurately the presence or progression of burn wound sepsis due to poor correlation between the surface flora and colonisation of the eschar and subcutaneous tissue (Bretano L & Gravens AC 1967, Clarkson JG, Ward CG & Polk HC 1967, Colebrook L, Lowbary EJL and Hrust L 1960, Georgiade NG et al 1966). Multiple eschar biopsy obtained serially from representative area of burn wound and culture
quantitatively and qualitatively furnish valuable information about bacterial growth. The literature recommends site a bacterial count or $10^5$/gm of tissue as the upper limit for minimal penetration to deeper tissue level (Moncrief AJ, Teplitz C 1964, Krupps, Barchler M, Bille J 1985). If the concentration of the bacteria is $>10^5$ bacteria/gram of tissue, burn wound sepsis is generally present. Under such conditions skin grafts are often autolysed by infection. Therefore, prior to skin grafting, aggressive wound treatment must be instituted and continued until the bacterial concentration of wound biopsies falls $<10^5$ bacteria/gram tissue (Artz, Moncrief & Pruitt 1979, Parks, Linares & Thomson 1981, Robson, Krizek & Heggers 1975, Steen 1983, Teplitz 1969, Teplitz 1974).

Considering all the above facts we used PVP+ Neosporin powder topically in all patients. This combination forms an almost complete barrier against microbials (Sinha et al 1987). Neosporin powder contains polymyxin, neomycin and bacitracin. The combination of povidone iodine and neosporin was basically selected for both its physical and bacteriological properties. Povidone iodine has wide antibacterial, anti-fungal, sporicidal and viricidal properties (Zellner and Bugyi 1985, Robson CM,
Schaerf RHM, Krizep TJ 1974, Law EJ, McMillan BC 1972, William WM, Bruce F 1987, Georgiade NG, Harris WA 1973, Peter Zellner 1985). Neomycin and bacitracin supplement the gram positive antibacterial properties and polymyxin protects against P.aeruginosa (Georgiade NS, Harris WA 1973) but not against staphylococcus aureus and haemolytic streptococci (Jackson DM, Lowbury EJZ, Topley E 1951). The tanning effect of PVP has an added advantage on dead layers of skin, creating a demarcation between viable and nonviable areas. The tanned second or third degree burns were never transformed into sticky necroses. In addition, the tanned skin is less likely to produce infected material that can be transported into lymphatic and blood vessels during surgical scraping (Peter Zellner 1976), so by this effect PVP keeps the surface dry holding colonization to low levels and also permits early surgery (Zellner PR, Bugyl S 1985).

Daily spray of PVP lotion and neosporin powder on the wound forms a "crust" which sets up a barrier to colonization of bacteria, helps in healing with limitation of infection. Because the 'crusting' effect ensures that the locally applied drug remains at the burn site for a prolonged period and at the same time because subsequent application are applied
over the previous ones, patient does not feel any pain after the first application. Even the first application entails only minimal pain. In our opinion, if a burn patient can be given an option of pain free local burn wound management, it would be of immense psychological relief to the patient. In addition, nursing personnel time is reduced by about 70 to 80% on average and therefore treatment cost is less.

By the above discussion, the presence of infection should be a consideration in deciding on the optimal time for surgery. If there is no or minimal infection, we can postpone the surgery and can grant time for removal of eschar in their normal course instead of doing surgical escherectomy and can prevent haemorrhage. Deep burns start to lose their eschar in 2 to 4 weeks. In the presence of infection, eschar separation is early because of collagenase production from bacteria, but because of poor presence of granulation tissue and $>10^5$ bacteria/gram of tissue skin graft can not be applied to covered the wound.

