INTRODUCTION

Burn injuries account for 100,000 hospital admissions and approximately 12,000 deaths per year in USA. The management of burns has always been a challenge. The fear of disfigurement from burns is more traumatizing than the knowledge of the actual incidence of mortality associated with it. Inhalational burn is a more serious condition. Pulmonary insufficiency is responsible for more than 75% of fire-related deaths. Heat generated during combustion causes significant thermal injury to the upper airway. Particulate matter like soot mechanically clogs and irritates the airways resulting in reflex bronchoconstriction. There is release of noxious asphyxiant substances by thermal decomposition and combustion of furniture, cotton (aldehydes), rubber and plastics producing chlorine gas, ammonia, hydrocarbons, various acids, ketones, carbon monoxide (CO) and hydrogen cyanide. Extensive efforts are required with aggressive intensive care management. Relative prognosis remains grave with significant morbidity even in developed countries despite adequate facilities for inhalational burns.

This study was conducted to determine the clinico-pathological profile and outcome in terms of mortality for inhalational burns in a specialized burns unit.

METHODOLOGY

The study was conducted on 19 patients of inhalational burns, which occurred onboard a sea ship in Karachi due to a massive fire in a large closed cabin in March 2005.
2005. The patients were initially resuscitated at a tertiary care hospital in Karachi and later evacuated by air to specialized Army Burn Centre at Combined Military Hospital, Kharian within 30 hours of the accident.

Body parts affected by burns were noted, with special emphasis to face, hands and genitalia, including perineum. Total Body Surface Area (TBSA) involved in burns was calculated according to the Lund and Browder charts.

Baseline investigations including complete blood count, renal profile in terms of serum urea, creatinine and electrolytes, along with serum albumin and total proteins, were done at the time of admission and repeated after 48 hours as and when required. Portable chest radiographs were also done in all the cases, while bronchoscopic examination was conducted only in selected cases.

Central venous access was maintained in the subclavian vein and Central Venous Pressure (CVP) was monitored. Foley catheterization was also done for urine output measurement. Tetanus prophylaxis, intravenous ranitidine was given along with subcutaneous clexane. Analgesics were advised as and when required. Injectable antibiotics like co-amoxiclav, amikacin and ceftazidime were given according to recommended dosage regimen.

Maintenance intravenous fluid resuscitation was done and monitored by CVP line as initial emergency fluid resuscitation had already been carried out at Karachi. Fresh Frozen Plasma (FFP) and intravenous albumin were given to all the patients.

Aseptic dressings were done on alternate days using silver sulphadiazine and bactigrass. Polyfax ointment was used over the facial burns. Escarotomies were carried out where required. Burn wounds were covered with split thickess skin grafts using dermatome and mesher. Full thickness grafting was done only in selected cases. Any wound infection/pus was subjected to microbiological culture and sensitivity and antibiotics were modified accordingly.

Meticulous chest physiotherapy, salbutamol nebulization and incentive spirometry was carried out. Ventilatory support was used on the advice of consultant anaesthetists according to the clinical condition of the patient. The patients mortality was noted and the probable etiology was also considered.

Data was entered and analysed using SPSS version 10.0. Descriptive statistics like frequency, percentage, mean and standard deviation were computed and the association between quantitative variables in the surviving and fatal cases was determined using t-test, taking a p-value of < 0.05 as significant.

RESULTS

This study was carried out on 19 patients of inhalational burns who were physically fit, with no previous record of any chronic medical illness or surgical intervention. There were 8 (42%) patients with a fatal outcome. The mean age of patients was 29±5.75 with a range of 20-40 years. The mean hospital stay was 79±18 days.

The mean percentage of Total Body Surface Area (TBSA) involved in all the cases was 58.42±16.17% with a range of 32-87%. The mean TBSA in 11 patients who survived was 50±10.87 while the mean TBSA in fatal cases was 70±15.46. The face was involved in all the cases, along with burning of the nasal hairs. Both hands were affected in 94.7% cases, with involvement of genitalia and perineum in 31.5% patients (p < 0.05).

