Impact of thermal injury on hematological and biochemical parameters in burnt patients.

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ABSTRACT

Thermal injury is associated with anatomical, physiological, biochemical & immunological alterations which require specialized care. Burn shock is the first consequence of deep and extensive burns that constitutes the main cause of mortality if local and systemic treatments are not correct & timely. A prospective study was undertaken to determine hematological & biochemical changes occurring in severely burnt patients of different age groups. The study included 35 patients of 20-40 & 41-60 yrs age group all females and all with flame burns. Two day post burn samples were collected for hematological and biochemical parameters study. Leucocytosis was developed immediately after burn. Decrease in hemoglobin, & increase in PCV & ESR also the varying difference in both age groups were observed. In the biochemical parameters, increase in blood urea, serum creatinine, & serum potassium while slight decline in serum sodium i.e hyponatremia was observed.

Keywords: Burn, hematology, creatinine, urea, sodium, potassium.

INTRODUCTION

Burns are usually associated with the application of heat energy such as hot liquids, metals, gases, flames, radiations, electricity or chemicals which cause damage to different tissues. The degree of tissue damage is dependent on duration of contact, the degree of heat energy at the site of contact and sometimes the nature of the agent causing the burn. The most important factors influencing the incidence of burn injuries are age, sex, home environment and economic status. The resulting sequelae depend on depth of damage, total body surface involved, additional factors including smoke inhalation and potential for secondary systemic complication such as septicemia (Schwartz et al., 2004).

Burn shock is the first consequence of deep & extensive burns that constitutes the main cause of mortality if local & systemic treatments are not correct & timely. Burn shock, a type of hypovolaemic shock in the first stages is dominated by disturbances in membrane permeability accompanied by oedema, exudation & evaporation. Secondary manifestations of these process include plasma loss, haemoconcentration, increased blood viscosity & all haemodynamic consequences that these imply. Without intensive therapy, circulatory shock will follow (Dauti et al 1996). The patient with a major burn suffers one of the most severe forms of trauma. The pathological changes produced in the circulatory & respiratory systems are complex & failure to understand their progress and therapeutic management can cause the patient further problems. The hypermetabolic response after major burn is characterized by a hyperdynamic response with increased body temperature, oxygen consumption, CO2 production, glycogenesis, proteinolysis, lipolysis, and futile substrate cycling (Barret & Herndon 2003). This causes erosion of lean body mass, muscle weakness, immunodepression and poor wound healing. In no other disease or trauma is the hypermetabolic response as severe as it is after a thermal injury (Jeschke et al.2008).

Thermal injuries results in significant pathophysiological changes by interplay of various mediators in early stages (Bhagwat & Subrahmanyam 2007). The management of such burn patients remains a challenge for all those involved in their care. The improvement in survival have been attributed among other things to a better understanding of the pathophysiological nature of thermal injuries (Romas 2000).

The burn wounds have much higher incidence of sepsis as compared to other forms of trauma, the reasons are quite obvious; that is disruption of skin barrier and alteration in cellular and humoral responses (Lui et. al 2005). Burn injury results in significant
pathophysiological changes that have been attributed to the presence of detrimental plasma factor, also the serum of burn patients contain a substance that inhibits erythropoiesis. (Sonbaty et al., 1996). Cutaneous injury results in significant fluid loss as well as the release of multiple inflammatory mediators. The major cause of death in burn patient is multiple organ failure and infection and burn injury if not treated properly can also lead to permanent disability and deformity, hence it becomes important for clinician to understand the pathophysiology of burn injury and effects of various drugs on it. (Baris et al. 2004).

