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Research Article

Hematological and Inflammatory Biomarkers as Predictors of Burn Wound Healing in Iraq

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Abstract

Objective: Burn damage poses analytical trials, contributing to augmented death rates with late analyses. This study seeks to classify early risk influences and evaluate it impression on clinical consequences by exploratory hematological dynamics in burn cases. The effort contains age- sex-linked patterns, Total Body Surface Area (TBSA) damaged by burns, length of hospital stay (LOS) around between days to weeks, and variations in blood markers during burn wounds.
Methods: Data and Biochemical markers were documented, and statistical analysis was achieved using prism. A heightened primary immune response is recorded by white blood cell count on 1-7 days. Blood indicators, including hemoglobin, red cell count, and platelet count, presented dynamic outlines over the study durations.
Results: Peripheral differences in platelet numbers and intriguing patterns proposed potential values like circulated intravascular coagulation. The study affords a vital understanding of hematological responses to different burn wound degrees and sepsis. Early identification of biomarkers, particularly Interleukins1 and Tumor Necrosis Factor. Plasma procalcitonin level was related to sepsis, C-Reactive Protein patterns, and immune responses, informing clinicians about predicting results and managerial therapeutic interventions.
Conclusion: Regardless of restrictions, this work highlights the requirement for further extensive research to widely understand the complex associations between burn damage, hematological responses, and clinical results.

1. Introduction

Infections is the main cause of death among patients with burns [1]. Swift analysis and dealing with infection are linked with better results. Finding requires isolation of the microbe from the blood or the burn areas [2]. The degrees of the burn areas regulates the ways of treatment. In other words, the depth of the damage is assessed according to clinical findings [3]. Dermal and epidermal layers are the basis of increasing keratinocytes, which travel to the thrombotic and wound net, showing a key role in the healing procedure of burn areas. Burn damages can be identified depending on several features, including the surface area of skin, causes, and proportion of external region damage. All factors classified the grade of burn damage [4]. Burns recognized as fractional - full surface area of skin. If the injury is restricted to the epidermis and its surface part, without damaging the additional structures, recovery from burns typically occurs within 1–2 weeks, and the danger of harm Burnside skin is reduces. While the damaged area extends into the higher depth of layers of the dermis, with remarkable damage of additional structures, the epithelium requires a longer period to regenerate approximately 3–6 weeks with a high risk of hypertrophic scarring. Full-thickness burns are related to harm to all sheets of the skin and generally need medical interposition to confirm appropriate burn

health-giving [5]. The full-thickness burn wounds lead to coagulative necrosis of most layers cutaneous and subcutaneous. Because of skin lost its role as a physiological barrier to defensive underlying layers, this damage can be determined by several agents such as temperature, the energy diffused by the contributing agent, and the period of exposure [6]. The histological changes of skin burns is characterised as three zones. Region of clotting which characterizes the part of necrosis with permanent skin injury sustained at the period of injury. Region of stasis which surroundings the clotting area and is abstemiously injured with vascular simulations, raised narrowing of the blood vessels agents, besides limited inflammations responses, leads to diminished tissue perfusion. This zone may improve or might advance to an area of necrosis Based on the burns in the backgrounds. Region of hyperemia, with vasodilatation because of inflammation. It is described by augmented blood circulation to nearly good health skin without considerable risk of necrosis except if there is substantial sepsis or continued hypo-perfusion [7]. However, this study focuses on the hemodynamic changes, which appear as biomarkers for systemic dysfunction or recovery during different types of burns. The assessment of certain biomarkers has directed to a bright understanding of pathophysiology, whereas others have been employed either to measure the efficacy of specific treatments or for prognostic determinations.

The objective of this research is to evaluate the biological changes inculding inflammatory biomarkers factors in burn patients.

2. Material and Methods

Ethical Approval

Approval for this study was obtained from the Scientific and Ethical Committee, College of Pharmacy, University of Kerbala, Iraq (No: 2024An.12). The research was conducted on retrospective patient records. All identifying information was anonymized. Documented consent from parents was available for pediatric cases, and adult patients were included in accordance with the rules set out by the committee.

Study design and hospitals

This retrospective study analyzed 10 years (2014–2024) of medical records from patients with severe burn injuries treated in burn departments across several hospitals in Iraq.

