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Original Article

Changing Pattern of Burns In a Private Hospital

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ABSTRACT

Burn injuries pose a significant risk of infection due to their nature, immune-compromising effects, and prolonged hospital stays. Despite advancements in burn care, infections remain a major cause of morbidity and mortality. Understanding the evolving patterns of burns is crucial for effective management. This study aimed to analyze recent trends in burn cases, identifying demographic shifts and high-impact areas. Objectives included assessing the incidence and demographics of burns, investigating microbial colonization of wounds, and analyzing antibiotic resistance patterns. A demographic survey of 124 patients over 14 months included history, clinical evaluation, and wound management. Microbial colonization was studied through periodic wound swabs, cultured on various media. The study focused on identifying trends in burn etiology, patient demographics, and infection patterns. The study observed an increasing trend in burn cases, particularly domestic incidents, with a rise in scalds and electrical burns. Most affected were women. Pediatric burns and burns exceeding 20% total body surface area (TBSA) also increased. Urban burns were more prevalent but rural burns had higher mortality rates. Findings reflected changing infection patterns, with gram-negative organisms becoming predominant and multi-drug resistant. Variation in microbial sensitivity to antibiotics was noted, with Pseudomonas being most common. The study emphasized the importance of tailored antibiotic therapy based on antibiograms. In conclusion, the study provides valuable insights into evolving trends in burn injuries, emphasizing the need for proactive measures to address infection and antibiotic resistance. The findings contribute to improving burn care strategies and patient outcomes.

INTRODUCTION

Burn patients are at a high risk for infection as a result of the nature of the burn injury itself, the immune-compromising effects of burn, prolonged hospital stays, and intensive diagnostic and therapeutic procedures. Despite many advances in the care of burn patients, infection remains an important cause of morbidity and death. Furthermore, wound invasion, the immunocompromising effects of burn, prolonged hospital stays, and diagnostic and therapeutic procedures further increase the burden of morbidity of burn patients.

The importance of preventing infection in burn wounds cannot be overstated, as infections can significantly impede the healing process and lead to severe complications. Infections in burn patients can rapidly escalate from local wound infections to systemic infections, including sepsis, which can be life-threatening. The increasing severity of infections is often due to the presence of multi-drug resistant organisms, which are more prevalent in hospital settings. These resistant infections are more difficult to treat and require stronger, sometimes more toxic, antibiotics.

Effective infection control measures are crucial in promoting proper wound healing and improving overall outcomes for burn patients. This includes stringent hygiene practices, timely and appropriate use of antibiotics, and regular monitoring of wounds for signs of infection. Additionally, advanced therapeutic interventions such as the use of antimicrobial dressings and therapies targeting resistant organisms are essential in managing and preventing severe infections in burn patients. [1-3]

The objectives of the study were to find out the recent pattern of burns in a private hospital and to see if there are increasing and decreasing trends in the demography of burns and accordingly focus our attention to high impact areas.

METHODOLOGY:

A demographic survey with history, clinical evaluation, investigations and preliminary management of the burn patient was performed. A total of 124 patients were taken for the study in the duration of 14 months, starting from November 2019 to December 2020. Inclusion criteria were the admitted patients who presented at out centre. All age groups were included. First degree burns were excluded.

The microbial colonization of wounds was studied weekly from the date of admission up to the 4th week of hospitalization. Periodic wound swabs were collected at 1st, 2nd, 3rd, and 4th weeks of hospital stay or till the resurfacing was done.

The bandages after removal, the topical antimicrobial agent remnants were cleaned and the wounds swabs were taken. Then after washing, topical antimicrobial agents were applied. Sterile cotton-tipped swabs were used to collect the pus and or any other purulent discharge. Specimens were then transferred to sterile test-tubes. In case of collection of samples from dry surface, swabs were moistened with sterile normal saline. Gram stain and culture are performed.

