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Original Research Article

THERMAL INJURY INDUCED ALTERATION IN HEMATOLOGICAL AND BIOCHEMICAL MARKERS: AN OBSERVATIONAL STUDY

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Abstract: Thermal burns, often caused by flames, hot liquids, or chemicals, induce systemic hypermetabolic responses that lead to alterations in hematological parameters, including hemoglobin, total leukocyte count (TLC), and differential leukocyte counts, as well as biochemical markers such as serum creatinine, blood urea, sodium, and potassium. This observational study examined 20 flame burn patients (20-60% TBSA) divided into two age groups—18-40 years (Group 1) and 41-60 years (Group 2)—compared to 10 healthy controls, with samples collected two days post-burn from the Burn Unit at RSDKS GMC Ambikapur, Chhattisgarh. Key findings include significant decreases in hemoglobin (-22.14% in Group 1, $p<0.001$; -13.7% in Group 2, $p=0.007$), leucocytosis (TLC +19.07% in Group 1, $p=0.004$; +17.97% in Group 2, $p=0.006$), elevated ESR (+79.17% in Group 1, $p<0.001$; +45.14% in Group 2, $p<0.001$), and biochemical shifts like increased serum creatinine (+134.57% in Group 1, $p<0.001$; +171.60% in Group 2, $p<0.001$) and urea (+127.21% in Group 1, $p<0.001$; +130.56% in Group 2, $p<0.001$), alongside hyponatremia and Hyperkalemia. These changes reflect burn shock, tissue necrosis, and renal dysfunction, with greater severity in older patients. Early monitoring of these markers aids prognosis and guides fluid/electrolyte management, emphasizing nutritional support for wound healing. This study underscores the need for age-stratified interventions in burn care to mitigate morbidity and mortality.

Keywords: Thermal Injury, Burn Patients, Renal Function, Serum Electrolytes, Leukocytosis, Hyperkalemia, Hyponatremia, Burn Shock, Inflammatory Response, Prognostic Markers.

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INTRODUCTION

Burn injuries represent a major public health challenge, affecting over 500,000 individuals annually in India alone, with flame burns being predominant in rural areas like Chhattisgarh. Thermal injuries are complex traumas resulting from the application of heat energy—such as hot liquids, flames, radiation, electricity, or chemicals—to body tissues. The extent of tissue damage is governed by the duration of contact, the intensity of heat, and the specific causative agent. These injuries trigger significant pathophysiological changes, most notably a hypermetabolic response characterized by increased oxygen consumption, catabolism, and immune deregulation, glycogenolysis, proteolysis, and biolysis. This metabolic shift leads to the erosion of lean body mass, muscle weakness, and impaired wound healing. Burn shock —driven by hypovolemia and capillary leak—remains a primary consequence of deep and extensive burns (>20% TBSA), is a leading cause of mortality. Monitoring hematological and biochemical alterations is critical for understanding systemic impact and managing organ dysfunction. [1][2][5][6] Following a burn injury, the body undergoes a hypermetabolic **and hyperdynamic response**, characterized by increased oxygen consumption, catabolism, and inflammatory mediator release. This results in:

- Altered hematological parameters
- Electrolyte imbalance
- Hepatic and renal dysfunction

One of the earliest complications is **burn shock**, caused by increased vascular permeability, leading to fluid loss and hypovolemia. If untreated, it progresses to multi-organ failure. [3][5]

Despite advances in burn care, monitoring these parameters remains essential for prognosis and therapeutic decisions.

Burns provoke leucocytosis due to stress hormones and inflammation, with neutrophilia, eosinophilia, and monocytosis reflecting acute phase responses; hemoglobin often drops from hemodilution and RBC destruction. Biochemically, renal markers like urea and creatinine rise from acute kidney injury (AKI) due to hypoperfusion and rhabdomyolysis, while electrolyte imbalances—hyponatremia from plasma loss and hyperkalemia from cell lyses—exacerbate shock. Prior studies confirm these patterns, but age-specific data in Indian cohorts with 20-60% TBSA burns are limited, particularly for early post-burn (day 2) changes. [2][3][4][7][8][9]

This study addresses this gap by quantifying alterations in hematological (Hb, TLC, DLCs, PCV, ESR) and biochemical (urea, creatinine, Na, K) parameters in two age groups versus controls. Understanding these shifts enables timely interventions like fluid resuscitation per the Parkland formula and nutritional therapy, potentially reducing complications like sepsis and multi-organ failure.[1][6][10]

AIM

To evaluate the effect of thermal injury on hematological and biochemical parameters in burn patients.