The haemoglobin (Hb) at the time of admission was raised in the patients with a mean Hb of 15.8±1.6 g/dL, ranging from 13-19.1 g/dL due to haemoconcentration. After 48 hours of fluid resuscitation, the Hb became 11.4±1.5 g/dL. However, no significant difference was seen in the mean initial Hb or after 48 hours of admission in surviving and fatal cases.

The mean Total Leucocyte Count (TLC) amongst all patients was 6.2±6.3x10^9/L and varied significantly between surviving and fatal cases. The mean TLC in surviving patients was 9.6±6.1x10^9/L. On the other hand, fatal cases had a mean TLC of 1.5±2.3x10^9/L (p=0.001). There was only one patient with a low TLC of 1.0x10^9/L who survived. Granulocyte colony stimulating factor (Neupogen G-CSF) was given to all the patients with a low TLC.

The mean platelet count of surviving patients was 205±63x10^12/L, while fatal cases had a mean platelet count of 58±48x10^12/L (p < 0.05). The surviving 11 patients had a normal platelet count, except for 2 cases.

The serum urea levels in surviving patients was 4.3±2 mmol/L, while in fatal cases it was 8.6±0.9 mmol/L. The serum creatinine levels were 98.2±16.5 µmol/L in the survivor group while the mortality group had a serum creatinine of 249.5±76 µmol/L (p < 0.05). The total serum protein in surviving patients was 63±8 g/L, while in mortality cases, it was 57±7 g/L. Similarly, serum albumin in the survivor group was 36.7±5 g/L and 35±4 g/L in fatal cases. No significant difference in the means of surviving and fatal cases was seen for serum protein and albumin levels.

Table I shows the summary of the clinico-pathological features and difference in the means of surviving and mortality cases.

Chest radiographs showed variable features. They were unremarkable in 6 patients who survived, while pneumonic shadows were present in 3 cases and prominence of bilateral hilar shadows was seen in the remaining 2 patients. In the fatal cases, bilateral hilar prominences were seen in 3 patients while bilateral soft opacities were seen in 4 patients and one case had right sided pneumothorax for which thoracostomy was done.
Inhalational burns were performed in 2 surviving patients and one patient in the mortality group. Bronchoscopic findings in the 3 patients showed inflamed bronchioles with marked inflammation of the lungs. Bronchial lavage fluid yielded polymorph nuclear cells on microscopy and growth of *Staphylococcus aureus* on culture.

Escharotomies were performed in 5 (26.3%) patients. It was done in both hands of 3 patients, and one patient each required it in the right and left hand respectively. Skin grafting was performed in all 11 patients who survived. The earliest grafting was done on the seventh post-admission day. There were 12 patients (63.1%) who had wound infection with positive culture. Culture and sensitivity of pus showed growth of *Pseudomonas aerogenosa* in 11 patients, while growth of *Staphylococcus aureus* was seen in 4 patients. The pattern of sensitivity was similar in all the cases. *Pseudomonas aerogenosa* was found sensitive to ciprofloxacin, ofloxacin, meronem, imipenam and tazocin. *Staphylococcus aureus* was not resistant and showed similar sensitivity.

The patients were placed on ventilatory support according to their clinical condition. The earliest time required by a patient for requiring this support was 47 hours after admission. However, one patient was placed on ventilator as late as 230 hours (9 days) after admission. All the patients placed on a ventilator ultimately had a fatal outcome. They remained on this support for 3-48 hours and only one remained alive on the ventilator for 158 hours.

Among the fatal cases, all of them had acute respiratory distress syndrome. Acute renal failure with multi-organ failure occurred in 6 patients. One patient died due to pulmonary embolism with sepsis and disseminated intravascular coagulation.

**DISCUSSION**

Inhalation injuries occur in approximately one-third of all major burns and account for a significant number of deaths in the burn patients each year. They are difficult to manage because of infrequent exposures and unknown toxic agents. Pulmonary edema which is a major determinant in the outcome of smoke-inhalation injury is related to the products of incomplete combustion.

Accidental burns due to liquids are the commonest (48%) cause of burns followed by flame injury burns in 37%. Inhalational burns being uncommon constitute only 1.4%. Only 15% of these burn injuries are occupational.