In a view of the fact that numerous complications can occur in burn patients, a prospective study was undertaken to determine hematological and biochemical changes in burnt patients. Hematologic data provides information which is useful in both detection of infectious disease and in monitoring of patients diagnosed with bacterial, viral, or fungal infections. Infection, typically accompanied by inflammation, results in changes within the hematopoietic system that can be observed as qualitative or quantitative changes in 3 cell lines: leucocytes, erythrocytes and thrombocytes. During burn injury leukocyte count may increase or decrease depending on the type of infection, point in the time of infectious process, age and chronic health of patient. Peripheral blood phagocytic cells like granulocytes & monocytes may also be influenced with serious consequences for infection resistance, which is known to deteriorate in burn injury. Variation in biochemical parameters like urea and creatinine, sodium and potassium was also seen as hydroelectrolytic and metabolic disturbances are seen in burn patients.

MATERIALS AND METHODS: The present study included 35 burn victims all females, all with flame burns ranging from 30% to 65% TBSA (total body surface area). Two groups of varying age were taken. First group having patients within 20-40 years age while Second comprises of 41-60 years age. In addition to the study groups, there was control group consisting of ten healthy volunteers of the same age.

The samples of study groups were taken from Burn unit of Shriram Hospital, Akola (M.S) and were proceeded in Biochemistry Department of Shri Shivaji College, Akola. (M.S) Determination of hematological parameters like Hb%, Total Leucocytes Count, Differential Leucocytes Count, Packed Cell Volume and Erythrocyte Sedimentation Rate were checked in two day post burn patients. Similarly biochemical parameters like blood urea, serum creatinine, serum sodium, and serum potassium were checked. The samples were processed on Robonik make Prietest Touch Auto Biochemistry autoanalyser. The standard tests used for investigation are:

Total leucocyte count & differential leucocyte counts were done by method of Romanowsky (Ochie & Kolatkar 2007) Hemoglobin percentage was determined by Sahlis Hemoglobinometer, (Ochie & Kolatkar 2007) while PCV & ESR were determined by using Wintrob method. (Ochie & Kolatkar 2007). Serum creatinine was estimated by Fix-Time Kinetic alkaline picrate method (Bartels et. al 1972) while blood urea level was determined by Berthelot method. (Patton et. al 1977) Serum electrolytes (Na+, K+) were estimated on AVL 9180 electrolyte analyzer.

RESULTS AND DISCUSSION:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Group I (20-40yrs) % change</th>
<th>Group II (40-60 yrs) % change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb%</td>
<td>12.92</td>
<td>-22.14</td>
<td>11.15</td>
</tr>
<tr>
<td>TLC</td>
<td>8460</td>
<td>10073.34 +18.6</td>
<td>9980 +17.9</td>
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<tr>
<td>DLC-N</td>
<td>64.7</td>
<td>70.66 +9.22</td>
<td>73.30 +13.2</td>
</tr>
<tr>
<td>DLC-L</td>
<td>21.30</td>
<td>25.87 +23.14</td>
<td>22.6 +6.1</td>
</tr>
<tr>
<td>DLC-E</td>
<td>3.2</td>
<td>4.26 +87</td>
<td>4.0 +25</td>
</tr>
<tr>
<td>DLC-M</td>
<td>1.1</td>
<td>2.068 +87.2</td>
<td>2.06 +100</td>
</tr>
<tr>
<td>PCV</td>
<td>35</td>
<td>38.74 +10.6</td>
<td>38.40 +6.9</td>
</tr>
<tr>
<td>ESR</td>
<td>14.2</td>
<td>25.8 +81.6</td>
<td>20.9 +47.1</td>
</tr>
</tbody>
</table>
The findings of the present study demonstrate the impact of thermal injury on certain hematological and biochemical parameters. Thermal injury resulted in the alterations in both the hematological and biochemical parameters. Hemoglobin concentration showed significantly low levels in both groups. 22% decrease was observed in group I as compared to control while the decrease was 13.7% in group II. Leucocytosis was observed in both the post burn groups.

