ROLE OF LEUKOCYTES IN ACUTE CEREBROVASCULAR STROKE (CVS)


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ABSTRACT

50 patients presenting with acute CVS and 30 controls were studied for the possible role of leukocytes in such an illness. They were tested for total and differential leukocytic count together with leukocytic aggregation. Results were correlated with stroke subtype (ischaemic or haemorrhagic); brain insult size (large or small), initial neurological deficit (initial Glasgow coma score and ADL score) and finally with short term outcome (one month follow up ADL score). We found significant differences between patients and controls regarding leukocytic count and aggregation being higher in patients. Furthermore, leukocytic count and aggregation were directly proportional to stroke severity and poor outcome.

Also, significantly, leukocytic count and aggregation were higher in patients with ischaemic than haemorrhagic strokes.

We conclude that leukocytic count and aggregation would be an easy but valuable parameter in assessing stroke severity and predicting its outcome.

INTRODUCTION

Recently, attention has been focused on the role of leukocytes in acute CVS (Hallenbeck and Dutka, 1990). The visco-elastic, functional and morphological properties of these cells qualify them for a central role in acute CVS especially ischaemic one (Mercuri, et al., 1990).

Nevertheless, the exact role and mechanism of leukocytes in such an illness has not been fully elucidated.

PATIENTS AND METHODS

Patients presenting with acute CVS admitted into Al-Hussein University Hospital in the period between Nov. 1993 and April 1994 were included in the study.

Inclusion criteria:
- A focal neurological deficit(s) of sudden onset, lasting for more than 24 hours.
- A readily positive brain CT for an infarct or intracranial haemorrhage.
- Admission into Al-Hussein University Hospital in the second day after stroke onset.
- A stay for one month, at least, unless terminated by death or discharge for unexpected reasons.

Exclusion criteria:
- A known inflammatory disease e.g. Behcet's disease.
- A recent infection e.g. urinary tract or chest infection.
- A haematological disorder e.g. leukaemia.
- A disabling previous disease e.g. hemiplegia or paraplegia.
- All patients were subjected to:
  - History and clinical neurological and general assessment including glasgow coma and Barthel ADL scales, initially and one month later.
  - Brain CT scanning.
  - Routine laboratory investigations: ESR; blood glucose and urea; serum creatinine; liver functions and urine & stool analysis.
  - C.B.C. including total and differential leukocytic count.
  - Leukocytic aggregation.
  - ECG and X-ray chest.
  - Special investigations whenever required.

Stroke outcome was evaluated by Barthel ADL scale on admission and one month later.

30 healthy subjects were chosen with similar age and sex to patients. They had no history of inflammatory disease, recent infection or haematological disorder.

They were subjected to:
- History and clinical assessment.
- Total and differential leukocytic count.
- Leukocytic aggregation.

RESULTS

A total of 80 cases were included in the study.
They comprised three groups:

Group I: 35 patients presented with acute cerebral infarction (18 were males and 17 were females), their age ranged between 45 - 70 years with a mean of 58.91 ± 8.79.

Group II: 15 patients presented with acute haemorrhagic stroke, 9 with ICH (intracerebral haemorrhage) and 7 with SAH (subarachnoid haemorrhage), 8 were males
and 7 were females. Their age ranged between 45 - 70 years with a mean of 52.93 ± 7.19 years.

Group III: Control group; 30 normal subjects, 15 were males and 15 were females. Their age ranged between 45 - 70 with a mean of 56.8 ± 8.13 years.

As regard age, there was no significant difference between patients and control Table (1).

<table>
<thead>
<tr>
<th>Table (1) : Age of cases.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
</tr>
<tr>
<td>Range</td>
</tr>
<tr>
<td>Mean ± SD</td>
</tr>
</tbody>
</table>

There was no significant difference between age of the patients who died or survived in both infarct and haemorrhagic groups. There was no significant correlation between age in each of the studied groups and total, differential leukocytic count or aggregation.

Mean ± SD total leukocytic count and aggregation were higher in infarction (11.76 ± 3.03 for the leukocytic count and 9.08 ± 2.0 for aggregation) and hemorrhagic (12.62 ± 7.05 for leukocytic count and 5.33 ± 1.29 for aggregation) groups than control (6.26 ± 1.04 for leukocytic count and 1.37 ± 0.82 for aggregation) group and this was of highly significant value (P < 0.001). There was no significant difference between infarct and haemorrhagic groups regarding total leukocytic count but there was highly significant difference regarding leukocytic aggregation being higher in infarct group (P < 0.001) (Fig. 1).

