Some Hematological and Biochemical Changes in Burn Patients

A Thesis

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By

Hayder Abdul Hussain Aziz

M.B.Ch.B.

Supervisor

Assist. Prof. Dr. Muhammad Obaid AL- Muhammadi

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بعض التغيرات الدموية والكيميوحيوية لمرضى الحروق

رسالة تقدم بها

حيدر عبد الحسين عزيز

بكالوريوس طب وجراحة عامة

إلى

مجلس كلية الطب / جامعة بابل

وهي جزء من متطلبات نيل درجة ماجستير علوم في علم الفسلجة الطبية

إشراف

أ.م.د. محمد عبيد المحمدي

شعبان1429 هـ أب 2008 م

﴿ يَرْفَعِ اللهُ الَّذِينَ آمَنُوا مِنْكُمْ وَالَّذِينَ أُوتُوا العِلْمَ دَرَجَاتٍ وَاللهُ بِمَا تَعْمَلُونَ خَبِيرٌ ﴾

{المجادلة:11}

Abstract

This study was designed to estimate some hematological and biochemical changes to the burn patients ,since the levels of these substances are very important in letting the health care team to know how the body is responding to the different therapies that being provided and this will help the medical staff for proper management with less morbidity and mortality .

The study lasted from November/2007 to May/2008. There are 125 patients and 100 healthy controls (clinically assessed by specialist doctor) are taken in this study .The patients are classified in 5 groups .The ages of these groups were divided as follows: the first group (1-<3 years) ; the second group (3-<5 years) ; the third group (5-<10 years) ;the fourth group (10 - <18 years);and the fifth group (18 - 58 years old) . Those patients were admitted to the burn unit at Al-Hilla General Teaching Hospital. They were suffering from second to third degree (flame and scald) burn injury .

Concerning the hematological parameters , it is found that red blood cells count (RBCs) ,hemoglobin (Hb) and packed cell volume (PCV) shows significant decrease in comparison with healthy controls. The white blood cells (WBCs) count shows significant increase in comparison with healthy controls . The differential WBCs count shows significant increase in neutrophil percentage and significant decrease in lymphocyte percentage , while monocyte , eosinophil and basophil percentages elicits insignificant differences in comparison with healthy controls . The platelets counts shows insignificant decrease in comparison with healthy controls .

Regarding the biochemical parameters ,it was found that the total serum protein , albumin and globulin of male and female burn

patients shows significant decrease for all age groups except the males of fourth and fifth groups and the females of fifth group which show significant decrease for globulin only . The results of liver enzymes including serum GOT, GPT and alkaline phosphatase , and serum of total, direct and indirect bilirubin of male and female burn patients shows significant increase for all age groups . The values of serum total cholesterol , high density lipoprotein (HDL) , low density lipoprotein (LDL) and triglycerides (TG) of male and female burn patients shows significant decrease for all age groups. The level of random blood sugar (RBS) shows significant increase in comparison with healthy controls .

Regarding the serum electrolytes , serum sodium shows a significant decrease before resuscitation and significant increase within 2 days duration after resuscitation while the serum potassium exhibits significant increase before resuscitation and shows significant decrease within 2 days duration after resuscitation for male and female burn patients of all age groups . Finally , the serum of calcium ,copper and zinc shows significant decrease for males and females of all age groups except the males of first and fourth groups and the females of first and fifth group which show significant decrease for zinc only in comparison with healthy controls .

In view of the changes summarized , the increase or decrease in some hematological and biochemical parameters may be attributed to hypermetabolic state which arises mainly due to increase of adrenaline release ,loss of fluid and electrolytes , hemolysis and sepsis .

List of Abbreviations

ALP	Alkaline phosphatase
ANP	Atrio –natriuretic peptide
apo-AI	Apolipoprotein AI
ATP	Adenosine triphosphate
Ca^{+2}	Calcium ion
C°	Centigrade
CD	Cluster of differentiation
Cu	Cupper
EDTA	Ethylenediamine tetra-Acetic Acid
Fl	Femto liter
GH	Growth hormone
GPT	Glutamic pyruvic transaminase
GOT	Glutamic oxaloacitic transaminase
Hb	Hemoglobin
HDL	High density lipoprotein
IL-1β	Interleukin-1 beta
LDL	Low density lipoprotein
LPS	Lipopolysaccharide
mm	Millimeter
mm^3	Cubic millimeter
m^2	Square meter
Ml	Milliliter
µ	Micro liter
meq/l	Milliequielent per liter
mmol	Millimole
µmol	Micromole
Na^+-K^+ ATPase	Sodium –potassium Adenosine triphosphatase
NADPH	Reduced nicotinamide adenine dinucleotide-phosphate
nm	Nanometer
PCV	Packed cell volume
Pg	pictogram
PMN	Polymorphonuclear leukocytes
RBCs	Red blood cells
SE	Standard error
TLRs	Toll-like receptors

TBSA	Total body surface area
TE	Trace element
TG	Triglyceride
T3	Thyronine
T4	Thyroxin
$TGF\beta_1$	Transforming growth factor beta-1
WBCs	White blood cells
Zn	Zinc

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Hayder ,2008

Certification

I certify that this thesis was prepared under our supervision at the Department of physiology , College of Medicine, University of Babylon as partial fulfillment of the requirements for the Degree of Master of Science in medical physiology .

Ass. Prof. Dr. Muhammad Obaid AL-Muhammadi

College of Medicine/ University of Babylon
Supervisor
/ / 2008

In view of the available recommendation, I present this thesis for evaluation by the Examining Committee.

Ass. Prof. Dr. Abudl Kareem J. AL-Bermany

Head of Dep. of Medical Physiology and Physics

College of Medicine/ University of Babylon

/ / 2008

4.2 Biochemical studies :

4.2.1 Total Serum Protein , Albumin and Globulin :

The values of total serum protein , albumin and globulin within 2 days duration for male and female burn patients in comparison with control are shown in the table 4 .

4.2.1.1 Total serum protein :

The values of total serum protein for males burn`s patients of all age groups are : 44.923 ± 1.559 ; 44.076 ± 0.824 ; 46.846 ± 1.556 ; 45.846 ± 3.606 and 44.769 ± 1.925 g/dl respectively. These results are significantly ($p < 0.01$) decrease in comparison with healthy control . The results of total serum protein for female burns patients for all age groups are : 47.75 ± 1.508 ; 47.83 ± 1.696 ; 46.75 ± 1.388 ; 46.667 ± 2.13 and 52.833 ± 2.633 g/dl respectively . These results also show significant ($p < 0.01$) decrease in comparison with healthy controls .

4.2.1.2 Serum Albumin :

The values of serum albumin for all age groups of male burn patients are : 20.769 ± 1.105 ; 25.133 ± 0.945 ; 22.077 ± 0.977 ; 19 ± 1.41 and 19.308 ± 1.379 g/dl respectively .Regarding the results of female burn patients for all age groups are : 22.75 ± 1.634 ; 21.615 ± 1.206 ; 20.25 ± 1.008 ; 21.5 ± 1.564 and 24.667 ± 1.662 g/dl respectively . These results of both males and females for all age groups show significant ($p < 0.01$) decrease in comparison with healthy controls .

4.2.1.3 Serum Globulin :

The results of Serum globulin of males burns` patients for all age groups are : 24.153 ± 0.9187 ; 22.7 ± 1.566; 24.769 ± 0.865 ; 26.077 ± 0.812 and 24.923 ± 0.644 g/dl respectively and for the females are : 25 ± 0.835 ; 22.462 ± 1.333 ; 26.5 ± 0.744 ; 24.333 ± 0.873 and 26.417 ± 1.227 g/dl respectively . The results of male burns' patients show significant ($p < 0.01$) decrease in all age groups except the fourth and fifth groups which show significant decrease at ($p<0.05$) in comparison with healthy controls . The females also show significant ($p<0.01$) decrease except the fifth group which is only significant at ($p < 0.05$) .

4.2.2 Liver enzymes determination :

In this study , the liver enzymes which are estimated for male and female burn patients within 2 days duration are: serum GOT ;GPT and ALP and their values are illustrated in the table 5 .

4.2.2.1 Serum GOT. :

It is found in this study , the level of serum GOT for male burn patients are : 19.845 ± 1.334 ; 18.083 ± 0.596 ; 19.385 ± 0.712 ; 17.846 ± 0.839 and 18.75 ± 0.87 international unit (I.U.) respectively and for females are : 19.167 ± 1.029 ; 17.231 ± 0.833 ; 18.667 ± 0.838 ; 18.167 ± 0.716 and 24.231 ± 3.643 I.U. respectively .Both of male and female burn patients results are significantly ($p < 0.01$) increase for all age groups in comparison with healthy control .

4.2.2.2 Serum GPT :

The values of GPT for male patients are : 28.538 ± 1.264 ; 27.25 ± 1.548 ; 29.23 ± 1.11 ; 27.923 ± 0.937 and 29.667 ± 0.772 I.U. respectively , and for females are : 28.25 ± 1.309 ; 24.308 ± 1.603 ; 28.25 ± 1.162 ; 28.5 ± 0.793 and 43.308 ± 7.096 I.U. respectively .It is found that there is significant ($p < 0.01$) increase for males and females of all age groups in comparison with healthy controls .

4.2.2.3 Serum Alkaline phosphatase :

The values of ALP male burn patients are : 63.3 ± 4.524 ; 64.917 ± 4.2 ; 69.23 ± 2.977 ;71.25 ± 5.118 and 71 ± 4.023 I.U. respectively and the results of female burn patients are : 63.1± 4.051 ; 79.385 ± 8.974 ; 61.417 ± 4.369 ; 63.167 ± 3.96 and 71.231 ± 3.03 I.U. respectively . All these values are significantly increase at ($p < 0.01$) for males and females of all age groups in comparison with controls .

4.2.3 Determination of serum bilirubin (total , direct and indirect) :

The results of total , direct and indirect bilirubin within 2 days duration of male and female burn patients in comparison with control are depicted in figure 2 .

4.2.3.1 Total serum bilirubin :

The values of total serum bilirubin for male burn patients are : 10.231 ± 0.352 ; 10.385 ± 0.351 ; 10.208 ± 0.319 ; 10.385 ± 0.276 and 10.217 ± 0.367 μ mol /L respectively and for females are : 10.092 ± 0.37 ; 9.954 ± 0.312 ; 10.542 ± 0.28 ;

10.275 ± 0.253 and 10.392 ± 0.261 µ mol /L respectively . Both their values show significant ($p < 0.01$) increase in comparison with healthy control .

4.2.3.2 Direct bilirubin :

The values of direct serum bilirubin for males are : 2.962 ± 0.139 ; 2.992 ± 0.1 ; 2.869 ± 0.06 ; 3.085 ± 0.093 and 2.842 ± 0.057 µ mol /L respectively , and for females are : 3 ± 0.14 ; 2.992 ± 0.112 ; 3.108 ± 0.088 ; 3.25 ± 0.097 and 3 ± 0.112 µ mol /L respectively . Both their values show significant ($p < 0.01$) increase for all age groups in comparison with control .

4.2.3.3 Indirect bilirubin :

The results of serum indirect bilirubin is found as follows for males are : 7.308 ± 0.258 ; 7.367 ± 0.275 ; 7.262 ± 0.286 ; 7.3 ± 0.253 ; 7.275 ± 0.204 µ mol /L respectively , and for females are : 7.13 ± 0.289 ; 6.962 ± 0.901 ; 7.433 ± 0.225 ; 7.025 ± 0.247 ; 7.315 ± 0.234 µ mol /L respectively .Both of their values for all age groups are significant ($p < 0.01$) increase in comparison with healthy controls .

4.2.4 Determination of serum lipid :

It include for cholesterol , HDL and LDL and triglyceride (TG) within 2 days duration of male and female burn patients in comparison with control as shown in table 6 .

4.2.4.1 Serum triglyceride :

The values of TG for male burn patients are: 0.916 ± 0.088 ; 0.715 ± 0.041 ; 0.7 ± 0.038 ; 0.808 ± 0.064 and 0.85 ± 0.062

mmol/ l respectively and for females are: 0.75 ± 0.072 ; 0.858 ± 0.06 ; 0.792 ± 0.06 ; 0.838 ± 0.05 and 0.754 ± 0.063 mmol/l respectively . The values of triglyceride of males and females are significantly($p < 0.05$) decrease in comparison with controls.

4.2.4.2 Serum cholesterol :

The level of cholesterol in this study for male burn patients for all age groups are : 2.762 ± 0.795 ; 2.769 ± 0.056 ; 2.823 ± 0.043 ; 2.8 ± 0.044 and 2.958 ± 0.099 mmol/l respectively , and for females are : 2.758 ± 0.058 ; 2.683 ± 0.061 ; 2.725 ± 0.051 ; 2.708 ± 0.053 and 2.977 ± 0.063 mmol/l respectively . The values of cholesterol of males and females for all age groups show significant ($p < 0.01$) decrease in comparison with healthy controls .

4.2.4.3 Serum HDL :

The result of serum HDL for all age groups of male burn patients are: 0.746 ± 0.013 ; 0.75 ± 0.044 ; 0.692 ± 0.045 ; 0.7 ± 0.036 and 0.658 ± 0.042 mmol/l respectively and for females are : 0.758 ± 0.047 ; 0.766 ± 0.636 ; 0.742 ± 0.034 ; 0.742 ± 0.036 and 0.7 ± 0.042 mmol/ l respectively . The results of HDL for males and females are significantly ($p < 0.01$) decrease in comparison with controls .

4.2.4.4 Serum LDL :

The level of LDL for males are : 1.754 ± 0.089 ; 1.876 ± 0.064 : 2.006 ± 0.013 : 1.938 ± 0.066 and 2.138 ± 0.11 ; mmol / l respectively and for females are: 1.85 ± 0.056 ;

1.745 ± 0.076 ; 1.825 ± 0.058 ; 1.802 ± 0.048 and 2.126 ± 0.082 mmol / l respectively . The results of both males and females for all age groups are significantly ($p < 0.01$) decrease in comparison with controls .

4.2.5 Serum glucose :

The values of random blood sugar (figure3)within 2 days duration of all age groups for males burn`s victims are : 9.492 ± 0.432 ; 10.458 ± 0.073 ; 11.108 ± 0.207 and 13.217 ± 0.342 mmol / l respectively , and for females burn`s victims are: 9.317 ± 0.383 ; 10.808 ± 0.301 ; 10.675 ± 0.297 ; 10.9 ± 0.47 and 13.246 ± 0.303 mmol / l respectively. The values of both male and female burn patients for all age groups are significantly ($p < 0.01$) increase in comparison with healthy controls .

4.2.6 Serum urea and creatinine :

The values of urea and creatinine within 2 days duration of male and female burn patients are shown in table 7 .

4.2.6.1 Serum urea :

The urea results that obtained from this study for male burn patients are: 4.225 ± 0.185 ;4.0± 0.121;4.385 ± 0.3 ; 4.454 ± 0.296 and 3.908 ± 0.257 mmol / l respectively , and for females are : 3.917 ± 0.196;4.169 ± 0.321 ; 4.285 ± 0.276; 3.95 ± 0.297 and 4.158 ± 0.179 mmol / l respectively . The urea values are insignificantly ($p > 0.05$) increase for all age groups of males and females in comparison with controls .

4.2.6.2 Serum creatinine:

The values of creatinine for males are : 72.4615± 1.175; 71.4167± 1.27 ;71 ± 1.000 ;72.2308± 1.63 and 70.75 ± 1.62 µmol/l respectively , and for females 67.833± 2.415 ; 66± 0.77; 66.9167± 1.422 ; 62.750 ± 1.629 and 68.692 ± 1.273 µmol / l respectively .The results for males and females for all age groups are insignificantly increase at ($p > 0.05$) in comparison with control .

4.2.7 Serum sodium and serum potassium :

The figure 2 and 3 show the values of serum sodium and serum potassium before and within 2 days after resuscitation respectively .

4.2.7.1 Sodium :

4.2.7.1.1 Before resuscitation :

The values of serum Na before resuscitation for male burn patients are : 114.769 ± 1.638 ; 116.667 ± 1.524 : 116.769 ± 1.392 ;114.615 ± 1.61 and 115.167 ± 1.618 meql / l respectively , and for females are : 117.167 ± 1.342 ; 114.154 ± 1.523 : 115.83 ± 1.694 : 115.667 ± 1.635 and 115.15 ± 1.556 meql/l respectively. Both their results are significantly ($p < 0.01$) decrease for all age groups in comparison with healthy controls .

4.2.7.1.2 After resuscitation

The results of Na within 2 days duration after resuscitation for male burn patients are : 151.838 ± 1.164 ; 150.167 ± 1.043 : 151.538 ± 1.164 : 152.462 ± 1.113 and 151.333 ± 1.245 meql / l respectively , and for females are : 150.917 ± 1.708 ;

150.077 ± 1.238 : 148.462 ± 1.426 : 149.75 ± 1.371 and 148.077 ± 1.456 meql / l respectively . Both their results show significant (p < 0.01) increase in comparison with controls .

4.2.7.2 Serum potassium :

4.2.7.2.1 Before resuscitation :

The results of serum potassium before resuscitation for males burn patients are : 5.377 ± 0.089 ; 5.283 ± 0.099 : 4.696 ± 0.586 : 5.215 ± 0.367 and 5.233 ± 0.126 meql / l respectively , and for females are : 5.567 ± 0.0 ; 5.192 ± 0.156 : 5.208 ± 0.145 ; 5.161 ± 0.16 and 5.154 ± 0.176 meql / l respectively . Both of males and females potassium values show significant (p < 0.01) increase for all age groups in comparison with healthy controls .

4.2.7.2.2 After resuscitation :

The values of serum potassium within 2 days duration after resuscitation for males are : 2.8 ± 0.083 ; 2.808 ± 0.078 ; 2.708 ± 0.09 ; 2.815 ± 0.078 and 2.842 ± 0.116 meql / l respectively , and for females are : 2.667 ± 0.098 ; 2.554 ± 0.115 ; 2.733 ± 0.144 ; 2.908 ± 0.1 and 2.762 ± 0.095 meql / l respectively . Both of males and females values after resuscitation are significantly (p < 0.01) decrease in comparison with healthy control .

4.2.8 Serum calcium ,copper and zinc :

The values of serum calcium ,copper and zinc within 7 days duration of male and female burns` victims are shown in table 8 .

4.2.8 .1 Serum calcium :

The values of serum calcium for males burn patients are: 1.823 ± 0.052 ; 1.825 ± 0.045 ; 1.815 ± 0.037 ; 1.792 ± 0.043 and 1.808 ± 0.43 mmol /l respectively , and for females are : 1.808 ± 0.061 ; 1.823 ± 0.034 ; 1.767 ± 0.043 ; 1.723 ± 0.053 and 1.746 ± 0.039 mmol /l respectively . Both their values are significant ($p< 0.01$) decrease in comparison with controls .

4.2.8.2 Serum copper

It is founded in this study the results of serum copper for male burn patients are : 0.0334 ± 0.022 ; 0.0352 ± 0.003 ; 0.0700 ± 0.006 ; 0.0860 ± 0.01 and 0.0700 ± 0.005 part per million and for females are : 0.0356±.002; 0.0728 ± 0.002 ; 0.0768 ± 0.005 ; 0.0860± 0.01 and 0.0356± 0.002 part per million . Both their values are significantly ($p < 0.01$) decrease in comparison with controls .

4.2.8.3 Serum zinc:

The values of serum zinc for male burn patients are: 0.0228±0.005 ;0.0554 ±0.006 ; 0.0514± 0.005 ;0.0240±0.004 and 0.0202±0.005 part per million and for females are: 0.0250 ± 0.003 ;0.0546 ± 0.006;0.0482 ± 0.004;0.0304 ± 0.005;0.0188 ± 0.003 part per million. Both their values are significantly ($p < 0.01$) decrease in comparison with controls .

5 . **Discussion**:

This study is aimed to estimate some hematological and biochemical changes to the burn patients , since the levels of these substances are very important in letting the health care team to know how the body is responding to the different therapies that being provided .