All the wound swab specimens were then inoculated on the Blood Agar (BA) plate, MacConkey agar (MA) and Nutrient agar (NA) and incubated at 37 degrees Celsius for about 18-24 hours. [4]

Gram negative rods were identified by performing few biochemical tests, as: the catalase test, the oxidase test, methyl-red (MR) test, Voges-Proskauer (VP) test, indole test, motility test, the oxidative-fermentative (OF) test, hydrogen sulphide (H_2S) production test, citrate utilization test, triple sugar iron (TSI) reactions and the urease test. [4]

Gram-positive cocci were identified based on their preference of growth on Blood Agar and Nutrient agar followed by catalase test, OF test, oxidase test and the coagulase test. [4]

Laboratory values of the patients were done initially at admission, then repeated when essential. The laboratory values of the patients were analyzed. The laboratory values were repeated as and when required, in situations when there was a clinical deterioration, a follow up of the previous values, as a marker of the efficiency of the management protocol that was followed and

or a decision-making tool for change in the lines of management. The clinical parameters, the condition of the wound, the laboratory values were all used in conjunction for decision making process for the patient.

RESULTS

There has been an increase in the number of patients presenting to the casualty in the last 5 years. (Average admissions per month 2011-2015 was 39 compared to 2016-2020 being 68), winters have highest rates. Domestic incidents have increased over time. Most affected patients in burns were women suffering burns due to domestic accidents (76%). We have observed increased incidence of house fires.

Two major patterns were observed: 1. During cooking 2. Due to heaters and heating coils. Most patients were in the young working age group (21-40yrs). Most patients were females with a ratio of 54% (69 patients). There has been an increasing trend in pediatric burns (21% in year 2011-2015 to 26%, 32 patients in year 2016-2020).The percentage of burns as per TBSA presenting has increased over the period of time, from average 28% TBSA in year 2011-2015 to 32% TBSA in year 2016-2020.

Large burns (>20% TBSA) are becoming more common in the recent times requiring more hospitalization, dressings, debridements, ICU care and surgeries. Urban burns are more common than rural burns (54%, 67 patients) but rural burns had higher mortality (12% higher)

Fig 1: Images showing large burns at our centre. Increasing incidence of large burns

Average duration of stay has increased (from 27 days in year 2011-2015 to 38 days in year 2016-2020) and which includes two separate timelines for the patient, (1) Initial: ICU care, fluid resuscitation, dressings, conservative management, debridement. (2) Late: Surgery: including resurfacing and managing post burn sequelae.Overall crude mortality has decreased by 5.3%, although the data was not significant ($p>0.05$).

