Original Article

Medical Investigation and Clinical Changes in the Early Phases of Post Burn Adult Inpatients as Indicators for Prompt Infection Diagnosis, in the Hospital for Burn and Plastic Surgery, Sulaimanyah, Iraq

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ABSTRACT

Background: Burn injury is one of the leading causes of morbidity and mortality worldwide. Burn related hospital acquired infections is one of the major problems in the management of these injuries.

Aim of the study: To elaborate on the early laboratory and microbial culture changes in adult burn inpatients, and to make use of subtle changes in the laboratory investigations, to predict and detect infections in burn victims, in its early phases.

Patients and Methods: A Clinical observational study included a random sample of one hundred adult burn inpatients from Sulaymaniyah Burn Hospital, admitted from April 2019 to January 2021, retrospectively and prospectively followed up during the course of the study.

Results: thirty one (31%) patients were male and 69% were female, all patients were adults and aged ≥18 years. 51 % of them had mix II-III degree burns, while 38% second and 11% third degree burns. Hematocrit decreased, total WBC count increased while platelet in most of the patients was normal. Blood urea was increased, Serum creatinine levels were normal in most of the patient in first three weeks of their admission. Eleven patients (11%) died. Whereas 36% of the patients who died had Total Body Surface Area burn of more than 60%, while 72% of them had II and III degree burns. Also 90% of the patients had infections, majority of the isolate results were Gram-negative bacteria (66.8%), and (33.2%) were Gram-positive. Pseudomonas aeruginosa (32.5%) was the most common gram negative. The Staphylococcus aureus (33.2%) was the gram positive organism recovered. While 66.6% of patients had fever during the first week, 55.5% of patients had fever in second week. Most of the patients, who had fever, were infected with S.aureus and Pseudomonas aeruginosa throughout the second week post burn.

Conclusion and recommendations; Many laboratories and microbial parameter changes occur in the early phases of post burning, of which Leukocytosis can be a good predictor for infection in burns, especially when it happens in the second week and beyond. Yet it cannot distinguish gram negative from gram positive bacterial infections. To detect any change in the back ground laboratory parameters, as soon as they happen, infections in burns can be detected or suspected.

Keywords burn, investigations, Bacterial infection , Septicemia, Adults

Introduction

The skin is the largest organ of the body, it functions as a sensory organ and protects against the invasion of foreign bodies and organisms.[1,2] It's burn is one of the most severe form of trauma that has affected mankind since the beginning of time. The development of since and medicine has led to more sufficient treatment and decreased mortality rate.[3] Thermal burns are the most common type of burn, composing about 86% of the burned patients that require hospitalization.[4-6]

Electrical, chemical, and sun are lesser but important caused of burns in human with variable depth of injury accordingly.[7-12]

Estimation of burn parts of the body may not be done accurately even by expert physicians making degree and severity calculations adressed by many formulations and categorization authorities.[13-19] Many studies conducted to elaborate on the clinical and laboratory changes happening in burn victims so that care givers predict infections and morbidities in as an early phases as possible and to tackle them as efficiently as possible.[20-24]

Inhalation is a major cause of death in the first 48 hours especially in older ages.[25-27]Several studies showed that size of burn has major effect on the mortality rate following burn injury at any age, the higher the TBSA% the higher risk of death.[28,29] In severe burn injuries the function and structure of almost every organ is impaired.[30-32]Critically burn patients are more likely to get infection. Bloodstream infections in the first week is called early BSI, and called late BSI during the second week and oncoming weeks.[33-39]

Patients and Methods

The study involved a random sample of 100 adult burn patients in Sulaimaniyah burn Hospital.

The data were obtained from hospital records from April 2019 to January 2021. Laboratory parameter changes were chronologically monitored from the first week of admission through the end of the third week post admission. The laboratory parameters were statistically analyzed by the statistical package for social sciences (SPSS V. 23) against back ground microbiological and clinical follow up charts so as to correlate changes in the parameters and clinical findings with possible infections. P value of less than 0.05 was considered statistically significant.

Descriptive analysis was used to explore frequency and percentage of the tables.

Results

Thirty one patients were male and 69 (69%) were female. The study showed that 41.8% of patient˅s ages were (18-28) years old, and 7% aged more than 48 years old. Most of them were married and lived in rural areas, (table 1 and 2).

Table 1: Demographic Data of Burn Patient enrolled in the study.

Variable		Frequency	Percent
Age range	18-28	46	46
	28-38	30	30
	38-48	14	14
	>48	7	7
	Total	97	97

Missing*	System	3	3
Total		100	100
Gender	Male	31	31
	Female	69	69
	Total	100	100
Total		100	100
Residency	City	44	44
	Rural	53	53
	Total	97	97
Missing*	System	3	3
Total		100	100
Marital status	Single	28	28
	Married	69	69
	Total	97	97
Missing*	System	3	3
Total		100	100

(* not recorded in the case sheet)

Degree and total body surface area

Our study showed that 25% of the patient had a TBSA of 40-50%, and 51 % of them had mixed II-III degree burns, 38% were second degree, and 11% were third degree burns.

Table 2: Total body surface area of burn.

Total body surface area	Frequency	Percentage
< 20%	17	17%
20-30%	23	23%
30-40%	21	21%
40-50%	25	25%
50-60%	6	6%
>60%	8	8%

Table 3: Degree of burn.