Patient selection and subgroups

Patient data included age, sex, TBSA, type and location of burn, length of hospital stay, and outcome (survivor or non-survivor).
Main cohort: A total of 70 patients with complete demographic information, TBSA, and clinical course data.
Biomarker subgroup: 44 patients with PCT, CRP, and cytokine measurement results in their files; 15 of these had a record of sepsis/septic shock (Table 1, Figure 3 and 6).
Mild burn subgroup: Cases who stayed in the hospital for $<$48 hours and had D1–D3–D7 hemogram results in their files Table 2.
Necrotic area comparison: Records of patients aged 10–15 and 50–70 with the same TBSA (10%) Figure 2.

Inclusion/exclusion criteria

Inclusion: Patients hospitalized due to second- and/or third-degree thermal burns with complete records in their files.
Exclusion: Files with incomplete basic clinical data; causes of skin necrosis other than burns.

Definitions

TBSA (Total Body Surface Area): Values calculated using the Lund–Browder method for patients under 15 years of age and the Rule of Nines for patients 15 years of age and older were used in the records. In our cohort, the extent of TBSA involvement ranged from 15% (moderate burns) to 55% (severe burns).
Sepsis/septic shock: Defined as the presence of clinical signs of infection with laboratory abnormalities and/or positive culture results. Specific cut-off values included leukocyte counts $<$3000 or $>$15,000/mm^3 and temperature $<$35.5 °C or $>$38.5 °C; cases requiring hemodynamic support were classified as septic shock.
Burn type and location: According to the records, most burns were scald burns, most commonly affecting the abdomen, chest, and lower extremities Figure 1.

Time points and recorded measurements

Laboratory results in patient files were reviewed:
Biomarkers:
PCT (ng/mL): Values recorded at baseline and follow-up Figure 3.
CRP (mg/L): Records at D1, D7, and D14 Figure 6.
Cytokines (pg/mL): Measurements of IL-1α, IL-1β, and TNF-α (within the first week, Table 1).
Hemogram and NLR: Values recorded on D1 (application), D3, and D7 Table 2.
Platelet: D1, D3, D7, D14, and D21; values reported according to TBSA percentage (20–30%, 31–40%, 41–50%) Figure 5.

Laboratory methods

All measurements were performed routinely for diagnostic purposes in the laboratories of the relevant hospitals and were obtained from file records. Commercial ELISA kits were used for cytokines, immunoturbidimetry for CRP, and immunochemistry methods for PCT, as indicated in the files.

Microbiological examination

The culture results recorded in the files were evaluated. It was noted that biopsy samples taken from the burn bed were weighed, serially diluted, and cultured in aerobic/anaerobic environments, and the results were reported in CFU/g. Fungal agents were identified at the species level, and viral agents were not routinely screened for and therefore not reported in the records Figure 6.

Statistical analysis

Normality for continuous variables was assessed using Shapiro–Wilk. Two independent groups were compared using the Student t-test or, if the distribution was not appropriate, the Mann–Whitney U test. Multiple group comparisons were performed using one-way ANOVA (repeated measures ANOVA where appropriate). The data were stated as mean ± SD. The significance threshold was set at $p<0.05$. Analyses were performed using GraphPad Prism software.

3. Results

3.1. Measurement of the necrotic area within in patient's age

Mature was multifaceted, multifactorial singularity. Hormones, mitochondrial DNA, inherited substances, free radicals, inflammation, and immunosenescence all have an important function in the development of aging (Jin, 2010). However, this study was achieved to associate burn patients from two groups depending on age: one younger 10-15 years as well as the other elderly person 50 years of age for patients suffering from burn wounds with necrotizing. Burns encompassing 20% of the TBSA were accompanying with delayed recovery of only 20% in young adults in good health, whereas older individuals with identical burn sizes observed 75%. Necrosis in wound burn has a main impact on skin regeneration, functional, and pleasing results.

Incidences of burns in age group in Kerbala, Iraq.

As shown in Figure 1, when the distribution of burn cases by age group was examined, the highest incidence was found to be in the 16–23 age group. In the early childhood (0–7 years) and 8–15 age groups, female cases showed a higher rate compared to males. In contrast, in the 31–70 age group, the rate of male patients was higher than that of females. These findings indicate that burns exhibit a different epidemiological distribution according to age and gender.