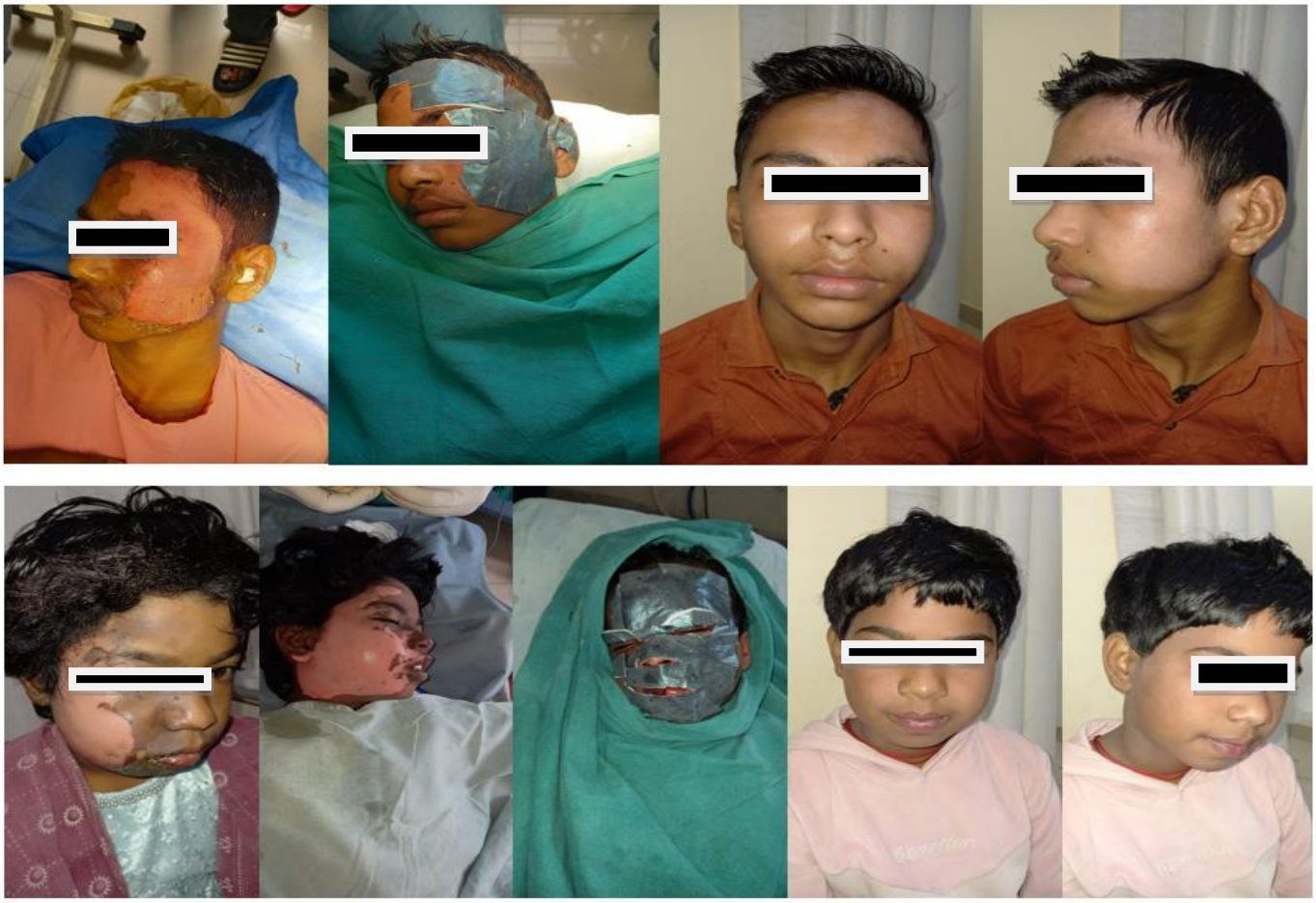

Fig 2: Early Debridement and cover with Collagen based dressing

Type of burn: Etiology (Fig 3)

- Scald: on a rising trend, mostly pediatric population. About 42%
- Thermal (excluding Scalds): On a decreasing trend, 47%
- Electrical: On an increasing trend, 8%
- Chemical: 2%

Fig 3: Etiology of burns at our centre

- **Infection:** As large burns have increased with deeper burns, late referrals, inadequate primary management, patient related causes with improper judgement, injudicious use of antibiotics, the burn wound is colonized and teaming with more resistant strains of microorganisms with much wider spectrum than previously seen, presenting with different sequelae and pathologies. (Fig 4,5)

Fig 4: Infected burns presenting in casualty

Fig 5: Burns and their sequelae with different pathologies

Bacteriology: The bacteriology of the burns is represented in the following image. (Fig 6)

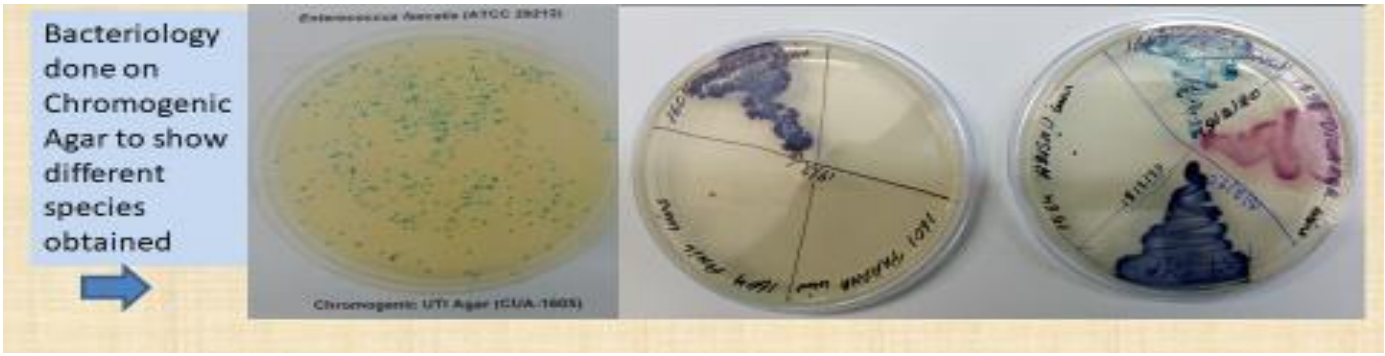

Fig 6: Bacteriology of burns, Chromogenic agar Petri dish, different colonies on the dish