5.1 Hematological Studies :

5 .1.1 RBCs count :

The values of RBCs count for males and females of all age groups are significantly decreased in comparison with healthy controls (table1). This results of RBCs count are in agreement with Wesly , (2000) who states that hematological changes are manifested by early destruction, injury, and loss of red blood cells. As much as 8% to 19% of the red blood cell pool may be destroyed by heat. Subsequent loss secondary to wound debridement (as much as one unit every 3– 4 days), phlebotomy for frequent laboratory studies, clearance of damaged red blood cells by the reticuloendothelial system, and decrease in hematopoiesis cause anemia that persists until the wound is closed. Other study pointed out that red blood cell count showed significantly high levels immediately after the burn, especially in the non-survivors. This high level decreased gradually to below control level by day 4 post-burn in the non-survivors and by day 6 post-burn day in the survivors(EI-Sonbaty & EI-0tiefy ,1996).

5.1.2 Hb and PCV :

The values of Hb concentration and packed cell volume (PCV) of male and female burn patients are significantly decrease in comparison with control (table 1) .This study agrees with EI-Sonbaty & EI-0tiefy (1996) , who pointed out that hemoglobin concentrations

and hematocrit showed significantly high levels immediately after the burn, especially in the non-survivors. This high level decreased gradually to below control level by day 4 post-burn in the non-survivors and by day 6 post-bum day in the survivors. As well as other study states that the high hematocrit values are common in the first 24 hours after serious burn injuries, even with adequate fluid administration. The decreasing of hematocrit are to be expected with adequate fluid resuscitation, but may also be a hallmark of occult bleeding (Stewart,1998) . Demling *et al* . , (2004) found that hematocrit either increased due to plasma volume decreases while circulating red blood cell volume remains relatively constant ; or hematocrit decreased because of either plasma volume replacement in case of hemolysis from prolonged heat exposure and/or major loss of blood from non-burn injury ; pre-existing anemia ; or hypervolemia .

5.1.3 WBCs count

The values of total WBCs are significantly increase in comparison with controls (figure 1). This results is consistent with the results of other study who states that there was a highly significant leukoeytosis in burn patients . Leukocytosis in the survivors remained constantly at high levels, while in the non survivors it showed oscillating levels until death . Significant leukocytosis was noticed in both survivors and non-survivors in this study, with constant high levels in survivors (EI-Sonbaty and., EI-0tiefy ,1996). As well as , Wesly , (2000) stated that hematological changes in burn injury include early leukocytosis and development of a hypercoagulable state. Other study pointed out that the total leukocyte counts were significantly increased in burn animals with infection (Gamelli *et al* .,1998) . Although most clinicians consider an increment in WBCs or left-shift to be associated with the presence of infection (Dunham *et al*., 2003) . Initial white blood cell

count may be high, normal, or low, depending on the magnitude of the stress response and white cell sequestration into the burn (Demling,2004).

5.1.4 The differential WBCs count

The differential WBCs count was illustrated in table 2 which the percentages of neutrophil ,eosinophil ,basophil ,lymphocyte and monocytes . The results of Dunham et al., (2003) is in agreement with our study ,since they found that there were significant increase in neutrophile percentage . Cavallini *et al*. , (1994) pointed out that Leucocytes showed rapid increases to as much as twice the normal values. Early post-burn leucocytosis generally implied high levels of neutrophil .

As well as our results are in agreement with Fujimi *et al .,* (2006) who pointed out that percentages of neutrophiles was increased after thermal injury in mice compared with control mice. While , the results of lymphocyte percentage recorded significant decrease in comparison with controls(table 2). These results were in agreement with Steinstraesser, *et al* . ,(2005)who states that lymphocytes were significantly lower after treatment for the burn and in the infected group in compared to control .

5.1.5 The platelets count :

The values of platelets count for both males and females show insignificant decrease in comparison with controls(table 3).This study is in agreement with Fujimi *et al .,* (2006) who pointed out that burn injury-induced change in the number of circulating platelets or platelet-derived products or alterations in platelet function may be detrimental. This has been suggested by the clinical observation that platelet deficiency correlates with a higher mortality after severe trauma and after sepsis. They demonstrate that platelets play a significant

protective role in survival following injury and also show that injury leads to measurable changes in the numbers of circulating platelets. Other study support this study states that all patients developed a mild thrombocytopenia (Murphy *et al*., 1999). While Pallua *et al .,*(2003) pointed out that platelet numbers decreased significantly in comparison with the other groups .George *et al.,(* 2001) showed that minor burns may present thrombocytopenia as an acute major complications in the presence of other thrombocytopenic factors like trauma and sepsis and thrombocytopenia by it self can be a good indicator for sub-clinical infection. The platelet count showed a significant decrease in levels below control, reaching very low levels in the non-survivors, while it increased steadily in the survivors after day 4 post-burn, reaching levels above control by day 7 post-burn and thereafter . This increase may be considered as a good prognostic parameter during the treatment of severely burned patients. In contrast, there was a significant and progressive decrease in the platelet count in the non-survivors until death ensued. This explanation for the thrombocytopenia is usually due to the increase of platelet consumption or the decrease of production by bone marrow. They were concluding that a decline in the platelet count preceded other signs of sepsis in all cases (EI-Sonbaty & EI-0tiefy ,1996) **.** As well as other explanation , burn patients present significant thrombocytopenia, attributed to the peripheral destruction or utilization of platelets in the burn area, and toxic depression of the blood-forming organs in response to products arising from tissue destruction and bacterial infection (Kamel *et al* . ,1999) .

5.2 Biochemical studies :

5.2.1 Total serum protein ,albumin ,and globulin :

Total serum protein ,albumin ,and globulin were studied in this work (table4) .

5.2.1 .1 Total serum protein :

It was found that there was significant decrease in the total serum protein for all age groups for both male and female burn patients in comparison with healthy controls . This study was supported by Demling ,(2004) who recorded that a marked decreased in plasma proteins occur early postburn . Other study also pointed out that burns injury results in dramatic changes in plasma proteins in which the concentration of protein was significantly lower in serum of patient than the control (Vemula *et al* . , 2004) . As well as , this study is consistent with Samuelsson *et al* . ,(2006) who states that there is loss of homeostatic control as a result of massive losses of fluid and protein during the first 24 hours. The massive amount of fluid needed during resuscitation, particularly in larger burns, creates a generalized edema that is caused both by the volume of fluid itself and the decreased colloid osmotic pressure that will develop secondary to the resuscitation fluid given and to proteins lost from the circulation. This may compromise tissue perfusion in both injured and uninjured tissues of the burn-injured patients. These results occur because local inflammatory cytokines enter the circulation and result in systemic inflammatory response. As burns approach 25 % of TBSA , this will lead the microvascular leak to become generalized and permit the loss of fluid and protein from the intravascular compartment to the extravascular compartment and finally they are lost through the wound (Brunicardi *et al* . ,2005).

5.2.1.2 Serum albumin :

The results of serum albumin of both males and females for all age groups show significant decrease in comparison with healthy controls(table 4) .These results are in agreement with Ge *et al* . , (1996) who states that serum albumin concentration decreased gradually after resuscitation. As well as, other study hypothesized that initial serum albumin level may be useful as an indicator for prognosis and severity of injury in burned patients. Depressed serum albumin level is associated with an increase in mortalityr ate in the major burn patients. In addition, hypoalbuminemia may result in impaired wound healing and predisposing to sepsis. However, it has not been clear whether hypoalbuminemia itself can be used as an independent predictor of mortality in major burns (Ho Kim *et al*. , 2003). The reason for obtaining these results is thought that stress (ex.trauma, infection or others) has always been associated with hypoalbuminemia in either animals or humans (Fearon *et al* . ,1998; Voisin *et al* . ,1998). A reduction of plasma albumin concentration in general can be the consequence of various factors, including a change in its rate of synthesis, an increased catabolic rate, and/or a redistribution of albumin from plasma to interstitial compartment. While in burn , plasma albumin is well known to decrease in response to inflammation (Ruot *et al* ., 2000) . They are also refer to that the depressed plasma levels of albumin were not associated with a reduced hepatic albumin synthesis. Such results could have potential clinical implications. Since the intravascular compartment which is easily accessible , represents <35% of the total exchangeable albumin pool and they observed no decrease of plasma albumin synthesis rate during the acute phase , hypoalbuminemia could be due to an increase in either catabolism or escape of the protein from the plasma

pool in the extravascular space (Ruot *et al* ., 2000). Therefore in the colloid fluid resuscitation for burn patients ,protein should not be given between 8 to12 hours postburn because of the massive fluid shifts during this periods , after this the protein should be used (Brunicardi *et al* ., 2005) .

5.2.1.3 Serum globulin :

The results of s. globulin of both male and female burn patients show significant decrease in comparison with control(table 4) . This study also consistent with Rothenbach *et al*. ,(2004) who pointed that burn injury induced a rapid decrease in globulin values. As well as, the production of immunoglobulin G (IgG) in response to T-cell-dependent antigens is also impaired after serious injury (Church *et al* . , 2006).

Our results are in disagreement with Sheridan *et al* .,(1997) who state compensatory increase in acute-phase proteins reflected in plasma globulin. This is only true when we take the serum globulin as percentage to the total serum protein .

5.2.2 Liver enzymes :

5.2.2 .1 Serum GOT and GPT:

The results of serum GOT and GPT of male and female burn patients for all age groups are significantly increase in comparison with healthy control (table 5) . These results agree with Price *et al* .,(2007) who pointed out that the alteration of liver function tests is extremely common following major burns. An incidence as high as 50% has been described when there is transient elevation of the aspartate aminotransferase . He also states that the precise etiology of these changes is not known, but they usually are benign in character and resolve spontaneously. Other study states that serum enzyme levels in post burn period show interesting trends. It was found that the mean

levels of GOT and GPT were increased on 5th day thereafter they declined on 10th day. However, the levels were higher than the level at the time of admission. This trend in the values shows that the transaminases rise transiently in response to the thermal injury and they shortly fall back to original level (Bhagwat *et al* ., 2007) . The explanation for these results as follows :as the magnitude of a burn increases ,so does the likelihood of early postburn hepatic dysfunction. An initial increase in the hepatic aminotransferase is common following the burns of more than 50%of the body surface area . This mostly due to the acute reduction in cardiac output, increased blood viscosity, and associated splanchnic vasoconstriction that occur immediately following thermal injury. Following successful fluid resuscitation ,the hepatic enzyme promptly return to normal in most patient .The magnitude of initial enzyme derangement has not been predictive of outcome(Bongard and Sue ,2002) .

5.2.2 .2 Serum alkaline phosphatase ALP :

The results of serum alkaline phosphatase (ALP) of male and female burn patients for all age groups are significantly increase in comparison with healthy control (table 5) .

These results also are supported by Price *et al*. (2007) who stated that there are increased in liver enzymes and particularly ALP. As well as it is found that Alkaline phosphatase activities continued to rise till day 10. It is found that the reason for obtaining these results that functional disturbances occur in liver after thermal injury. Monitoring serum ALP in postburn period has valuable prognostic importance (Bhagwat *et al* ., 2007).Other study states that ALP were significantly elevated over the first 3 weeks post-burn, normalizing over time(Jeschke & Herndon ,2002).

5.2.3 Serum bilirubin :

The values of total serum bilirubin, direct and the indirect bilirubin show significant increase in comparison with healthy control (figure2).

Price *et al* .,(2007) found that patients who show elevation of bilirubin, usually have some associated complication of their burns, such as burn wound sepsis, and in these patients, the prognosis is guarded .The reason for obtaining these results could be due to increase in the conversion of cholesterol into bile acids combined with the decreased breakdown of the bile acids could be a potential mechanism for solublizing fatty acids and increasing their catabolism in the gut through the formation of mixed micelles this will lead to increased in the direct bilirubin level (Vemula *et al* . , 2004) . While the increased indirect bilirubin could be due to increased in production. The increase in both direct and indirect bilirubin will ultimately lead to increase in the total bilirubin (Andreoli *et al* .,2004).

5.2.4 Lipid profile :

The values of serum total cholesterol ,HDL, LDL and triglycerides of male and female show significant decrease for all age group in comparison with healthy controls(table6). Hypocholesterolemia is an important observation following trauma. In a study of critically ill trauma patients, mean cholesterol levels were significantly lower .Since lipoproteins can bind and neutralize lipopolysaccharide, hypocholesterolemia can lead to poor outcome. New therapies directed at increasing low cholesterol levels may become important options for the treatment of sepsis (Dunham *et al*. , 2001).Other investigators observed in their study that a profound decrease in cholesterol levels within a few days of the burn, with the lowest values occurring between the days 6 and 10. They noted that both LDL and HDL, which carry over 80% of the total cholesterol in humans, were

both decreased(Wilson *et al*. ,2003). Clinical support was provided by Gordon *et al*. , (2001) who noted that patients with clinical and/or laboratory evidence of infection had significantly lower mean high-density lipoprotein concentrations and the lowest cholesterol and lipoprotein levels also predicted a poor clinical outcome. Other study pointed out that serum cholesterol values were sequentially monitored in critically injured patients. A decrease in serum cholesterol or persistent hypocholesterolemia also suggests the onset of a new, serious infection. In contrast, WBCs responses were less consistent and less sensitive in predicting the onset of infection. A decrement in cholesterol also indicates a progression in organ failure. Conversely, an increase in cholesterol suggests that organ failure is resolving. These data support the notion that serial serum cholesterol values parallel clinical improvement or deterioration in critically ill trauma patients (Wilson *et a*l., 2003) .

While, the results of serum triglycerides (TG) are supported by Vemula *et al* . , (2004) who states that the decrease in TG by 24 hours postburn could be due to their increased export into the bloodstream and enhanced hydrolysis to produce fatty acid. The explanation fot export of TG into the bloodstream after burn injury is supported by the data on enhanced expression of apolipoprotein AI(apo-AI), a major constituent of very low density lipoprotein (VLDL) particles along with the TG, as well as prior reports on increased levels of TG in the blood plasma after burn injury .

5.2.5 Serum glucose :

The values of serum glucose of both male and female burn patients for all age groups are significantly increase in comparison with healthy controls (figure 3) . The results of serum glucose are supported by

other studies who pointed out that glucose metabolism in patients with severe burn is almost always abnormal. Patients that are victims of sepsis, burn, or trauma commonly enter a hypermetabolic stress state that is associated with changes in carbohydrate metabolism, such as enhanced peripheral glucose uptake and utilization, hyperlactatemia, increased glucose production, depressed glycogenesis, glucose intolerance, and insulin resistance (Mizock,1995 ; Price *et al* .,2007). As well as ,the burn also elicits the general trauma response, including the increase in blood glucose concentrations as a result of gluconeogenesis, glycogenolysis , insulin-resistance, and lipolysis. In this study, blood glucose concentration was influenced by trauma-induced insulin resistance, which peaked on day two and then gradually decreased to a lower but still increased value. (Samuelsson *et al*.,2006). The explanation for these results are the increased release of catecholamine in burns patients often leads to hyperglycemia . In elderly and diabetic patients , insulin may be required ;some glucose should be infused at this time as well. Infants are prone to hypoglycemia as a results of decreased glucose stores(Dauglas *et al*.,2003). In addition to, other a study showed that there is a marked stress response predominately involving increases in catecholamines, cortisol, vasopressin, and glucagon (Rainer *et al* . , 1999) .

5.2.6 Serum creatinine and serum urea :

It is shown that serum creatinine and serum urea values of male and female for all age groups are insignificant decrease in comparison with controls (table7) . Serum creatinine results are supported by Chauhan *et al* . ,(2004)which his study also showed similar findings to our results . Regarding the urea results show

agreement with Rrainer ,*et a*l., (1999) who found in their study there is no significant difference in serum urea value in comparison with control . Serum creatinine was found to be the most important prognostic indicator for patients going into acute renal failure followed by serum potassium. Blood urea is not an useful independent indicator as it is increased in non renal conditions such as dehydration and high protein diet. (Chauhan,*et al* ., 2004) . Zhewei Fei, *et al* (2003) shows that unilateral nephrectomy in burns' rabbits did not produce a significant alteration in serum creatinine and blood urea nitrogen (BUN).

5.2.7 The serum sodium:

The serum sodium of male and female burn patients before resuscitation are significantly decrease in comparison with healthy control (figure 4) . Hinton *et al* . , (2003) who states that the hyponatraemia in these cases results rarely from sodium deficit but usually from excess water retention and entry of sodium into the cells. These changes may in some patients be reversed by blood-transfusion, by insulin and glucose infusions, or by both together. Improvements in supportive therapy have reduced morbidity and mortality . While the results after resuscitation which is done more than 48 hours , it is found that there is significant increased in comparison with controls (figure4). This results are consistent with Huang *et al* . ,(1995)who states that during the first 3 days after burn, serum sodium concentrations were moderately elevated in the patients .As well as, these results were supported by Ge *et al* .(1996) who pointed out that serum Na^+ decreased post-burn and increased after resuscitation . Other study found that the initial resuscitation period below 36 hours characterized by hyponatraemia. The explanation for these results are in major burns, intravascular volume is lost in

burned and unburned tissues: this process is due to an increase in vascular permeability, increased interstitial osmotic pressure in burn tissue. and cellular oedema. The most significant shifts occurring in the first hours. Hyponatraemia is frequent, and the restoration of sodium losses in the burn tissue is therefore essential. While the hypernatraemia which is occur later on is caused by several mechanisms: intracellular sodium mobilization, reabsotption of cellular oedema, urinary retention of sodium (because of the increase in renin, angiotensin. and ADH), and the use of isotonic or hypertonic fluids in the resuscitation phase (Ramos, 2000).

5.2.8 The serum potassium:

The results of serum potassium of males and females are significantly increase before resuscitation in comparison with healthy controls (figure 5). Our results is supported by other study which states that in major burns , the initial resuscitation period (between 0 and 36 h) characterized by hyperkalaemia because of the massive tissue necrosis(Ramos ,2000).As well as, . Demling *et al* . , (2004)state that potassium ions will increase if severe hemolysis has occurred or renal impairment is present.

While our values of the serum potassium after resuscitation are significantly decrease in comparison with healthy control (figure5).This results are in agreement with Rainer *et al* . , (1999) who pointed out that hypokalaemia is well recognized after stress states and is due to a combination of the effect of adrenaline and insulin. Adrenaline stimulates receptors on skeletal muscle with consequent uptake of potassium from the circulation. It is probable that total body potassium is not reduced. As well as ,other study showed that the early post-

resuscitation period between 2-6 days of burns' patients characterized by hypokalaemia. It may be due to increased potassium losses (urinary-, gastric. faecal) and the intracellular shift of potassium because of the administration of carbohydrates (Ramos ,2000).

5.2.9 Serum Calcium:

The results of serum calcium ,copper and zinc in males and females for all age groups are significantly decrease in comparison with healthy controls (table 8).This is supported by Snider *et al.* (1996)who pointed out that the mean serum calcium were significantly lower at the initial time of study than at discharge. Other study who states that there is decreased in serum calcium and they have shown that vitamin D metabolism is disturbed after burn injury. Vitamin D is essential for calcium and phosphorus homeostasis and skeletal bone integrity (Wray *et al* .,2002) .As well as, serum calcium concentrations remained significantly lower than values measured in shams 8 days after burn trauma in the absence of sepsis,(Jureta *et al* .,2006). This study is consistent with Yang *et al* .,(1999) calcium contents were significantly decreased compared to control group. It is found that Ca^{2+}-ATPase activities, calcium uptake function and left ventricular contractile function decreased markedly. They conclude The cardiac renin-angiotensin system is activated rapidly after severe burns and inhibits the calcium transport function which may play an important role in cardiac contractile dysfunction following burns. Hypocalcemia is common among critically ill patients. It has been shown to correlate with increased mortality . Hypocalcaemia may be iatrogenically induced due to chelation of calcium by high concentrations of citrate in blood derived colloid (blood, fresh frozen plasma, and human albumin solution). It may be a result of change in calcium binding due to

change in blood pH, elevation of fatty acids, sepsis , hypoalbuminemia, renal failure and hypomagnesaemia (Saleh *et al* ., 2008) .