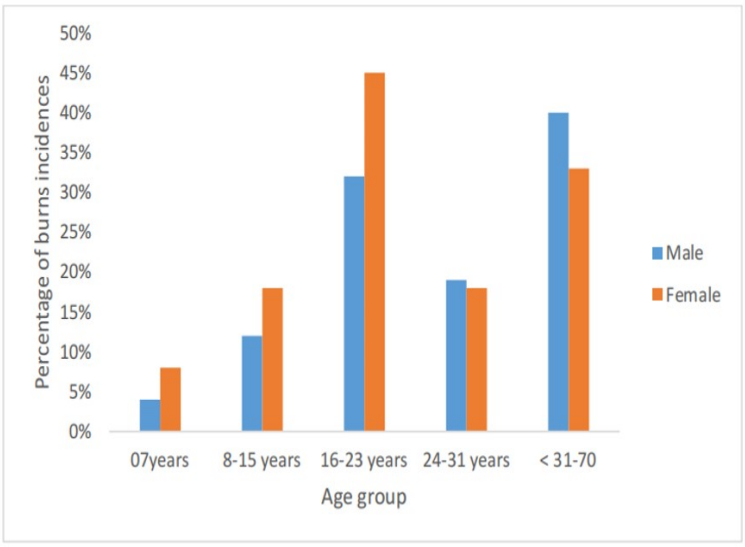

Figure 1: Distribution of burn cases by age group and gender (%)

As shown in Figure 2, in burns with the same TBSA (10%) value, the necrotic area ratio in the elderly group (50–70 years) is significantly higher than in the young group (10–15 years). This finding reveals that advanced age negatively affects tissue regeneration capacity and delays the healing process.

3.2. Measurement of inflammatory biomarkers in patients suffering from burns

Radioimmunoassay was used to quantify the levels of interleukins (IL)-1β and -1α as well as tumor necrosis factor (TNF-α) in plasma samples IL-1 concentrations were measured by using ELISA.

Of 44, 15 patients were in septic shock Table 1. This research also examined the relationship between the plasma procalcitonin (PCT) level and burn size, sepsis, and CRP in patients who had suffered severe burns during the course of their treatment. Figure 3 shows an increasing level of CPT in both second and third-degree burns about the mean of 15.7 ng/ml and in sepsis cases averaged 63.35 ng/ml compared to first-day (2.9 ng/ml) admission to the hospital with an electrical burn.

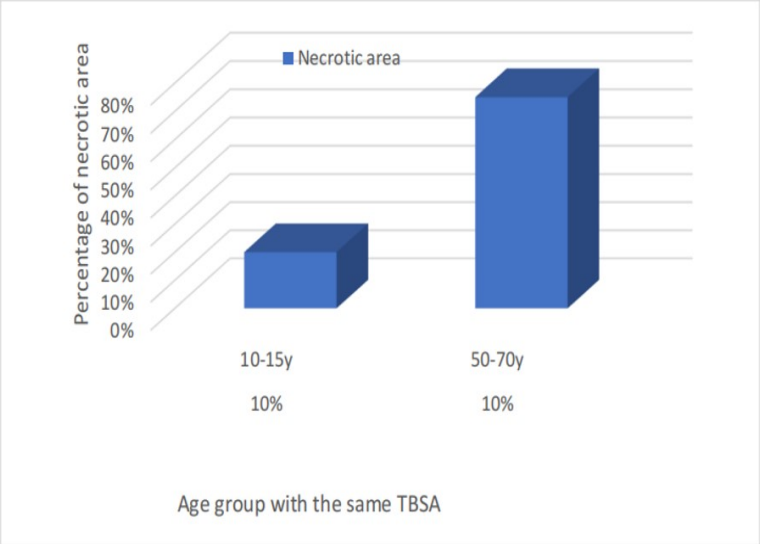

Figure 2: Comparison of the percentage of necrotic area according to age groups in patients with the same total body surface area (TBSA, 10%)

Significant differences in proinflammatory cytokine levels were observed depending on burn severity and sepsis development. As shown in Table 1, IL-α levels were 20 pg/ml, IL-1β levels were $<$70 pg/ml, and TNF-α levels were 69 ± 27 pg/ml in patients with only second- and third-degree burns. In contrast, these values increased significantly in patients who developed sepsis; IL-α was 40 pg/ml, IL-1β was 120 ± 17 pg/ml, and TNF-α was 119 ± 30 pg/ml. These findings indicate that the systemic inflammatory response is significantly enhanced in sepsis and that cytokines such as IL-1β and TNF-α may be valuable biomarkers for sepsis diagnosis and prognosis prediction.

