Evaluation of Prognostic Value of Adhesion Molecules in Septic Critically Ill Patients


Departments of Chest Diseases*, Microbiology and Immunology**, Clinical Pathology***, and Critical Care Medicine****, Faculty of Medicine, University of Alexandria.

Septic shock is the most frequent cause of death in intensive care units. Despite major advances in antimicrobial therapy, critical care and surgical techniques, there has been little improvement in morbidity or mortality due to sepsis or septic shock. The aim of this study was to evaluate the role of vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1) in sepsis, septic shock, haemodynamic changes and outcome. Thirty intensive care unit patients suffering from sepsis with or without shock were subjected to blood culture, culture from the site of infection if possible, blood gases analysis, acute physiology and chronic health evaluation score (APACHE II) at baseline, multiple organ failure score on day one (MOF1), cumulative organ failure score (MOFC) on day 5, haemodynamic measurements, as well as serum VCAM-1 and ICAM-1 levels for 5 days after admission. Ten healthy control subjects were also included in the study. The most common site of infection was the chest, the isolates were mostly Gram negative (60% of cases), 9 patients (30%) had positive blood cultures. Serum ICAM-1 and VCAM-1 levels gradually increased from a baseline till day 5 of the study and were significantly higher in patients on admission (62 ± 20.21, 404.67 ± 130.85 ng/ml, respectively) than in the control group (14.0 ± 4.71, 128.0 ± 34.9 ng/ml respectively), (P=0.00). They were higher in shocked than in non-shocked patients, and significantly so in non-survivors than in survivors and in patients with positive blood cultures than in those with negative blood cultures, throughout the study period (P=0.00). A significant positive correlation was observed between serum ICAM-1 and VCAM-1 levels on one hand, and APACHE II as well as both organ failure scores for the 1st day or cumulative on the other hand. We conclude that these adhesion molecules could be measured in critically ill septic patients to predict prognosis and guide therapy.

INTRODUCTION

Severe sepsis is a systemic inflammatory response to infection, which involves a characteristic spectrum of pathologic changes in many host systems. It is one of the most devastating problems for patients who are critically ill as it is the most common cause of shock and is a leading cause of death in most intensive care units (ICUs). (1) Despite major advances in antimicrobial therapy, critical care and surgical techniques, severe sepsis remains the dominant challenge in the care of critically ill patients. (2)

On infection, the pathophysiologic sequence for sepsis involves cytokine release, and endothelial and neutrophil activation, initiating a cascade of leukocyte-endothelium interactions and adhesions. This is followed by transendothelial migration and subsequent microvascular and tissue injury, leading ultimately to multiple organ failure (MOF). (3) Adhesion of neutrophils to the endothelium is regulated by at least three adhesion molecule families (selectins, integrins, and the immunoglobulin superfamily which includes vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1)), and by chemotactic signals. (3,8)

Besides various neutrophil- and endothelial-bound adhesion molecules, soluble forms of endothelial-derived adhesion molecules have been detected in the circulating blood and seem to be good markers of endothelial damage. Their importance in the critically ill has been subject to investigation. (4,5,6)

The aim of this study was to evaluate the role of vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1) in sepsis, septic shock, haemodynamic changes and outcome.

MATERIAL

This study was carried out on a group of 30 patients with sepsis, with or without shock attending the ICU of Alexandria University Hospital. They were selected according to the following criteria: 1- Systemic inflammatory response syndrome as indicated by the presence of at least two of the following: Body temperature > 38°C or <
36°C, heart rate > 90/min, respiratory rate > 20 breath/min, and WBCs > 12,000 cells/mm³ or 10% immature neutrophils.

2- Clinical and/or microbiological evidence of infection as the likely cause of inflammation.

3- Septic shock, defined as sepsis associated with hypotension and organ hypoperfusion (manifested by one or more of lactic acidosis, oliguria <30 ml/h, and acute alteration in mental status unexplained by other conditions).

Ten normal healthy subjects were included in the study to serve as a control group.

**METHODS**

The selected patients were subjected to the following:

1) History taking including age, sex and circumstantial data.

2) Clinical measurements: pulse rate, temperature, mean arterial blood pressure, and respiratory rate.

3) Routine laboratory measurements: Total and differential white cell count, serum sodium and potassium levels, serum urea and creatinine levels.