As regard differential leukocytic count, there was no significant difference between percentage of lymphocyte and monocyte in control, infant and hemorrhagic groups, however neutrophil percentage was higher in infarct and hemorrhagic groups than control group and this was of highly significant value (P < 0.001) (Table 2). Also neutrophil percentage was higher in infarct group than

<table>
<thead>
<tr>
<th>Table (2) : Differential leukocytic count among different groups.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group Mean ± SD of differential leukocyte count</td>
</tr>
<tr>
<td>Neutrophil</td>
</tr>
<tr>
<td>Lymphocyte</td>
</tr>
<tr>
<td>Monocyte</td>
</tr>
</tbody>
</table>
haemorrhagic group and this was of significant value ($P < 0.02$).

**In infarction group:**

There was significant correlation between leukocytic count and aggregation and the Barthel ADL score on admission (Fig. 2 & 6) that had become highly significant after one month (Fig. 4 & 8).

There was significant inverse correlation between Glasgow coma scale and both leukocytic count and aggregation (Fig. 12 & 14).

Leukocytic count and aggregation were higher among patients with large sized infarcts than those with small sized infarct and this was of highly significant value ($P < 0.001$) (Table 3).

**Table (3) : Relation between leukocytic count, aggregation and infarct size among infarct patients.**

<table>
<thead>
<tr>
<th>Infarct size</th>
<th>Leukocytic count &amp; aggregation</th>
<th>Small sized infarction (17 patients)</th>
<th>Large sized infarction (18 patients)</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leukocytic count</td>
<td>9.79 ± 2.21</td>
<td>13.62 ± 2.49</td>
<td>$&lt; 0.001$</td>
<td></td>
</tr>
<tr>
<td>Leukocytic aggregation</td>
<td>7.58 ± 1.8</td>
<td>10.5 ± 0.78</td>
<td>$&lt; 0.001$</td>
<td></td>
</tr>
</tbody>
</table>

N.B. : * Large Sized infarction = The hypodense area is including whole territory - cortical and subcortical - supplied by one of the major cerebral arteries.

* Small sized infarction = The hypodense area is including cortical or subcortical region alone.

Also, there was highly significant correlation between initial Glasgow coma scale and Barthel ADL score after one month (Fig. 10).

Leukocytic count and aggregation were higher in patients who died, than in patients who survived and this was of highly significant value ($P < 0.001$) (Table 4).

**Table (4) : Age, leukocytic count and aggregation in survived and died subjects in infarction group.**

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of cases</th>
<th>Age of cases</th>
<th>Leukocytic count</th>
<th>Leukocytic aggregation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Died</td>
<td>7 (20%)</td>
<td>62.14 ± 6.76</td>
<td>16.31 ± 1.31</td>
<td>11.28 ± 0.48</td>
</tr>
<tr>
<td>Survived</td>
<td>28 (80%)</td>
<td>58.10 ± 9.16</td>
<td>10.62 ± 2.1</td>
<td>8.53 ± 1.85</td>
</tr>
<tr>
<td>$P$ value</td>
<td></td>
<td>$&gt; 0.10$</td>
<td>$&lt; 0.001$</td>
<td>$&lt; 0.001$</td>
</tr>
</tbody>
</table>
In haemorrhagic group:

Patients with subarachnoid haemorrhage had higher leukocytic count and aggregation than those patients with intracerebral haemorrhage and this was statistically highly significant ($P < 0.001$) (Table 5).

Table (5): Leukocytic count and aggregation in patients with subarachnoid and intracerebral haemorrhage.

<table>
<thead>
<tr>
<th>Intracranial haemorrhage type</th>
<th>Leukocytic count</th>
<th>Leukocytic aggregation</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICH no = 9 (60%)</td>
<td>7.65 ± 3.29</td>
<td>4.55 ± 1.01</td>
</tr>
<tr>
<td>SAH no = 6 (40%)</td>
<td>20.08 ± 3.28</td>
<td>6.5 ± 0.547</td>
</tr>
</tbody>
</table>

There was highly significant correlation between leukocytic count and aggregation and Barthel ADL score on admission and after one month (Fig. 3, 5, 7, 9).