Antibiotics

MAR Index (Multi Antibiotic Resistance Index): MAR index is calculated as the ratio between the number of antibiotics that an isolate is resistant to and the total number of antibiotics the organism is exposed to. Most patients (65%) had **multi antibiotic resistant infections**, mostly **Pseudomonas** with other strains making it **multi-bacterial infection**. (Fig 7)

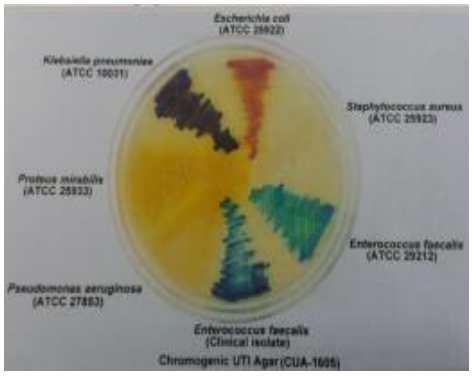

Fig 7: A Petri dish depicting different strains of bacteria found in the study

Due to **late referrals** (>72hrs) the patients harbour significant infection and **improper use** of antibiotics resorting to **resistant growths**. Depending on the **culture** and resorted to **higher class of antibiotics** (3rd or 4th Generation cephalosporins), Anti-ESBL antibiotics, as Meropenem, Imipenem, with cilastinetc with lesser used Aminoglycosides as Netilmycin was used as strategy to control infection and debridements at proper times. (Fig 8)

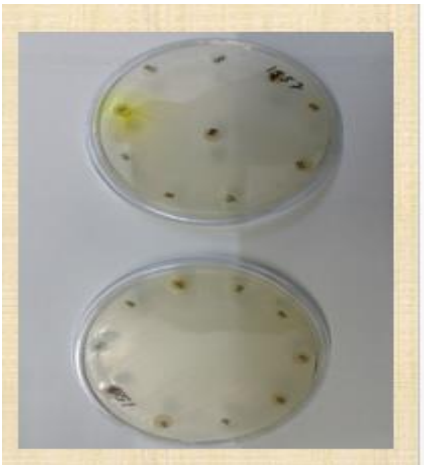

Fig 8 :A Petri dish showing different antibiotic inoculates. The therapy is targeted as per the antibiogram.

Markers of Sepsis in Burns

Sepsis in burn patients is a critical condition that requires prompt diagnosis and treatment. Several biomarkers are used to identify and monitor sepsis in these patients. Following are the key laboratory values that we found to be relevant in monitoring the burn patients. We mapped the sensitivity and specificity values in percentage for the different markers.

- *Procalcitonin (PCT)*
  Procalcitonin levels increase significantly during bacterial infections, making it a useful marker for diagnosing sepsis. Elevated PCT levels can indicate the presence and severity of a bacterial infection in burn patients.

- *C-Reactive Protein (CRP)*
  CRP is an acute-phase protein that rises in response to inflammation. Elevated CRP levels are indicative of infection and inflammation, making it a valuable marker for sepsis.

- *White Blood Cell Count (WBC)*
  An elevated or decreased WBC count can indicate an infection. Leukocytosis or leukopenia can be signs of sepsis in burn patients.

- *Lactate*
  Elevated lactate levels can indicate tissue hypoperfusion and sepsis. It is often used to assess the severity and progression of sepsis in burn patients.

- *Prothrombin Time (PT) and Activated Partial Thromboplastin Time (aPTT)*
  These coagulation markers can be prolonged in sepsis, indicating coagulopathy, which is common in severe sepsis and septic shock.

- *Serum Creatinine*
  Elevated levels of serum creatinine can indicate kidney dysfunction, which is a common complication in severe sepsis.

- *Platelet Count*
  Thrombocytopenia (low platelet count) is often observed in septic patients and can indicate the severity of the condition.

- *Serum Albumin*
  Low serum albumin levels can indicate a poor nutritional state and are often associated with a higher risk of sepsis.