5.2.10 Trace elements:

Our results of serum copper and zinc are supported by Marvaki *et al* . , (2001) who they states that mean plasma concentrations of Cu and Zn were low at admission and discharge. Urinary Zn was elevated at admission, whereas Cu was elevated at both times. Wound Cu and Zn concentrations exceeded plasma concentrations, suggesting that inflammatory wound exudate was a primary route of loss. They demonstrate that burn injury in children results in low plasma levels of Cu and Zn that are inadequately compensated during hospitalization . These micronutrients are essential for bone matrix formation, linear growth, and wound healing. Other study consistent with our results found that there is a significant difference was shown. Irrespective of etiology, the serum trace element levels were lower in the burn patients than in the healthy individuals. Despite the marked difference in the percentage of body burns, trace element levels changed as a result of the systemic effect of the burns. The explanation for these results could be after the burn, trace element excretion was shown to occur from the wound surface and in the urine. (Safran *et al* .,1999).

Chapter Four

The Results

4.1 Hematological studies :

4.1.1 Red blood cells (RBCs)count , hemoglobin (Hb) and packed cell volume (PCV) :

The table 1 shows the RBCs count, Hb and PCV values for male and female burn patients within 7 days duration and their statistical analysis .

4.1.1.1 RBCs count :

The values of RBCs count for all the age groups (1-<3 ; 3-<5 ; 5-<10 ; 10-<18 and 18- 58 years old) of male burn patients are : 4.208 ± 0.213 ; 3.6 ± 0.175 ; 3.329 ± 0.175 ; 4.046 ± 0.214 and 3.525 ± 0.152 millions/mm^3 respectively , and for females are: 3.904 ± 0.17 ; 3.196 ± 0.114 ; 3.673 ± 0.198 ; 3.467 ± 0.107 and 3.573 ± 0.193 millions/mm^3 respectively .Both their values of males and females are significantly ($p< 0.01$) decrease in comparison with healthy controls .

4.1.1.2 Hb :

The results of Hb for all age groups of male burn patients are : 11.523 ± 0.613 ; 9.525 ± 0.527 ; 9.36 ± 0.552 ; 11.289 ± 0.665 and 9.8 ± 0.51 g/dl respectively and for females are: 10.459 ± 0.514 ; 8.8 ± 0.288 ; 10.038 ± 0.599 ; 9.221 ± 0.339 and 9.531 ± 0.515 g/dl respectively . Both their values for all age groups of male and female burn patients are significantly ($p< 0.01$) decreased in comparison with healthy controls .

4.1.1.3 PCV :

The values of PCV for all age group of male burn patients are : 0.343 ± 0.018 ; 0.28 ± 0.016 ; 0.272 ± 0.016 ; 0.33 ± 0.019 and 0.284 ± 0.014 L/L respectively , and for females are : 0.313 ± 0.016 ; 0.255 ± 0.009 ; 0.288 ± 0.018 ; 0.27 ± 0.009 and 0.278 ± 0.016 L/L respectively .All the values of male and female burn patients for all age group are significantly ($p< 0.01$) decrease in comparison with healthy controls .

4.1.2 Total white blood cells (WBCs) count :

As shown in the figure 1 the values of WBCs for male burn patients are : 9.531 ± 0.961; 9.733 ± 0.881; 8.931 ± 0.603 ; 9.654 ± 0.483 and 10.175 ± 0.459 $\times 10^3$ mm^3respectively and for females are : 8.383 ± 0.538 ; 8.738 ± 0.597 ; 8.792 ± 0.44 ; 9.917 ±0.431 and 10.277 ± 0.52 $\times$ 10^3 mm^3 respectively within 7 days duration . Both of their values shows significant ($p< 0.01$) increase in comparison with healthy control .

4.1.3 Differential WBCs Count :

As illustrated in table 2, this includes neutrophil , lymphocyte , monocytes , basophil and eosinophil percentages for male and female burn patients within 7 days duration .

4.1.3.1 Neutrophil :

The mean percentage of male burn patients are : 71.308 ± 0.957 ; 71.916 ± 0.988 ; 72.154 ± 1.255 ; 71.846 ± 1.636 and 72 ± 1.605 % respectively , and for females the mean percentage are : 70.75 ± 1.031 ; 72.769 ± 0.9 ; 71.75 ± 0.906 ; 75.583 ± 3.662 and 72.769 ± 1.105 % respectively. The

percentages of the males and females are significantly (p < 0.01) increase in comparison with controls .

4.1.3.2 Eosinophil :

The values of eosinophil percentage for male burn patients are : 1.462 ± 0.183 ; 1.25 ± 0.131 ; 1.385 ± 0.14 ; 1.231 ± 0.122 ; and 1.417 ± 0.149 % respectively , and for females are: 1.417 ± 0.149 ; 1.308 ± 0.208 ; 1.167 ± 0.167 ; 1.088 ± 0.083 and 1.462 ± 0.144 % respectively .Both their values are insignificant (p> 0.05) in comparison with healthy controls .

4.1.3.3 Basophil :

The values of basophil percentage for male burn patients are: 0.462 ± 0.14 ; 0.5 ± 0.151 ; 0.231 ± 0.122 ; 0.385 ± 0.14 and 0.333 ± 0.142 % respectively , and for females are : 0.583 ± 0.149 ; 0.308 ± 0.133 ; 0.417 ± 0.149 ; 0.5 ± 0.151 and 0.308 ± 0.133 % respectively .Both of their percentage are insignificant (p > 0.05)in comparison with controls .

4.1.3.4 Lymphocyte :

The percentage of lymphocyte for male burn patients for all age groups are : 19.923 ± 1.135 ; 19.417 ± 0.892 ; 18.769 ± 1.063 ; 19.615 ± 1.38 and 19.083 ± 1.658 % respectively , and for females are : 19 ± 1.052 ; 18.538 ± 0.882 ; 18.833 ± 0.903 ; 18.5 ± 1.07 and 18.616 ± 0.979 % respectively . The percentages of males and females' values are significantly decrease for all age groups at p < 0.01 in comparison with healthy control .

4.1.3.5 Monocyte :

The percentage of monocytes for male burn patients for all age groups are : 6.846 ± 0.274 ; 7.083 ± 0.288 ; 7.385 ± 0.24 ; 7 ± 0.3 and 7.167 ± 0.271 % respectively , and for females are : 8.25 ± 0.279 ; 7.231 ± 0.257 ; 7.917 ± 0.336 ; 7.333 ± 0.31 and 6.923 ± 0.383 % respectively . The percentage of males and females' values are insignificantly decrease for all age groups at $p > 0.05$ in comparison with controls .

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4.1.4 Platelets counts :

Table 3 shows the values of platelets count within 7 days duration for male burn patients are : 337.692 ± 15.974 ; 318.5 ± 18.213 ; 331.5 ± 12.943 ; 352.5 ± 19.71and 359 ± 13.454 $\times 10^3/mm^3$ respectively , and for females are: 298.5 ± 5.965 ;324.5 ± 19.895 ;350 ± 14.832 ; 298 ± 8.794 and 355.833 ± 16.627$\times 10^3/ mm^3$ respectively . Both their values show insignificantly ($p > 0.05$) decrease in comparison with controls .

1 .1 INTRODUCTION :

The skin is one of the largest and most versatile organs of the body .It has surface area of 1.5 to 2 square meters , and form the major interface between the internal organs and the external environment . As the body the first line of defense, the skin is continuously subjected to potentially harmful environmental agents ,including solid matter , liquids ,gases, sunlight ,and microorganisms . The skin also serves as immunological barrier . The langerhans cells detects foreign antigens, playing an important part in allergic skin conditions and skin graft rejection (Abston *et al* .,2000; Porth , 2007) .

Burn is one of the most common and devastating forms of trauma (Church *et al*. , 2006) . It is an injury to the skin that damages or destroys skin cells and tissue. It is generally caused when skin makes contact with flames, chemicals, electricity , or radiation. Thermal burns are caused by intense external sources of heat, such as flames, scalding liquids, or steam. Burns resulting from an impaired driving crash are most likely thermal burns (Abston *et al* . , 2000) .

The severity of any burn injury is related to the size and depth of the burn, and to the part of the body that has been burned . Burns are the only truly quantifiable form of trauma. The single most important factor in predicting burn-related mortality, need for specialized care, and the type and likelihood of complications is the overall size of the burn as a proportion of the patient's total body surface area (TBSA) (Church *et al*. , 2006). Burns are classified according to increasing depth as epidermal ,first-degree **;** superficial and deep partial-thickness ,second degree **;** full-

thickness third-degree ; and full thickness with underlying structure fourth-degree (Brunicardi *et al*.,2005).

Although burn injuries are frequent in our society, many surgeons feel uncomfortable in managing patients with major thermal trauma because of high morbidity and mortality. Advances in trauma and burn management over the past three decades have resulted in improved survival and reduced morbidity from major burns. Improved results are due to advancements in resuscitation, surgical techniques, infection control and nutritional support (Abston *et al*. ,2000) .

Thermal injury is among the most severe forms of trauma and its effects are both local and systemic. Response to thermal injury includes cellular protection mechanisms, inflammation, hypermetabolism, prolonged catabolism, organ dysfunction and immuno-suppression (Yang *et al*.,2007) .

Burn patients commonly manifest an inflammatory process involving the entire organisms ;the term systemic inflammatory response syndrome (SIRS) summarizes these conditions. SIRS with infection (ie. ,sepsis syndrome) is a major factor determining morbidity and mortality in thermally injured patients(Brunicardi, *et al*., 2005).

Severe burn injury is characterized by a marked hypermetabolic response and hypermetabolism and even more markedly by loss of lean body mass. This hypermetabolic response is accompanied by a progressive decline of host defenses via immunological abnormalities (Marvaki, *et al*.,2001). Significant thermal injuries induce a state of immunosuppression that predisposes burn patients to infectious complications (Church *et al*..,2006).

1.2 The Aim of the study : -

This study is aimed to estimate some hematological and biochemical changes to the burn patients who are admitted to the burn unit at Al-Hilla General Teaching Hospital because this will help the medical staff for proper management with less morbidity and mortality . So this study is designed to determine the following :-

1. Total red blood cells (RBCs)count .
2. Total leukocytes count and the differential counts .
3. Platelets count .
4. Serum of total protein , albumin and globulin.
5. Serum of GOT ,GPT, alkaline phosphatase and bilirubin and how they are important in certain cases .
6. Serum total cholesterol, HDL ,LDL and triglycerides .
7. Random blood sugar and their important in management.
8. Serum of sodium and potassium and their benefits of estimation in fluid resuscitation .
9. Serum calcium, zinc and copper .

Chapter three

Materials & Methods

3.1 Materials :

3.1.1 Subject of the study :

This study lasted from November/2007 to May / 2008. There are 125 patients and 100 healthy controls (clinically assessed by specialist doctor) are taken in this study . The patients are classified into 5 groups and each group composes of 25 patients , while each control group composed of 20 subjects .The ages of these groups were divided as follows: the first group (1-<3 years) ; the second group (3-<5 years) ; the third group (5-<10 years) ; the fourth group (10- <18 years);and the fifth group including the adults (18- 58) years old (Behrman *et al* ,2004) (Gill & Obrien ,1988) . Those patients were admitted to the burn unit at Al-Hilla General Teaching Hospital. They were suffering from second to third degree ,(flame and scald) burn injury, with TBSA range between (20 -35) % and with no infection or sepsis .

3.1.2 Chemicals : the chemicals materials sources used in this work were as follow :

No.	Chemical material	sources
1	Formal citrate solution	Crescent ,Saudi Arabia
2	Hydrochloric acid	Segma ,chemical.
3	Glacial acetic acid	Crescent ,Saudi Arabia
4	ammonium oxalate	Crescent ,Saudi Arabia

5	Leishman stain solution	Crescent ,Saudi Arabia
6	HNO_3	Segma, chemical.
7	Bilirubin kit	Biomerieux ,France
8	Creatinine Kit	Human ,Germany.
9	Glucose Kit	Human ,Germany .
10	SGOT kit	Randox, UK.
11	SGPT kit	Randox, UK.
12	Alkaline phosphatase kit	Biomerieux ,France
13	Total protein kit	Human, Germany
14	Albumin kit	Human, Germany
15	Total cholesterol kit	Human, Germany
16	TG kit	Human, Germany
17	HDL kit	Biolabo ,France
18	Total calcium kit	Human, Germany
19	Potassium kit	Human, Germany
20	Sodium kit	Human, Germany

3.1.3 **Instrument**s : The following table shows the main instrument used in this work and their sources :

No.	Instruments	sources
1	Hematocrit centrifuge	Hettich ,Germeny
2	Hemocytometer	Osaka , Japan.
3	Light Microscope	Olympus .Japan.
4	Water bath	Memmert, Germeny
5	Centrifuge	Universal ,Germany.

6	Hemoglobinometer	Optima, 202 , Japan.
7	Spectrophotometer	Cecil.England.
8	Plain tube	Afma – Dispo, Jordan.
9	EDTA tube	Afma – Dispo, Jordan.
10	Micro – pipette 100 -1000 µl	Oxford ,USA.
11	Micro – pipette 50 µl	Oxford ,USA.
12	Disposal syringe	Asia medical instrument .
13	Microscope slide	Sail Brand ,China
14	Microscope Cover slip	Ataco, China
15	Refrigerator	Concord ,Lebanon
16	Manual differential counter.	Erma ,Japan
17	Atomic Absorbiton Flame Emission Spectrophotometer AA6200.	Shemadzu , Japan
18	Incubator	Memmert ,Germany

3.2 Methods :

3.2.1 Blood collection:

The collection of blood was done in burns` ward in Hilla teaching hospitals at 9 A.M. . Five ml. of blood are drawn for each hematological and biochemical studies .The blood samples for biochemical studies are drawn within 2 days duration after burn , while for hematological studies are drawn within 7 days duration . Two groups of labeled tubes were used ; the first tubes contain EDTA as anti- coagulants to prevent clotting of blood to be used for hematological studies. The second group tubes were without

anti-coagulant as plain tubes, for blood to be used for preparing sera for subsequent biochemical tests(Bishop *et al*., 2000). Each sample was labeled and given a serial number together with the patient name. The serum samples were frozen at -20°C for biochemical analysis (Lewis *et al.* , 2006).

3.2.2 Hemotological Studies :

3.2.2.1 Red blood cells count (RBCs count) :

Blood was diluted with formal citrate solution (l ml formalin ,3.8 gm tri-sodium citrate,99 ml distilled water).Blood is drawn up to mark 0.5 in the RBCs pipette ,without letting any bubble as into the pipette by holding the pipette almost horizontally . The pipette must be clean and dry .Then the tip is cleaned and the diluting fluid is drawn up to 101 mark (dilution 1 : 200). The content is mixed for three minutes by gently rotating the pipette to obtain good mixing . The counting chamber (neubaur hemocytometer) was filled by holding the pipette at an angle 45 degree and touching the space between the coverslip and the chamber by the point of the of the pipette , an appropriate drop of the mixture is allowed to run under the coverslip by capillary action . The chamber is examined under 40X objective lens of the microscope to count in RBCs counting area of the chamber (in the four corners and center tertiary squares of the RBCs) ,and the depth of the field is 0. 1 mm (Lewis *et al.* , 2006).

3.2.2.2 Estimation of hemoglobin (Hb) :

A cyanomethemoglobin method was used to estimate the hemoglobin contents of the blood. The method was based on Drabkins cyanide- ferriccyanide solution . Twenty micro liter (µL)

of blood was added for 5 ml of Drabkin 's solution mixing, and incubated for at least 5 minutes at 37°C and then the results were estimated by using Hb meter at 540 nanometer (nm) wave length (Markarem,1974).

3.2.2.3 Determination of packed cells volume (PCV) :

Microhematocrit method was used to determine PCV . Heparinized capillary tubes used, and blood was filled to approximately three quarters of their lengths then the unmarked end is closed with modeling clay and put in the microhematocrit centrifuge. After centrifugation for 15 minutes, the red blood cells were separated from plasma and remain a band of buffy coat at the interface between them consisting of leukocytes and blood platelets (Lewis *et al.*, 2006) .

3.2.2.4 White blood cells count (WBCs count):

Blood was diluted with Turk's solution . Blood is drawn in a clean and dry WBC pipette up to the mark 0.5 and outside of the pipette is wiped off with gauze . Then diluting fluid (Turk's solution) is drawn up to mark 11 (dilution 1:20) the contents are mixed for three minutes ; the counting chamber a (Neubaur hemocytometer) was filled . It is waited for three minutes to let the cells for setting down and then the chamber is examined under 40X objective lens of the microscope to count WBCs in the four corners secondary squares (Lewis *et al.*,2006).

3.2.2.5 The platelets count :

This is done by taking 0.38 ml of diluting fluid (1% ammonium oxalate which hydrolyze the RBCs,) and added to it 0.02 ml of blood taken by using hemoglobin pipette after having wiped the tip of the pipette ,and then we charge the chamber with the help of a Pasteur's pipette and placing the counting chamber in a petridish containing a moistened filter paper and let stand for 15 minutes(Lewis *et al.*,2006).

3.2.3 The biochemical studies:

3.2.3.1 Measurement of total serum protein :

Cupric ions react with protein in alkaline solution to form a purpule complex . The absorbance of this complex is proportional to the protein concentration in the sample. The intensity of color was measured photometricaly at 546 nm wave length, by using spectrophotometer (according to procedure recommended by the total serum protein kit from Human company ,Germany) (Josephson *et al* . ,1957 ; Burits & Ashwood , 1999).

3.2.3.1.1 Determination of serum albumin:

Albumin in the presence of bormocresol green at slightly acid pH, produces a color change of the indicator from yellow - green to green blue. The intensity of the color is proportional to the albumin concentration in the sample. The intensity of color was measured by using spectrophotometer at 630 nm wave length (according to procedure recommended by albumin kit from the Human

company ,Germany) (Josephson *et al* .,1957; Burits & Ashwood , 1999).

3.2.3 .1.2 Determination of serum globulin:

Serum globulin was determined by subtracting albumin from total serum protein and the results represented the values of serum globulin (Bishop *et a*l., 2000) .

3.2.3.2 Determination of glutamic oxaloacetic transaminase (GOT):

Colorimetric method (Reitman and Frankel, 1957; Burits & Ashwood , 1999) was used to measure the amount of oxaloacetic acid produced under fix conditions. A reddish brown hydrazone formed when 2,4 dinitrophanyl hydrazine in alkaline solution, is added which can be measured by using spectrophotometer at 546 nm (according to procedure recommended by the GOT kit from Randox company, UK).

3.2.3.3 Glutamic pyruvic transaminas (GPT) determination :

Colorimetric method (Britman and Frankel , 1957; Burits & Ashwood , 1999) was used for the determination of GPT reaction with 2,4 dinitrophenyl hydrazin in alkaine solution, a brown colored hydrazone is formed which can be measured by using spectrophotometer at 505 nm (according to procedure recommended by GPT kit from the Biomaghreb company ,Germany).

3.2.3.4 Determination of alkaine phosphatase (ALP) :

The method used was that in which phenyl phosphate is hydrolyzed with the liberation of phenol and formation of phosphate. The amount of phenol which formed is estimated

colorimetrically by using spectrophotometer at 510 nm(according to procedure recommended by ALP kit from the Biomerieux company ,France) (Kind and King, 1954; Burits & Ashwood , 1999).

3.2.3.5 Measurement of total serum bilirubin:

Working solution was prepared by mixing reagent 1 (R1) (sulfanilic acid, hydrochloric acid and dimethyl sulfoxide), with reagent 3(R3) (sodium nitrate) .Mix well and incubate exactly for 5 minutes at 37 C° and read wave length 555 nm by using spectrophotometer (according to procedure recommended by the bilirubin kit from the Human company ,Germany) (Walter and Gerarde, 1970; Burits & Ashwood , 1999).

3.2.3.5.1 Measurement of direct serum bilirubin :

Working solution was prepared by mixing reagent 2 (R2) (sulfanilic acid and hydro chloric acid) with R3 (sodium nitrate) . Mix well and incubate exactly for 5 minutes at 37 C°. After that read at wave length 555 nm by using spectrophotometer (according to procedure recommended by the bilirubin kit from the Human company ,Germany) (Walter and Gerarde, 1970; Burits & Ashwood , 1999).

3.2.3.5.2 Determination of indirect serum bilirubin concentration:

It was obtained by the substraction of the values of the direct bilirubin from the values of total bilirubin (Walter and Gerard,1970; Burits & Ashwood , 1999).