There was highly significant inverse correlation between leukocytic count and Glasgow coma scale, and less significant correlation between leukocytic aggregation and Glasgow coma scale (Fig. 13, 15).

Table (6): Age, leukocytic count and aggregation in survived and died subjects in haemorrhagic group.

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of cases</th>
<th>Age of cases</th>
<th>Leukocytic count</th>
<th>Leukocytic aggregation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Died</td>
<td>5 (33.3%)</td>
<td>56 ± 8.63</td>
<td>20.48 ± 3.50</td>
<td>6.6 ± 0.54</td>
</tr>
<tr>
<td>Survived</td>
<td>10 (66.6%)</td>
<td>51.4 ± 5.96</td>
<td>8.7 ± 4.53</td>
<td>4.7 ± 1.05</td>
</tr>
<tr>
<td>P value</td>
<td></td>
<td>&gt; 0.10</td>
<td>&lt; 0.001</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

There was highly significant correlation between initial Glasgow coma scale and Barthel ADL score after one month (Fig. 11).

Leukocytic count and aggregation were higher in patients who died than in patients who survived and this was of highly significant value ($P < 0.001$) (Table 6).
Figure (12) Correlation between serial Glasgow coma score and total leukocyte count in infarction group.

Figure (13) Correlation between initial Glasgow coma score and total leukocyte count in hemorrhagic group.

Figure (14) Correlation between initial Glasgow coma score and leukocyte aggregation in infarction group.

Figure (15) Correlation between initial Glasgow coma score and leukocyte aggregation in hemorrhagic group.
Role of leukocytes in acute ...
Figure 2: Correlation between total leucocyte count and the Barthel ADL score on admission in infarction group.

Figure 3: Correlation between total leucocyte count and the Barthel ADL score on admission in hemorrhagic group.

Figure 4: Correlation between total leucocyte count and the Barthel ADL score after one month in infarction group.

Figure 5: Correlation between total leucocyte count and the Barthel ADL score after one month in hemorrhagic group.
DISCUSSION

Prentice et al., (1982) & Harrison and Marshall, (1987) stated that, a high total leukocytic count is considered to be a predictor of stroke and T.I.A. In our study, we found that there is a marked increase of total leukocytic count in the early stage of ischaemic stroke. Also an obvious correlation has been found between this leukocytosis and the degree of unconsciousness judged by Glasgow Coma scale, (Fig. 12) and Barthel ADL score within the 1st. 24 hours (Fig. 2) as well as at the end of the 1st. month follow up, (Figure 4) and this agrees with the finding of Pozzilli et al., (1985), and Galante et al., (1992), and disagrees with the finding of Rowland, (1989) who concluded that, leukocytic count is usually normal in patients with ischaemic stroke.

The present study showed that, those patients who died had a higher leukocytic count than that of those who survived, with the difference between them was statistically highly significant, (Table 4), and this agrees with the finding of Lowe et al., (1983), Pozzilli et al., (1985), and Galante et al., (1992).

There was a significant positive correlation between the leukocytic count and the extent of infarction in C.T. scan and this agrees with the study of Pozzilli et al., (1985).

As regard the differential leukocytic count, neutrophils were significantly higher in patients than controls, (Table 2) and this agreed with the finding of, Bednar et al., (1991). Also Sornas et al., (1972) found a transitory increase of neutrophil in cerebrospinal fluid during 3 to 4 days after the stroke which could not be done in our study.

The elevation of leukocytes especially neutrophils both in the peripheral blood and CSF in the acute stage after infarction and the direct relation to size of the infarction may denote some degree of inflammatory responses to the local cerebral damage, (Pozzilli et al., 1985) and may be due to sympathetic over activity with secondary release of catecholamines and corticosteroids after the stress of stroke i.e. part of a generalized stress reaction in response to stroke (Galante et al., 1992).