Degree	Frequency	Percentage
II	38	38%
III	11	11%
II-III	51	51%

Effect of burn on HCT, Total WBC and PLT during the first three weeks (fig 1-3)

Hematocrit level decreased in the admitted burn patients in the first three week, especially in the second and third week.
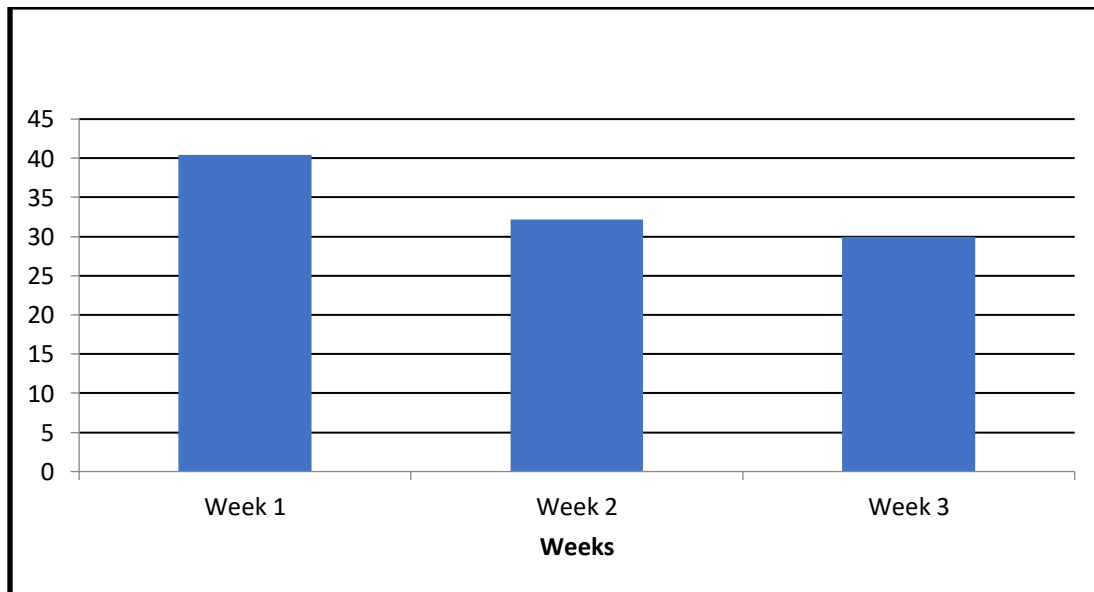

Figure 1: HCT value during first three weeks.

Total WBC counts were increased in the burn patients in all three weeks, a statistically not significant finding (p value more than 0.05).
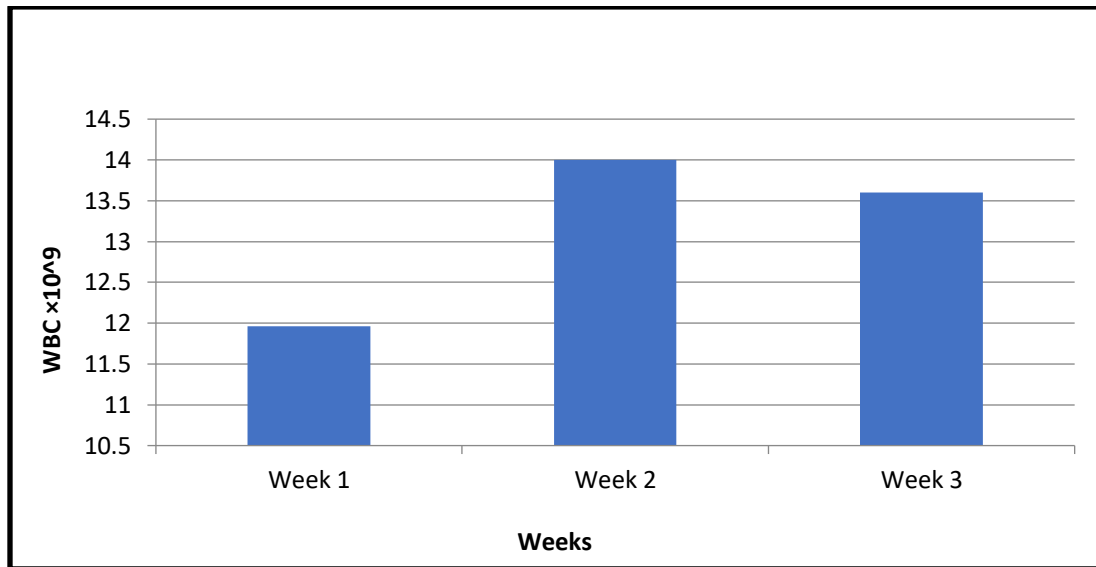
Figure 2: Total WBC count during first three weeks.

Platelet count levels decreased in the first week then increased in second and third week, but overall stayed in the normal range (fig 3).
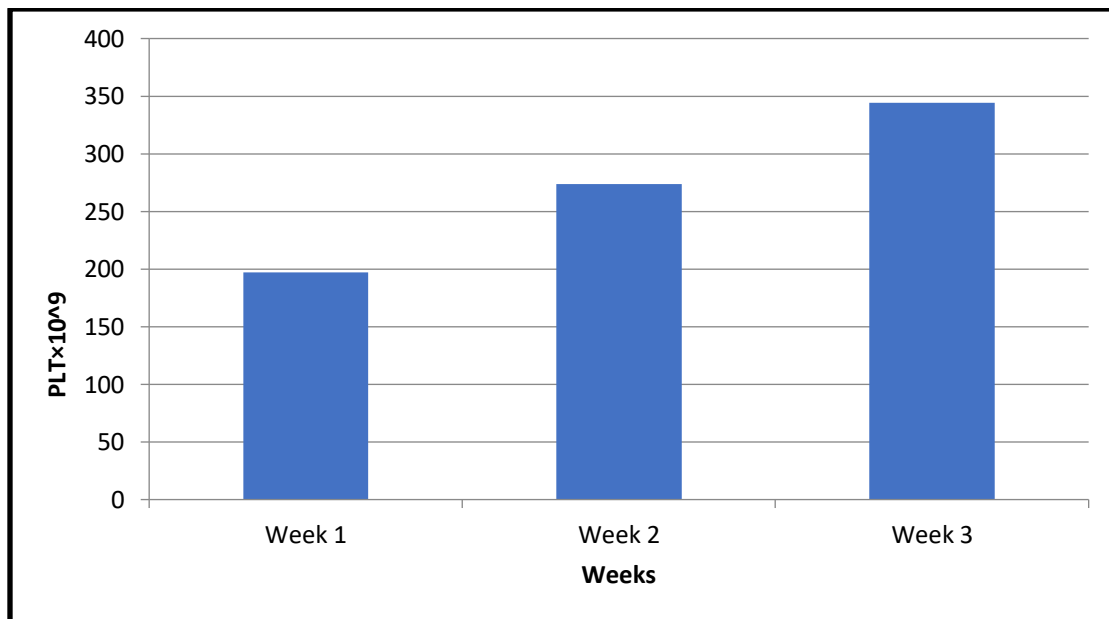