Table 1: Comparison of proinflammatory biomarker levels in patients with second- and third-degree burns and in patients with sepsis

Biochemical Marker	**Second- and third-degree burns**	**Septic patients**
Interleukin-α (IL-α, pg/mL)	20	40
Interleukin-1β (IL-1β, pg/mL)	$<$70	120± 17
Tumor Necrosis Factor-α (TNF-α, pg/mL)	69 ± 27	119 ± 30

As shown in Figure 3, procalcitonin (PCT) levels were found to be quite low on the first day of hospitalization, but showed a significant increase in cases of second- and third-degree burns. In patients who developed sepsis, PCT values reached their highest levels, creating a distinct difference. These findings indicate that PCT levels increase in direct proportion to burn severity and the presence of sepsis, and may serve as a valuable biomarker for predicting the early development of sepsis.

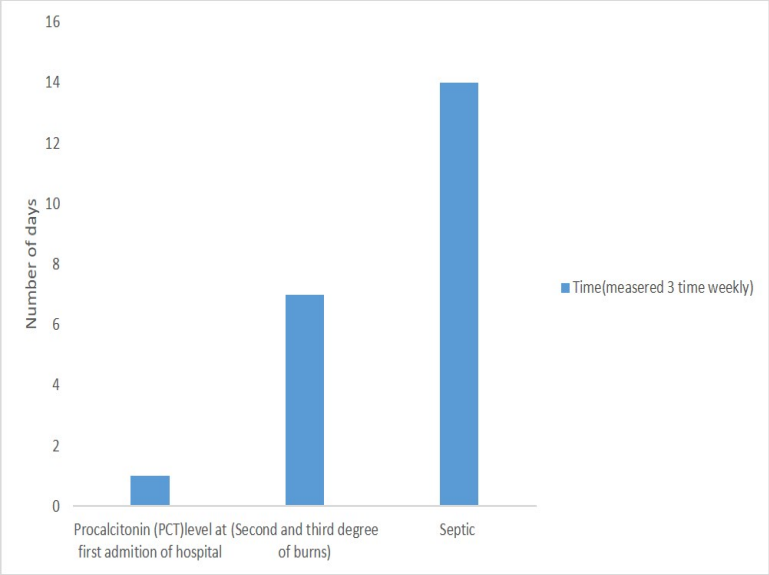

Figure 3: PCT levels in burn patients and septic cases

As shown in Figure 4 C-reactive protein (CRP) levels fluctuated over time in burn cases with 27% TBSA. On the first day of hospitalization, CRP levels were significantly higher in patients with sepsis compared to those with second- and third-degree burns. By the seventh day, CRP levels were low in both groups, but an increase was observed again on the 14th day. During this period, CRP levels in patients with sepsis reached an average of 90 mg/L, while in burn cases alone, they remained at approximately 70 mg/L. These findings suggest that CRP may be an important biomarker for distinguishing the presence of sepsis, particularly in the late period (D14), and for indicating the severity of the inflammatory response.

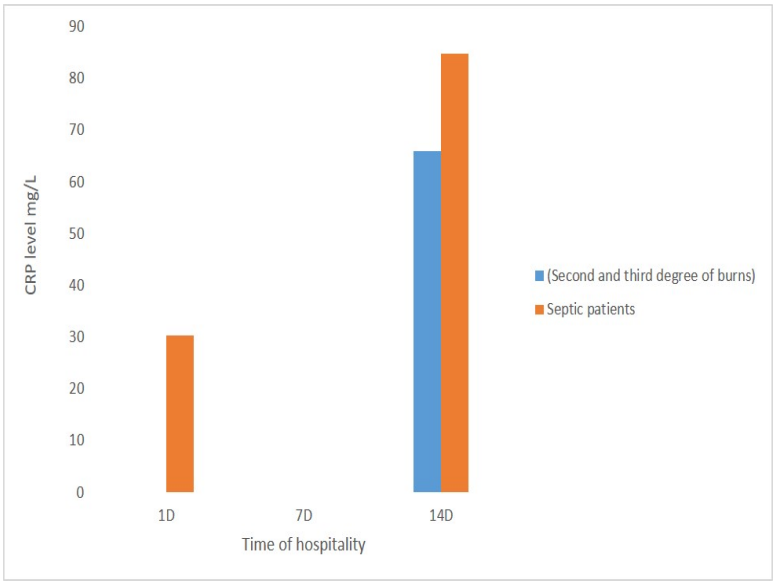

Figure 4: CRP values over time with the same burn size of 27% of TBSA. Huge burns have meaningfully and determinedly higher C-RP levels starting 2-7 days postburn, $p<0.05$