4) Blood gases analysis (arterial oxygen tension, arterial carbon dioxide tension and arterial pH).

5) Blood culture and culture from the site of infection whenever possible.(9)

6) Scoring at baseline using the APACHE II score system.(10)

7) Organ failure was determined, multiple organ failure score on day 1 (MOF1) and cumulative organ failure score on day 5 (MOFC).(11) Death during hospitalisation or survival to discharge from hospital were also recorded.

8) Haemodynamic measurements including cardiac index (CI), central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), and systemic vascular resistance (SVR).

9) Adhesion molecule serum levels: circulating ICAM-1 and VCAM-1 levels were estimated using ELISA.(12)

The kits were supplied by Diaclone Research Company and purchased by ABCO-France Company. The assay was performed and the results were estimated by strictly adhering to the manufacturer’s instructions.

**RESULTS**

The study was conducted on 30 septic critically ill patients; 16 with septic shock and 14 with sepsis. Their ages ranged between 18 and 77 with a mean of 51.17±14.96 years. They were 20 males (66.67%) and 10 females (33.33%). The male to female ratio was 2:1. There were 16 (53.4%) survivors and 14 (46.6%) non-survivors with a mortality rate of 46.6%.

The most common site of infection was the chest which was seen in 43% of the patients followed by abdominal sepsis (17% of cases), multiple infections (13%), and urosepsis (10%). Gram–ve organisms represented most of isolated pathogens (60%) whereas gram+ve organisms represented 13% of cases. The most common pathogen was Klebsiella (20%) followed by Pseudomonas...
and Proteus (13% for each) and then E.coli and Staphylococci (10% for each). Blood culture was +ve in 9 cases (30%). The organisms isolated from blood were Proteus, Klebsiella, Staphylococci, Pseudomonas and E. coli.

Figure 1, represents the different levels of serum ICAM-1 from the baseline to the 5th day of the study in all patients in ng/ml. At baseline, sICAM-1 level ranged between 32-96 ng/ml with a mean of 62.6±20.21 ng/ml and increased gradually and significantly till day 5 of the study where serum level of ICAM-1 ranged between 52-193 ng/ml with a mean of 94.87±33.3 ng/ml (P=0.000).

Figure 2, represents the levels of serum VCAM-1 in all patients along the duration of the study. The levels gradually increased significantly from a baseline mean of 404.67±130.85 ng/ml to the mean of 589.67±105.82 ng/ml on day 5 of the study (P=0.000).

Table 1 shows the comparison between patients with sepsis on admission and the control group as regards serum levels of ICAM-1 and VCAM-1. There was a statistically significant difference between the studied group on admission and the control group as regards serum levels of sICAM-1 and VCAM-1 which were higher among patients (62±20.21, 404.67±130.85 ng/ml, respectively) than in the control group (14.0±4.71, 128.0±34.9 ng/ml, respectively), (P=0.000).

We also found serum levels of sICAM-1 and VCAM-1 to be significantly higher among patients with a positive blood culture compared to those with a negative blood culture in all days of the study, P<0.05.

Table 2 demonstrates a non-significant difference between shocked and non-shocked patients as regards mean serum level of ICAM-1 at baseline, day one, day two and three. However, on days four and five there were statistically significant differences between shocked and non-shocked patients as regards ICAM-1 serum level which were higher in shocked patients (99.44±33.12, 107.38±32.17 ng/ml) than in non-shocked patients (76.86±27.12, 80.57±28.93 ng/ml respectively), (P = 0.024, P=0.000 respectively).

Table 3 demonstrates a statistically significant difference between shocked and non-shocked patients as regards VCAM-1 serum level on day two, three, four and five being higher in shocked than in non-shocked patients (P=0.017, 0.005, 0.008 and 0.000 respectively). However, on baseline and day one of the study the differences were insignificant (P=0.091 and 0.088, respectively).

Table 4 shows a statistically significant difference between survivors (16 patients) and non-survivors (14 patients) as regards ICAM-1 serum level. It appeared to be higher among non-survivors in all days of the study from baseline to day five than among the other group, (P=0.000). The same also applied to VCAM-1 serum levels which were significantly higher among non-survivors compared to survivors from baseline to day five, (P=0.000) (Table 5). Survival was defined as survival throughout the period of ICU admission.