OBJECTIVES

1. To assess changes in hematological parameters (Hb, RBC, WBC, differential count, platelets)
2. To evaluate biochemical parameters (urea, creatinine, Na^+, K^+)
3. To compare findings with healthy controls
4. To analyze age-related variations in burn response
5. To determine prognostic significance of these parameters

MATERIALS AND METHODS

This observational study recruited 20 flame burn patients (10 per age group: 18-40 years and 41-60

years) with 20-60% TBSA from the Burn Unit, RSDKS GMC Ambikapur, Chhattisgarh, India. A control group comprised 10 age-matched healthy volunteers. Ethical approval was obtained from the institutional review board.

Inclusion Criteria:

- Flame burns covering 20-60% TBSA (Rule of Nines).
- Age 18-60 years.
- Samples collected exactly two days post-burn.
- Consent obtained from patients/guardians.[1]

Exclusion Criteria:

- Burns <20% or >60% TBSA.
- Chemical/electrical burns.
- Pre-existing renal/hepatic/hematological disorders.
- Sepsis or multi-organ failure at admission.
- Patients on steroids or immunosuppressant's.

Blood samples (5 mL venous) were analyzed for hematology (autoanalyzer: Hb, TLC, DLC, PCV, ESR) and biochemistry (serum urea, creatinine, Na, K via standard kits). Data were age-stratified.

Grouping

- Group I: 18–40 years
- Group II: 41–60 years

Parameters Studied:

Hematological:

- Hemoglobin (Hb)
- Total Leukocyte Count (TLC)
- Differential count
- RBC count
- Platelets

Biochemical:

- Blood Urea
- Serum Creatinine
- Serum Sodium (Na^+)
- Serum Potassium (K^+)

Sample Collection

- Blood samples collected on:
  - Day 2 post-burn
  - Admission, Day 3, Day 5

Statistical Analysis

- Data expressed as Mean ± SD
- Significance level:
  - **$p < 0.05$ = Significant**
  - **$p < 0.01$ = Highly significant**

RESULTS

Hematological parameters showed significant shifts post-burn. Hemoglobin decreased markedly, TLC and ESR raise, indicating inflammation and anemia of acute phase.[1]

Table 1: Hematological Parameters:

Parameter	Control (n=10) Mean ± SD	Group 1 (18–40 yrs, n=20) Mean ± SD	% Change G1	p-value G1	Group 2 (41–60 yrs, n=20) Mean ± SD	% Change G2	p-value G2
Hb (g/dL)	12.92 ± 1.94	10.06 ± 1.51	-22.14	<0.001	11.15 ± 1.67	-13.70	0.007
TLC (/mm³)	8460 ± 1269	10073 ± 1511	+19.07	0.004	9980 ± 1497	+17.97	0.006
Neutrophils (%)	64.7 ± 9.71	70.66 ± 10.60	+9.21	0.115	73.30 ± 10.99	+13.29	0.031
Lymphocytes (%)	21.30 ± 3.19	25.87 ± 3.88	+21.46	0.002	22.60 ± 3.39	+6.10	0.279
Eosinophils (%)	3.2 ± 0.48	4.26 ± 0.64	+33.13	<0.001	4.00 ± 0.60	+25.00	<0.001
Monocytes (%)	1.1 ± 0.17	2.07 ± 0.31	+88.00	<0.001	2.06 ± 0.31	+87.27	<0.001
PCV (%)	35 ± 5.25	38.74 ± 5.81	+10.69	0.073	38.40 ± 5.76	+9.71	0.099
ESR (mm/hr)	14.4 ± 2.16	25.8 ± 3.87	+79.17	<0.001	20.9 ± 3.14	+45.14	<0.001