The numeral of platelets in one or more areas of 1 mm 2 was measured as follows:

$$\text{Platelet count}/\text{l} = \frac{\text{Number of cells counted} \times \text{dilution} \times 10^6}{\text{Volume counted}}$$

In this research, all the patient with burns was tracked for 3 weeks (with no death occurring) as well as the level of platelet numbers was calculated. In survivors, a steady trend in platelet numbers was detected without the increase of septicemia.

As shown in Figure 5, platelet counts fluctuated over time in different TBSA groups (20–30%, 31–40%, and 41–50%). A significant increase was observed in all groups on the 3rd day of hospitalization, while platelet levels decreased on the 7th day and showed a tendency to recover again between the 14th and 21st days. This fluctuation was not directly related to the burn surface area but was considered to be more closely associated with the development of sepsis. These findings suggest that platelet counts may be used as an indicator of the post-burn immune response and, in particular, the presence of sepsis.

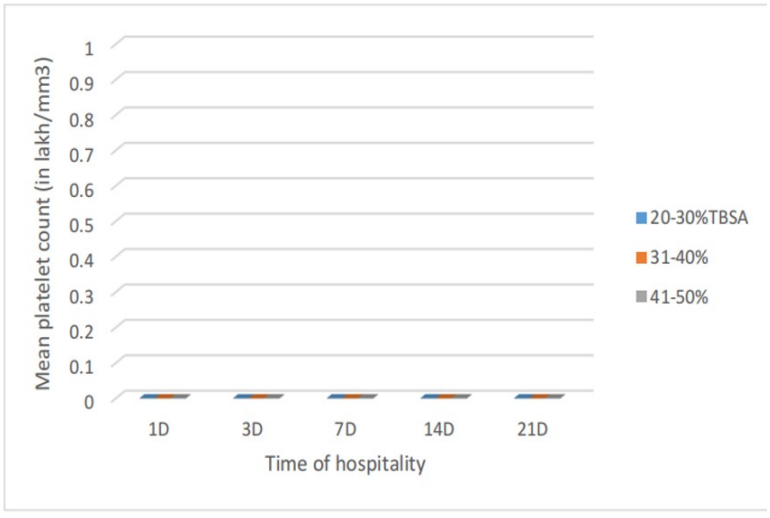

Figure 5: The variation in platelet amount was not related to the percentage of burns but may be linked with the presence of sepsis in the burn patient

The results of blood tests followed up routine tests included white blood cell count.

As shown in Table 2, hematological parameters showed significant fluctuations within the first 7 days in cases with minor burns who stayed in the hospital for less than 48 hours. On the day of admission (D1), white blood cell (WBC: 18.92 ± 8.06 $\times 10^9$/L) and neutrophil (16.33 ± 7.39 $\times 10^9$/L) counts were elevated, but these values decreased significantly by the third day (D3) (WBC: 11.57 ± 5. 33 $\times 10^9$/L; neutrophil: 9.58 ± 4.83 $\times 10^9$/L), and showed an upward trend again on the seventh day (D7) (WBC: 13.77 ± 6.35 $\times 10^9$/L; neutrophil: 11.49 ± 5.86 $\times 10^9$/L). Lymphocyte counts remained at low levels and decreased during the first three days (1.43 ± 1.04 → 1.10 ± 0.56 $\times 10^9$/L), showing a limited increase on the seventh day (1.27 ± 0.82 $\times 10^9$/L). The neutrophil/lymphocyte ratio (NLR) followed a similar pattern; it was quite high on D1 (14.45 ± 9.46), decreased on D3 (9.58 ± 5.22), and increased again on D7 (10.55 ± 7.45). In contrast, red blood cell (RBC) and hemoglobin levels decreased gradually from the outset; hemoglobin levels decreased from 154.53 ± 29.04 g/L at D1 to 105.97 ± 20.48 g/L at D7. These findings indicate that a significant inflammatory response develops even in mild burns in the early stages, but fluctuations in leukocyte and neutrophil values are observed during follow-up; additionally, the continuous decrease in erythrocyte and hemoglobin levels indicates the development of anemia after burns.