A statistically significant positive correlation between sICAM-1 level and APACHE II score (P<0.05) is demonstrated in Figure 3. Similarly, a positive significant correlation between sICAM-1 level and multiple organ failure score (for day 1 and cumulative) was detected (Figure 4). Figure 5 demonstrates a statistically significant positive correlation between VCAM-1 serum level and APACHE II score, (r =0.662). Also there was a statistically significant positive correlation between VCAM-1 serum level and both organ failure scores for the 1st day (r =0.489) and the cumulative (r =0.725), (P<0.05) as seen in figure 6.

Upon comparing between the different parameters under investigation in relation to outcome, logistic regression showed that the best prognostic parameters were MOFC, followed by ICAM-1 and then VCAM-1 serum levels (P=0.022, 0.039, and 0.047 respectively), while APACHE II score was insignificant (P=0.105).

Regarding haemodynamic parameters, a statistically significant negative correlation was detected between sICAM-1 levels and SVR, sICAM-1 levels and CVP, and between serum VCAM-1 levels and CVP, (P<0.05).
Table 1. Comparison between the sepsis group on admission and the control group as regards serum levels of s-ICAM-1 and s-VCAM-1

<table>
<thead>
<tr>
<th></th>
<th>ICAM-1 (ng/ml)</th>
<th>VCAM-1 (ng/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Patients</td>
<td>Controls</td>
</tr>
<tr>
<td><strong>Range</strong></td>
<td>32 - 96</td>
<td>7 - 21</td>
</tr>
<tr>
<td><strong>Mean ± SD</strong></td>
<td>62.60 ± 20.21</td>
<td>14.00 ± 4.71</td>
</tr>
<tr>
<td><strong>t-test</strong></td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
</tr>
</tbody>
</table>

P is significant if < 0.05
* = significant

Table 2. Comparison between shocked and non-shocked patients as regards serum s-ICAM-1 levels (ng/ml)

<table>
<thead>
<tr>
<th></th>
<th>Base (on admission)</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Min</strong></td>
<td>35</td>
<td>46</td>
<td>47</td>
<td>52</td>
<td>59</td>
<td>59</td>
</tr>
<tr>
<td><strong>Max</strong></td>
<td>95</td>
<td>107</td>
<td>122</td>
<td>150</td>
<td>193</td>
<td>193</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td>65.38</td>
<td>72.56</td>
<td>81.88</td>
<td>90.88</td>
<td>99.44</td>
<td>107.38</td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td>19.40</td>
<td>19.92</td>
<td>19.88</td>
<td>19.88</td>
<td>19.44</td>
<td>20.57</td>
</tr>
<tr>
<td><strong>t-test</strong></td>
<td>P = 0.431</td>
<td>P = 0.250</td>
<td>P = 0.075</td>
<td>P = 0.053</td>
<td>P = 0.024*</td>
<td>P = 0.000*</td>
</tr>
</tbody>
</table>

P is significant if < 0.05
* = significant

Table 3. Comparison between shocked and non-shocked patients as regards soluble s-VCAM-1 levels (ng/ml)

<table>
<thead>
<tr>
<th></th>
<th>Base (on admission)</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Min</strong></td>
<td>240</td>
<td>250</td>
<td>250</td>
<td>250</td>
<td>400</td>
<td>410</td>
</tr>
<tr>
<td><strong>Max</strong></td>
<td>660</td>
<td>780</td>
<td>910</td>
<td>630</td>
<td>640</td>
<td>650</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td>442.50</td>
<td>536.25</td>
<td>587.50</td>
<td>640.63</td>
<td>671.25</td>
<td>496.43</td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td>123.21</td>
<td>140.33</td>
<td>161.18</td>
<td>159.85</td>
<td>184.93</td>
<td>141.45</td>
</tr>
<tr>
<td><strong>t-test</strong></td>
<td>P = 0.091</td>
<td>P = 0.088</td>
<td>P = 0.017*</td>
<td>P = 0.005*</td>
<td>P = 0.008*</td>
<td>P = 0.000*</td>
</tr>
</tbody>
</table>

P is significant if < 0.05
* = significant
Table 4. Comparison between survivors and non-survivors as regards serum s-ICAM-1 levels (ng/ml) throughout the days of the study period