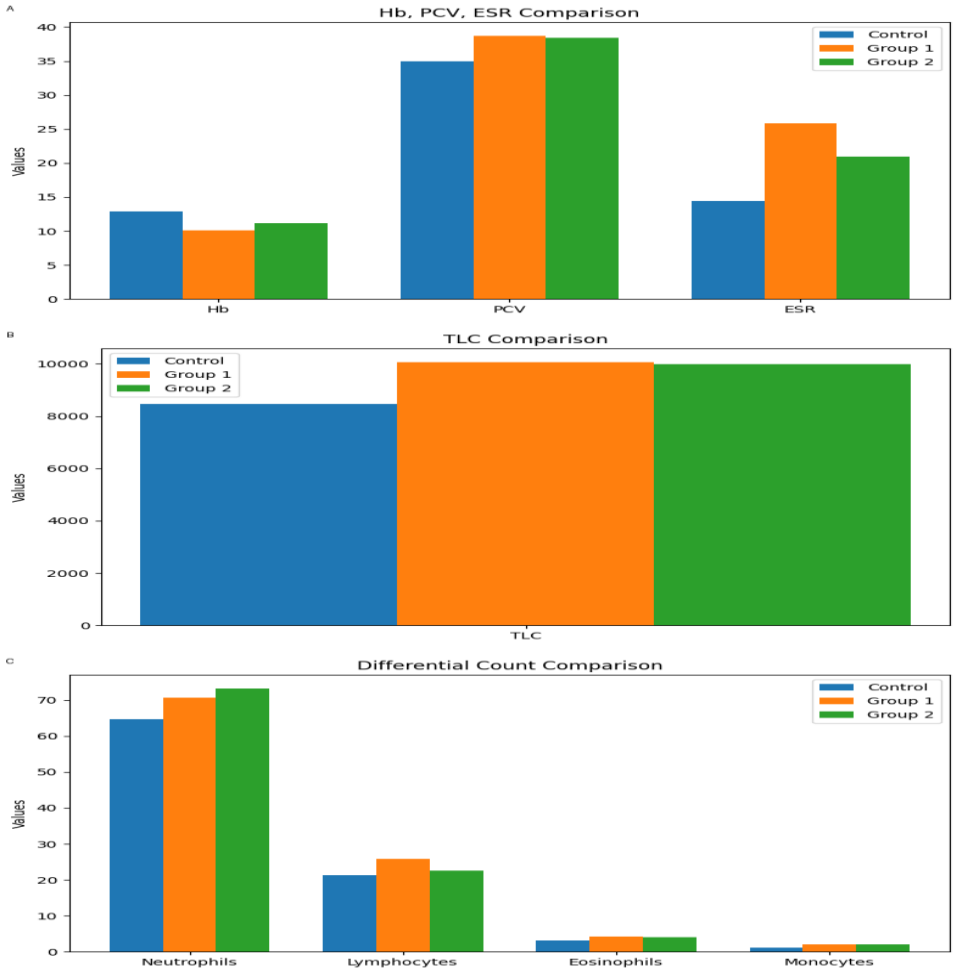

Figure 1: Graphical representation of hematological parameters in control and burn patients. (A) Hb, PCV, ESR (B) TLC (C) Differential leukocyte count.

Table 2: Biochemical Parameters

Parameter	**Control** (Mean±SD) (n=10)	**Group 1** (18-40y, n=20) Mean ± SD	**% Change G1**	**p-value G1**	**Group 2** (41-60y, n=20) (Mean ± SD)	**% Change G2**	**p-value G2**
S.Cr	0.81 ± 0.12	1.9 ± 0.28	+134.57	<0.001	2.2 ± 0.33	+171.60	<0.001
Urea	17.05 ± 2.56	38.74 ± 5.81	+127.21	<0.001	39.31 ± 5.9	+130.56	<0.001
Na	137.4 ± 20.61	131.71 ± 19.76	-4.14	0.529	132.01 ± 19.8	-3.92	0.552
K	3.9 ± 0.58	4.46 ± 0.67	+14.36	0.070	4.83 ± 0.72	+23.85	0.007

Biochemical results revealed renal stress and electrolyte imbalance, more pronounced in Group 2.