Table 2: Hematological parameters of patients with mild burns (hospital stay <48 h)*

Blood parameters	Day 1 (D1)	Day 3 (D3)	Day 7 (D7)
WBC ($\times 10^9$/L)	18.92±8.06	11.57±5.33	13.77±6.35
Neutrophils ($\times 10^9$/L)	16.33±7.39	9.58±4.83	11.49±5.86
Lymphocytes ($\times 10^9$/L)	1.43±1.04	1.10±0.56	1.27±0.82
NLR	14.45±9.46	9.58±5.22	10.55±7.45
RBC ($\times 10^{12}$/L)	5.05±0.91	4.30±0.81	3.51±0.64
Hemoglobin (g/L)	154.53±29.04	130.62±26.25	105.97±20.48

*Patients with mild burns and hospitalization <48 hours, with follow-up data available up to day 7.

3.3. Measurement of type of microbes in different burns locational

Anatomically, the skin is an organ triggered by numerous physiological damage like thermal, chemical, and electrical which prevents sustaining homeostasis, excretion, and protection from exterior agents. In this condition, the skin has lost its physical barrier to fight pathogen attacks [8]. Burn wound disrupts the epidermal wall, resulting in down-regulation of the immune system. Leading to, such wounds convert an ideal environment for a breeding ground for pathogens. Samples were obtained and then weighed, standardized, consecutively diluted, and cultivated on agar media under both aerobic and anaerobic conditions to offer measurable and qualitative data [9]. Tissue biopsies are collected at appropriate times (after the 7^{th}, 14^{th}, 21^{st}, and $\geq 28^{th}$) days for laboratory analysis of fungal contaminations [10] Figure 6.

$$\text{CFUs} \times \log \text{reciprocal} \times 2 = \frac{\text{Colony count}}{\text{Tissue weight (g)}}$$

As shown in Figure 6, the most frequently isolated pathogens in microbiological culture results obtained from burn wounds were Escherichia coli (30 cases) and Staphylococcus aureus (26 cases). Fungal growth was detected in a smaller number of cases (10 cases), whereas no viral pathogens were identified in any of the cases. These findings indicate that burn wounds are an important source of infection, particularly with regard to Gram-negative and Gram-positive bacteria, and that fungi may play a secondary role.

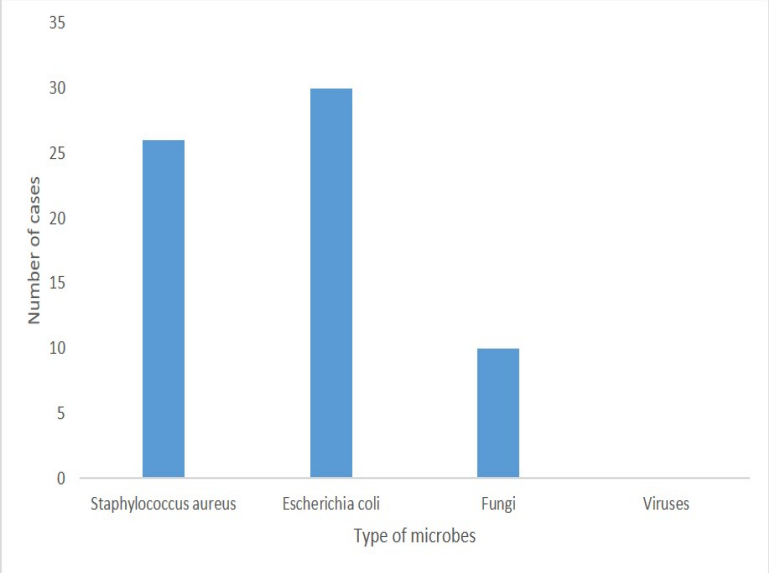

Figure 6: Burn injury infection microbes, showing dominance of E.coli, Staphylococcus aureus species, and fungus. Without a diagnosis of viruses