Charles R Baxter et al in 1973 first used antibacterial therapy in the escharal plane by subescharoclysis. This method permits the delivery of high concentration of specific antibiotics into
the avascular burn wound interface by multiple subescharal injection. Later William F McManus et al (1982) also used antibiotics in the subescharal plane. But one thing was common in all studies that antibiotics solution was used in the presence of infection i.e. bacterial count >10^5 gm i.e. when the patient was critically ill none of them used them prophylactically or in the earlier course of wound infection. Sinha et al in 1988 in his study used injection of PVP subescharally first time in all patients of deep burn. That time also a controversy was there as to whether such a procedure (routine PVP-I subescharal injection) was helpful or not? Subescharal PVP injections were attempted basically because PVP has been shown to have beneficial antibacterial effects when used simultaneously, intrapleurally or intraperitonealy without any serious iodine toxicity (Zomoral J 1984). The concentration of .25% PVP may seem to be too low for it to be effective but it has been mentioned that with this concentration there is an increase in free iodine and antibacterial activity (Zomora LJ 1984). In our study we found that patients treated with multiple subescharal injections of PVP showed remarkably reduced subescharal bacterial colony count as compared to the control group. That subescharal injection of PVP was beneficial was
evident from the result; only one septicaemic mortality occurred in the test group of patients. The second beneficial effect is that it opens up a subescharal plane by tanning of non-viable tissue & reduced sticky necrosis thus helping in early escharoclysis and decreasing bleeding. Additionally tanned skin produced least infected material so that transport in lymphatic and blood vessels was minimum. The burn wound in most of the patients were grafted immediately after the eschar separation because most of the patients showed a colony count far less than $10^5$ /gm which is the upper limit below which grafting can easily be done without much fear of graft rejection. In the control group a considerable period of time was spent in limiting the infection at the burn site and infection and less granulation tissue was the cause of graft rejection.

However, reservations have been expressed by some authors that the large quantity of PVP-I absorbed during treatment can have an effect on the thyroid gland in non-euthyroid patients and can cause thyrotoxicosis and thyroid dysfunction. Law EJ and McMillan BG 1974 reported thyroid toxicity in 2 patients after using PVP lotion (1-% available iodine). The high serum iodine concentration can also trigger renal insufficiency but Zellner PR and
Bugyi S in 1985 in the study of approx. 1500 patients never saw thyroid & renal dysfunction in patients treated with PVP lotion, who were examined several times over a long period of time. In our study non of the patients showed any clinical evidence of iodine toxicity.

Zellner PR and Bugyi S in their study with PVP-I in burn patients, measured T₃, T₄, TSH and the iodine level in blood and urine over a period of 21 days. The result showed an initial steep increase in the iodine level of blood and a parallel rise in urine. This increase reached their maximum on second or third day. The high level of iodine is an indicator of the good penetration of PVP-I through the burn wound. Thereafter, the serum iodine concentration falls and a little later the urine concentration level falls as well, despite continued administration. An explanation for this phenomenon was given that it could be that either the application of the ointment was less or that the healing process was preventing penetration. Most probably penetration was less because of the tanning effect. The iodine levels returned to normal about one week after withdrawal of treatment. Initial decrease in T₃ and T₄ pointed to transient hypothyroidism due to inhibition of iodine deposition because of high serum iodine
concentration. However, a gradual decrease in T₃ and T₄ was also observed in burn patients by Becker et al 1980 who had never received topical treatment with iodine so one can say that this change can be brought about by different kinds of stress or disease (Burger et al 1976). Furthermore, the thyroid hormones bound to proteins also become lost in large quantities because of the high protein loss in burn patients due to exudate or oedema.

They have also shown the absence any thyroid or renal toxicity with PVP application. Even the component of neosporin, especially polymyxin and neomycin, which all have serious systemic toxicity, did not show any toxicity because they are applied locally and there is limited absorption, preventing systemic toxicity. In addition, the combination of PVP + N also reduce costs and improve patient compliance, which to our mind are very important aspects of the management of burn patients.

Sometimes PVP-I injection causes slight pain to patient when we injected either in wrong plane or too much amount is injected or the eschar is attached too tightly.

Hence it can safely be concluded that subescharal injection of PVP is effective in decreasing bacterial count in subescharal plane,
early scar separation and adding possibility of early grafting and it's better acceptance.

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