<table>
<thead>
<tr>
<th>Base admission (on admission)</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surv. (n=16) Non Surv. (n=14)</td>
<td>Surv. (n=16) Non Surv. (n=14)</td>
<td>Surv. (n=16) Non Surv. (n=14)</td>
<td>Surv. (n=16) Non Surv. (n=14)</td>
<td>Surv. (n=16) Non Surv. (n=14)</td>
<td>Surv. (n=16) Non Surv. (n=14)</td>
</tr>
<tr>
<td>Min</td>
<td>32</td>
<td>45</td>
<td>41</td>
<td>54</td>
<td>44</td>
</tr>
<tr>
<td>Max</td>
<td>86</td>
<td>96</td>
<td>86</td>
<td>107</td>
<td>95</td>
</tr>
<tr>
<td>Mean</td>
<td>50.44</td>
<td>76.50</td>
<td>55.25</td>
<td>83.50</td>
<td>57.75</td>
</tr>
<tr>
<td>SD</td>
<td>14.16</td>
<td>17.02</td>
<td>12.95</td>
<td>17.49</td>
<td>14.83</td>
</tr>
<tr>
<td>t-test</td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
</tr>
<tr>
<td>t-test between level of ICAM-1 and its basal level</td>
<td>P = 0.003*</td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
</tr>
</tbody>
</table>

P is significant if < 0.05
* = significant

Table 5. Comparison between survivors and non-survivors as regards serum s-VCAM-1 levels (ng/ml) throughout the days of the study period

<table>
<thead>
<tr>
<th>Base admission (on admission)</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surv. (n=16) Non Surv. (n=14)</td>
<td>Surv. (n=16) Non Surv. (n=14)</td>
<td>Surv. (n=16) Non Surv. (n=14)</td>
<td>Surv. (n=16) Non Surv. (n=14)</td>
<td>Surv. (n=16) Non Surv. (n=14)</td>
<td>Surv. (n=16) Non Surv. (n=14)</td>
</tr>
<tr>
<td>Min</td>
<td>220</td>
<td>360</td>
<td>240</td>
<td>370</td>
<td>250</td>
</tr>
<tr>
<td>Max</td>
<td>590</td>
<td>660</td>
<td>590</td>
<td>740</td>
<td>620</td>
</tr>
<tr>
<td>Mean</td>
<td>318.75</td>
<td>502.86</td>
<td>326.88</td>
<td>524.29</td>
<td>371.25</td>
</tr>
<tr>
<td>SD</td>
<td>99.19</td>
<td>85.88</td>
<td>97.14</td>
<td>97.25</td>
<td>104.49</td>
</tr>
<tr>
<td>t-test</td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
<td>P = 0.000*</td>
</tr>
<tr>
<td>t-test between level of ICAM-1 and its basal level</td>
<td>P = 0.001*</td>
<td>P = 0.004*</td>
<td>P = 0.000*</td>
<td>P = 0.002*</td>
<td>P = 0.000*</td>
</tr>
</tbody>
</table>

P is significant if < 0.05
* = significant
Figure 1: Comparison between sICAM values in cases at different times of the study

Figure 2: Comparison between sVCAM values in cases at different times of the study
Figure 3: Correlation between sICAM level and APACHE II

Figure 4: Correlation between sICAM level and MOFC
Figure 5: Correlation between sVCAM level and APACHE II

Figure 6: Correlation between sVCAM level and MOFC
DISCUSSION

Severe sepsis remains the dominant challenge in the care of critically ill patients. Septic shock is difficult to understand because of the heterogeneity of patients affected, that is, widely different clinical manifestations occur in postoperative, posttraumatic, urologic and general internal medicine patients and in those with respiratory failure.

It is a systemic response to severe bacterial infections, generally caused by Gram negative bacterial endotoxins, with multiple manifestations such as hypotension, tissue injury, disseminated intravascular coagulation, and multi-organ failure. All these effects, are induced by the generation of pro-inflammatory and vasodilator mediators, cell adhesion molecules, coagulation factors, and acute-phase proteins.