4. Discussion

Numerous experimental and human investigations have revealed that microcirculatory modifications are recurrent in harshly. Severely burned patients with poor systemic health, mostly in the skin [11]. The existing understanding of burn injuries involve 3 regions of damage: region of clotting, stasis, and hyperemia [12].The zone of coagulation characterizes tissue that was demolished at once of injury. This is enclosed by a region of stasis, with swelling, redness, and rest signs of inflammation within low levels of perfusion. Regularly the zone of stasis will develop and turn to necrotic within the early 48 hours next thermal injury. This study has investigated recovery burn injury by measuring necrotic areas with the same TBSA for two groups Figure 1, showing developments in necrotic areas for the elderly group while reduced necrotic areas for the young group. The range of tissue necrosis distended could be the inhibitory influence in the wound healing procedure, leading to tissue progressive wound delay of healing [13]. The development of primary necrosis can increase through heat injury and cell ischemia. These events can ultimately result in a disruption of ion homeostasis, swelling, disruption of the cellular membranes, and release of detrimental lysosomal enzymes and inflammatory factors [14]. All systemic responses to heat injury dramatically show extensively in elderly burn patients compared to the youngest and that agreed with this study's finding with other researchers that shows elderly burn patients increase hospital in patient mortality rate compared to youngest groups with the same TBSA [15–17]. Table 1 shows an increase in pro-inflammatory biomarkers levels in both second and third-burn degree and septic patients, this finding explains cellular mechanisms during heat damage that will predict and improve systemic treatment. This finding agreed with many researchers, showing the tendency of acidosis in the metabolism was inadequate in harshness, and the promotion of pro-inflammantory cytokines like TNF-α and IL- 6 was increased considerably [18]. TNF-α also activated the anti-microbial by stimulation of neutrophils Table 2, other blood parameters as well as, has the ability to prompt the excretion of additional pro-inflammatory cytokines that agreed to other researchers [19, 20]. There was a remarkable rise the carnitine palmitoyle transferase (CPT serum) of sepsis in burnt individuals Figure 3. It is a marker for systemic infection and sepsis in burn patients [1]. CPT proteins analyse can be a supportive assistant to clinical judgement of sepsis and potential as a method for reducing antibiotic exposure in the critically ill patient. The complex cellular mechanisms of burn injury are gradually coming to light, and this could be a helpful technique in the future to combat the systemic effects of thermal injury. However, understanding these biological pathways and the numerous research that have followed have frequently not resulted in better clinical care for burn patients.

5. Conclusion

The broad understanding of burn injury has improved during the past few years. The complex mechanisms involved in the system's response to burning are now being studied scientifically. By examining distinct course patterns and their connections to patient outcomes, our work advances knowledge of blood components in varying burn severity and sepsis in Iraqi patients. Additionally, it highlights the predictive value of several biomarker types within this exact clinical context. conversion of findings from simple research publications into improved therapeutic treatments for burn patients.

Article Information

Disclaimer (Artificial Intelligence): The author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.), and text-to-image generators have been used during writing or editing of manuscripts.

Competing Interests: Authors have declared that no competing interests exist.

References

[1] M. Barati, F. Alinejad, M. A. Bahar, M. S. Tabrisi, A. R. Shamshiri, N. Bodouhi, and H. Karimi. Comparison of WBC, ESR, CRP and PCT serum levels in septic and non-septic burn cases. *Burns*, 34(6):770–4, 2008. doi: 10.1016/j.burns.2008.01.014.

[2] M. G. Dehne, A. Sablotzki, A. Hoffmann, J. Mühling, F. E. Dietrich, and G. Hempelmann. Alterations of acute phase reaction and cytokine production in patients following severe burn injury. *Burns*, 28(6):535–42, 2002. doi:10.1016/S0305-4179(02)00050-5.

[3] A. M. I. Watts, M. P. H. Tyler, M. E. Perry, A. H. N. Roberts, and D. A. McGrouther. Burn depth and its histological measurement. *Burns*, 27(2):154–60, 2001. doi:10.1016/S0305-4179(00)00079-6.

[4] A. Shpichka, D. Butnaru, E. A. Bezrukov, R. B. Sukhanov, A. Atala, V. Burdukovskii, Y. Zhang, and P. Timashev. Skin tissue regeneration for burn injury. *Stem Cell Res Ther*, 10(1):94, 2019. doi:10.1186/s13287-019-1203-3.