3.2.3.6 Serum creatinine :

Reagent mixture was prepared by mixing 1ml picric acid with 1 ml sodium hydroxide.Mix and let stand for 2 minutes at 25 C° .

After read at 550 nm by using spectrophotometer (according to procedure recommended by the company Human ,Germany) (Schirmeister,J.,1964; Burits & Ashwood , 1999).

3.2.3.7 Serum urea determination :

Working solution was prepared by mixing the content of reagent 2 (urease) to the bottle of reagent 3 (phosphate buffer sodium salicylate, sodium nitroprusside and EDTA), and incubate for 5 minutes at 37 C°, and read at 580 nm by using spectrophotometer(according to procedure recommended by the bilirubin kit from the Biomerieux company , France) (Fawcett and Scott,1960; Burits & Ashwood , 1999).

3.2.3.8 Determination of total serum cholesterol :

The cholesterol is determined after enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4-amino-antipyrine in the presence of phenol and peroxidase. The quantity of this red dye quinoneimine formed is proportional to the cholesterol concentration, and was measured photometrically by using spectrophotometer at 505 nm wave length (according to procedure recommended by the total serum cholesterol kit from the Human company ,Germany) (Trinder,1969; Burits & Ashwood , 1999).

3.2.3.9 Determination of serum HDL :

Determination of HDL is occurred by specimen preparation by mixing with precipitant(phosphotungstic acid and Magnesium chloride) vigorously and let it to stand for 10 minute at room temperature and then centrifuge for 15 minutes at 3500-4000

round per minute (RPM). The supernatant is mixed with total cholesterol reagent and let it stand for 5 minutes at 37 C° and then record the absorbances at 500 nm against reagent blank .The reagent of the kit (precipitants) is only for treatment of specimens before determination of HDL with a reagent for total cholesterol . LDL, VLDL and chylomicrons from specimen are precipitated phosphotungstic acid and Magnesium chloride .The HDL obtained in supernatant after centrifugation then measured with total cholesterol reagent (according to procedure recommended by HDL kit from the Biolabo company , France) (Tietz,2006).

3.2.3.10 Determination of serum LDL :

The LDL concentration is calculated as follows :

LDL=Total cholesterol –(HDL+TG/5) (Andreoli *et al* ., 2004)

3.2.3.11Determination of serum triglycerides :

The triglycerides are enzymetically hydrolyzed to glycerol and fatty acids according to the following reaction:

$$\text{Triglycerides} \xrightarrow{\text{Lipoprotein lipase}} \text{glycerol + fatty acids}$$

So the TG are determined after enzymatic hydrolysis with lipases . Indicator is quinoneimine formed from hydrogen peroxide ,4-aminotipyrine and 4 –chlorophenol under the catalytic influence of peroxidase .The reagent (4 - aminotipyrine , 4 –chlorophenol,Magnesium ions ,ATP ,lipases ,peroxidase ,buffer (pH 7.5) glycerol kinase and glycerol -3- phosphate oxidase) was mixed with sample and standard and incubated for 5 minutes at 37 °C, and then measured photometrically by using

spectrophotometer at 500 nm wave length (according to procedure recommended by TG kit from the Human company, Germany) (Trinder,1969; Burits & Ashwood , 1999).

3.2.3.12 Determination of serum glucose :

The glucose is determined after enzymatic oxidation in the presence of glucose oxidase . The formed hydrogen peroxide reacts under catalysis of peroxidase with phenol and 4 – aminophenazone to a red – violet quinoneimine dye as indicator . the absorbance of standards and samples are measured against reagent blank at 546 nm (according to procedure recommended by the glucose kit from Human company ,Germany)(Barham and Trinder ,1972; Burits & Ashwood , 1999).

3.2.3.13 Determination of serum sodium:

Sodium is precipitated with Mg - uranyl acetate ; the uranyl ions remaining in suspension form a yellow-brown complex with thioglycolic acid (according to procedure recommended by the sodium kit from the Human company ,Germany) (Trinder ,1969; Burits & Ashwood , 1999).

3.2.3.14 Determination of serum potassium :

Potassium ions in a protein-free alkaline medium react with sodium tetraphenylboron to produce a finely turbid suspension of potassium tetraphenylboron. The turbidity produced is proportional to the potassium concentration and read photometricaly (according to procedure recommended by the potassium kit from

Human company ,Germany)(Hillman,1967; Burits & Ashwood , 1999) .

3.2.3.15 Determination of total serum calcium :

Calcium in the sample, reacts with O-cresolphtaleine at alkaline pH. The colored complex formed is proportional to the amount of calcium present in the sample. The intensity of the color was measured photometrically by using spectrophotometer at 570 nm wave length (according to procedure recommended by the serum calcium from Human company ,Germany) (Barnett,R.N.,1973; Burits & Ashwood , 1999).

3.2.3.16 Determination of serum copper and zinc :

Atomic absorption spectrophotometer method was used to determine the trace elements(copper and zinc) in serum samples. The serum samples were diluted (1:4) by adding distilled water and then digested with addition of concentrated nitric acid (HNO_3).The samples were injected into auto sample cup of atomic absorption spectrophotometer to read emitted lights. Standard curves were done for both copper and zinc. (Burits and Ashwood, 1999).

3.2.4 Statistical analysis:

All values were expressed as means ± SE. The data were analyzed by using of computer SPSS program and taking $p < 0.05$ as the lowest limit of significant . Student's t - test was used to examine the differences between different groups. Both t test and ANOVA test were applied to determine the differences between group and another and among all group and within group (Daniel, 1999) .

CHAPTER TWO

LITERATURE REVIEW

2.1 Anatomy and Histology of the Skin :

The skin is one of the largest organs in the human body . It is derived from ectoderm and mesoderm and has two anatomic layers : the epidermis or outermost nonvascular layer consists of several layers of epidermal cells that vary in thickness over various body surfaces, and the dermis or corium is largely made of collagen and contains the microcirculation, a complex vascular plexus of arterioles, venules, and capillaries. The two skin layers are bound together by a complex mechanism that is essential for normal function (Pendegrass *et al* ., 2006). While regarding the size and weight of the human skin, it is found that in an adult male, the skin weighs between 6 and 10 kg and the average adult skin surface area is about 1.5 to 2.0 m^2, in contrast to that of a newborn, whose skin surface area is only 0.2 to 0.3 m^2 . The two skin layers together are up to several millimeters thick, but both epidermal and dermal thickness varies depending on the body site. The epidermis is the thinnest (0.05 mm) over the eyelid but thicker (up to 1 mm) over the soles of the feet . The dermis is thickest on the back. Males generally have thicker skin than females. General skin thickness peaks in midlife and gradually thins as part of the aging process. Infants, young children, and elderly adults have a much thinner dermal layer to their skin, resulting in an increased propensity for deeper burn injury(Wysocki , 2002 **;** Church *et al* . , 2006).

Epidermal appendages are distributed throughout the dermis layer, including the sweat glands, sebaceous glands, and hair follicles. The dermal layer is capable of producing new epithelial cells to replace those

lost from the epidermis by burning or other injury to the skin because the shafts of these appendages are lined with epithelial cells. Nerve endings occur throughout both skin layers, and the connective tissue of the dermis also provides a firm structural base for the skin. Burn injury is a very painful form of trauma because of the multitude of pain receptors and nerves that traverse the skin layers. Beneath the skin lie the subcutaneous tissues, muscle, and bone (Church *et al* . , 2006) .

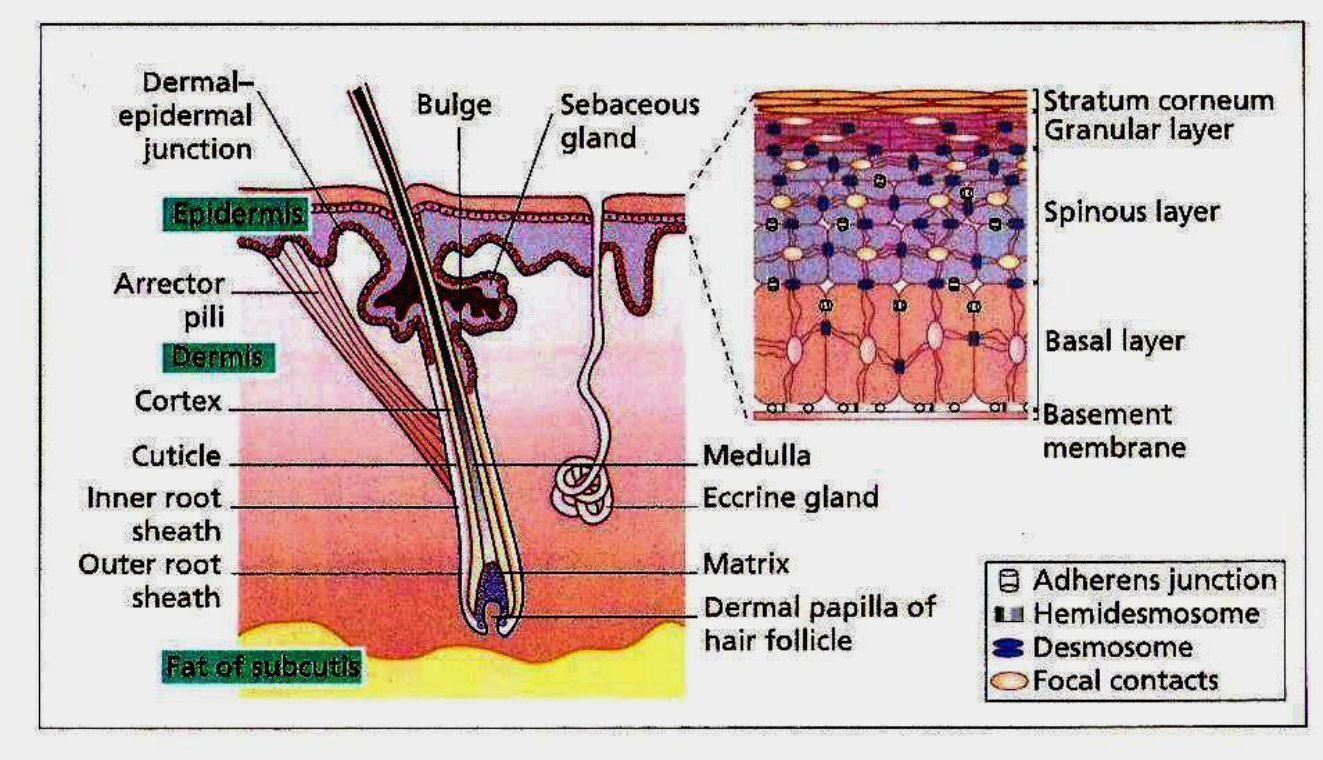


Figure 2.1 : Structure of the skin and subcutaneous tissues(Church *et al* . , 2006) .

2 .2 The Physiology of the Skin :

An intact human skin surface is vital to the preservation of body fluid homeostasis, thermoregulation, and the host's protection against infection. The skin also has immunological, neurosensory, and metabolic functions such as vitamin D metabolism. Thermal injury creates a breach in the surface of the skin. A basic knowledge of skin anatomy and physiology is

required to understand emergency burn assessment and approaches to burn care (DeBoer & Connor , 2004) . Epidermal cells are constantly being shed and replaced every month through a process that continually pushes new cells to the surface. This natural process is designed to continually replenish and heal breaches in the outermost protective skin barrier, be it from the microtraumas sustained as part of daily living or from overt injury. The epidermis therefore heals itself after superficial injury. Several important physiological functions of the skin are altered by thermal injury (Church *et al* . ,2006).

2.3 **Burns and their etiology :**

Burns are coagulative necrosis of skin and subcutaneous tissue that caused by the application of heat, cold, chemicals, electricity or radiation to the skin. When heat is applied to the skin, the depth of injury is proportional to the temperature applied, duration of contact, and thickness of the skin (Abston , 2000 ;Brunicardi *et al* . , 2005).

2.3.1 **Scald burns** :

Scalds, usually from hot water, are the most common cause of burns in civilian practice. Water at 60 degree centigrade (C°)creates a deep partial-thickness or full-thickness burn in 3 seconds. At 69 C°, the same burn occurs in 1 second. Boiling water always causes deep burns ; likewise, thick soups and sauces, which remain in contact with the skin longer, invariably cause deep burns. Exposed areas of skin tend to be burned less deeply than clothed areas, as the clothing retains the heat and keeps the hot liquid in contact with the skin for a longer period of time. Immersion scalds are always deep, severe burns (Yeoh *et al* . ,1994) .The liquid causing an immersion scald may not be as hot as with a spill scald ; however, the duration of contact with the skin is longer

during immersion, and these burns frequently occur in small children or elderly patients who have thinner skin . Scald burns from grease or hot oil are usually deep partial thickness or full - thickness burns, as the oil or grease may be in the range of 200 C° (Whitaker & Oliver , 2002).

2.3.2 **Flame burns** :

Flame burns are the second most common mechanism of thermal injury. They are caused by house fires , improper use of flammable liquids, motor vehicle collisions, and ignition of clothing by stoves or space heaters also are responsible for flame burns . Patients whose bedding or clothes have been on fire rarely escape without some full-thickness burns (Brunicardi *et al* . , 2005).

2.3.3 **Flash burns:**

Flash burns are next in frequency. Explosions of natural gas, propane, butane, petroleum distillates, alcohols, and other combustible liquids, as well as electrical arcs cause intense heat for a brief period of time (Still *et al* . , 1997 ; Yarbrough ,1998). Flash burns generally have a distribution over all exposed skin, with the deepest areas facing the source of ignition . Flash burns are typically epidermal or partial thickness, their depth depending on the amount and kind of fuel that explodes, but the burns generally cover a large total body surface area (TBSA) (Barnes *et al* . , 1989).

2.3.4 Contact burns :

Contact burns result from contact with hot metals, plastic, glass, or hot coals. They are usually limited in extent, but are invariably deep. Toddlers who touch or fall with outstretched hands against irons , ovens, and wood-burning stoves are likely to suffer deep burns of the palms (Sheridan *et al* . ,1999 ;Yen *et al* . , 2001) . Contact buns are often fourth-degree burns, especially those in unconscious or postictal patients, and those caused by molten materials (Margulies *et al* . ,1998).

2.4 Burn Severity:

There are many factors that determine the severity of a burn injury including the size of the burn which is the major factor, the depth of the injury, the age and general health of the patient and the presence or absence of inhalational injury (Glasheen *et al*., 1983).

A general idea of the burn size can be made by using the **rule of nines** for adult patients(figure 2.2) in which

- each upper extremity accounts for 9% of the TBSA .
- each lower extremity accounts for 18% .
- the anterior and posterior trunk each account for 18% .
- the head and neck account for 9% .
- the perineum accounts for 1 % .

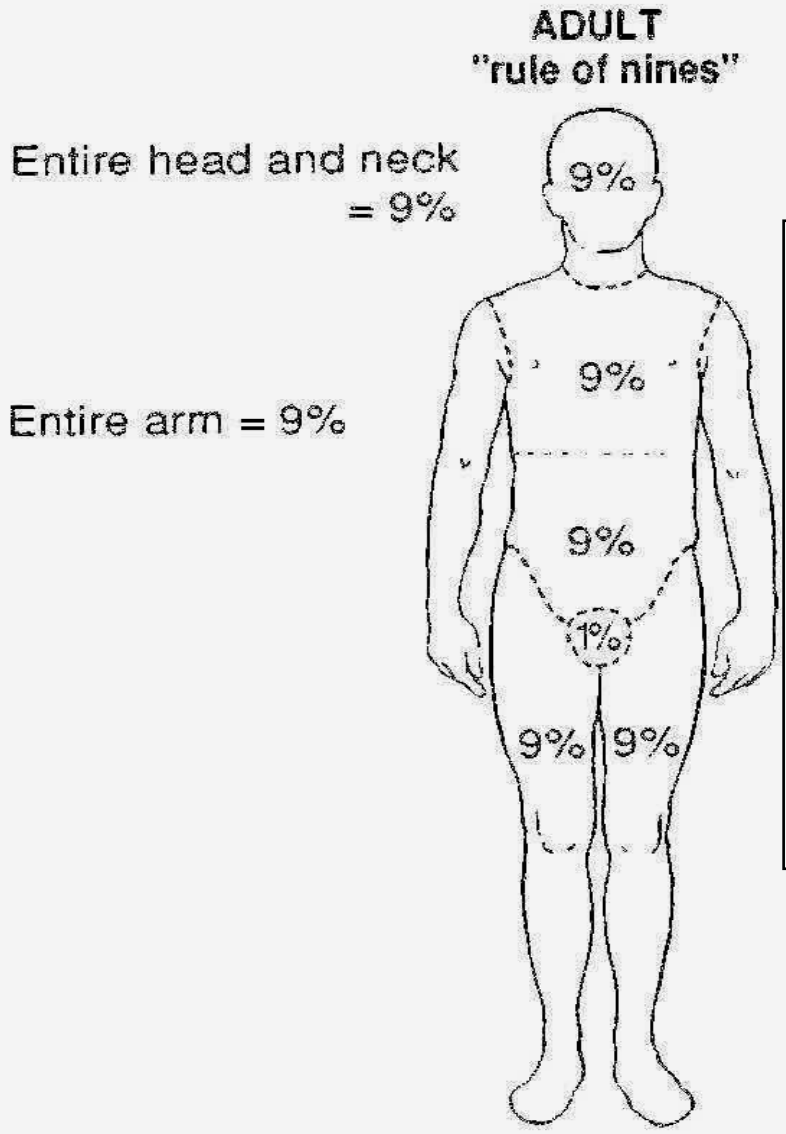


Figure 2.2: showing the distribution of body surface area by anatomic parts in the adults (Modified from Bongard and Sue,2002) .

While by using Lund - Browder charts (figure 2.3) that are particularly helpful in assessing pediatric burns because children under 4 years of age have much larger heads and smaller thighs in proportion to total body size than do adults. In infants the head accounts for nearly 20% of the TBSA ; a child's body proportions do not fully reach adult percentages until adolescence.

BURN ESTIMATE AND DIAGRAM

AGE vs AREA

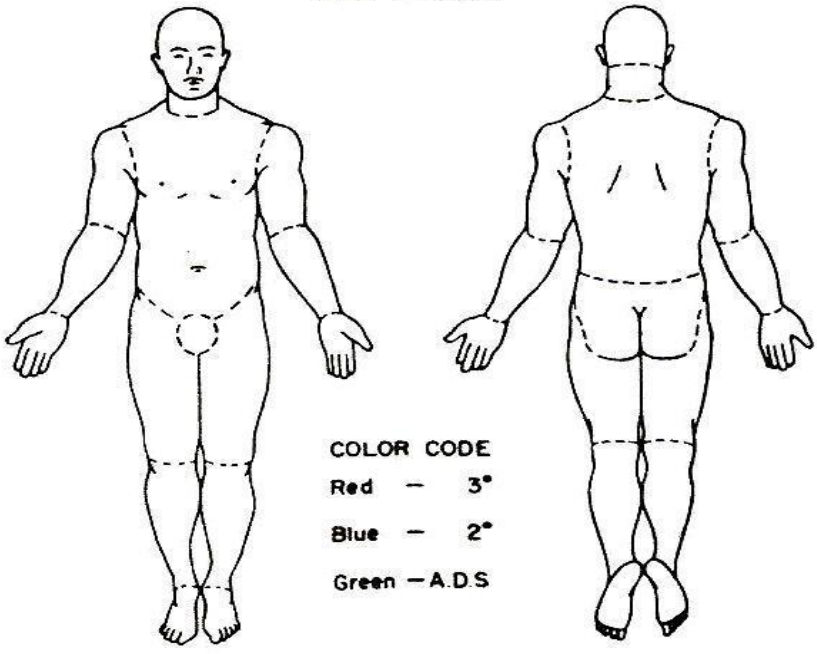
Area	Birth 1 yr	1–4 yr	5–9 yr	10–14 yr	15 yr	Adult	2°	3°	Total	Donor Areas
Head	19	17	13	11	9	7				
Neck	2	2	2	2	2	2				
Ant Trunk	13	13	13	13	13	13				
Post Trunk	13	13	13	13	13	13				
R Buttock	2½	2½	2½	2½	2½	2½				
L. Buttock	2½	2½	2½	2½	2½	2½				
Genitalia	1	1	1	1	1	1				
R U Arm	4	4	4	4	4	4				
L.U. Arm	4	4	4	4	4	4				
R L Arm	3	3	3	3	3	3				
L L Arm	3	3	3	3	3	3				
R Hand	2½	2½	2½	2½	2½	2½				
L Hand	2½	2½	2½	2½	2½	2½				
R Thigh	5½	6½	8	8½	9	9½				
L. Thigh	5½	6½	8	8½	9	9½				
R. Leg	5	5	5½	6	6½	7				
L. Leg	5	5	5½	6	6½	7				
R Foot	3½	3½	3½	3½	3½	3½				
L. Foot	3½	3½	3½	3½	3½	3½				
						TOTAL				

BURN DIAGRAM

AGE _____

SEX _____

WEIGHT _____



COLOR CODE
Red — 3°
Blue — 2°
Green — A.D.S

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Figure(2.3) : the use of burn diagram permit amore exact estimation of the extent of the burn .note that the surface areas of the head and lower extremities change significantly with age (Modified from Bongard and Sue,2002).