Figure 3: PLT count levels in first three weeks

Effect of burn on kidney function tests

Our study showed that blood urea levels were increased in burn patients. The levels were decreasing gradually throughout the three weeks (fig. 4).
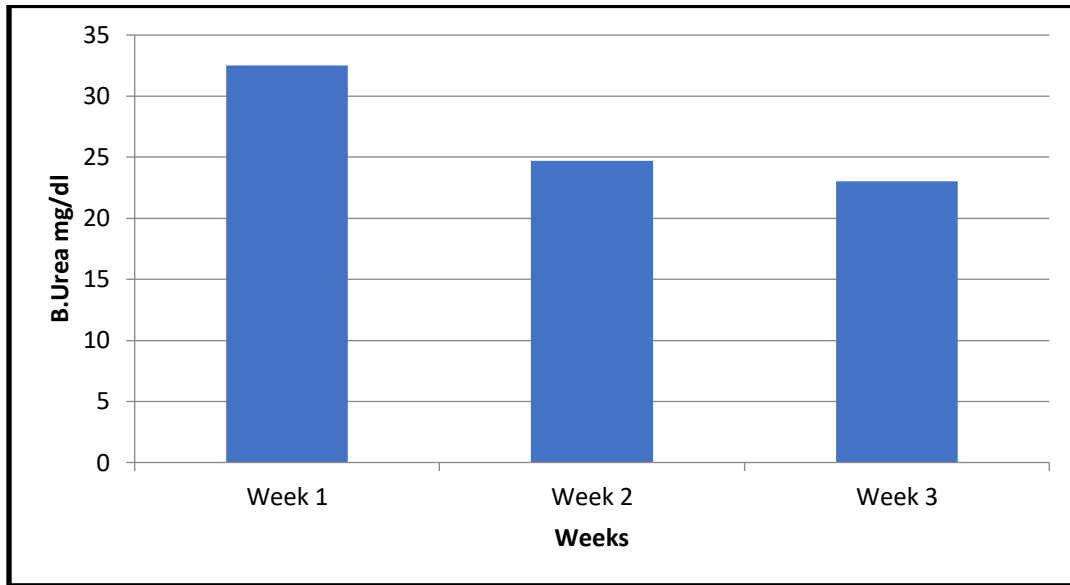
Figure 4: Blood Urea level during first three weeks.

Serum creatinine level was normal in most of the patients, and it stayed normal in all three weeks (fig. 5).
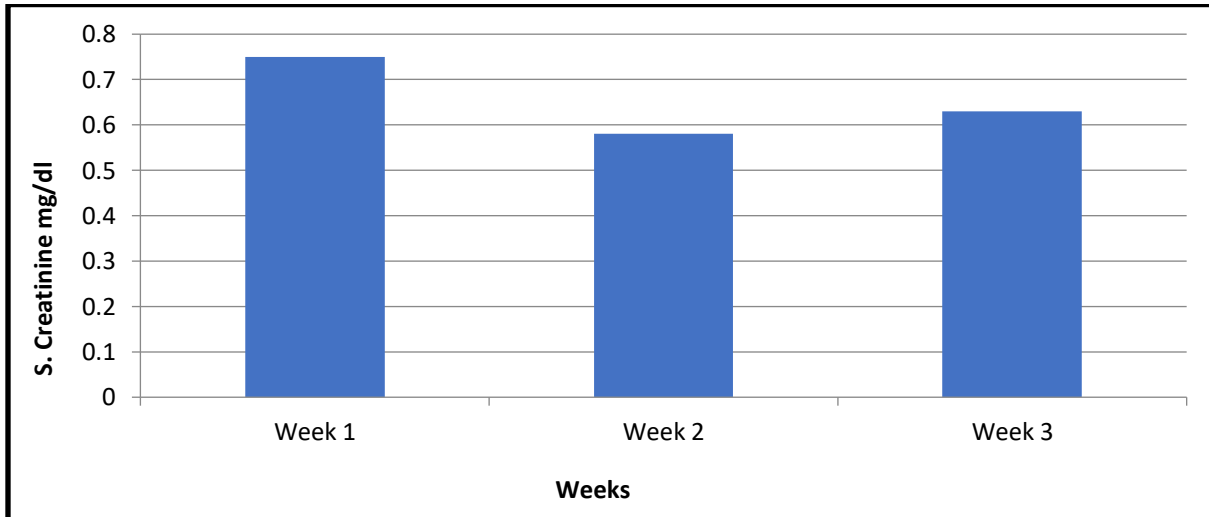

Figure 5: Serum creatinine level during first three weeks.

Effect of burn on total serum protein and serum albumin

Our results showed that levels of total serum protein are decreased in burn patients in all three weeks (fig. 6). The same finding is a statistically significant result in the non survivors.
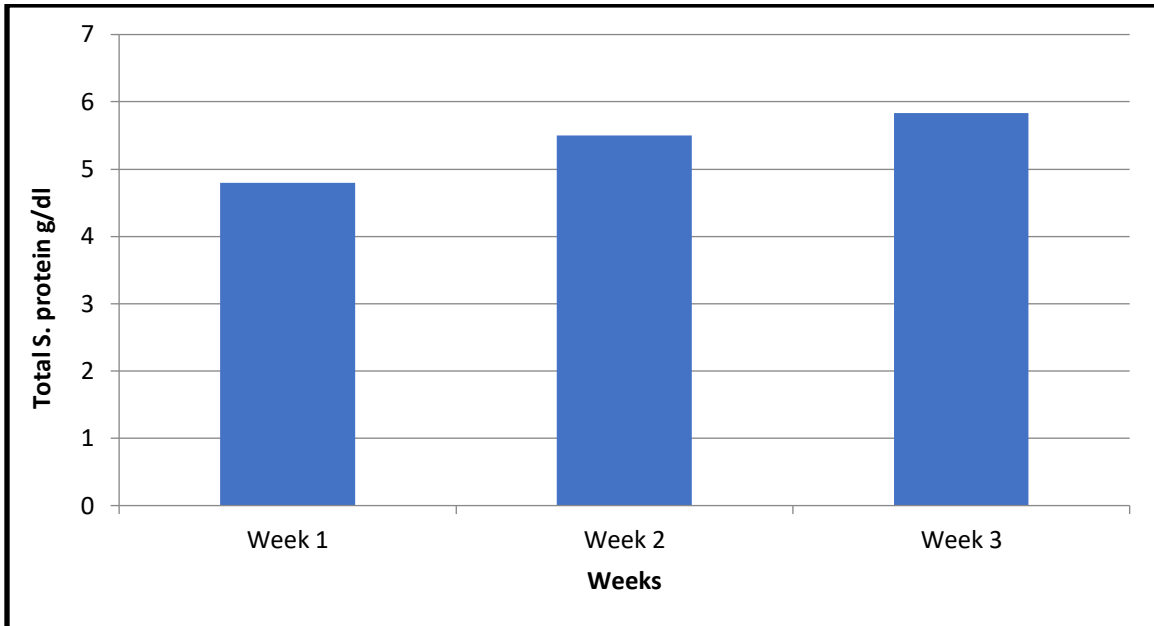
Figure 6: Total serum protein levels during three weeks.