[5] T. Schaefer and K. Szymanski. *StatPearls*. StatPearls Publishing, 2021.

[6] S. Hettiaratchy and P. Dziewulski. Pathophysiology and types of burns. *BMJ*, 328(7453):1427–9, 2004. doi:10.1136/bmj.328.7453.1427.

[7] T. Lund and R. K. Reed. Acute hemodynamic effects of thermal skin injury in the rat. *Circ Shock*, 20(2):105–14, 1986. URL http://www.ncbi.nlm.nih.gov/pubmed/3779904.

[8] S. Mohapatra, A. Gupta, K. Agrawal, H. Choudhry, and M. Deb. Bacteriological profiles in burn patients within first twenty-four hours of injury. *Int J Med Microbiol Trop Dis*, 2(2):71, 2016. doi:10.5958/2455-6807.2016.00008.8.

[9] N. Pallua, P. Fuchs, B. Hafemann, U. Völpel, M. Noah, and R. Lütticken. A new technique for quantitative bacterial assessment on burn wounds by modified dermabrasion. *J Hosp Infect*, 42(4):329–37, 1999. doi:10.1053/jhin.1998.0594.

[10] M. Capoor, S. Sarabahi, V. Tiwari, and R. Narayanan. Fungal infections in burns: Diagnosis and management. *Indian J Plast Surg*, 43 (3):37, 2010. doi:10.4103/0970-0358.70718.

[11] T. O. Meier, M. Guggenheim, S. T. Vetter, M. Husmann, S. R. Haile, and B. R. Amann-Vesti. Microvascular regeneration in meshed skin transplants after severe burns. *Burns*, 37(6):1010–4, 2011. doi:10.1016/j.burns.2011.01.001.

[12] A. Hussain and K. W. Dunn. Predicting length of stay in thermal burns: A systematic review of prognostic factors. *Burns*, 39(7): 1331–40, 2013. doi:10.1016/j.burns.2013.04.026.

[13] S. Lu, J. Xiang, C. Qing, S. Jin, Z. Liao, and J. Shi. Effect of necrotic tissue on progressive injury in deep partial thickness burn wounds. *Chin Med J (Engl)*, 115(3):323–5, 2002. URL http://www.ncbi.nlm.nih.gov/pubmed/11940355.

[14] M. Henriquez, R. Armisen, A. Stutzin, and A. Quest. Cell Death by Necrosis, a Regulated Way to Go. *Curr Mol Med*, 8(3):187–206, 2008. doi:10.2174/156652408784221289.

[15] R. W. Griffiths and J. E. Laing. A burn formula in clinical practice. *Ann R Coll Surg Engl*, 63(1):50–3, 1981. URL http://www.ncbi.nlm.nih.gov/pubmed/7247262.

[16] E. J. Kovacs. Aging, traumatic injury, and estrogen treatment. *Exp Gerontol*, 40(7):549–55, 2005. doi:10.1016/j.exger.2005.04.009.

[17] D. B. Lombard, K. F. Chua, R. Mostoslavsky, S. Franco, M. Gostissa, and F. W. Alt. DNA Repair, Genome Stability, and Aging. *Cell*, 120(4):497–512, 2005. doi:10.1016/j.cell.2005.01.028.

[18] F. Ravat, J. Payre, P. Peslages, M. Fontaine, and N. Sens. [Burn: An inflammatory process]. *Pathol Biol (Paris)*, 59(3):e63–72, 2011. doi:10.1016/j.patbio.2009.12.001.

[19] D. Agay, M. Andriollo-Sanchez, R. Claeyssen, L. Touvard, J. Denis, A.-M. Roussel, and Chancerelle Y. Interleukin-6. TNF-alpha and interleukin-1 beta levels in blood and tissue in severely burned rats. *Eur Cytokine Netw*, 19(1):1–7, 2008. doi:10.1684/ecn.2008.0113.

[20] M. Barati, F. Alinejad, M. A. Bahar, M. S. Z. Tabrisi, N. Bodohi, and H. Karimi. Procalcitonin, C-reactive Protein ESR and WBC Count: Marker of Sepsis in Burn Patients. *Int J Infect Dis*, 12:e461, 2008. doi:10.1016/j.ijid.2008.05.1309.