For smaller burns, an accurate assessment of burn size can be made by using the patients palmer hand surface including the digits, which accounts to approximately 1% of TBSA (Hettiaratchy & Papini, 2004).

Burn wound depth is a significant determinant of patient's treatment and morbidity. According to their increasing depth, burn wounds are classified into four categories: epidermal(first degree burn), superficial and deep partial thickness(second degree burn), full thickness (third degree burn) and full thickness with underlying structure (fourth degree burns) (Johnson & Richard, 2003). Superficial burns and superficial partial thickness burns typically heal without any need for surgical excision and grafting. Dressing changes and daily wound care can remove necrotic debris and facilitate healing with minimal scaring. For deep partial thickness and full thickness burns, however, operative debridement with subsequent skin graft coverage is necessary(Klein *et al*., 2004). As well as , Kao & Garner,(2000) and Hettiaratchy & Papini,(2004) **classified the burn** in to four degrees which are :

First degree burns are minor epithelial damage of epidermis exists with redness, tenderness and pain but without blistering.

Second degree burns are superficial partial and deep partial thickness burns in which some portion of the skin appendages remains viable, allowing epithelial repair of the burn wound without skin grafting. The superficial partial thickness burn involves the epidermis and superficial (papillary) dermis. They are pink, moist, soft and tender, healing in approximately 2-3 weeks usually without scarring by outgrowth of epithelial buds from the viable pilosebaceous units and sweat glands residing in papillary and reticular dermis. Deep partial thickness burns extend into the reticular dermis and heal within 3-6 weeks, but with scarring. Skin graft is preferred for this type of burn.

Third degree burns are full-thickness that destroy both epidermis and dermis. Skin grafting is always necessary to resurface the injured area.

Fourth degree burns cause full thickness destruction of skin and subcutaneous tissue with involvement of underlying fascia, muscle, bone or other structures .

2.5 Pathology and Pathophysiology of thermal injuries:

The breached skin barrier is the hallmark of thermal injury. The body tries to maintain homeostasis by initiating a process of contraction, retraction, and coagulation of blood vessels immediately after a burn injury. Three distinct zones have been defined within the burn wound : (i) **The zone of coagulation**, which comprises the dead tissues that form the burn eschar that is located at the center of the wound nearest to the heat source.

(ii) **The zone of stasis**, which comprises tissues adjacent to the area of burn necrosis that is still viable but at risk for ongoing ischemic damage due to decreased perfusion.

(iii) **The zone of hyperemia**, which comprises normal skin with minimal cellular injury that has predominant vasodilation and increased blood flow as a response to injury (Gibran & Heimbach , 2000).

A rapid loss of intravascular fluid and protein occur through the heat - injured capillaries. The volume loss is greatest in the first 6 - 8 hours, with capillary integrity returning toward normal by 36 - 48 hours. In addition, there is an increase in interstitial osmotic pressure that accentuates the edema. A transient increase in vascular permeability also occurs in non burned tissues, probably as a result of the initial release of vasoactive mediators. However, the edema that develops in non burned tissues during resuscitation appears to be due in large part to the marked hypoproteinemia caused by protein loss into the burn itself . A generalized

decrease in cell energy and membrane potential occurs as a result of the early decrease in tissue perfusion. This leads to a shift of extracellular sodium and water into the intracellular space, which in turn increases fluid requirements. This process is also corrected as hemodynamic stability is restored (Bongard & Sue,2002).

Inhalation injury , a complex and deadly disease process , occur when the heat and toxins in smoke that formed in the site of accident make contact with airway mucosa and alveoli . The degree of injury depends on the composition of smoke ,which varies according to its source heat affect primarily the supraglottic area and causes edema and upper airway obstruction ,whereas the gas and particle components of the smoke affect primarily the airway mucosa and cause the actual chemical burn . Smoke inhalation markedly increases the hemodynamic instability, fluid requirements, and mortality rates (Church *et al* . , 2006).

2.6 HYPERMETABOLISM:

Energy expenditure after burn injury can be as much as 100 %above the prediction based on standerd calculation for size , age , sex and weight . Some debate persist regarding the genisis of this phenomenon ,but increased heat loss from the burn wound and increase β-adrenergic stimulation are probably primary factors.(Kelemen *et al* . ,1996). Radiant heat loss is increased from the burn wound secondary to increased blood flow and integumentary loss (Yu *et al*. ,1999). Glucose is elevated in almost all critically ill patients , including those with burn injuries .Gluconeogesis and glycogenolysis are increased in burn patients. In addition , plasma insulin levels are typically elevated in burn patients .The basal rate of glucose production is elevated despite this hyperinsulinemic state , which can be defined as hepatic insulin resistance ,since the cortisol decrease the binding of insulin to insulin

receptors in muscles and adipose tissue (Vemula *et al* . ,2004). Hyperglycemia complicate the acute management of many significant burn and may be related to poor outcomes ,specifically increased mortality and decreased graft take (Gore *et al* . , 2001). Further hyperglycemia may exacerbate muscle catabolism in burn patient while not influencing energy expenditure (Gore *et al* . , 2002). Exogenous insulin administration to achieve euglycemia has been shown to decrease donor site healing time and decrease length of stay , while ameliorating skeletal muscle catabolism (Pierre *et al* ., 1998; Thomas *et al* ., 1999).

Lipolysis occur at a rate in excess of the requirement for fatty acids as an energy source due to alterations in substrate cycling .In burn patients , the majority of the released fatty acids are not oxidized , but rather resterified in to triglyceride ,resulting in fat accumulation in the liver . The acute and long term consequences of this hepatic steatosis are unclear . However in pediatric autopsy study , 80% of fatalities over 10 years had hepatic steatosis ,and it applied to correlated with sepsis (Barret *et al* . ,2001). The β - blockade by using propranolol appear promising as a means to manipulate peripheral lipolysis and potentially prevent hepatic steatosis , although clinical outcome data with respect to fatty metabolism are still lacking.

Proteolysis is increased in burn patient as compared to normal individual who are fed isonitrogenous , isocaloric diets . Following utilization , protein is exerted as urea .This may be result from increased efflux of amino acid from skeletal muscle pool , including gluconeogenic amino acids . In particular , alaninic and glutamine are released at increased rate . Muscle protein breakdown is accelerated while acute phase proteins are produce at increased rate in the liver (Barret *et al*. ,2001).

Wound healing requires enhanced protein synthesis and increased immunologic activity . Protein intake greater than 1 g/Kg B.W. per day has been recommended for all thermally injured patient and for burn patient with normal renal function ; the recommended protein intake is 2 g/kg B.W. per day (Wischmeyer *et al* .,2001; Brunicardi *et al* . , 2005). Enteral ornithine α-ketoglutarate (OKG) supplementation was associated with increased plasma glutamine levels ,enhanced wound healing ,and improved nitrogen metabolism (Coudray-Lucas *et al* . , 2000). The anabolic steroid oxandrolone also has shown to improve donor site healing time ,diminish weight loss and blunt protein catabolism during acute phase of wound healing (Demling & Orgill , 2000).

2.7 Neuroendocrine response in burn injury:

Catecholamines are massively elevated following burn injury ,and appear to be the major endocrine mediators of the hypermetaolic response in thermally injured patients . Pharmacological beta- blockade utilizing propranolol diminishes the intensity of postburn hypermetabolism in pediatric patients as demonstrated by improved skeletal muscle protein kinetics with diminished oxygen consumption ;however , clinical outcome data are still lacking , and these results have yet to be demonstrated in adult burn patients (Herndon *et al* . ,2001).

Conversely , growth hormone (GH) levels are attenuated following thermal injury . Although early studies of exogenous GH as an anticatabolic agent were promising in the pediatric population , ultimately its use has been supplanted by less expensive , safer ,and equally effective pharmacotherapies (Murphy *et al* . , 2003) . Propranolol has been shown to be superior to GH ,while oxandrolone is equally as effective as GH in ameliorating catabolism . Both propranolol and oxandrolone has lower significant side effects than GH , and oxandrolone

appears to be equally effective in adults and children (Demling & Orgill ,2000 ; Hart *et al* . ,2002) .

Serum thyroid hormone concentration are altered in patients with large burn .Total thyronine (T_3) and thyroxin (T_4)concentration are reduced , and reverse T_3 concentrations are elevated ,while cellular concentration are likely normal . Concentration of free T_3 and T_4 fall markedly in the presence of sepsis in burned patients (Vaughan *&* Pruitt ,1993).

Burn injury abolish the normal diurnal variatuion in glucocorticoid secretion, producing persistent hypercortisolemia . Although catabolic , cortisol does not appear to appreciably influence metabolic activity alone ,but acts additively and synergistically with the catecholamine and glucagons . glucagons concentrations are related directly to metabolic rate and appear to exert effects via insulin and insulin like growth factors-1 modulation (Nygren *et al* . ,1995) .

2 .8 **Immunological response in burn injury:**

A number of immunologic abnormalities in burn patients predispose to infection. Serum immunoglobulin A and serum immunoglobulin M, are frequently depressed, reflecting depressed B cell function. Cell-mediated immunity or T cell function is impaired, as demonstrated by prolonged survival of homografts and xenografts. A decrease in interleukin-2 production due to circulating mediators may be responsible .An excess of suppressor T cell activity is seen in burned patients, and the degree of activity has been found to be a good predictor of sepsis and eventual fatality (church *et al* . , 2006). Polymorphonuclear (PMN) leukocytes chemotactic activity is suppressed. This has been attributed by some to a circulating inhibitory factor released from the burn wound. A decrease in chemotaxis predates evidence of clinical sepsis by several days (Rosenthal *et al* .,1998). Decreased oxygen consumption and impaired

bacterial killing have also been demonstrated in PMNs. Depressed killing is probably due to decreased production of hydrogen peroxide and superoxide; this has been demonstrated by decreased PMN chemiluminescent activity in burn patients (Sabeh *et al* . , 1998).

2.9 Hematological studies :

2.9. 1 Red blood cells(RBCs):

They are biconcave discs, that are manufactured in the bone marrow in response to erythropoietin stimulation via erythropoiesis process (Ogawa, 1993). In mammals, they lose their nuclei and mitochondria before entering in to blood circulation, and survive for average of 120 days. The vital function of RBCs is to carry oxygen to different parts of the body because they contain hemoglobin by which oxygen is transported (Broudy,1997). The mature red blood cell derives its energy in the form of adenosine triphosphate (ATP) from anaerobic glycolysis by conversion of glucose to lactate. Adenosine triphosphate generated by glycolysis is essential for integrity and stability of the cell membrane, in part this achieved by maintaining the concentration gradients of cations across cell membrane through Na^+-K^+ ATPase (Deluise & Flier, 1983; McCarrol, 1995). The 2,3-diphosphoglycerate is an intermediate metabolite of glycolysis, It is important compound because it binds with hemoglobin and dissociates of oxygen (Zere & Tanaka,1989) . Pentose phosphate pathway in which reduced nucleotides such as reduced nicotinamide adenine dinucleotide - phosphate (NADPH) are produced and used in reduction reactions that occurring in RBCs (Hsia, 1998) . Destruction of red blood cells following thermal injury occur to an extent proportion to the size and depth of burn .In area of full thickness burn, the coagulation occur in the involved microvasculature. There is continuing red cell mass loss per day caused by continued lysis of cells damaged by heat ,

microvascular thrombosis in zones of ischemia that subsequently become necrotic ,and repeated blood sampling. In the early postburn period , platelets number and fibrinogen levels are depressed ,with a corresponding rise in fibrin split products. Following resuscitation, platelets and serum levels of fibrinogen and factors V and VIII rapidly increase to supranormal levels . Erythropoietin levels are increased coincident with the anemia following thermal injury . Recent studies have suggested that the rate of erythropoiesis may be further increased by administration of recombinant erythropoietin and iron . However ,a decrease in transfusion requirement has yet to be demonstrated (Bongard and Sue,2002). It is well known that a severely burned patient presents the greatest deregulation of homeostasis of any injury . It was found that a general relationship exists between the extent of deep burn and the amount of red cell destruction. There was a shorter life span of red blood cells and about 10% of the total red cell mass is injured during the burn process. All these changes have been attributed to the presence of some type of detrimental plasma factor, because when the red cells are injected into a normal person they survive a normal length of time .Also, the serum of burn patients contains a substance that inhibits erythropoiesis (EI-Sonbaty & EI-0tiefy ,1996) .

2.9.2 Hemoglobin(Hb)

Hb is oxygen transporter in erythrocytes. Hemoglobin consists of four polypeptide chains, two of one kind and two of another .The four polypeptide chains are held together by non covalent attractions. Each contains a heme group, which consists of porphyrin ring with iron resides in the center (Turgeon, 2005). The values of Hb are normally within 13.5-17.5 for males and 12-16 for females (Andreoli *et al* ., 2004). In burn patient hemoglobin concentrations showed significantly high levels

immediately after the burn, especially in the non-survivors. This high level decreased gradually to below control level by day 4 post-burn in the non-survivors and by day 6 postburn day in the survivors(EI-Sonbaty and EI-0tiefy ,1996). Although it is helpful to monitor the hemoglobin concentration ,changes in these values may not accurately reflect the changes in blood volume . The rate of plasma loss often exceeds the rate of whole blood loss , which means that the hematocrit may be normal even in the face of severe volume depletion ; consequently ,blood loss can easily be underestimated .If hemoglobin declines in the absences of hemolysis, this clear indication that there is significant source of blood loss somewhere. After large burns , normalization of blood volume is almost impossible until 24 to 48 hours after burn (Dauglas,*et al* . ,2003) .

2.9.3 Packed cell volume (PCV)or Hematocrit:

PCV, is the ratio of blood volume of RBCs to the volume of whole blood. PCV is usually expressed as a percentage (Lewis *et al* . , 2006). The values of PCV are normally within 39 - 49 % and for male 35-45 % for females (Andreoli *et al* ., 2004). Other study illustrated that one of the parameters that used to judge the efficacy of fluid replacement is hematocrit . Hematocrit, blood pressure and pulse have significant limitations as indicators of shock in the burned patient. It is often quite difficult to obtain an accurate pulse or blood pressure through the thick tough eschar of a severe burn. The blood pressure in children and young adults is often stable until late in the clinical picture of shock. Hypertension may be found frequently in severely burned children . With the increased metabolic rates associated with thermal trauma, a pulse in excess of 100 is often found and is compatible with adequate fluid resuscitation (Stewart,1998).

2.9.4 White blood cells (WBCs) or leukocytes:

Leukocytes are nucleated cells of blood tissue. The granulocytes (polymorphonuclear leukocytes)are the most numerous, since, these cells have multilobed nuclei as the cell grow older (Borregaard and Cowland, 1997). Most of them contain neutrophilic granules (neutrophils). But a few contain granules that stain with acidic dyes (eosinophils)and some have basophilic granules (basophils) (Metcalf, 1991). The other two cell types found normally in peripheral blood are: lymphocytes, which have large round nuclei with scanty cytoplasm; and monocytes, which have kidney- shaped nuclei . By acting together, these cells provide the body with powerful defenses against tumors, viral, bacterial, fungal, and parasite infections (Hoff brands *et al*., 1999).). The values of WBC are normally within 4.5-11 * 10^3 cell /µL and the percentages of differential counts are for neutrophile (54-62%) ,lymphocytes (23-33%) , monocytes (3-7%) ,eosinophile (1-3%) and basophile (0-0.75) (Andreoli *et al* ., 2004).

Catecholamines are massively elevated following burn injury ,and appear to be the major endocrine mediators of the hypermetabolic response in thermally injured patients (Herndon *et al* . ,2001). So it is found that adrenaline injection causes an increase the leukocyte count ; here , too increases in the numbers of all major types of leukocytes (and platelets) occur, possibly reflecting the extent of the reservoir of mature blood cells present not only in the bone marrow and spleen but also in other tissue and organs of the body . Emotion may possibly cause an increase in the leukocyte count in a similar way (Lewis *et al*. ,2006).

2.9.5. Platelets counts:

Platelets are the smallest but most abundant cell type found in the circulation and range in numbers from 150 x 10^9/L to 400 x 10^9/L in humans (Klinger *&* Jelkmann , 2002) . They are enucleated cells derived

from the fragmentation of megakaryocytes and contain preformed compartmentalized proteins as well as messenger RNA (George, 2000). While platelets primarily function to maintain hemostasis, they also participate in tissue repair and wound remodeling and in antimicrobial host defense(Szpaderska *et al* .,2003; Weyrich *&* Zimmerman ,2004). Thus, platelets can be perceived as nomadic "sentinels" capable of responding instantly to chemical changes in their environment and acting as a first line of defense after injury or bacterial invasion (fumjii *et al* . ,2006) .The activation of platelets in response to injury or bacterial infection initiates the formation of platelet aggregates and the expression of cell adhesion molecule receptors and costimulatory molecules such as P-selectin (CD62P), CD40, and CD154 (Danese *et al* .,2004 ; Weyrich *&* Zimmerman . *,* 2004). Activated platelets are also capable of releasing proinflammatory cytokines such as interleukin -1 beta (IL-1β) and immune - regulatory cytokines such as transforming growth factor beta-1 ($TGF\beta_1$) (Klinger *&* Jelkmann ,2002*)*. Platelets also play a role in the innate immune response, since they express the pattern recognition molecules referred to as toll-like receptors (TLRs), which recognize a broad range of microbial antigens (Shiraki *et al .,* 2004 ; Cognasse *et al*. , 2005). The ability of platelets to respond to their environment and their subsequent expression and release of cell surface molecules and cytokines suggest that they may play a role in inflammatory responses. In support of this idea, platelets have been shown to participate in inflammation of the vessel wall during the development of atherosclerosis where they interact with the endothelium and infiltrating monocytes, in the synovium during arthritis development, and in the mucosal capillaries during active inflammatory bowel disease (Danese *et al* .,2003 ;Angeli *et al .,*2004).

As well as , it is found that platelets may participate in the intercellular communication network between injured tissue and the immune system.

This may occur as part of the innate immune response, through the expression of platelet TLRs, or through other specific ligand-receptor interactions. Thus, an injury-induced change in the number of circulating platelets or platelet-derived products or alterations in platelet function may be detrimental. This has been suggested by the clinical observation that platelet deficiency correlates with a higher mortality after severe trauma and after sepsis (Takashima *et al .,* 1997;George *et al* ., 2001).

2.10 Biochemical studies :

2.10.1 Total serum proteins TSP:

Proteins are the structural and the functional components of a cell. The functional components serve as biocatalysts (enzymes) ,regulator of metabolism (hormones) . The normal value of total serum protein lies between 60 -80 g / L(Andreoli *et al* ., 2004).Plasma proteins and tissue proteins share the same amino acid pool building blocks of proteins, and thus alterations in one group will eventually affect the other. So, the plasma proteins can be hydrolyzed to amino acids, which can be used for production of energy (Hershko & Ciehanover, 1992).