Our results showed that levels of serum albumin are decreased in all three weeks. The same finding was statistically not significant in the deceased patients (P value more than 0.05).
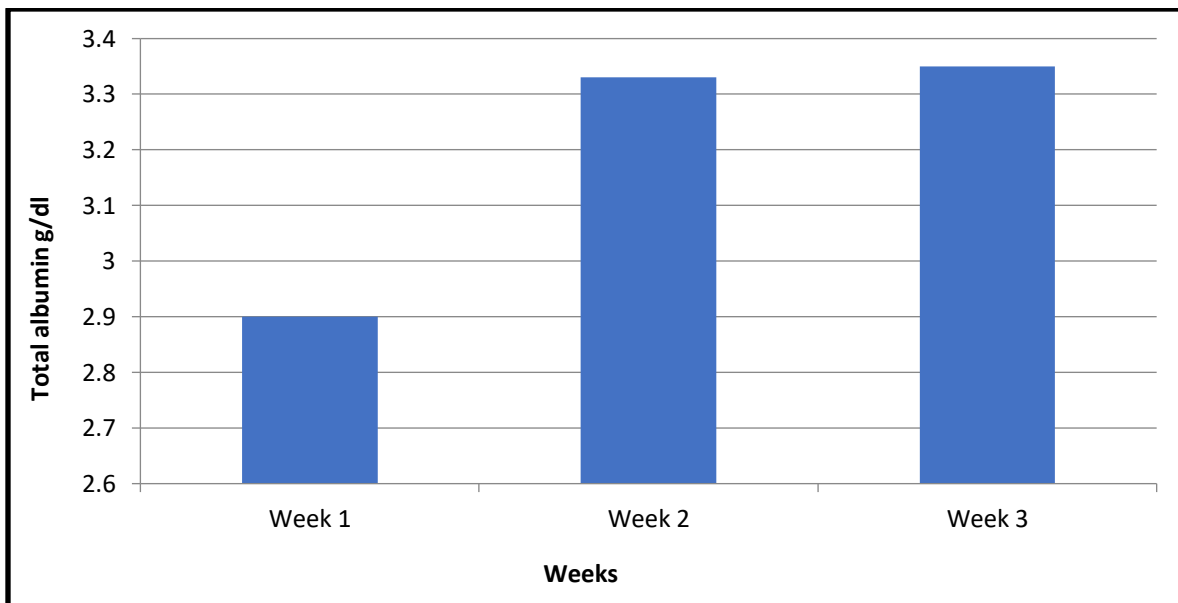
Figure 7: Serum albumin levels during three weeks.

Relation between Total WBC count changes and Total body surface area (fig. 8-10)

To detect the relation, we collected the result of total WBC during the three weeks and compared each week to the total body surface area, change in the Total WBC count has occurred, but it has no relation with total body surface area, $(P > 0.05)$ in other words it is not statistically significant.

Figure 8: Total body surface area and total WBC count change in the first week.

Figure 9: Total body surface area and total WBC count change in the second week.

Figure10: Total body surface area and total WBC count change in the third week.