- *Bilirubin*
  Elevated bilirubin levels can indicate liver dysfunction, which can be a complication of sepsis.

The following table (Table 1) denotes some of the key laboratory markers used in assessing sepsis in burns at our centre. The table also denotes their specificity and sensitivity values in percentage in the determination of sepsis in burns.

Table 1: Laboratory markers of sepsis with their sensitivity and specificity for sepsis

Marker	Normal Range	Elevated Level (Sepsis)	Severity Levels	Specificity and Sensitivity in %
Procalcitonin (PCT)	< 0.05 ng/mL	> 0.5 ng/mL	Mild (0.5-2 ng/mL), Severe (>2 ng/mL)	**Sensitivity**: 85% **Specificity**: 87%
C-Reactive Protein (CRP)	< 10 mg/L	> 50 mg/L	Moderate (50-100 mg/L), Severe (>100 mg/L)	**Sensitivity**: 76% **Specificity**: 62%
White Blood Cell Count (WBC)	4,000-11,000 cells/µL	< 4,000 or > 12,000 cells/µL	Mild (12,000-20,000 cells/µL), Severe (>20,000 cells/µL)	**Sensitivity**: 56% **Specificity**: 57%
Lactate	0.5-1 mmol/L	> 2 mmol/L	Moderate (2-4 mmol/L), Severe (>4 mmol/L)	**Sensitivity**: 62% **Specificity**: 52%
Prothrombin Time (PT)	11-13.5 seconds	> 15 seconds	Moderate (15-20 seconds), Severe (>20 seconds)	**Sensitivity**: 64% **Specificity**: 54%
Activated Partial Thromboplastin Time (aPTT)	30-40 seconds	> 45 seconds	Moderate (45-60 seconds), Severe (>60 seconds)	**Sensitivity**: 63% **Specificity**: 52%
Serum Creatinine	0.6-1.2 mg/dL	> 1.5 mg/dL	Moderate (1.5-3 mg/dL), Severe (>3 mg/dL)	**Sensitivity**: 66% **Specificity**: 73%
Platelet Count	150,000-450,000 cells/µL	< 100,000 cells/µL	Moderate (100,000-150,000 cells/µL), Severe (<100,000 cells/µL)	**Sensitivity**: 56% **Specificity**: 76%
Serum Albumin	3.5-5.0 g/dL	< 2.5 g/dL	Moderate (2.5-3.5 g/dL), Severe (<2.5 g/dL)	**Sensitivity**: 53% **Specificity**: 66%
Bilirubin	0.1-1.2 mg/dL	> 2 mg/dL	Moderate (2-3 mg/dL), Severe (>3 mg/dL)	**Sensitivity**: 53% **Specificity**: 66%

DISCUSSION

Many studies such as Fouzia et al, Patil et al and Anwar et al have shown the changing pattern in burns. Gram positive organisms are initially prevalent during hospital stay of patients; then gradually become superseded by gram negative opportunists that appear to have a greater propensity to invade. Over the period of time the MAR index has been increasing. Similar results were found in other studies. [5-8]

Fouzia et al reported Staphylococcus aureus as the most common infective agent, whereas our study reported Pseudomonas species as the most common agent. [6]

Pseudomonas species were found most sensitive to Polymyxin B. Staphylococcus aureus was

found most sensitive to Vancomycin. Although high levels of sensitivity were found with Levofloxacin. Coagulase negative staphylococci showed similar sensitivity as Staphylococcus aureus. Acinetobacter species, Klebsiella species, E. coli was most sensitive to Amikacin. Citrobacter species, Enterobacter species and Proteus species were extremely sensitive to Gentamycin apart from Amikacin. Ciprofloxacin and Levofloxacin were extremely effective against Proteus and Citrobacter species. Netilmycin was used in cases resistant to Amikacin and Gentamycin. Similar pattern of sensitivity was found by Anwar et al and Rajbahak et al. [8,9]

Our study not only had some similar characteristics of the previous mentioned studies but also reflected a regional variation in some of the features. These variations are unique to every set up managing burns at a defined demographic region but at the same time also contain some that are common to all centres. Younger working age group (21-40 yrs) are most commonly burnt. The age bracket has decreased as seen by other studies. Large burns (>20% total body surface area) have become more common which is in contrast to Anwar et al where they reported a statistically significant trend of admitting lower percentage of burns. [8]