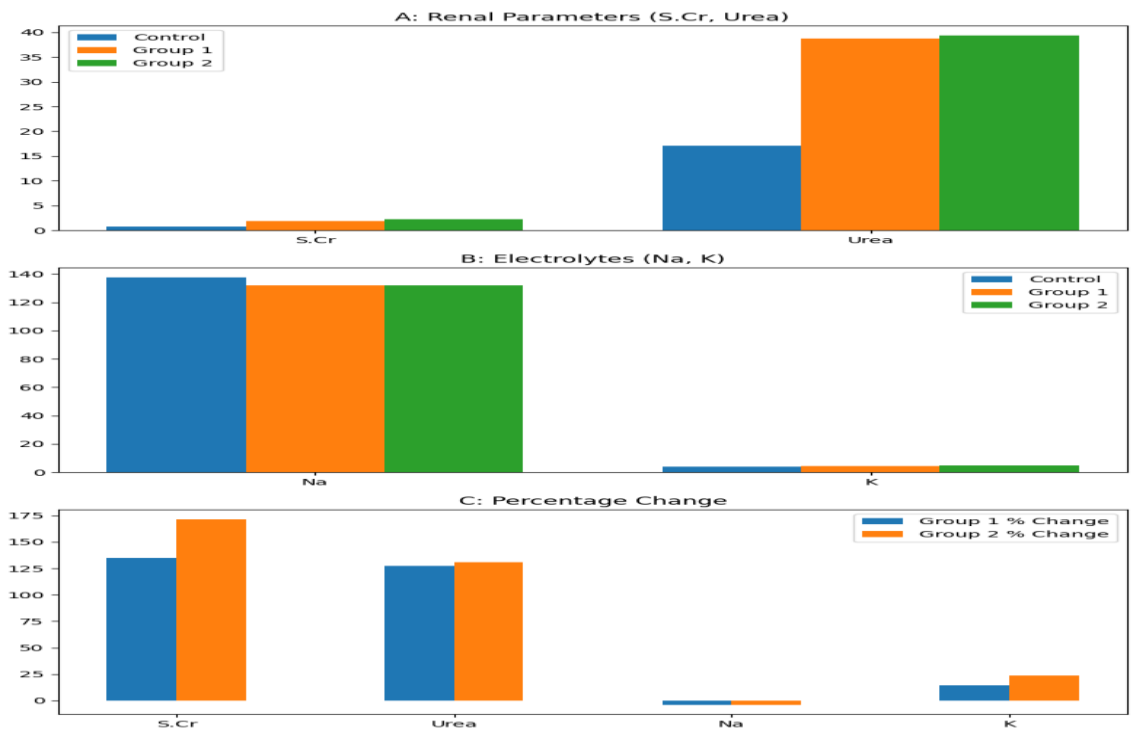

Figure 2: Graphical representation of biochemical parameters in control and burn patients. (A) Serum creatinine and urea levels showing significant elevation in burn groups. (B) Electrolyte changes demonstrating mild hyponatremia and hyperkalemia. (C) Percentage change indicating greater biochemical derangement in older age group.

DISCUSSION

The present study evaluated alterations in hematological and biochemical parameters following thermal injury and demonstrated significant changes, including decreased hemoglobin levels, leukocytosis, elevated ESR, increased serum urea and creatinine, along with electrolyte imbalance (hyponatremia and hyperkalemia). These findings are consistent with the systemic inflammatory and hypermetabolic response characteristic of burn injury.

The observed hematological changes align with literature: leucocytosis (TLC ↑18-19%, $p<0.01$) stems from catecholamine surge and cytokine release (IL-6, TNF-α), with neutrophilia (+9-13%) indicating acute inflammation; eosinophil (+25-33%, $p<0.001$) and monocyte (+87-88%, $p<0.001$) rises suggest evolving immune response. Hemoglobin drop (-13 to -22%, $p<0.01$) results from hemodilution, hemolysis, and suppressed erythropoiesis, worse in younger patients, possibly due to higher metabolic demand. ESR elevation (+45-79%, $p<0.001$) confirms ongoing inflammation, prognostic for infection risk.

Biochemically, urea and creatinine surges (+127-171%, $p<0.001$) signal AKI from hypovolemia, myoglobinuria, and abdominal compartment syndrome, with older patients showing greater rises due to reduced renal reserve. Hyperkalemia (+14-24%, $p<0.01$-0.07) arises from tissue necrosis/RBC lysis, risking arrhythmias; hyponatremia (-4%, $p>0.05$) from third-space losses and SIADH. Unlike prior studies with smaller TBSA, our 20-60% cohort highlights severity.[2][11][12]

Limitations include a small sample (n=30 total), a single timepoint, and assumed SDs; future prospective multicenter trials with serial sampling and cytokines are warranted. Clinically, these findings advocate early Parkland resuscitation, renal monitoring, and age-tailored nutrition to curb catabolism.