Common pathogen and bacterial infection in burn patients (table 4)
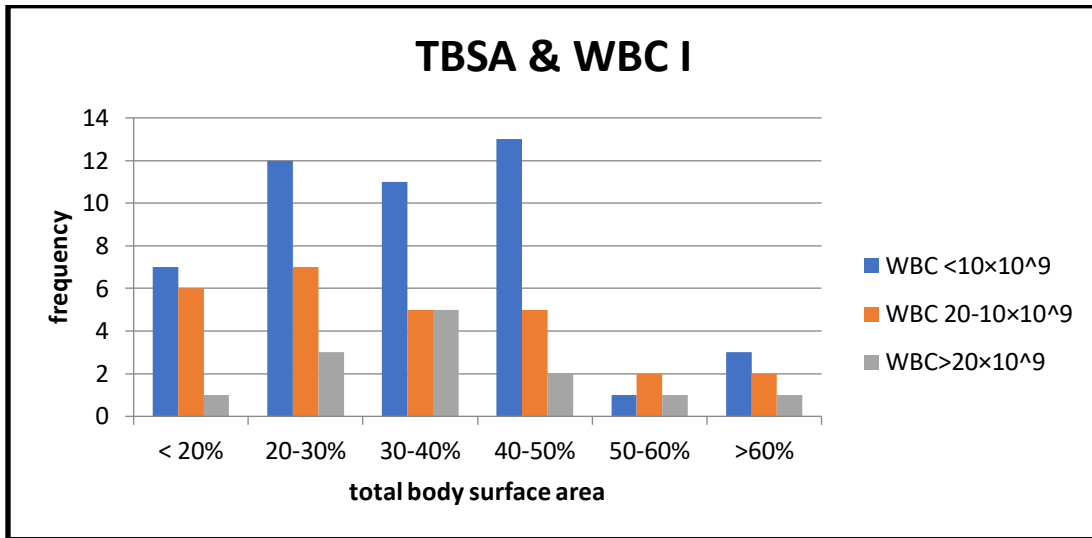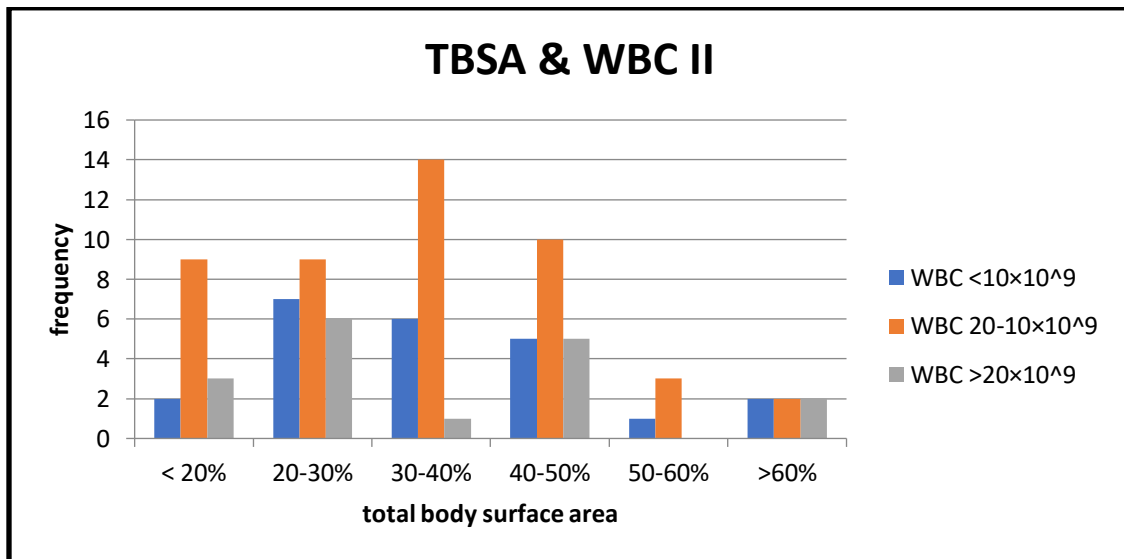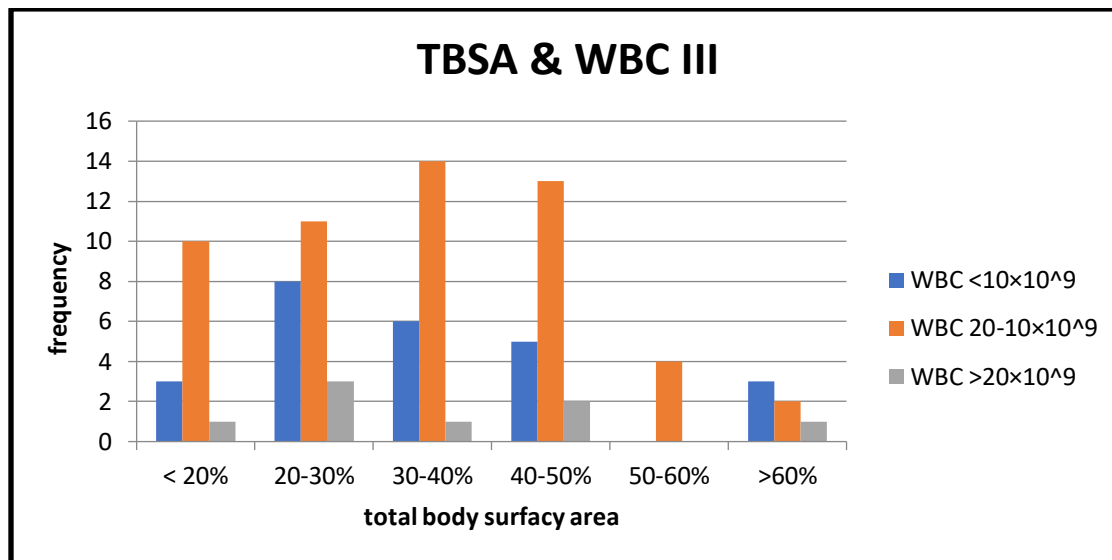
90% of the patients had infections. A total of 14 different types of bacteria were isolated. A majority of the results were Gram-negative bacteria (66.8%), and (33.2%) were Gram-posative. Pseudomonas aeruginosa (32.5%), and Klebsiella pneumonia (11%) and Acinetobacter baumannii (9.5%) were the most common gram negative organisms, and Staphylococcus aureus (33.2%) was the only gram positive organism recovered.

Table 4: Types of bacteria.

Type of bacteria	Frequency	Percentage
Acinetobacter baumannii	16	9.5
SS. aureus	57	33.2
Enterobacter cloacae	9	5.2
Escherichia Coli	6	3.4
Klebsiella pneumoniae	19	11
Pseudomonas aeruginosa	56	32.5
Raultella (Kleb) terrigene	1	0.5
Burkholderia (Pseudo)	1	0.5
Cepacia (CDCEO-1 gene I)	1	0.5
Enterobacter aeruginosa	1	0.5
Serratitaliquefaciens	1	0.5
Pseudomonas luteola	1	0.5
Klebsiella oxytoca	1	0.5
Enterobacter skazakii	1	0.5

Types of the bacteria that cause fever in burn patients (table 5-7)

The result show that 66.6% of patients had fever during the first week, 55.5% of them had fever in second week.

Table 5: Fever of the infected patients.

Week	First week	Second week
Fever	66.6%	55.5%
Not fever	12.2%	22.2%
Data missing	21.1%	22.2%

Table 6. Types of the bacteria that cause fever in first week.