2.10.2 Serum albumin:

Albumin is the protein present in highest concentration in the serum. It is synthesized in the liver. The normal value of serum albumin lies between 35 -50 g / L(Andreoli *et al* ., 2004). Albumin has two well-known functions, one is the contribution albumin makes to the colloid osmotic pressure of intravascular fluids, due to its high concentration, which maintains the appropriate fluids in the tissues (Geveart & Vandekerchohove,2000). The other prime function is its propensity to bind various substances in the blood such as albumin binds bilirubin, fatty acids, hormones, metals, drugs and ions (Murray *et al*., 2003) It is found by Chen *et al* . ,(2003) that early albumin resuscitation aggravated the

burn - induced gut damaged . Lee-Wei *et al* . , (2005) found that thermal injury induced lung damage when restoration of extracellular fluid in early burn shock with albumin markedly augmented the lung neutrophiles deposition, lung permeability increase . In both of these studies , it is found that albumin administration and inhibition of the inducible isoform of nitric oxide synthase decreased burn -induced gut barrier dysfunction and reversed the damaging effect of albumin on gut barrier and decreased bacterial translocation and reversed its damaging effects on thermal injury-induced lung dysfunction to beneficial ones. Ruot *et al* ., (2000)suggest that hypoalbuminemia is not due to reduced albumin synthesis during sepsis. Therefore, albumin synthesis measured in the plasma is a good indicator of liver albumin synthesis .

2.10.3 Serum globulin:

The globulin fraction is subdivided into numerous components. One classification divide it into alpha l, alpha 2, beta1 , beta 2 and gamma globulins. The normal value of serum globulin lies between 24 -37 g / L(Andreoli *et al* ., 2004). The immunoglobulin are synthesized in the reticuloendothelial system, and other globulins are synthesized in the paranchymal liver cells (Pepys & Hirchfield , 2003). The alpha-globulins carry metals, lipids, whereas glycoproteins are a group of carbohydrate containing proteins which are important constituents of the alpha glycoproteins (Brockhausen,1993). Beta-globulin are as apolipoproteins that combined with cholesterol and other lipids, and also, as individual binding globulins, carries fat soluble vitamins and hormones (Myers *et al*., 1994).

2.10.4 Serum cholesterol (total , HDL and LDL) :

Cholesterol is unsaturated steroid alcohol of high molecular weight. In its esterified form ,it contains one fatty acid molecule(Javitt,1995). The normal values lie between (3.9 -6.5) , (0.9-1.4) (1.8-4.3) mmol/L for total serum cholesterol , HDL and LDL respectively (Andreoli *et al* ., 2004) .The high solubility of cholesterol blood is due to plasma lipoprotein mainly low-density lipoprotein (LDL)and very low density lipoprotein (VLDL) that have the ability to bind and thereby solubilize large amount of cholesterol (Sundaram *et al*.,1997). LDL delivers cholesterol to various tissues that require cholesterol for membrane structure or steroid hormone synthesis. While high density lipoprotein (HDL), which is rich in cholesterol and has a low triglyceride content, is the main vehicle for carrying excess cholesterol from peripheral tissues to the liver, where it can be excreted in bile either directly in the form of cholesterol or after conversion to bile (Sassolas & Cartier, 1999; Andreoli *et al*., 2004).The cholesterol biosynthetic pathway was suppressed following burn injury, while cholesterol was increasingly imported and converted into bile acids(Vemula *et al* . ,2004). The hypocholesterolemia seen in critically ill and injured patients, especially those with sepsis. It is found that the importance of this is related to the ability of lipids and lipoproteins to bind to and neutralize bacterial endotoxin lipopolysaccharide (LPS) (Wilson *et al* . ,2003).

2.10.5 Serum triglycerides (TG) :

Triglycerides molecule comprises one molecule of glycerol with their fatty acids including both saturated and unsaturated fatty acids. The source of triglycerides in the body can be either dietary and /or synthesized in liver and other tissues (Warnik, 1991). The normal value of serum TG. lies between (0.9 - 2.4) mmol/L (Andreoli *et al* ., 2004).

Chylomicrons are the largest of the lipoprotein particles and represent the major carriers of exogenous dietary triglycerides. Very low density lipoprotein , like, chylomicrons, are also rich in triglycerides. They are the major carriers of endogenous (liver synthesized) triglycerides (Hussain 1996). Triglycerides may be enzymatically hydrolyzes to release the fatty acids and glycerol. Lipoprotein lipase hydrolyzes triglycerides in the circulation and , this enzyme is located on the surface of endothelial cells of capillaries. Hormone-sensitive lipase, the other enzyme, acts inside fat cells to release free fatty acids from triglycerides. This enzyme is stimulated by epinephrine ,cortisol and thyroxine hormone (Cachefo *et al*. ,2001). Vemula et al . ,(2004) pointed out that fatty acid (FA) and triglyceride biosynthesis in the liver were unchanged, whereas TG utilization, FA import, and ß-oxidation increased after burn injury. The increased FA pools after burn injury appear to serve as substrates for ATP production. The FA are increasingly imported and oxidized in the liver to meet the enhanced energy demands arising from an inflammatory response during the first 24 h after burn injury.

2.10.6 Liver enzymes :

The liver plays an important role in the body's response to thermal injury. It is the principal organ responsible for producing acute-phase proteins and modulating the systemic inflammatory response.(Jeschke, 2002). Following thermal injury, the acute-phase response brings about the activation of the coagulation , complement cascades, granulocytes, , monocytes ,macrophages as well as platelets, (Xia *et al* ., 1992) and induces the liver to synthesize and release proteins that exert effects on a variety of tissues. After major trauma, such as a severe burn, hepatic protein synthesis shifts from hepatic constitutive proteins, such as albumin, prealbumin, transferrin, and retinol-binding protein, to acute

phase proteins, which serve as mediators of the inflammatory process, function as transport proteins, and participate in burn wound healing. (Tilg *et al* . ,1997) . It was demonstrated that the acute-phase response persists for longer duration than previously thought, which suggests that any compromise in liver function as seen in patients with chronic liver disease has both short-term and long-term detrimental effects on the body's ability to respond to burn injury (Jeschke *et al* ., 2004).). The normal values lie between (<20) , (<20) and (30-85) IU for s.GOT,s. GPT and s.ALP respectively (Andreoli *et al* ., 2004) .

2.10.6.1 Transaminases :

Transaminases are enzymes which are responsible for the transfer of amino group from one amino acid to keto acid which is important for kreb s cycle .Two clinically important enzymes within this group are :glutamic oxaloacitic transaminase (GOT) and the second enzyme glutamic pyruvic transaminase (GPT) . It is found that the distribution of enzyme GOT is widely with high level in liver ,heart ,skeletal muscles and red blood cells .Hence ,any damage to these tissues leads to increase the level of this enzyme ,GPT is normally presented in the same tissues in spite of its concentration in the liver which is higher than other tissues(Murray *et al*. ,2003) .

Serum GOT and serum GPT (aminotransferases) are intracellular amino transferring enzymes present in large quantities in hepatocytes .After injury or death of liver cells , they are released into the circulation . In general ,the serum aminotransferases are sensitive tests of liver damage , the height of serum aminotransferase activity level reflects the severity of hepatic necrosis, with important exceptions .For instance , both enzymes require pyridoxal 5 – phosphate as a cofactor , and the relatively low serum aminotransferase values seen in patients with

alcoholic hepatitis may reflect deficiency of this cofactor . Although aminotransferase level are increased in a wide array of liver diseases, high levels (>15 times the upper limit of normal) generally indicate acute hepatocellular necrosis from viral or toxic causes or less frequently indicate acute bile duct obstruction or hepatic ischemia . Patients who present with isolated asymptomatic elevation of GOT and GPT (as in burn patients) may have nonalcoholic fatty liver disease caused by obesity ,insulin resistance diabetese or hyperlipedimia . ; alchohol – induced liver disease or hepatocellular disease ,such as hemochromatosis or chronic viral (Andreoli *et al*., ,2004).

2.10.6.2 Serum Alkaline Phosphatase (ALP):

The phosphatases are a group of enzymes which promote the hydrolysis of organic phosphates with liberation of phosphate ions .These enzymes classification depending on wether the enzyme has maximum activity in alkaline or in acid medium (Bikle ,1997).

ALP has a major concern with bone metabolism , and produced in many cells of the body ,one important site of secretion is the osteoblasts . The main other alkaline phosphatase isoenzymes come from liver ,placenta , and sometimes from intestinal mucosa (Biscoveanu and Hasinsk,2000).

2.10.7 Serum Sodium :

Sodium, a blood electrolyte, is the most abundant cation and the chief base of the blood. Its primary functions in the body are to chemically maintain osmotic pressure, acid -base balance, and to transmit nerve impulses (Kumar & Tomas, 1998). The normal value of serum potassium lies between (136 -146) mEql/L (Andreoli *et al* ., 2004) A multiple regulatory mechanisms have been evolved to control excretion of this ion.

Sodium concentration is under control of the kidneys and the central nervous system acting through the endocrine system (Adrogue & Medians, 2000). Adrenal mineralocorticoids such as aldosterone increase tubular reabsorption of sodium ion in association with excretion of potassium and hydrogen ions (White, 1994). Atrionatriuretic peptide (ANP),a peptide hormone, secreted by atrial myocytes enhances excretion of sodium ions by the kidneys (Maack, 1992). Ge *et al* . ,(1996) showed that serum sodium ions decreased post-burn and increased after resuscitation in burns patients . In the early post-resuscitation period between 2 – 6 days in which hypernatraemia presents in various forms, depending on the amount of water retained: peripheral oedema, ascites, pleural effusion, and interstitial or alveolar oedema (with possible impaired ventilation) may dominate, or alternatively manifestations of dehydration may be more significant . .

2.10.8 Serum Potassium:

Potassium is the principal cation in the cells, and it is present in relatively low concentration in the extracellular fluids. Potassium plays important role in nerve conduction and muscle function. Moreover, it helps maintain acid - base balance and osmotic pressure (Gennari, 2002)). The normal value of serum potassium lies between (3.5 - 5.1) mEql/L (Andreoli *et al* ., 2004). The kidneys are important in the regulation of potassium balance. Initially, the proximal tubules reabsorbed nearly all the potassium ions, then, under the influence of aldosterone, additional potassium ions are secreted in to urine in exchange for sodium ions in both distal tubules and colleting ducts (Whany & Sims, 2000). Early hypokalaemia had corrected towards normal within a few hours, without potassium supplementation. In adults, hypokalaemia is well recognised after stress states and is due to a combination of the effect of adrenaline

and insulin. Adrenaline stimulates β-receptors on skeletal muscle with consequent uptake of potassium from the circulation(Raineret *et al* . ,1999).

2.10.9 Serum Calcium:

More than 99% of calcium in the body is part of bones and teeth .The remaining 1% is mostly in the blood and other extra cellular fluids. Very little amount is in the cytosol of most cells(Bushinsky & Monk, 1998). The normal value of serum potassium lies between (2.1 - 2.55) mmol/L (Andreoli *et al* ., 2004) The plasma calcium is found in two States partly bound to protein and partly diffusible. It is the free ionized calcium in the body fluids, that is vital second messengers and is necessary for blood coagulation, muscle contraction, and nerve function. Calcium is absorbed from small intestine by Ca^{+2} - H^{+} ATPase pump and passive diffusion (Wandrup,1989). Three hormones probably operate to maintain the constancy of the calcium level in the body are parathyroid hormone, vitamine-D3,and calcitonin (Glenn, 1995; Nissenson, 2000) . Murphey *et al* . , (2000) pointed out that their findings are consistent with up-regulation of the parathyroid calcium-sensing receptor (Ca R) and a related decrease in set-point for calcium suppression of parathyroid hormone secretion that may contribute to the previously reported postburn hypoparathyroidism and hypocalcemia. Hypovitaminosis D observed in burn injury correlates with serum calcium and phosphorus abnormalities. Early after injury (<1 week) there was no observed correlation between vitamin D and other variables possibly because of the effects of burn shock. After 1 week, vitamin D appears to significantly effect phosphorus homeostasis. The relationship between vitamin D and magnesium is not well established. These results may indicate a role for

vitamin D replacement therapy during the initial phase of burn resuscitation (Wray *et al* ., 2002).

2.10.10 Trace elements:

A few elements are present in the body in small quantities that they are called trace elements. Usually, the amount of these in the foods are also minute. Those believed to be essential for life at normal levels. Conversely, some of them can be toxic when present in excess (Standstead, 1995). Trace elements are inorganic substances found in human blood in the order of micrograms. These elements are incorporated into the structures of proteins, enzymes, and complex carbohydrates. They take part in biochemical reactions together with enzymes. Zinc (Zn) and copper (Cu) are especially active in metabolic and biochemical processes in the recovery phase of wounds. It is known that the trace elements are necessary for the continuous systematic functioning of the immune system . In burns, there is an increase in certain metabolic processes, such as tissue repair, wound healing, microcirculation, and oxygenation. These trace elements that take part in these processes are of primary importance to the burn patient.(Safran,1999).

2.10 .10 .1 Serum Copper

Copper is an essential trace element. It is required in the diet because it is the metal cofactor for a variety of enzymes including ceruloplasmin, cytochrome oxidase, superoxide dismutase, dopamine beta-hydroxylase, ascorbate oxidase, lysyl oxidase, and tyrosinase (Walshe, 1995). Copper is absorbed in the stomach and upper small intestine, after diffusion across luminal membrane, copper is bound to metallothionein in the cytosol of enterocytes. Metallothionein also found in other cells of the body to provide protection of this metal available to generate free radicals (Harris ,2000) .In liver, part of copper is excreted in the bile, and the

other part leaves the liver attached to ceruloplasmin, which is synthesized in that organ. Ceruloplasmin is an alpha2-globulin and has a high copper content and carries 90% of the copper and the remainder 10% is carried with albumin (Saari & Schuschke ,1999) .There is now evidence that the alteration of the immunological status in burn patients is strongly related to the trace element (TE) .Since it is found deficit in TE in these patients. Trace element , especially Ca, Se, and Zn, play a key role in many metabolic and immune pathways and are involved in both humoral and cellular immunity. Their deficiency may lead to decreased antibody production, reduced T-cell counts, decreased neutrophil function, and decreased natural killer cell activity .The increased in amounts of micronutrients given to burned patients in the early period following injury reduce the incidence of infections (Marvaki *et al* .,2001) . Several factors affect the return of the reduced serum trace element levels to normal. First of all, the increased intake of the elements, nutritional support, parenterally administered fluids, plasma, albumin, and globulin and blood transfusions affect the blood levels of the elements. The most important factor however, is the healing of the burn wound. (Safran et al .,1999).

2.10.10 .2 Serum Zinc :

The physiological functions of zinc are based largerly on its presence as an essential component of many enzymes involved in virtually all aspects of metabolism(King,1990).Over 100 enzymes are known to requir zinc as part of their prosthetic groups .These include alkaline phosphatase, super oxide dismutase carbonic anhydrase, alcohol dehydrogenase, and DNA polymeras (Standstead,1995). Zinc containing enzymes are essential to growth, wound healing,reproductive function, the immune system , and protection from free radical damage(Prasad,1996).Absorption of zinc is inhibited by increased dietary

Lund-Browder chart : shows the use of

content of phytate (inositol phosphate). Moreover, zinc absorption appear to induce the synthesis of metallothionein in the intestinal mucosa, and its absorption proportionate to level of metallothionein, and competes for absorption with iron and copper . Zinc is transported in the circulation by albumin and alpha2 macroglobulin. It is found primarily in prostate, RBCs, glands,muscles, bones, and liver, (Dursun *et al*., 1995).

Chapter One

Introduction

Chapter Three

Materials & Methods

Chapter five

Discussion

References

Chapter Two

Litrature Review

Chapter four

Results

Conclusions

From the data of the present study ,we conclude that the increase or decrease in some hematological and biochemical parameters may be attributed to hypermetabolic state which arise mainly due to increase of adrenaline release ,loss of fluid and electrolytes , hemolysis and sepsis . This may be due to the breach that are created by thermal injury in the surface of the skin which lead to affection of its function in the preservation of body fluid homeostasis, thermoregulation, and the host's protection against infection . As well as , immunological, neurosensory, and metabolic functions of the skin such as vitamin D metabolism are affected .

Recommendation

1. Baseline hematocrit and hemoglobin is useful to monitor, although changes in the values may not accurately reflect changes in blood volume due to the selective loss of the plasma component of blood but , it can be used to judge the efficacy of fluid replacement because the decreasing hematocrits may be expected with adequate fluid resuscitation.

2. The monitoring of the platelet count is of great importance during the resuscitation and care of severely burned patients. Whenever the platelet count begins to decline, all measures to support the general condition of the burned patient should be initiated, including the administration of intravenous fluids and antibiotics, optimal care of the burn wound, debridement or escharectomy, and blood transfusion .

3. Early enteral nutrition provided optimal preservation of the nutritional state of burn patients, maintaining nutritional markers within the normal range, and enhanced their immunological response. It was found that total serum proteins ,albumin and serum globulin increase on early entral nutrition. so that protein intake greater than 1 g/Kg per days has been recommended for all thermally injured patients , and for burns patients with normal renal function ,the recommended protein intake is 2g/ Kg per days. This entral nutrition for burn patients is needed to study .

4. Baseline values for serum creatinine and urea may help rule out intrinsic renal disease ,which impairs the reliability of urine output as an index of perfusion .

5. In elderly and diabetic patients ,insulin may be required ;some glucose should be infused at this time as well . Infants are prone to hypoglycemia as a result of decrease glucose stores .

6. The checking of liver function is very important to exclude underlying liver disease because liver impairment worsens the prognosis in patients with thermal injury.

Contents

List of Tables

List of Figures

Table 4 : The changes in total serum protein , serum albumin and serum globulin of males and females for burn patients within 2 days duration and controls .

AGE YEAR	SEX	Total serum protein g/L		Serum albumin g/L		Serum globulin g/L	
		Patients	Controls	Patients	Controls	Patients	Controls
(1 – < 3)	M	* 44.923 ± 1.559	* 70.5 ± 1.36	* 20.769 ± 1.105	* 40.6 ± 1.127	* 24.153 ± 0.9187	* 28.9 ± 1.129
	F	* 47.75 ± 1.508	* 70.9 ± 1.178	* 22.75 ± 1.634	* 41.4 ± 3.92	* 25 ± 0.835	* 31.2 ± 1.254
(3 – < 5)	M	* 44.076 ±0.824	* 70.5 ±1.708	* 25.133 ± 0.945	* 41.7 ± 1.528	* 22.7 ± 1.566	* 28.8 ± 0.573
	F	* 47.83 ±1.696	* 70.8 ± 1.254	* 21.615 ± 1.206	* 40.4 ± 1.284	* 22.462 ± 1.333	* 30.4 ± 0.792
(5 – <10)	M	* 46.846 ± 1.556	* 71.7 ± 1.136	* 22.077 ± 0.977	* 41.3 ± 1.044	* 24.769 ± 0.856	* 30.4 ± 0.67
	F	* 46.75 ± 1.388	* 71.1 ± 1.703	* 20.25 ± 1.008	* 41.6 ± 1.194	* 26.5 ± 0.744	* 30.5 ± 1.327
(10 –<18)	M	* 45.846 ± 3.606	* 70.4 ± 1.477	* 19 ± 1.41	* 40.7 ± 0.858	** 26.077 ± 0.812	** 29.7 ± 0.882
	F	* 46.667 ± 2.13	* 71.2 ± 1.205	* 21. 5 ± 1.564	* 40.7 ± 1.359	* 24.333 ± 0.873	* 30.5 ± 0.778
(18 -58)	M	* 44.769 ± 1.925	* 70.5 ± 1.833	* 19.308 ± 1.379	* 42.5 ± 1.5	** 24.923 ± 0.644	** 28 ± 0.83
	F	* 52.833 ± 2.633	* 70.7 ± 1.932	* 24.667 ± 1.662	* 40.9 ± 1.362	** 26.417± 1.227	** 29.8 ± 1.052

- Values are mean ± SE .
- * p < 0.01 .
- ** at p < 0.05 .

Table 5 : The changes in Serum of GOT, GPT and Alkaline phosphatase(ALP) for males and females burn patients within 2 days duration and controls .