However, the lack of protective action of neutropenia in the development of ischaemic stroke in experimental animals as reported by Aspey et al., (1989), who suggested that the total leukocytic count is not the only relevant factor to explain leukocytes role. Therefore a functional alteration of leukocyte may be an important factor than the total leukocytic count and this is strengthened by the known mechanisms of leukocytes in pathogenesis of ischaemic stroke, (Harlan, 1985, Moncade and Higgs, 1986, Ernst et al., 1987, Schonbein, 1987, Hallenbeck and Dutka, 1990, and Tanaka et al., 1993) and/or through it's role in potentiating or precipitating the already present risk factors such as diabetes mellitus (Wilson, 1991) ischaemic heart disease (Berliner et al., 1986 and Tanaka et al., 1993), atherosclerosis, (Light, 1986, Nathan, 1987, Ross, 1988) and viscosity, (Sung et al., 1982, Braide et al., 1986, Shonbcin, 1987 & Sutton and Schonbcin, 1990).
The result of this study in addition to confirm the association between leukocytosis and ischaemic stroke also showed that leukocytic aggregation was significantly higher in patients than controls (Fig.1). Also there is a positive correlation between leukocytic aggregation and the degree of unconsciousness judged by Glasgow coma scale (Fig. 14) and Barthel ADL score within the 1st. 24 hours, (Fig. 6) as well as after one month, (Fig.8) and this figure is comparable to the finding of, (Galante et al., 1992) and (Galante et al., 1993).

The present study showed that, those patients who died had a higher leukocyte aggregation than that of those who survived, with the difference between them was statistically highly significant, (Table, 4) and this agree with the finding of (Galante et al., 1992).

The significant positive correlation found between leukocytic aggregation and the extent of infarct area showed by C.T. scan in this study, (Table 3) agreed with the finding of, (Berliner et al., 1986) who found a positive correlation between the size of the ischaemic lesion and the percentage of leukocytic aggregation in patients with myocardial infarction.

However, the strict relationship between the level of hyperaggregability and the clinical course suggests that the former is not a non-specific or non-influential response. It could, at least, play a role in the evolution of an ischaemic event, perhaps by obstructing collateral circulation and facilitating the no reflow phenomenon as (Mercuri, et al., 1990) found. This hypothesis is supported by experimental studies showing that activated leukocytes can obstruct individual capillaries in ischaemic injury area (Engler et al., 1983 and Schonbein 1987). This may be more likely to occur with higher leukocytic count.

In haemorrhagic stroke:

In our study, leukocytic count was significantly higher in patients than controls (Fig. 1) and this figure is comparable to that reported by (Rowland, 1989).

The present study also showed an obvious correlation between the leukocytosis and degree of unconsciousness judged by Glasgow coma scale, (Fig. 3) and Barthel ADL score within the 1st. 24 hours, (Fig. 3) as well as at the end of the 1st. month follow up, (Fig. 5), and mortality, (Table 6) and this agrees with the finding of (Dwyer and Cruickshank, 1974 & Parkinson and Stephensen, 1984).

As regard differential leukocytic count, neutrophil count was significantly higher in patients with haemorrhagic stroke than controls, (Table 2) and this agrees with finding of (Dwyer and Cruickshank, 1974) who suggested that, this increase may reflect the degree of the tissue necrosis and / or a stress situation with increased catecholamine and corticosteroid activity which probably lead to cerebral arterial spasm. But they found that, in patients with subarachnoid haemorrhage, the spasm and prearterial spasm phases significantly higher leukocytic count and neutrophil percent than those with non spasm and this in the same line with, (Kubota et al., 1993) who concluded that cellular and humoral immunity play a role in the pathogenesis of cerebral vasospasm after subarachnoid haemorrhage and this also with the same line with our study which showed significantly higher leukocyte aggregation in pa-
tients with haemorrhagic stroke than controls (Fig. 1) and the degree of the increase was highly significant in relation to the severity of illness as regard consciousness, (Fig. 15) and Barthel ADL score, (Fig. 7) outcome as regard Barthel ADL after one month, (Fig. 9) and mortality, (Table 6) and this in agreement with the study of, (Perry and Granger, 1992) who found reduction in leukocyte rolling velocity and increase in leukocyte adherence and emigration in haemorrhage induced ischaemia.

In this study, we found that, leukocytic aggregation was significantly higher in patients with subarachnoid haemorrhage than that with intracerebral haemorrhage (Table 5). Also, leukocytic aggregation was significantly higher in patients with ischaemic stroke than haemorrhagic stroke, (Fig. 1) and this was expected where there was more cellular alteration in ischaemic stroke as regard function.

As regard level of consciousness, 20, (40%) of acute stroke patients suffered an alteration in their level of consciousness at the onset of admission and this agreed with (Wade et al., 1985).