First week		P value 0.043
Type of Bacteria	**Fever**	
Acinetobacter baumannii (MDRO)	4.5%	
S. aureus (MDRO)	1.1%	
Acinobacterbaumannii	9%	
Escherichia Coli	1.1%	
Klebsiella pneumoniae	9%	
Klebsiella pneumoniae (MDRO)	1.1%	
Pseudomonas aeruginosa	17%	
Pseudomonas aeruginosa (MDRO)	13.6%	
SS. aureus	13.6%	
S.aureus (MRSA)	20%	
Burkholderia (Pseudo)	1.1%	
Cepacia (CDCEO-1 gene I)	1.1%	
Enterobacter cloacae (ESBL)	2.3 %	
Enterobacter cloacae	1.1%	
Raultella (Kleb) terrigene	1.1%	
Enterobacter skazakii (MDRO)	1.1%	
Klebsiella oxytoca	1.1%	

Table 7: Types of bacteria that cause fever in second week.

P value 0.089	Second week	
	Fever	**Types of Bacteria**
	2.7 %	Acinetobacter baumannii (MDRO)
	2.7 %	S. aureus (MDRO)
	2.7 %	Acinobacterbaumannii
	2.7 %	Escherichia Coli (ESBL)
	13.5 %	Klebsiella pneumoniae
	24.3 %	Pseudomonas aeruginosa
	18.9 %	Pseudomonas aeruginosa (MDRO)
	5.4 %	S.S. aureus
	27 %	S.aureus (MRSA)
	2.7 %	Serratitaliquefaciens

Table 8: Leukocytosis in the first week.

Leukocytosis in first week		
Types of bacteria	**Frequency**	**Percentage**
SS.aureus	23	32.3
Pseudomonas aeruginosa	29	40
Klebsiella pneumoniae	7	10
Acinobacterbaumannii	3	4
Enterobacter cloacae	5	7
Escherichia Coli	4	6

Leukocytosis in burn patients who had infection

Our study showed that most of the patients who had leukocytosis were infected with pseudomonas aeruginosa and staphylococcus aureus.

Table 9: Leukocytosis in the second week.

Leukocytosis in second week		
ypes of bacteria	Frequency	Percentage
SS.aureus	33	35
Pseudomonas aeruginosa	35	37
Klebsiella pneumoniae	10	10.6
Acinobacterbaumannii	6	6.3
Enterobacter cloacae	6	6.3
Escherichia Coli	4	4.3

Table 10: Leukocytosis in the third week.

Leukocytosis in third week		
Types of bacteria	Frequency	Percentage
SS.aureus	30	34.4
Pseudomonas aeruginosa	32	36.7
Klebsiella pneumoniae	10	11.5
Acinobacterbaumannii	9	10.3
Enterobacter cloacae	3	3.44
Escherichia Coli	3	3.44

DISCUSSION

Burn injuries are a significant cause of both mortality and morbidity, leaving the victims with lifelong physical, psychological and emotional disability.

In our study female to male ratio of the burn patients was nearly(2:1), this result correlates with a study undertaken at the Al-Fayhaa Burn Centre in Basra city,[40] and it is contrast to demographic research done in Karachi that reported the ratio of the burn patients was (1:1),[41] and to another study that was conducted in Alabama University in USA,[42] that reported (76.3%) of the patients were male and 382 (23.7%) were female.

The reasons why females have higher risk of burn in our study may be due to their association with cooking, using unsafe cooking stove which can ignite loose clothing, and self-inflicted or interpersonal violence are also factors.

Our study showed that 41.8% of patient ̓s ages were (18-28) years old, and 7% aged more than 48 years old , a study by Gupta et al reported that the majority (79%) of the cases were in the 15-45 ages group,[43] another study in Karachi reported that (58.2%) of the cases were 16-30 years followed by those aged between 31-45 age group.[41]Therefore we can say that people aged between 16-45 years are more likely to suffer from burn injuries than any other age group.

The results showed that 25% of the patient had a TBSA of 40-50% and 51% of them had II-III degree burns.In most cases (28.7%), total body surface area burned was 16-30%, mean percentage of total body surface area burned was 35.49 ± 27.276%, which is consistent with a study by Lari et al., that reported mean body surface area burned to be 30.6%.[44]

In our study Hematocrit level was deceased, especially in the second and third week, that agrees with the study conducted in USA,[20] and another one in Egypt.[22]

In our study Total WBC count was increased in all three weeks, this correlates with the study in Egypt,[22] that reported significant leukocytosis from the first day after burn, also agrees with another study in Iraq,[21] and Korea.[23] WBC level dramatically changes post burn injury, due to physiological trauma

to the body and infection. According to our results there is no statistically significant relation between WBC count and TBSA%, but this result is constant to a studies conducted in Iraq,[21] and Korea.[23]

Platelet count levels showed a decreased level during the first week, and then increased in second and third week, but still stayed at normal range in all three weeks, this result is contrast with a study in Egypt,[22] and a study in Korea.[23]

In our study the results showed that B.urea levels were increased, and Serum creatinine level was normal, a study in Egypt that reported that B urea level was increased which stays aline with our result, but the level of Creatinine was also increased in their study, which is contrast with our result of serum creatinine.[22]

Results of total serum protein and serum albumins were decreased; this correlates with a study conducted in Hilla,[24] and another one in Germany.[45]

According to the results of our total of 100 patients, 11 of them have died, and 89 of them survived, which is a low rate of mortality, this result correlates with a study in Basra City.[40] One of the causes that has effect on increasing mortality rate is TBSA%, according to our resultsthe higher the TBSA%, the higher the mortality rate, this relates with studies conducted in Italy,[28] and USA.[29]

We compared some parameters between the survived and non survived groups to determine which one has effect on mortality rate, according to our results total serum protein showed a statistically significant decrease in non survivors in all three weeks,

this result agrees with a study conducted in India.[46] Although the level of our results on serum albumin showed a decrease in the first week, there was no statistically significant relation between the two groups, which is constant with the results of the same study in India,[46] and another study in Mexico.[47]

Serum creatinine levels showed statistically significant increase in non survivors compared to the survivors in all three weeks. Levels of blood urea were increased in non survivors compared to the survivors in the first and third week, but decreased in the second week, but ultimately there was no statistically significant relation.The levels of WBC count showed a decreased level in the non survivors compared to the survivors in all three weeks, but there was no statistically significant relation according to the p-value.