AGE YEAR	SEX	Serum GOT I.U.		Serum GPT I.U.		Serum ALP I.U.	
		Patients	Control	Patients	Control	Patients	Control
(1 – <3)	M	* 19.846 ± 1.334	* 11.562 ± 0.436	* 28.538 ± 1.264	* 12.231 ± 0.382	* 63.3 ± 4.524	* 41.4 ± 3.605
	F	* 19.167 ± 1.029	* 10.8 ± 0.485	* 28.25 ± 1.309	* 11.8 ± 0.291	* 63.1 ±4.051	* 46.778 ± 3.639
(3 –< 5)	M	* 18.083 ± 0.596	* 10.892 ± 0.423	* 27.25 ± 1.548	* 11.8 ± 1.236	* 64.917 ± 4.2	* 38.9 ± 2.057
	F	* 17.231 ± 0.833	* 10.485 ± 0.49	* 24.308 ± 1.603	* 11.5 ± 0.279	* 79.385 ±8.974	* 34.3 ± 3.992
(5 –< 10)	M	* 19.385 ± 0.712	* 10.838 ± 0.584	* 29.23 ± 1.11	* 12.818 ± 0.288	* 69.23±2.977	* 34.546 ± 1.856
	F	* 18.667 ± 0.838	* 10.525 ± 0.495	* 28.25 ± 1.162	* 13.15 ± 0.325	* 61.417 ±4.369	* 34.4 ± 2.566
(10 –<18)	M	* 17.846 ± 0.839	* 11 ± 0.44	* 27.923 ± 0.937	* 13.25 ± 0.583	* 71.25± 5.118	* 32.5 ± 2.067
	F	* 18.167 ± 0.716	* 9.723 ± 0.399	* 28.5 ± 0.793	* 11.51 ± 0.269	* 63.167 ±3.96	* 36.7 ± 2.04
(18 -58)	M	* 18.75 ± 0.87	* 11.9 ± 0.421	* 29.667 ± 0.772	* 13.4 ± 0.562	* 71 ±4.023	* 31.3 ± 2.261
	F	* 24.231 ± 3.643	* 11.7 ± 0.305	* 43.308 ± 7.096	* 13.65 ± 0.5	* 71.231 ±3.03	* 27.3 ± 1.461

- **Values are mean ± SE .**
- *** p < 0.01 .**

Table 6 : The changes in serum of triglyceride, cholesterol, HDL and LDL for males and females burn patients within 2 days duration and controls .

AGE Year	SEX	Serum triglyceride mmol/l		Serum cholesterol mmol/l		Serum HDL mmol/l		Serum LDL mmol/l	
		Patients	controls	patients	controls	Patients	controls	Patients	controls
(1-<3)	M	*	*	*	*	*	*	*	*
	F	0.916 ± 0.088	1.33 ± 0.048	2.762 ± 0.795	4.185 ± 0.091	0.746 ± 0.013	1.165 ± 0.046	1.754 ± 0.089	2.754 ± 0.061
		*	*	*	*	*	*	*	*
		0.75 ± 0.072	1.294 ± 0.047	2.758 ± 0.058	4.37 ± 0.14	0.758 ± 0.047	1.125 ± 0.05	1.85 ± 0.056	2.986 ± 0.126
(3-<5)	M	*	*	*	*	*	*	*	*
	F	0.715 ± 0.041	1.305 ± 0.06	2.769 ± 0.056	4.52 ± 0.121	0.75 ± 0.044	1.185 ± 0.045	1.876 ± 0.064	3.084 ± 0.138
		*	*	*	*	*	*	*	*
		0.858 ± 0.06	1.37 ± 0.045	2.683 ± 0.061	4.52 ± 0.121	0.766 ± 0.036	1.165 ± 0.049	1.745 ± 0.076	3.081 ± 0.106
(5-<10)	M	*	*	*	*	*	*	*	*
	F	0.7 ± 0.038	1.33 ± 0.388	2.823 ± 0.043	4.57 ± 0.051	0.692 ± 0.045	1.155 ± 0.05	2.006 ± 0.013	3.149 ± 0.172
		*	*	*	*	*	*	*	*
		0.792 ± 0.06	1.38 ± 0.042	2.725 ± 0.051	4.59 ± 0.127	0.742 ± 0.034	1.195 ± 0.057	1.825 ± 0.058	3.119 ± 0.146
(10-<18)	M	*	*	*	*	*	*	*	*
	F	0.808 ± 0.064	1.225 ± 0.028	2.8 ± 0.044	4.5 ± 0.529	0.7 ± 0.036	1.12 ± 0.042	1.938 ± 0.066	3.135 ± 0.117
		*	*	*	*	*	*	*	*
		0.838 ± 0.05	1.32 ± 0.051	2.708 ± 0.053	4.54 ± 0.183	0.742 ± 0.036	1.12 ± 0.065	1.802 ± 0.048	3.156 ± 0.217
(18 -58)	M	*	*	*	*	*	*	*	*
	F	0.85 ± 0.062	1.51 ± 0.11	2.958 ± 0.099	4.8 ± 0.137	0.658 ± 0.042	1.16 ± 0.056	2.138 ± 0.11	3.338 ± 0.131
		*	*	*	*	*	*	*	*
		0.754 ± 0.063	1.83 ± 0.073	2.977 ± 0.063	4.727 ± 0.161	0.7 ± 0.042	1.215 ± 0.055	2.126 ± 0.082	3.272 ± 0.183

- Values are mean ± SE .
- * $p < 0.01$.

Table 7 : The changes in serum urea and serum creatinine of males and females for burn patients within 2 days duration and controls .

AGE YEAR	SEX	Serum urea mmol/l		Serum creatinine µmol / l	
		Patients	Control	Patients	Control
(1 – <3)	M	3.908 ± 0.257	3.4 ± 0.19	72.4615± 1.175	70.5000± 1.319
	F	3.917 ± 0.196	3.458 ± 0.205	67.833± 2.145	65.5000± 1.463
(3 –< 5)	M	4.0± 0.121	3.683 ±0.155	71.4167± 1.27	68.4167± 1.564
	F	4.169 ± 0.321	3.946 ± 0.204	66± 0.77	64.3571± 1.067
(5 –< 10)	M	4.385 ± 0.3	3.931 ± 0.279	71± 1.000	67.7692± 1.051
	F	4.158 ± 0.179	3.5 ± 0.169	66.9167± 1.422	65.2500± 1.42
((10-<18))	M	4.454 ± 0.296	3.885 ± 0.277	72.2308± 1.63	69.2308± 1.695
	F	3.95 ± 0.297	3.292 ± 0.275	62.750 ± 1.629	59.6667± 1.384
(18 -58)	M	4.225 ± 0.185	3.58 ± 0.246	70.7500± 1.62	67.4167± 1.52
	F	4.285 ± 0.276	3.717 ± 0.303	68.692 ± 1.273	66.231 ± 1.41

- Values are mean ± SE .
- Means without any * are insignificant at p > 0.05 .

Table 8 : The changes in serum of calcium, copper and zinc of males and females for burn patients within 2 days duration and controls .

AGE Year	SEX	Serum calcium mmol /l		Serum copper part per million		Serum zinc part per million	
		Patients	controls	Patients	controls	Patients	controls
(1 – <3)	M	* 1.823 ± 0.052	* 2.408 ± 0.049	* 0.0334 ± 0.022	* 0.0646±0.002	** 0.0228±0.005	** 0.0736±0.011
	F	* 1.808 ± 0.061	* 3.379 ± 0.05	* 0.0356 ±.002	* 0.0660±0.005	** 0.0250±0.003	** 0.0640±0.008
(3 – <5)	M	* 1.825 ± 0.045	* 2.358 ± 0.036	* 0.0352 ± 0.003	* 0.0654±0.005	* 0.0554±0.006	* 0.0778±0.004
	F	* 1.823 ± 0.034	* 2.462 ± 0.013	* 0.0356 ± 0.002	* 0.0646±0.006	* 0.0546±0.006	* 0.0784±0.004
(5 – <10)	M	* 1.815 ± 0.037	* 2.415 ± 0.027	* 0.0700 ± 0.006	* 0.1198±0.012	* 0.0514±0.005	* 0.0888±0.004
	F	* 1.767 ± 0.043	* 2.283 ± 0.051	* 0.0768 ± 0.005	* 0.1220±0.012	** 0.0482±0.004	** 0.0840±0.0035
((10-<18))	M	* 1.792 ± 0.043	* 2.331 ± 0.063	* 0.0860 ± 0.01	* 0.1430±0.1430	** 0.0240±0.004	** 0.0650±0.003
	F	* 1.723 ± 0.053	* 2.317 ± 0.071	* 0.0860 ± 0.01	* 0.1458 ± 0.018	* 0.0304±0.005	* 0.0628±0.0026
(18 -58)	M	* 1.808 ± 0.043	* 2.317 ± 0.061	* 0.0700±0.005	* 0.1700±0.018	* 0.0202±0.005	* 0.0720±0.005
	F	* 1.746 ± 0.039	* 2.223 ± 0.062	* 0.0728±0.002	* 0.1610±0.013	* 0.0188±0.003	* 0.0710±0.0048

- Values are mean ± SE .
- * (p < 0.01).
- * (p < 0.05).

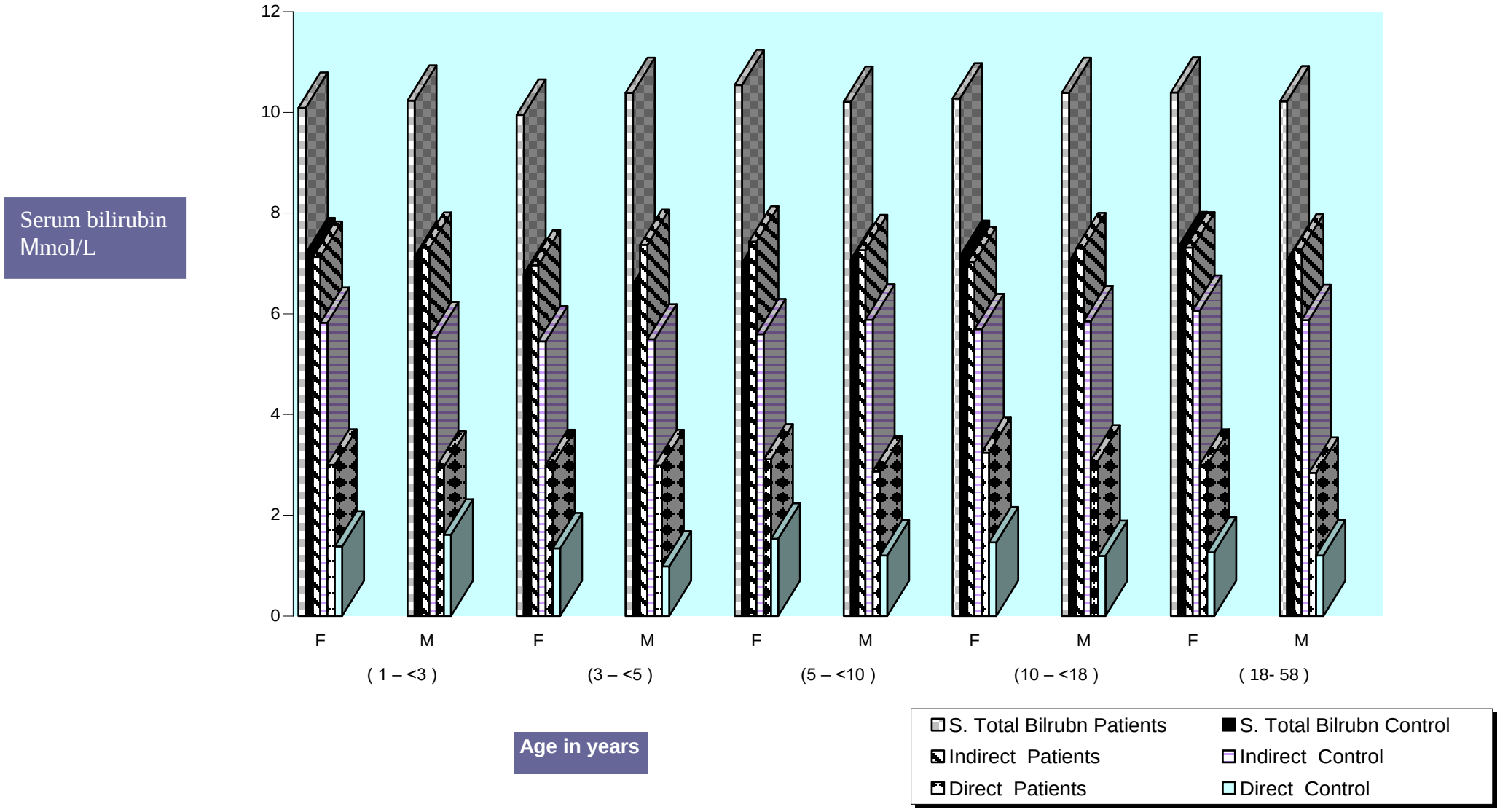

Figure 4.2 :The changes in serum bilirubin (total ,direct & indirect) within 2 days duration for male & female burn patients in comparison with controls .

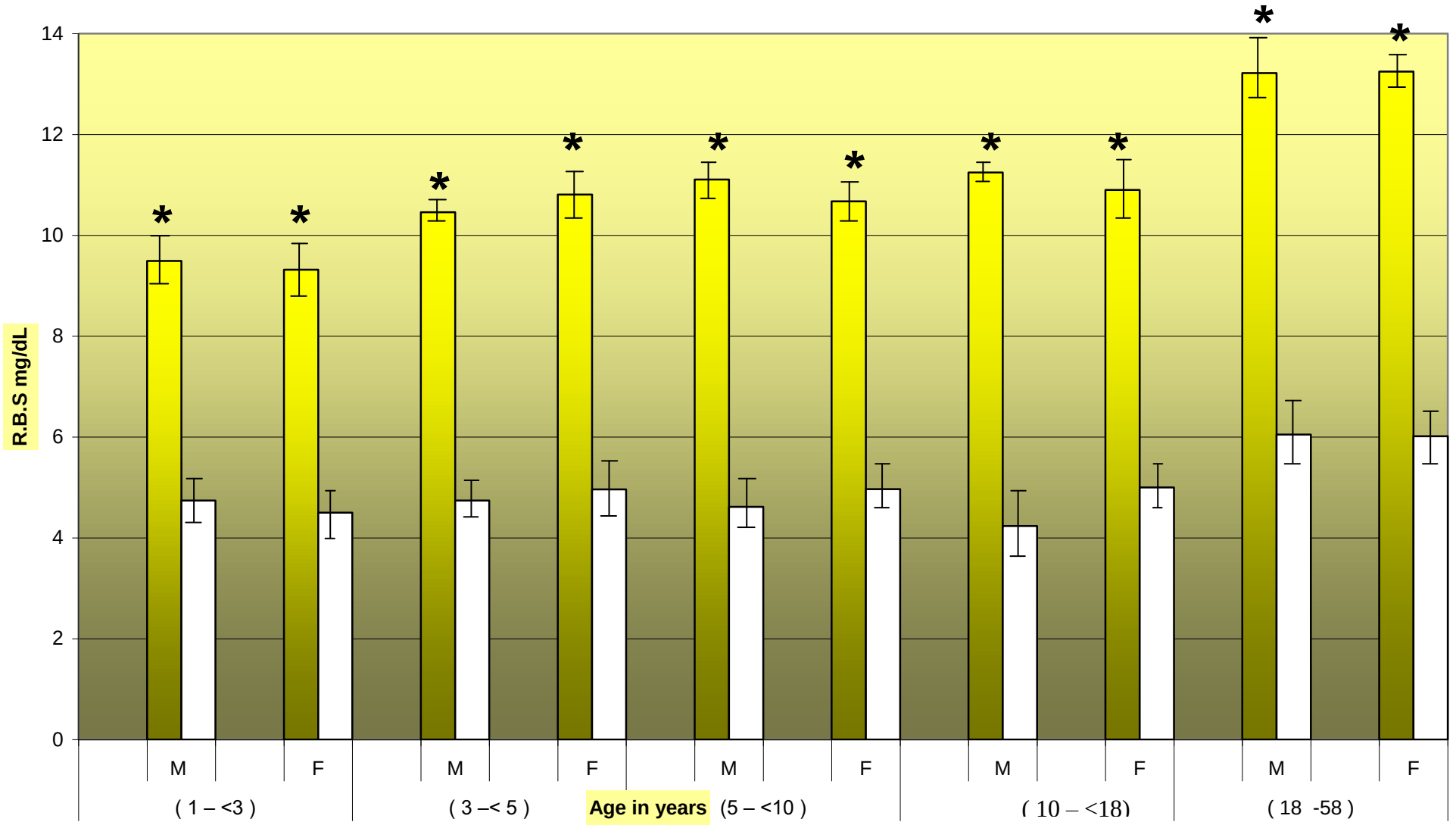

Figure 4.3 :The changes in random blood sugar (R.B.S) within 2 days for male & female burn patients in comparison with controls .

* P < 0.01

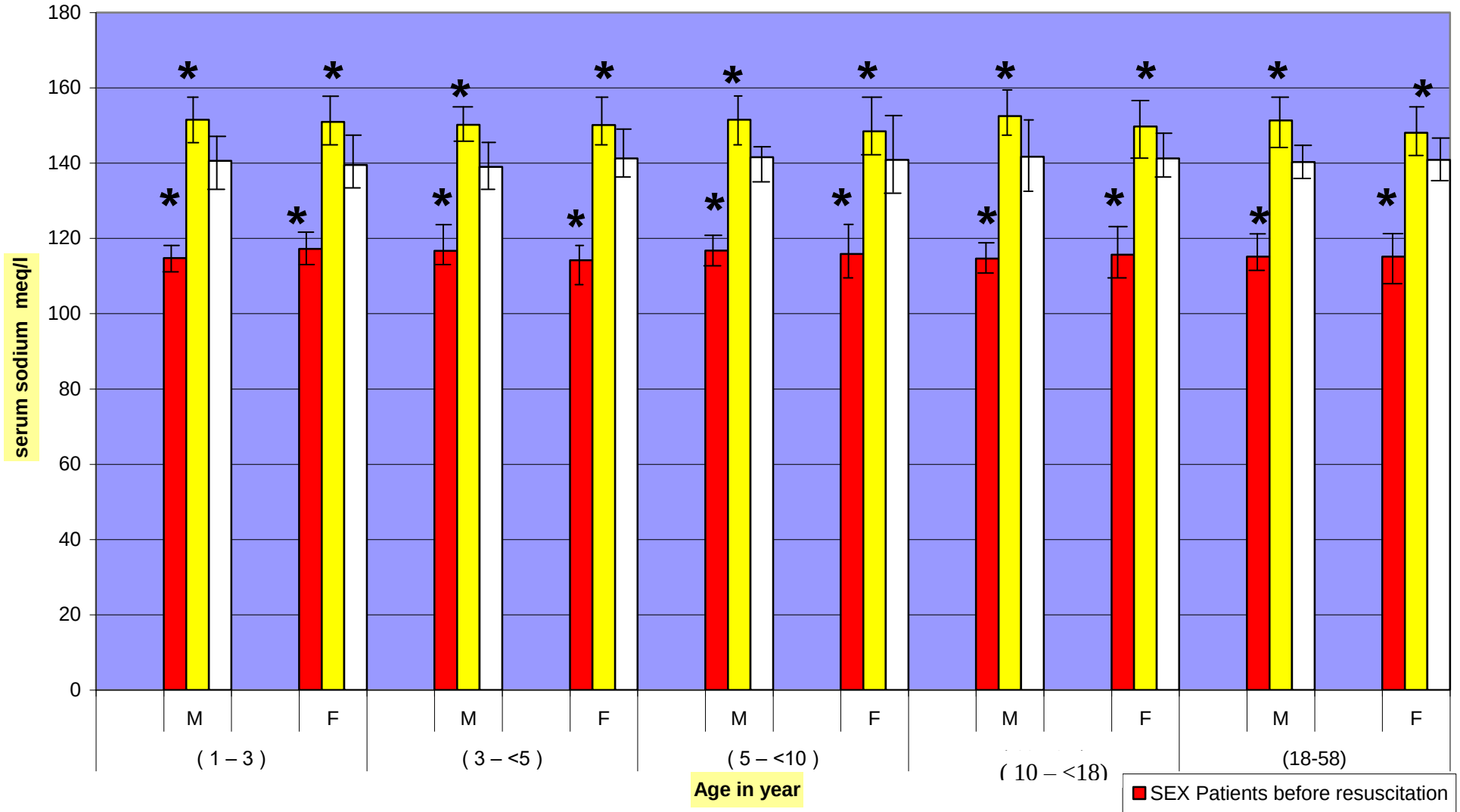

Figure 4.4 :The changes in serum sodium before& within 2 days after resuscitation for male & female burn patients in comparison with controls .