The following table (Table 2) depicts the types of infection in burn patients as per Rajbahak et al. Similar were the findings at our centre.[9]

Table 2: Types of infection in burns

Types of infection					
Organisms	**Mixed**		**Organisms**	**Single**	
	No.	**%**		**No.**	**%**
P. aeruginosa +*Acinetobacter* spp.	19	12.9	*P. aeruginosa*	47	32.0
P. aeruginosa + *S. aureus*	10	6.8	*S. aureus*	20	13.6
P. aeruginosa+ CONS	7	4.8	CONS	3	2.0
P. aeruginosa+ *E. coli*	3	2.0	*Acinetobacter* spp.	10	6.8
P. aeruginosa + *Klebsiella* spp.	4	2.7	*Klebsiella* spp.	0	0
P. aeruginosa + *Citrobacter* spp.	2	1.4	*E. coli*	0	0
P. aeruginosa + *Proteus* spp.	4	2.7	*Citrobater* spp.	0	0
P. aeruginosa + *Enterobacter* spp.	1	0.7	*Enterobacter* spp.	0	0
P. aeruginosa +*S. aureus* + *Klebsiella* spp.	1	0.7	*Proteus* spp.	0	0
S. aureus + *Acinetobacter* spp.	4	2.7			
S. aureus + CONS	1	0.7			
S. aureus + *E. coli*	1	0.7			
S. aureus + *Proteus* spp.	1	0.7			
S. aureus + *Klebsiella* spp.	3	2.0			
Acinetobacter spp. + CONS	1	0.7			
Acinetobacter spp. + *Enterobacter* spp.	1	0.7			
Acinetobacter spp. + *Citrobacter* spp.	1	0.7			
Acinetobacter spp. + *E. coli*	2	1.4			
Proteus spp. + *E. coli*	1	0.7			
Total	**67**	**45.6**		**80**	**54.4**

The markers of sepsis in burns have been extensively studied. Studies have mentioned expert consensus by the American Burn Association, BURN-6 and SEPSIS 3 criteria. Most sensitive laboratory value was Procalcitonin followed by C-RP. Most specific marker of sepsis was Procalcitonin followed by platelet counts. Similar markers were used in the above-mentioned studies. [10-12].

The Sequential Organ Failure Assessment (SOFA) score is a tool used to track a patient's status during a stay in the intensive care unit (ICU) to determine the extent of a person's organ function or rate of failure. This scoring system is particularly useful in assessing patients with sepsis, including burn patients who develop sepsis. The SOFA score is calculated based on six different scores, one each for the respiratory, cardiovascular, hepatic, coagulation, renal, and neurological systems. Following is the scoring system used in Intensive care setting. We have used it to monitor the patients with burn sepsis. [13]

Table 3: The 'SOFA' - Sequential Organ Failure Assessment scoring system

Organ System/Parameter	**1 Point**	**2 Points**	**3 Points**	**4 Points**
Respiratory/ PaO2/FiO2 [mmHg]	< 400	< 300	< 200 with respiratory support	< 100 with respiratory support
Cardiovascular/ Hypotension MAP [mmHg]	< 70	nor-/epinephrine ≤ 0.05 µg/kg/min	nor-/epinephrine ≤ 0.1 µg/kg/min	nor-/epinephrine > 0.1 µg/kg/min or multiple vasopressors
Coagulation/ Platelets [x10^3/mm^3]	< 150	< 100	< 50	< 20
Renal/ Creatinine or urine output	1.2–1.9 mg/dL (110–170 µmol/L)	2.0–3.4 mg/dL (171–299 µmol/L)	3.5–4.9 mg/dL (300–440 µmol/L) or < 500 mL/day	≥ 5 mg/dL (> 440 µmol/L) or < 200 mL/day
Metabolism/ Hyperglycaemia	plasma glucose > 200 mg/dL (untreated)	> 25%/24 h increase of insulin/h i.v. drip	> 50%/24 h increase of insulin/h i.v. drip	persistent plasma glucose > 200 mg/dL despite insulin bolus + continuous therapy
CNS/Glasgow Coma Scale (points), not sedated	13–14	10–12	6–9	< 6
CNS/Glasgow Coma Scale (points, sedated) Intestines/ Enteral Feeding Intolerance	distended abdomen	gastric residual volume of 100% of feeding rate	gastric residual volume of 200% of feeding rate or inability of gastric feeding > 24 h	inability of gastric feeding > 48 h

This table provides a structured scoring system for evaluating the severity of organ dysfunction in patients, particularly useful in the context of sepsis or critical care scenarios. Each parameter is scored from 1 to 4 points, with higher scores indicating more severe dysfunction.