Our understanding of the pathogenesis of severe sepsis continues to grow. Expression of membrane surface molecules such as toll-like receptors, adhesion molecules and cytokine receptors induces a high degree of redundancy and amplification. Despite the several adhesion-dependent phenomena that occur in septic shock, the role of adhesion molecules in this process has not been fully investigated and is still the subject of research.

Soluble adhesion molecules are detectable at low levels in healthy people but are increased in patients with various disorders. They may reflect an immune response resulting from tissue distraction or chronic inflammation, or they may serve as immunomodulators of subclinical inflammation or arise as a consequence of inflammation.

In the present work, sICAM-1 was found to be significantly higher in patients with sepsis (62.6±20.21 ng/ml) than in the control group (14.0±4.71 ng/ml). Our data were in concordance with Sessler et al (3) who stated that sICAM plasma levels were significantly elevated in patients suffering from septic shock. Several other clinical trials have also reported extensive increase in the circulating concentrations of some adhesion molecules in sepsis. An increase in circulating adhesion molecules e.g. ICAM-1 may either result from a cytokine-induced increased expression by endothelial cells, or from increased proteolytic cleavage of endothelial bound adhesion molecules secondary to endothelial damage, or both.

In our work, sVCAM-1 was found to be significantly higher in septic patients (404.67±130.85 ng/ml) than in controls (128.0±34.9 ng/ml). This was in agreement with Turner et al (21) and Boldt and associates (22). The latter showed that serum ICAM-1 and VCAM-1 levels increased in all groups of critically ill patients and that this increase was significantly highest in the sepsis group. Similar observations were noted by other workers in specific populations.

In the present study, sICAM-1 level ranged between 32-96 ng/ml and increased gradually till the 5th day of the study where it ranged between 52-193 ng/ml. Some authors stated that baseline sICAM-1, as a marker of endothelial cell activation, predicted disease severity and that it reflected the intensity of inflammation and tissue damage in late sepsis. As regards sVCAM-1, the levels gradually increased from a baseline mean of 404.67 ng/ml to 589.67 ng/ml on day 5 of the study. A similar persistent elevation was observed by other workers for as long as one week after admission.

We also demonstrated higher levels of sICAM-1 and sVCAM among shocked patients compared to non-shocked patients. Other workers showed similar findings.

Both adhesion molecules were higher among non-survivors compared to survivors thus suggesting that they could serve as an early prognostic marker for outcome in septic shock. This was confirmed by other investigators who obtained comparable results.
with sICAM-1 in particular. Another study, however, found sICAM-1 to be a relatively non-specific indicator for sepsis in contrast to sE-selectin which seemed to be a good and early predictor of the beginning of severe sepsis with MOF, and to have a prognostic value for the severity, possible course, and outcome of developing sepsis. One study carried out on mice suggests that ICAM-1 has an important pathophysiological role in the response to polymicrobial sepsis pointing out that absence of this molecule impairs the ability of PMNL to migrate into organ tissues and reduces consequent secondary organ damage resulting in improved clinical status and overall survival. However, further investigation into the effectiveness of ICAM-1 modulation in the treatment of sepsis is warranted. In our study, we also detected a statistically significant positive correlation between the studied adhesion molecules and APACHE II score as well as both organ failure scores for the 1st day or cumulative. It is speculated that endotoxin and inflammatory cytokines damage vascular endothelium as well as various other cells and produce, a large number of adhesion molecule, especially in patients with septic MOF, causing leakage of adhesion molecules into blood. Our findings were in agreement with Whalen et al. who showed that plasma ICAM-1 and VCAM-1 concentrations could independently predict the development of multiple organ failure as well as mortality. Similar findings, more or less were detected by others.

Regarding the significant negative correlation we detected between the studied adhesion molecules and some haemodynamic parameters (SVR, CVP), this could be attributed to the increased vasodilatation accompanying sepsis or septic shock with subsequent drop in vascular resistance and central venous pressure. The increase in inflammatory mediators stimulates the release of more adhesion molecules.

In view of our findings and the reviewed literature, we conclude that serum levels of ICAM-1 and VCAM-1 in critically ill patients may be useful in predicting the development of sepsis and assessing disease severity. They may serve as early prognostic markers for outcome in septic shock and as reliable investigations for recognition of patients at high risk of multiple organ dysfunction or failure. They might also provide a valuable means of monitoring and guidance of therapy.