The level of consciousness soon after stroke was an important prognostic factor for both survival and for short term functional recovery, where all patients being fully conscious on admission did not die during the follow up period and 12, (60%) of the 20 cases who has impaired consciousness on admission died during the follow up period, these results were in agreement with, (Matthews and Oxbury, 1975) who found that, all patients, who died within 3 weeks post stroke had impaired consciousness on admission and none of the patients who were admitted fully conscious died within the same interval of time.

Also all the survived cases who recovered fully one month post stroke among infarction group had no alteration in their level of consciousness, (Figure 10, 11). This was in agreement with (Matthews and Oxbury, 1975) who found that 48, (65%) of 74 stroke patients due to infarction who were alert on admission, recovered fully and became independent to 6 months follow up.

Incidence of intracerebral haemorrhage is frequent than subarachnoid haemorrhage, (Table 5) and this agreed with the finding of (Robbins and Baum, 1981) in which intracerebral haemorrhage was 6.3% and subarachnoid haemorrhage 5.9% among stroke patients and disagreed with the finding of, (Mohr et al., 1978) which concluded that, intracerebral haemorrhage occurred with equal frequency to subarachnoid haemorrhage.

The number of dead subjects among patients within the first month after the onset of stroke was 12, (24%) and this percentage was less than in the finding of (Herman, 1982) who suggested that about 30% of all acute stroke patients died within the first 4 weeks.

There was no significant difference in the mean age and stroke type between those who died and those who survived, (Table 4, 6) and this is consistent with the finding of Candelise et al., 1985).
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دور الخلايا البيض في السكتة الدماغية

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يهدف هذا البحث أساساً إلى تقديم دور الخلايا البيض من حيث عددها وتكميمها في مرضي السكتة الدماغية. وعندما يثيرنا هذا النمو في العين بالسكتة الدماغية خلال شهر من بدء حديث أجريت الدراسة على خمس مرضى يعانون من الإحتشاء الدماغي الحاد من بين المرضى على مستشفى الحسين الجامعي بكلية طب الأزهر خلال 24 ساعة من بدء حدوث السكتة الدماغية. وكان محدد اختيار حالات الدراسة هو إصابتها بقصور عصبي يؤدي إيجابياً أو سريع البداية موثقاً بنتائج الفحص المقطعي بالكمبيوتر لدماغ بالأشعة المهربة.

ولم يكن هناك تاريخ مرضي لهذه المرضي موضوع الدراسة لأي من أمراض الدم أو أي نوع من الإجهاضات أو سكتة دماغية أخرى أو تعاطي أي أدوية أخرى خلال 15 يوماً قبل حدوث السكتة وكذلك لم يكن هناك دليل على وجود إحتشاء قلبي مصاحب لهذا المرض.

وقد تراوحت أعمارهم بين 25 - 70 عاماً وكان من بينهم 35 مريضاً بالإحتشاء الدماغي الحاد بمتوسط عمر 69 عاماً من بينهم 17 ذكرًا و17 أنثى في حين أن 15 مريضاً 30% فقط أصيبوا بالنزف داخل الجمجمة، وتسعة أعمارهم 63 عاماً من بينهم 8 ذكور و27 إناث، وكان من 9 حالات أصيبوا بالنزف داخل المخ و11 حالة بالنزف تحت العنكبوتية.

وقد تم اختيار عينة ضامنة من ثلاثين فردًا من أقارب المرضى الأصحاء حيث تراوح أعمارهم بين 40 - 70 عاماً بمتوسط أعمارهم 47 تصفح من الذكور والنساء الأخر من الإناث ولم يكن لدي أي منهم تاريخ مرضي يرجع ترجمة لمرض عصبي أو أي من أمراض الدم أو أي نوع من الإجهاضات أو سكتات قلبية أو تعاطي أي أدوية خلال 15 يوماً قبل حدوث السكتة وكذلك لم يكن هناك دليل على وجود إحتشاء قلبي مصاحب لهذا المرض.