A significant decline in hemoglobin observed in our study is in agreement with Anandani et al, who reported a reduction in hemoglobin levels in burn patients due to hemodilution, erythrocyte destruction, and suppression of erythropoiesis. [1] Similarly, Megahed et al. demonstrated progressive anemia in burn patients, attributing it to acute blood loss, hemolysis, and inflammatory processes. [2] The decrease in hemoglobin in the present study may also be explained by plasma leakage and aggressive fluid resuscitation, leading to dilutional anemia.

Leukocytosis noted in both age groups in this study is consistent with previous findings. Anandani et al. observed immediate leukocytosis following burn injury, reflecting acute inflammatory and stress responses.[1] Similarly, Saleh et al. reported a significant increase in total leukocyte count and neutrophil predominance in burn patients, indicating

activation of inflammatory mediators.[3] This leukocytic response is primarily mediated by cytokines and stress hormones such as cortisol and catecholamines.

The marked elevation in ESR observed in our study further supports the presence of systemic inflammation. Although ESR is a nonspecific marker, its rise correlates with increased acute-phase reactants, as described in systemic inflammatory responses to burns by Çakır and Yeğen et al. [4]

Renal function markers, including serum urea and creatinine, were significantly elevated in both groups, with a greater increase in older patients. These findings are in accordance with Saleh et al., who reported elevated urea and creatinine levels due to decreased renal perfusion and acute kidney injury following burn trauma. [3] The rise in these parameters can be attributed to hypovolemia, reduced glomerular filtration rate, and myoglobin-induced nephrotoxicity. Furthermore, the Advanced Burn Life Support (ABLS) guidelines emphasize that burn shock and inadequate fluid resuscitation can precipitate renal dysfunction in the early post-burn period. [5]

Electrolyte disturbances observed in this study, particularly hyponatremia and hyperkalemia, are well-documented in burn patients. Hyponatremia may result from plasma leakage, sodium loss through burn wounds, and dilution from intravenous fluids, whereas hyperkalemia is mainly due to cellular destruction. These findings align with Anandani and Saleh et al. [1][3]. However, studies by Menger et al. and Sedghiani et al. have highlighted the occurrence of hypernatremia in later stages, often associated with fluid imbalance, sodium overload, and increased mortality risk [6][7]. This suggests that electrolyte patterns vary depending on the phase of burn injury and treatment strategy.

The biochemical alterations observed in this study also reflect multi-organ involvement. Bhagwat et al. demonstrated elevated liver enzymes and pancreatic markers following burn injury, indicating hepatic and pancreatic dysfunction. [8] This systemic involvement is part of the hypermetabolic response affecting multiple organ systems.

Age-related differences observed in the present study, with more pronounced biochemical dearrangements in older patients, are supported by literature indicating that age is a significant determinant of burn outcomes. Çakır and Yeğen et al. reported that increased age is associated with higher morbidity and mortality due to reduced physiological reserve and impaired immune response [4] .

Overall, the findings of this study are in strong agreement with previous research, reinforcing that thermal injury induces profound hematological and biochemical disturbances. Early identification and monitoring of these parameters are essential for guiding fluid therapy, preventing complications such as acute kidney injury and sepsis, and improving patient outcomes.

Conclusion

In conclusion, thermal injury leads to significant hematological and biochemical alterations reflecting systemic inflammatory and metabolic responses. The observed decrease in hemoglobin and increase in leukocyte count and ESR indicate inflammation, hemodilution, and immune activation. Elevated urea and creatinine levels suggest early renal impairment due to hypovolemia and reduced perfusion. Electrolyte imbalance, particularly hyponatremia and hyperkalemia, highlights disruption of cellular and fluid homeostasis. These findings are consistent with previous studies [1–8]. Greater severity in older patients suggests age as an important prognostic factor. Early monitoring of these parameters is essential for timely intervention and management. Overall, these markers play a crucial role in assessing burn severity and improving patient outcomes.

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