Out Of 100 burn patients, 90 of them had infection, a total of 14 different types of bacteria were isolated, majority of the results were Gram-negative bacteria (66.8%), and (33.2%) were Gram-posative, which correlates with a study carried out inVienna, Austria.[34]

Pseudomonas aeruginosa (32.5%), and Klebsiella pneumonia (11%) and Acinetobacter baumannii (9.5%) were the most common gram negative organisms, and staphylococcus aureus (33.2%) was the only gram positive organism recovered, this relates with a study in Iran.[35]

Our study show that 66.6% of the infected patients had fever during the first week, 55.5% of them had fever in second week, so according to our results we can predict presence of infection depending on fever in burn patients, this result is contrast with a

study done in USA,[48] that reports we can't depend on presence of fever to determine blood stream infection in severe burn patients.

20% of the patients who had fever infected with S.aureus, and 17% were infected with Pseudomonas aeruginosa in first week, but in the second week 27% infected with S.aureus and 24.3% of them were infected with Pseudomonas aeruginosa.

Our results on WBC count changes during three weeks showed that in the first week most of the patients had normal WBC count, but in the second week 64% of the patients had high WBC count (leukocytosis), and 68% had leucocytosis in the third week.

The number of the patients who had low WBC count (Leukopenia) was very low throughout the whole three week.We can say leucocytosis can be relied on in predicting infection in burn patients, but leucopenia can't, this result does not agree with a study conducted in USA.[48]

Our study showed that most of the patients who had leukocytosis in the first week were infected with pseudomonas aeruginosa (40%) and staphylococcus aureus (32.3). In the second week the P. aeruginosa isolation was (37%) and S. aureus was (35%). In the third week; the Pseudomonas aeruginosa was (36.7%) and S. aureus was (34.4%).

In the first week 6 patients had leucopenia, they were infected with S.aureus, Pseudomonas aeruginosa, Klebsiella pneumonia, Enterobacters kazakii. In the second week we had 3 cases that had leucopenia, they were infected with S.aureus, Enterobacter cloacae, Klebsiella pneumonia.

In the third week we had one patient who had leucopenia, infected by Serratia liquefaciens.

CONCLUSION

Leukocytosis can be a good predictor for infection in burns, especially when it happens in the second week and later. Still it's not the definite parameter distinguishing gram negative from gram positive infections.

Fever can be a prediction for infections, but again it cannot be used as significant finding to differentiate infection caused by gram positive or by negative bacteria. Also fever is mostly useful to be used as a sign of infection from second or 3rd week and later. Total serum protein and serum albumins are not good predictor that there is no or little change in their levels.

Kidney function test showed a statistically significant increase in non survived patients. Platelet count levels decreased during the first week, and then increased in second and third week. a significant decrease in platelet count was observed in patients with moderate and severe burn injuries.

Hematocrit level changes in the second and third weeks, but it cannot be a significant finding to differentiate gram positive from gram negative infection.

RECOMMENDATION

To have basic background parameters for the patients and do regular rechecks to detect any change in the laboratory parameters as soon as they happen.

Infections in burns can be detected or suspected when subtle changes happen in the background laboratory findings.

Better and more organized timely performing laboratory checks, tissue culture or swabs, so that more accurate correlations be found with the previous results.

Hospital staff and management must pay significant attention to data recordings so as to avoid missing important demographical and clinical parameters that are of paramount importance in future referencing and strategy making in patient management.

Conflict of Interests

The authors declared no conflict of interests.

REFERENCES

1. Kalafa EG, Hixona K, Kadakia P, Dunn A, Sell S. 9.1. 1 Skin anatomy and physiology. Electrospun Materials for Tissue Engineering and Biomedical Applications: Research, Design and Commercialization. 2017:179.
2. Wong R, Geyer S, Weninger W, Guimberteau JC, Wong JK. The dynamic anatomy and patterning of skin. Experimental dermatology. 2016;25(2):92-8.
3. Hettiaratchy S, Dziewulski P. ABC of burns: Introduction. BMJ: British Medical Journal. 2004;328(7452):1366.
4. Yesim Akpinar Kara. Burn Etiology and Pathogenesis. intechopen. May 23, 2018.
5. Phillip L Rice J, Dennis P Orgill. Assessment and classification of burn injury. uptodate. Jun 17, 2019.
6. Cobb Cynthia. Chemical Burns. Healthline. December 17, 2018.
7. Chwistek Mrcin. Electrical Burns and Injuries. Winchester Hospital. 2018.
8. Krisna Ananda. Electrical burn. DermNet NZ. September, 2015.
9. Trauma & Emergency Medicine. Types of Burns: Thermal, Electrical, and More. UPMC HealthBeat. September 1, 2017.
10. Laura C. Simko, Emily A. Ohrtman, Gretchen J. Carrougher, Nicole S. Gibran. Understanding a Burn Injury. 2018.
11. dreamsite.com. Skin burn. four degrees of burns. dreamsitecom.
12. Thom D. Appraising current methods for preclinical calculation of burn size–A pre-hospital perspective. Burns. 2017;43(1):127-36.
13. Hettiaratchy S, Papini R. Initial management of a major burn: II—assessment and resuscitation. Bmj. 2004;329(7457):101-3.
14. Moore RA, Waheed A, Burns B. Rule of Nines. StatPearls [Internet]: StatPearls Publishing; 2019.
15. Wachtel TL, Berry CC, Wachtel EE, Frank HA. The inter-rater reliability of estimating the size of burns from various burn area chart drawings. Burns. 2000;26(2):156-70.
16. SILLETT J. Remember, Remember... Burns and Scalds. east midlands emergency medicine educational media.
17. Hossein Mortazavi AK, Husain Ali Khan and Shahrokh C. Bagheri. Head and Neck Burn Injury. pocketdentisrtycom.
18. Garcia-Espinoza JA, Aguilar-Aragon VB, Ortiz-Villalobos EH, Garcia-Manzano RA , Antonio BA. Burns: Definition, Classification, Pathophysiology and Initial Approach. Genmed. September 22,2017.
19. Katie Frantz CGB. Thermal Injury. vetfoliocom. Mar 8, 2019.
20. Sen S, Hsei L, Tran N, Romanowski K, Palmieri T, Greenhalgh D, et al. Early clinical complete blood count changes in severe burn injuries. Burns. 2019;45(1):97-102.
21. Jasem MA, Mahmood AE, AL-Ubadi AE, AL-Bahadli A. SERUM PROCALCITONIN CONCENTRATION VS. TOTAL AND DIFFERENTIAL WHITE BLOOD CELL COUNT IN IRAQI BURN INJURY PATIENTS. European Journal of Biomedical. 2017;4(01):63-8.
22. Saleh SM. The effect of moderate and severe burn injuries on human liver, kidney & blood (Biochemical study). Zagazig Journal of Forensic Medicine. 2018;16(1):91-102.
23. Kim HS, Kwon HW, Yang HT, Chun W, Shin KS, Lee YK, et al. A Serial Study of Hematologic Change in Burned Patients. J Lab Med Qual Assur. 2011;33(1):9.
24. Al-Muhammadi MO, Azeez HA. Some physiological changes in burn patients. Medical Journal of Babylon. 2011;8(3):303-19.
25. Brusselaers N, Monstrey S, Vogelaers D, Hoste E, Blot S. Severe burn injury in Europe: a systematic review of the incidence, etiology, morbidity, and mortality. Critical care. 2010;14(5):R188.
26. Lam N, Duc N, Son N. Outcome and risk factors for death of elderly burn patients: a case series in Vietnam. Annals of burns and fire disasters. 2019;32(2):87.
27. Le HQ, Zamboni W, Eriksson E, Baldwin J. Burns in patients under 2 and over 70 years of age. Annals of plastic surgery. 1986;17(1):39-44.
28. Pavoni V, Gianesello L, Paparella L, Buoninsegni LT, Barboni E. Outcome predictors and quality of life of severe burn patients admitted to intensive care unit.