وقد تراوحت أعمارهم بين 40 - 70 عاماً وكان من بينهم 35 مريضاً بالإحتشاء الدماغي الحاد بمتوسط عمر 69 عاماً من بينهم 17 ذكرًا و17 أنثى في حين أن 15 مريضاً 30% فقط أصيبوا بالنزف داخل الجمجمة، وتسعة أعمارهم 63 عاماً من بينهم 8 ذكور و27 إناث، وكان من 9 حالات أصيبوا بالنزف داخل المخ و11 حالة بالنزف تحت العنكبوتية.
ولقد تم اختيار عينة شابة من ثلاثين فردًا من أقارب المرضى الأصحاء بحيث تتراوح أعمارهم بين 45 - 70 عامًا ومناينت أعمارهم 67 عامًا نصفهم من الذكور والنصف الآخر من الإناث ولم يكن لدى أي منهم تاريخ مرضي يرجع تعرفي لأمراض عصبية أو أي من أمراض الدم أو أي نوع من الإلغابات أو مساهمات قلبية أو تفاعلي أدية خلال الستة عشر يومًا السابقة لأخذ العينة. وقد كان الهدف من دراسة هذه المجموعة الضابطة هو مقارنة عدد الخلايا البيض الكلي والتفزيقي وكذا تكسيسها بنتائج مجموعة المرض موضوع الدراسة.

المجموعة الأولى: المرضى نوى الإحشاء الدماغي الحاد
المجموعة الثانية: المرضى نوى النزف داخل النسيج الدماغي تحت المنكوبة

ولقد تم تتبيع هؤلاء المرضى على مدى شهر من بدء السكتة فيما عدا الوفيات الذين بلغ عددهم 12 حالة (24).

ولقد كانت إجراءات تقويم الحالات كما يلي:
1- فحص إكلينيكي شامل مع التركيز على الفحص العصبي ووصفة خاصة قياس درجة وعي المريض عند بدء المرض باستخدام مقياس (جلاسجو) في حالات الشفوية وكذا تياس مدى الحقل الحركي عند بدء المرض وبعد شهر باستخدام قياس (بارسل).
2- قياس الدماغ بالأشعة النقطة المبرمجة بمساعدة الكمبيوتر.
3- قياس العد الكلي للخلايا البيض.
4- قياس العد التفزيقي للخلايا البيض.
5- تياس تكس خلايا البيض.
6- تياس تياس كهرمات.
7- إستقصاءات عملية روتينية.
8- إستقصاءات عملية خاصة عند الحاجة إليها.
9- تحديد حال المرض بعد شهر من بدنه وذلك باستخدام قياس (بارسل).
10- معاينة النتائج إحصائيًا ومقارنةها في ضوء ما أسفرت عنه الدراسات السابقة ومقارنتها بنتائج المجموعة الضابطة.
وأسفريت الدراسة عن النتائج التالية:

1. عدم وجود فرق ذات دلالة إحصائية بين مجموعتي المرضى والمجموعة الضابطة من ناحية العمر.
2. كان متوسط أعمار المرضى ذوي الإحصاء الدماغي الموات أعلى فارقًا في مرضى النزف سوءًا داخلي النسيج المخبي أو تحت العنكبوتية.
3. كان هناك زيادة ذات دلالة إحصائية في عدد الخلايا البيض الكلي والملة وكذا معدل تكس هذه الخلايا عند مجموعتي المرضى بمن حسب إجابات هذه الدالة ارتباطًا إيجابيًا بدرجة العجز العصبي وكذا حال المرضى.
4. كان عد الخلايا العيدية وكذا تكس الخلايا البيض أعلى في مرضى النزف سوءًا داخلي النسيج المخبي من بين مرضى النزف بمن حسب إجابات هذه الدالة عند مرضى النزف بمن حسب إجابات هذه الدالة.
5. كان عد الخلايا البيض الكلي وكذا تكس هذه الخلايا أعلى في مرضى النزف سوءًا داخلي النسيج المخبي بمقارنتهم بمرضى النزف داخلي النسيج المخبي.
6. كان عدد الخلايا البيض الكلي وكذا تكس هذه الخلايا أعلى في مرضى النزف سوءًا داخلي النسيج المخبي بمقارنة بمرضى النزف داخلي النسيج المخبي.
7. كان عدد الخلايا البيض الكلي وكذا تكسها أعلى في مرضى النزف سوءًا داخلي النسيج المخبي بمقارنة بمرضى النزف سواءًا داخلي النسيج المخبي.
8. مرتبة تدور درجة الوعي عند حدوث السكتة الدماغية بما فيها السليم (من حيث العجز والوقاية) خلال شهر من حصولها ارتباطًا سطحيًا.