We recommend further research to reveal important pathophysiological processes around which promising therapeutic strategies with potential benefits may be derived. Endothelium-white cell interactions might be an appropriate target for future interventions. Insights into the pathogenesis of severe sepsis and the knowledge of which adhesion molecules are involved in the distinct pathophysiology of disease opens the door to novel therapeutic strategies using monoclonal antibodies or the use of substances known to reduce endothelial damage.

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المتسلق العربي

تقييم دور جزيئات التلاصق بين الخلوى في التوقف المستقبلي لحالات التيفي الميكروبي الحرقة

محمد إبراهيم عبد الرؤوس، نتجية زكي سعد، منى صبحي سدراك، مها منير الغريباوي،
محمد إبراهيم محمود، تسير محمد زيوتن

يعتبر الصدم المتعاكس (النحافة) من أكثر أسباب الوفيات في الغرب، ويرجع ذلك إلى أن
التطور الكبير الذي حدث في العلاج بواسطة المضادات الحيوية والرعاية الحرقية والمراقبة
للاجل لم يطرأ أي تغيير كبير في تأثير تلك الصدم المتعاكس. من أجل الوقاية من الصدم أو
العجز، يمكن أن يكون الجهد من هذا البحث هو قياس مستوى جزيئات التلاصق بين الخلوى - 1 و-
جزيئات التلاصق الوعائية - 1 في مرضى الغرب الذين يعانون من الاستسلام مع صمود أو بدون.
وقد اتبعنا هذه الدراسة على حالات الاستسلام واعتقاداً بالدور الدموي وتحقيق المستقبلي للكمortal
30 فريقاً مصابًا بالاستسلام مع صمود أو بدون في وحدات الغربة المركز للمستشفى الرئيسي الجامعي
بالإلكترونية. وتم فحص جميع الحالات
عن طريق أخذ التاريخ المرضي وفحص الإكلينيكي الدقيق وعمل الفحوصات الفيروية والإنتاجية و-
غائزات الدم بالإضافة إلى مرضي الميكروبيات في جميع الحالات ومن مكان الأصابات.
وقد تم حساب نسبة الاستسلام في الأسابيع الأولى والأخيرة. كما شمل البحث قياس جزيئات التلاصق بين الخلوى - 1 و-
جزيئات التلاصق الوعائية - 1 عند دخول الغربة المركز وآليب الحبة التي تلتها كما تم مراقبة المرضي يومياً خلال
معينة وجدت بوجود الغربة المركزية. وقد كان أكثر الألماء ضعفي استسلام الميكروبيات هو الصحة و
كانت الميكروبيات سالبة الجرم في أكثر الميكروبيات التي عززت (30%) و كانت مرضي الميكروبيات في
تسع حالات (30%). و قد لوحظ ارتفاع مستوي جزيئات التلاصق بين الخلوى - 1 (1,201,720 نانوجرام في الميلين)،
و جزيئات التلاصق الوعائية - 1 (1,173,230 نانوجرام في الميلين) بالدم من تاريخ الدخول وظل في ازداد مضطردًا حتى اليوم الذي عززت ذلك زيادة دلالية معينة
في المرضي مقارنة بوجود الغربة المركزية (71,437,440, 1,401,847, 9,523, 201,840 نانوجرام في الميلين) على
الواقي. وقد كان الارتفاع في مستوي هذه جزيئات بدرجة أكبر في مرضى الاستسلام المصموم بصدمة و-
الحالات التي انتهت بالوفاة، و التي أظهرت التوقف في الوارد. كما هناك علاقة طردية ذات دلالية
معينة بين جزيئات التلاصق بين الخلوى - 1 و جزيئات التلاصق الوعائية - 1 من جهة و مقياس
الابتشي - 2 و مقياس عامل الأمعاء على الجانب الآخر. ونستنتج من هذا البحث أن قياس عوامل
الالتهاب الموجودة بالدم في مرضى الحالات الحرقية يمكن الاعتماد عليه في تشخيص ووضع حدوث
الاستسلام كما أنها تعتبر مؤشراً يمكن الاعتماد عليه في تشخيص المرضي المخصوصين لفشل الأمعاء و كذلك
النحو بشكل تلك الحالات و ما ينتج ذلك من فوارق في اتخاذ قرارات خاصة بالعلاج.