The increase in Total Leucocyte Count was slightly different in both the groups. 18.6% increase was observed in group I while it was 17.9% in group II. A significant increase in Differential Leucocyte Count varying from 7% - 25% was noticed in both the groups. Value of lymphocyte count was much higher in group I (23%) as compared to group II (6%). Similarly, eosinophil count was 87% higher as compared to control group in group I while group II shows only 25% increase. (Table 1). On the contrary, increase in monocyte count was 100% in group II as compared to group I where it is 87%. PCV indicates a slight increase of 10% and 7% in group I and group II respectively. ESR levels were much higher in group I (81%) as compared to group II. (47%).(Table 1)

In biochemical parameters a significant rise in blood urea and serum creatinine levels were observed. Serum creatinine level was 134% higher in group I with respect to control while it was much higher in group II, the magnitude being 171%. Similarly, blood urea level was higher in burn patients the percentage of increase was 120% and 130% respectively in group I and group II. A slight rise was also observed in serum potassium level. It was 14% higher in group I while group two shows 24% higher level in contrast a decrease in serum sodium level was observed. The decrease of 4% was equal in both group I and II. (Table 2)

Among hematological parameters, neutrophil count was found to increase instantaneously along with eosinophils, lymphocytes and monocytes. Leucocytosis happens during the onset of thermal injury. Burn injury leads not only to immune complications but also results in organ dysfunction. Our observations highlight a remarkable increase in blood urea and serum potassium levels in both the age groups while hyponatremia was also noticed.

In major burns, the initial resuscitation period is characterized by hyponatraemia and hyperkalaemia. This happens due to increase in vascular permeability, increased interstitial osmotic pressure in burn tissue & cellular oedema with most significant shifts occurring in first hours. Hyponatremia is frequent & the restoration of sodium losses in burn tissue is therefore essential. Hyperkalaemia is also characteristic of this period because of the massive tissue necrosis. The extent of this process depends on the severity of shock & can be minimized by early restoration of perfusion in the injured tissues. Failure to achieve this can cause widespread of organ dysfunction (Romas 2000). The value of serum creatinine increases with the increase in sepsicaemia.

Thermal injury of the skin is also an oxidation process, associated with biological & metabolic alterations; thermal injury generates free radicals from various cellular populations through many pathways & modulation of free radical generated activity with antioxidant seems to be an important part in pharmacological treatment of burns. Vitamin E effectively scavenges free radical within the cells & vit C could serve to scavenge free radical within extracellular space (Pinnell, 2003). These values can be made to reach a constant level by providing them a number of dietary supplements like high protein rich foods, milk based foods or food of high calorific value (McGregor et.al 1981). Nutrition is crucial to any wound healing which is important in case of burn victims. The correct and rational treatment of burn shock, fluid replacement and treatment of hydroelectrolytic and metabolic disturbances may improve the functioning of patient's body system leading to significant survival chances (Dauti et.al 1996). The prevention and successful treatment of complications after severe burns requires knowledge of the clinical periods of burn illness and a precise theoretical methodology for the evaluation of prognosis (Belba, 2002). Our findings revealed that hemoglobin percentage was decreased more in age group I as compared to age group II.

Our data also reveal that the high levels of urea and
creatinine, the markers of kidney function lead to renal failure in burn injury. Renal failure may be due to release of some toxins in blood circulation by burning of body fats. Similarly, hyperkalemia may be due to breakdown of RBCs as potassium concentration is much higher inside RBCs. Hyponatremia in both the group is due to loss of sodium through oozing of plasma from injuries and permeability of tissues. (Dauti et. al 1996).

The levels of urea and creatinine were high in both group I and group II. However in group I the extent of rise was less as compared to group II. which may be due to more tolerance of group I as compared to group II. These biochemical changes should be deliberately corrected only if there is good reason to suppose that they are dangerous and close observations should be made to detect any unexpected consequences of biochemical meddling. Thus, attention should be concentrated upon curative treatment & supportive therapy to protect tissue function, when such treatment is successful the patient will correct his biochemical & hematological disturbances efficiently.

REFERENCES