A large body surface area and older age are poor prognostic markers. The chances of mortality increase significantly after 70 years of age. In the present study, all the patients were young with a mean age of 29 years and without any underlying medical or surgical confounding factors which enabled to study the management sequel more critically.

There is a strong association between TBSA and mortality augmented by the severity of burns. In the present study, there was significant difference in the mean TBSA of surviving and fatal cases. Similar association has been found in a large series of 5000 patients of different types of burns where 5.4% mortality was seen with 20-39% burns of TBSA and 96% mortality in patients with > 90% TBSA of burns. Similarly 7.4% mortality was seen in patients with 62% TBSA burns.

Inhalational burns is an important co-morbid factor where mortality is higher in comparison to other burns. A mortality of 1.7% was seen in other burns patients in comparison to 34% cases with inhalational burns. Mortality in this study was 42%. However, mortality of 31% (88 out of 284) was documented by Smith *et al.* in their cases of inhalational burns in comparison to 4.3% (50 of 1163) in other burn patients. Tekin *et al.* documented their experience of a mass casualty incident resulting from a boiler room steam explosion aboard a cruise ship which caused 100% mortality as patients had extensive burns (> 80%) with steam inhalation. Acton *et al.* in their 15 years experience in inhalational burns observed reduced survival upto 40%. Inhalational burns prolong the duration of hospital stay in a highly unpredictable fashion as compared to patients with cutaneous burns. Average hospital stay is variable between 26 days to 13 days in patients of cutaneous burns only. In comparison, the average hospital stay of the studied patients was 79±18 days. Average hospital stay of 74 days in patients of inhalational burns was seen as compared to 23 days in patients of cutaneous burns independent of age and TBSA in the study by Tregdet *et al.*

There are limitations for accurate quantification of the magnitude of respiratory tract injury accompanying thermal trauma. Stridor and acute progressive respiratory distress are the two main symptoms which

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**Table I:** Clinico-pathological features and difference in mean of surviving (n=11) and fatal (n=8) cases of inhalational burns using student t-test.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Range</th>
<th>Mean</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total body surface area (percentage)</td>
<td>32-68</td>
<td>45-87</td>
<td>0.009</td>
</tr>
<tr>
<td>Haemoglobin (g/dL)</td>
<td>13-19.1</td>
<td>13.5-17.5</td>
<td>0.67</td>
</tr>
<tr>
<td>Haemoglobin after 48 hours (g/dL)</td>
<td>10-14.9</td>
<td>9-13</td>
<td>0.79</td>
</tr>
<tr>
<td>Total leucocyte count (x10^9/L)</td>
<td>1-20.4</td>
<td>0-7.1</td>
<td>0.001</td>
</tr>
<tr>
<td>Platelets (x10^11/L)</td>
<td>105-319</td>
<td>23-199</td>
<td>0.001</td>
</tr>
<tr>
<td>Urea (mmol/L)</td>
<td>2.9-10.5</td>
<td>6-9.7</td>
<td>0.001</td>
</tr>
<tr>
<td>Creatinine (µmol/L)</td>
<td>72-129</td>
<td>107-354</td>
<td>0.001</td>
</tr>
<tr>
<td>Protein (g/L)</td>
<td>52-74</td>
<td>48-72</td>
<td>0.13</td>
</tr>
<tr>
<td>Albumin (g/L)</td>
<td>30-43</td>
<td>30-42</td>
<td>0.42</td>
</tr>
</tbody>
</table>

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may be delayed up to 72 hours. \(^13\) Patients with soot in the oral cavity, facial burns, and/or body burns should be monitored closely because these findings indicate a higher likelihood of laryngeal edema and a need for intubation. \(^14\) Fibreoptic bronchoscopy is recommended as a bedside procedure to assess the inhalational injury respiratory burns so that an aggressive regime of respiratory support in the form of early intubation and PEEP ventilation could be suggested. \(^15\) It was practiced in a few patients who showed inflamed bronchioles with congestion and inflammation. Arterial blood gases are a useful tool in assessing the pulmonary status of the patient. Early diagnosis, best achieved by endoscopic bronchoscopy and \(^133\)Xenon ventilation perfusion scan, permits timely application of high-frequency ventilation that appears to reduce the incidence of pneumonia and to decrease mortality. \(^16\)