* $P < 0.01$

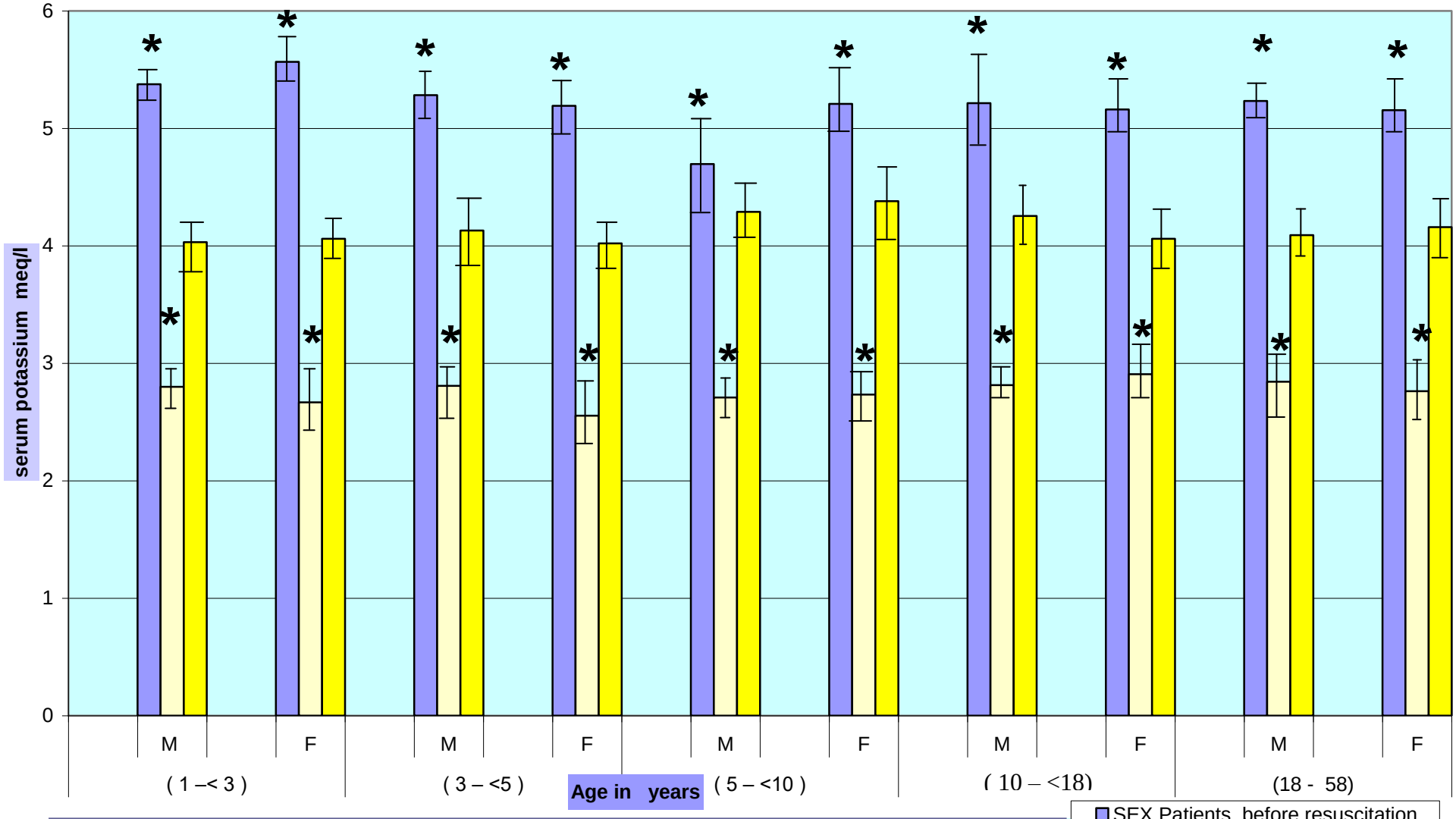

Figure 4.5 :The changes in serum potassium before& within 2 days after resuscitation for male & female burn patients in comparison with controls .

* P< 0.01

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Table 1: The changes in RBCs , Hb, PCV for burn patients within 7 days duration and controls .

AGE Years	SEX	RBCs count millions/mm³		Hb g/dl		PCV L/L	
		Patients	Control	Patients	Control	Patients	Control
(1 –< 3)	M	* 4.208 ± 0.213	* 4.87 ± 0.115	* 11.523 ± 0.613	* 13.65 ± 0.367	* 0.343 ± 0.018	* 0.399 ± 0.011
	F	* 3.904 ± 0.17	* 4.94 ± 0.1	* 10.459 ± 0.514	* 13.77 ± 0.332	* 0.313 ± 0.016	* 0.403 ± 0.01
(3 –< 5)	M	* 3.6 ± 0.212	* 4.832 ±0.133	* 9.525 ± 0.527	* 13.37 ± 0.365	* 0.28 ± 0.016	* 0.392 ± 0.011
	F	* 3.196 ± 0.114	* 4.89 ± 0.114	* 8.8 ± 0.288	* 13.38 ± 0.296	* 0.255 ± 0.009	* 0.393 ± 0.009
(5 –< 10)	M	* 3.329 ± 0.175	* 4.98 ± 0.124	* 9.36 ± 0.552	* 13.91 ± 0.398	* 0.272 ± 0.016	* 0.41 ± 0.012
	F	* 3.673 ± 0.198	* 5.009 ±0.121	* 10.038 ± 0.599	* 13.91 ± 0.462	* 0.288 ± 0.018	* 0.412 ± 0.014
(10 – <18)	M	* 4.046 ± 0.214	* 4.983 ±0.121	* 11.289 ± 0.665	* 13.84 ± 0.399	* 0.33 ± 0.019	* 0.408 ± 0.011
	F	* 3.467 ± 0.107	* 4.839 ±0.108	* 9.221 ± 0.339	* 13.05 ± 0.28	* 0.27 ± 0.009	* 0.386 ± 0.009
(18 - 58)	M	* 3.525 ± 0.152	* 5.188 ± 0.09	* 9.8 ± 0.51	* 14.47 ± 0.335	* 0.284 ± 0.014	* 0.429 ± 0.011
	F	* 3.573 ± 0.193	* 4.857 ± .071	* 9.531 ± 0.515	* 13.27 ± 0.251	* 0.278 ± 0.016	* 0.393 ± 0.007

- **Values are mean ± SE (standard error) .**
- *** p < 0 . 01 .**

Table 2 : The changes in differential WBC counts of males and females for burn patients within 7 days duration and controls .

AGE YEAR	SEX	Neutrophile %		Eosinophile %		Basophile%		Lymphocytes%		Monocytes%	
		Patients	Controls	Patients	Controls	Patients	Controls	Patients	Controls	Patients	Controls
(1 – <3)	M	* 71.31 ±0 .957	* 45.54 ± 0.882	1.462 ± 0.183	1.538 ± 1.83	0.462 ± 0.14	0.385 ± 0.144	* 19.92 ± 1.135	* 43.69 ± 0.873	8.077±.0.167	8.615 ± 0.29
	F	* 70.75 ± 1.031	* 46.67 ± 1.227	1.417 ± 0.149	1.417 ± 0.193	0.583 ± 0.149	0.667 ± 0.142	* 19 ± 1.052	* 42.25 ± 1.175	8.417± 0.26	9 ± 0.275
(3 – <5)	M	* 71.92± 0.988	* 60.17± 1.296	1.25 ± 0.131	1.417 ± 0.149	0.5 ± 0.151	0.667 ± 0.142	* 19.42 ± 0.892	* 29.25 ± 1.11	8.167± 0.207	8.583 ± 0.229
	F	* 72.769 ± 0.9	* 59.08 ± 0.977	1.308 ± 0.208	1.538 ± 0.183	0.31 ± 0.133	0.385 ± 0.140	* 18.54 ± 0.882	* 30.39 ± 1.071	8.308± 0.237	8.692 ± 0.175
(5 –<10)	M	* 72.15 ± 1.255	* 54.23 ± 1.178	1.385 ± 0.14	1.538 ± 0.183	0.231 ± 0.122	0.692 ± 0.133	* 18.77 ± 1.063	* 34.69 ± 1.327	8.462± 0.183	8.846 ± 0.274
	F	* 71.75 ± 0.906	* 58.5 ± 1.323	1.167 ± 0.167	1.417 ± 0.149	0.417 ± 0.149	0.417 ± 0.149	* 18.83 ± 0.903	* 31 ± 1.273	8.667± 0.225	8.667 ± 0.256
(10 –<18)	M	* 71.85± 1.636	* 60.9 ± 1.048	1.231 ± 0.122	1.5 ± 0.167	0.385 ± 0.14	0.67 ± 0.163	* 19.615 ± 1.38	* 28.4 ± 0.983	8.077 ±0.288	8.6 ± 0.267
	F	* 75.58± 3.662	* 58.46 ± 1.091	1.083 ± 0.083	1.818 ± 0.226	0.5 ± 0.151	0.545 ± 0.157	* 18.5 ± 1.07	* 31 ± 0.842	8.853± 0.246	8.18 ± 0.263
(18 -58)	M	* 72 ± 1.605	* 62.2 ± 0.8	1.417 ± 0.149	1.6 ± 0.221	0.333 ± 0.142	0.4 ± 0.163	* 19.08 ± 1.658	* 27.7 ± 0.87	7.833± 0.241	8.1 ± 0.233
	F	* 72.77 ± 1.105	* 62.3 ± 1.174	1.462 ± 0.144	1.5 ± 0.167	0.308 ± 0.133	0.5 ± 0.167	* 18.62 ± 0.979	* 24.91 ± 0.71	8.231± 0.281	8.2 ± 0.291

- Values are mean ± SE .
- * p < 0.01 .
-- Means without any * are insignificant at p > 0.05 .

Table 3 : the changes in platelets count of males and females for burn patients within 7 days duration and controls .

AGE YEAR	SEX	Platelets count $\times 10^3/mm^3$	
		Patients	Control
(1 – < 3)	M	337.692 ± 15.974	368.462± 15.974
	F	355.833 ± 16.627	379.167± 16.627
(3 – < 5)	M	318.5 ± 18.213	342.500± 18.03
	F	324.5 ± 19.895	340.000± 20.22
(5 – < 10)	M	331.5 ± 12.943	338.000± 15.29
	F	350 ± 14.832	361.000± 16.646
(10 – <18)	M	352.5 ± 19.71	362.000± 30.9
	F	298 ± 8.794	331.300± 18.812
(18 -58)	M	359 ± 13.454	367.500± 19.09
	F	298.5 ± 5.965	300.600± 8.38

- Values are mean ± SE .
- Means without any * are insignificant at (p > 0.05) .

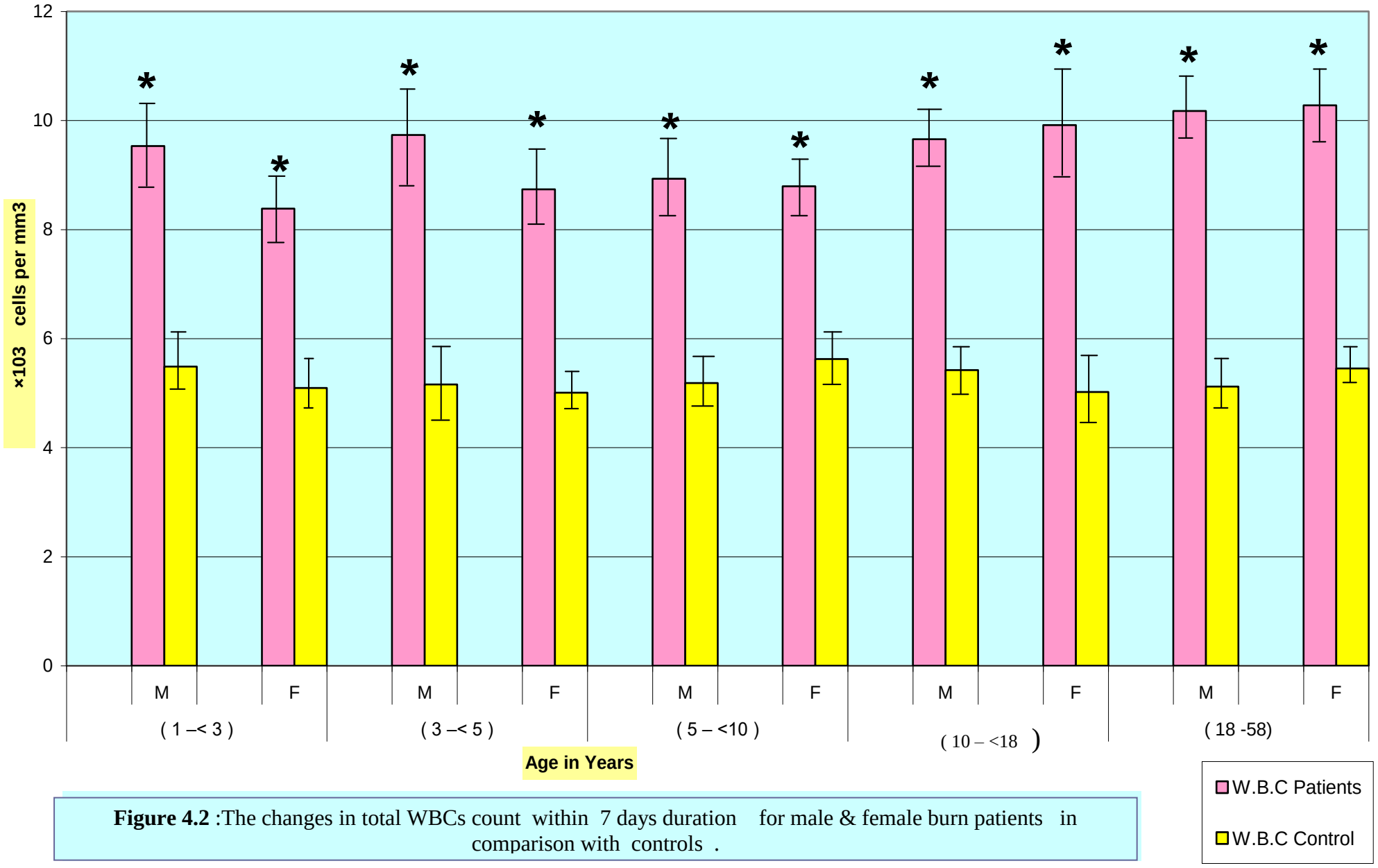

Figure 4.2 :The changes in total WBCs count within 7 days duration for male & female burn patients in comparison with controls .

* P < 0.01

We, the examining committee, certify that we have read the thesis entitled ***(Hematological and Biochemical changes in Burn Patients)*** and have examined the student ***(Hayder Abdul Husain Aziz)*** in its contents, and that in our opinion it is accepted as a thesis for the degree of Master of Science in Medical Physiology with excellent degree .

Prof. Dr. Yesar Mh. Al-shamaa
College of Medicine
University of Al-Kufa
Chairman

Dr. Ahmed A. Nawres
College of Medicine/ University of Babylon
Member

Ass. Prof. Dr . Saad M. Alaragi
College of Medicine/ University of Babylon
Member

Ass. Prof .Dr . Muhammed Obaid AL - Muhammadi
College of Medicine/ University of Babylon
Member and Supervisor

Approved for the College Committee of Graduate Studies

Ass. Prof .Dr . Ali K. Al- Shaali
Dean of College of Medicine
College of Medicine/ University of Babylon

الخلاصة

تضمنت هذه الدراسة معرفة التغييرات التي تحدث لبعض المعايير الدموية والكيميوحيوية لدى مرضى الحروق وذلك لأهميتها في مساعدة الفريق الطبي لمعرفة كيفية استجابة الجسم للعلاج المعطى مؤدياً إلى علاج أفضل .

استمرت هذه الدراسة من تشرين الثاني (2007) إلى أيار (2008) و قد تضمنت 125 مصابا بالحروق و100 من غير المصابين (السيطرة)من كلا الجنسين . و قد تم تقسيم المرضى المصابين بالحروق(بالمقارنة مع الأشخاص الأصحاء) إلى خمسة مجاميع عمرية حيث أن اعمار المجموعة الأولى تتراوح بين1- <3 سنة و المجموعة الثانية3-<5 سنة والمجموعة الثالثة 5-<10 سنة و المجموعة الرابعة10-<18 سنة و المجموعة الخامسة 18-58 سنة .حيث تم إدخال هؤلاء المرضى إلى ردهة الحروق في مستشفى الحلة التعليمي العام والذين يعانون من الحرق الحراري(حروق اللهب أو السماط) وبدرجة ثانية أو ثالثة.

تميز الأشخاص المصابين بالحروق فيما يخص المتغيرات الدموية بانخفاض معنوي ($p < 0.01$) لكل من العدد الكلي لخلايا الدم الحمر (RBCs) و خضاب الدم (Hb) وحجم الخلايا المضغوط (PCV) لكل المجاميع العمرية ومن كلا الجنسين عند مقارنتها بالأصحاء . كما لوحظ ارتفاع معنوي ($p < 0.01$) في العدد الكلي لخلايا الدم البيضاء(WBCs) و لنسبة الخلايا المتعادلة (neutrophil) و انخفاض معنوي لنسبة الخلايا اللمفية (lymphocyte) بينما لا توجد تغيرات معنوية لنسبة كل من الخلايا الأحادية (monocytes) والخلايا الحمضية (eosinophil) و الخلايا القاعدية (basophil) .كما لوحظ أيضا انخفاض غير معنوي لصفيحات الدموية .

أما بخصوص المتغيرات الكيميوحيوية فقد لوحظ انخفاض معنوي لقيم البروتين المصلي الكلي(total serum protein)و الألبومين (albumin)و الكلو بيولين (globulin) لكل المجاميع العمرية ومن كلا الجنسين (عدا كل من المجموعتين الرابعة و الخامسة من الذكور والمجموعة الخامسة من الإناث حيث شهدت انخفاضا معنويا على مستوى $p<0.05$ لمتغير الكلوبيولين فقط) عند مقارنتها بالأصحاء .أشرت قيم كل من الانزيمات الكبدية (GOT و GPT وALP) و معدل البلروبين الكلي وبنوعيه المباشر والغير مباشر (total bilirubin :direct and indirect) ارتفاعا معنويا في مصل الدم عند مقارنتها بالأصحاء.كما شهد معدل سكر الدم العشوائي(random blood sugar) في مصل الدم

أيضا ارتفاعا معنويا لكل المجاميع العمرية من كلا الجنسين. أظهرت الدراسة أيضا انخفاضا معنويا لكل من الكولسترول : الكلي و العالي الكثافة و المنخفض الكثافة (total cholesterol ,HDL and LDL) والكليسيرايد الثلاثي(triglycerides) في المصل. أما بخصوص قيم الصوديوم فقد وجد انخفاضا معنويا($p < 0.01$) قبل تزويد المرضى بالمحاليل الوريدية وارتفاعا معنويا بعد إعطائهم تلك المحاليل . بينما كانت قيم البوتاسيوم مرتفعة معنويا قبل تزويد المرضى بالمحاليل الوريدية و منخفضة معنويا بعد إعطائهم تلك المحاليل.كما شهدت العناصر النادرة (النحاس والزنك) والكالسيوم انخفاضا معنويا لكل المجاميع العمرية ومن كلا الجنسين (عدا كل من المجموعتين الأولى و الرابعة من الذكور والمجموعة الأولى و الخامسة من الإناث حيث شهدت انخفاضا معنويا $p<0.05$ لمتغير الزنك فقط) عند مقارنتها بالأصحاء .

إن التغييرات في المعايير الدموية و والكيميوحيوية الحاصلة لمرضى الحروق يمكن ان تعزى بصورة رئيسية إلى ارتفاع معدل الايض بسبب الإفراز العالي لهرمون الادرنالين و فقدان السوائل والأملاح وتكسر خلايا الدم الحمراء و تسمم الدم .