It was a helpful tool in assessing the protocol of management and the prognosis of a patient. Our setup has been receiving burns with a higher SOFA score compared to previous admissions over the years indicating a higher prevalence of sepsis, resistant strains of infective agents, highly morbid patients with increasing virulence of microorganisms.

CONCLUSION

The patterns of burn are changing as a dynamic process. Resorting to a single line of management over a sustained long period of time (>5-10Yrs, depending on the demography) may not be adequate.

The need of the hour is to reassess the problem, re-evaluate the present standards, re-formulate principles and re-strategize the management with changing needs of the burned patient.

REFERENCES

1. Santucci, S. G., Gobara, S. and Santos, C. R. 2003. Infections in a burn intensive care unit: experience of seven years. J Hosp Infect. 53: 6-13.
2. Lari, A. R. and Alaghehbandan, R. 2000. Nosocomial infections in an Iranian burn care center. Burns. 26(8): 737-740.
3. Pruitt, B.A. 1984. The diagnosis and treatment of infection in the burn patient. Burns. 11: 79.
4. Benson, F. 2001. Microbiological applications lab manual, 8th edn. McGraw-Hill, p. 50-70.
5. Sandhu R, Dahiya S, Sayal P. Evaluation of multiple antibiotic resistance (MAR) index and Doxycycline susceptibility of Acinetobacter species among inpatients. Indian J Microbiol Res 2016;3(3):299-304.
6. Fouzia, Begum et al. “Changing patterns of burn infections.” *IOSR Journal of Dental and Medical Sciences* 5 (2013): 11-14.
7. Patil, Sangeetha& Reddy, K. & Jeer, Mariraj& Krishna, Shiva. (2015). Aerobic Bacterial Isolates from Burn Wound Infection Patients and their Antimicrobial Susceptibility Pattern. Journal of Medical Science And clinical Research. 10.18535/jmscr/v3i9.10.
8. Anwar, U., Majumder, S., Austin, O., & Phipps, A.R. (2007). Changing Pattern of Adult Burn Referrals to a Regional Burns Centre. *Journal of Burn Care & Research*, *28*, 299-305.
9. Rajbahak, Sushma& Shrestha, C & Singh, A. (2015). Bacteriological changes of burn wounds with time and their antibiogram. Scientific world. 12. 70. 10.3126/sw.v12i12.13601.
10. Mann-Salinas, E.A.; Bann, M.M.; Meininger, J.C.; Murray, C.K.; Aden, J.K.; Wolf, S.E.; Wade, C.E. Novel predictors of sepsis outperform the American Burn Association sepsis criteria in the burn intensive care unit patient. J. Burn. Care Res. **2013**, 34, 31–43.
11. Hill, D.M.; Percy, M.D.; Velamuri, S.R.; Lanfranco, J.; Legro, R.I.; Sinclair, S.E.; Hickerson,W.L. Predictors for identifying burn sepsis and performance vs. existing criteria. J. Burn. Care Res. **2018**, 39, 982–988.
12. Singer, M.; Deutschmann, C.S.; Seymour, C.W.; Shankar-Hari, M.; Annane, D.; Bauer, M.; Bellomo, R.; Bernard, G.R.; Chiche, J.-D.;Coopersmith, C.M.; et al. The third international consensus definitions for sepsis and septic shock (Sepsis-3). JAMA **2016**, 315, 801–810.
13. Vincent, J.L.; Moreno, R.; Takala, J.; Willatts, S.; Mendonca, A.; Bruining, H.; Reinhart, C.K.; Suter, P.M.; Thijs, L.G. The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. Int. Care Med. **1996**, 22, 707–710.

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