Almost all haematological parameters are affected by severe burns in a biphasic manner. Initially, there is erythrocytosis and increase in hematocrit due to loss of plasma volume and hemoconcentration following thermal injury. However, in the long-term post-burns period, there is diminished red cell production. \(^17\) In this study, the mean haemoglobin at admission was 15.6 gm/dL due to hemoconcentration, which became 11 gm/dL after 48 hours due to volume replacement; erythrocyte loss and marrow suppression due to burns. No significant association was seen in the mean haemoglobin concentration in surviving and fatal cases. Acute plasma volume loss, demargination of mature neutrophils along blood vessels and rapid release of marrow reserves results in transient leucocytosis. However, this is followed by leucopenia due to trauma induced bone marrow suppression and superimposed gram negative sepsis. Leucopenia is also augmented by topical silver sulfadiazine, which is proportional to the size of burn wounds and also as a result of bone marrow and cellular toxicity. This leucopenia is self-limiting. \(^17\) In this study, there was a significant difference in the mean TLC of the surviving and fatal cases. Leucopenia was seen in all the fatal cases, with a mean TLC of 1.5x10⁹/L. In the fatal cases, there was up to 70% TBSA of burns and superimposed wound infections which were the likely causative factors in those patients.

Platelets are important in maintaining immune homeostasis after a major injury. Burn patients or those with severe trauma but having thrombocytopenia have a significantly high mortality. \(^18\) There was a significant difference in the mean platelet count of surviving and fatal cases in the study as well. Thrombocytopenia with a mean platelet count of 58x10⁹/L was seen in fatal cases with a p-value of <0.05 in the presence of a normal hematocrit in both the groups. Animal models in platelet-depleted mice with normal hematocrit, no abnormal necropsy findings and normal histology show that mortality did not occur as a consequence of hemorrhage or hypovolemic shock. It is the reduction in circulating platelet numbers which significantly influence survival following thermal injury and these findings are consistent with the clinical observations in burn and severe trauma patients as well. \(^19\) Platelet transfusions and/or immunotherapy with cytokines such as TGF β1 are suggested to be beneficial for the survival of such patients that develop thrombocytopenia.

A burn wound has a much higher incidence of sepsis as compared to other forms of trauma because of extensive skin barrier disruption and an alteration in the cellular and humoral immune responses. The distribution of micro-organisms changes over time in the individual patient and such changes can be ameliorated with appropriate management of the burn wound and the patient. \(^20\) The typical burn wound is initially colonized predominantly with gram-positive organisms, which are fairly quickly replaced by antibiotic susceptible gram-negative, usually within a week of the burn injury. However, if the wound closure is delayed and the patient becomes infected, requiring treatment with broad-spectrum antibiotics, this flora may be replaced by yeasts, fungi and antibiotic-resistant bacteria. Gram-positive organisms of particular concern include \(S. aureus\), enterococci, group A beta-hemolytic \(Streptococcus\) and coagulase negative \(Staphylococcus\). Gram-negative organisms especially \(Pseudomonas\) have long been known to cause serious infection in burns. \(^21\) Similar culture pattern was seen in this study without much resistance to common antibiotics.

The impact of inhalation injury on pneumonia is clinically important. Onset of pneumonia can either be early, generally within 7 days of the burn injury, or later in the burn course when it usually accompanies generalized systemic sepsis. \(^22\) Chest radiographs showed pneumonic shadows with prominence of hila in our patients and ventilatory support was required after 47 hours in our patients. Unfortunately, all of those patients had a fatal outcome due to acute respiratory distress syndrome associated with renal failure.

**CONCLUSION**

The inhalational burns injury in particular, presents as a challenge to manage which requires high vigilance and timely intervention to cater for the respiratory complications beforehand. Total body surface area involved in burns, total leucocyte count, mean platelet count, serum creatinine and blood urea levels are important indicators of prognosis.

**REFERENCES**