Scandinavian journal of trauma, resuscitation and emergency medicine. 2010;18(1):24.

29. Jeschke MG, Pinto R, Kraft R, Nathens AB, Finnerty CC, Gamelli RL, et al. Morbidity and survival probability in burn patients in modern burn care. Critical care medicine. 2015;43(4):808.

30. Kraft R, Herndon DN, Finnerty CC, Shahrokhi S, Jeschke MG. Occurrence of multi-organ dysfunction in pediatric burn patients-Incidence and clinical outcome. Annals of surgery. 2014;259(2):381.

31. Kallinen O, Maisniemi K, Böhling T, Tukiainen E, Koljonen V. Multiple organ failure as a cause of death in patients with severe burns. Journal of burn care & research. 2012;33(2):206-11.

32. Aikawa N, Shinozawa Y, Ishibiki K, Abe O, Yamamoto S, Motegi M, et al. Clinical analysis of multiple organ failure in burned patients. Burns. 1987;13(2):103-9.

33. Raz-Pasteur A, Hussein K, Finkelstein R, Ullmann Y, Egozi D. Blood stream infections (BSI) in severe burn patients—early and late BSI: a 9-year study. Burns. 2013;39(4):636-42.

34. Fochtmann-Frana A, Freystätter C, Vorstandlechner V, Barth A, Bolliger M, Presterl E, et al. Incidence of risk factors for bloodstream infections in patients with major burns receiving intensive care: A retrospective single-center cohort study. Burns. 2018;44(4):784-92.

35. Rezaei E, Safari H, Naderinasab M, Aliakbarian H. Common pathogens in burn wound and changes in their drug sensitivity. Burns. 2011;37(5):805-7.

36. Forson O, Ayanka E, Olu-Taiwo M, Pappoe-Ashong P, Ayeh-Kumi P. Bacterial infections in burn wound patients at a tertiary teaching hospital in Accra, Ghana. Annals of burns and fire disasters. 2017;30(2):116.

37. Rao SR, Lakshmi LJ, Pavani S, Kawle V, Prakash SJ. Bacteriological profile, Antibiogram of burn wound isolates and detection of MRSA and ESBL production at tertiary care hospital, Hyderabad. World Journal of Pharmacy and Pharmaceutical Sci. 2014;3(10):1691-8.

38. Papini R. Management of burn injuries of various depths. Bmj. 2004;329(7458):158-60.

39. Williams C. Successful assessment and management of burn injuries. Nursing Standard. 2009;23(32):53-9.

40. Al-Shamsi M, Othman N. The epidemiology of burns in Basra, Iraq. Annals of burns and fire disasters. 2017;30(3):167.

41. Ali S, Hamiz-ul-Fawwad S, Al-Ibran E, Ahmed G, Saleem A, Mustafa D, et al. Clinical and demographic features of burn injuries in Karachi: a six-year experience at the burns Centre, civil hospital, Karachi. Annals of burns and fire disasters. 2016;29(1):4.

42. McGwin Jr G, George RL, Cross JM, Reiff DA, Chaudry IH, Rue III LW. Gender differences in mortality following burn injury. Shock. 2002;18(4):311-5.

43. Gupta AK, Uppal S, Garg R, Gupta A, Pal R. A clinico-epidemiologic study of 892 patients with burn injuries at a tertiary care hospital in Punjab, India. Journal of emergencies, trauma and shock. 2011;4(1):7.

44. Lari AR, Alaghehbandan R, Nikui R. Epidemiological study of 3341 burns patients during three years in Tehran, Iran. Burns. 2000;26(1):49-53.

45. Lehnhardt M, Jafari HJ, Druecke D, Steinstraesser L, Steinau H, Klatte W, et al. A qualitative and quantitative analysis of protein loss in human burn wounds. Burns. 2005;31(2):159-67.

46. Gupta S, Bhattacharya S, Goyal P. Role of first day levels and subsequent trends of serum proteins in acute burns. Journal of Acute Disease. 2018;7(1):31.

47. Aguayo-Becerra OA, Torres-Garibay C, Macías-Amezcua MD, Fuentes-Orozco C, Chávez-Tostado MdG, Andalón-Dueñas E, et al. Serum albumin level as a risk factor for mortality in burn patients. Clinics. 2013;68(7):940-5.

48. Murray CK, Hoffmaster RM, Schmit DR, Hospenthal DR, Ward JA, Cancio LC, et al. Evaluation of white blood cell count, neutrophil percentage, and elevated temperature as predictors of bloodstream infection in burn patients. Archives of Surgery. 2007;142(7):639-42.