Prediction of Post-Stroke Dementia

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ABSTRACT

Background: Identification of predictors and significant risk factors of VaD is so important as it could provide an avenue for targeted therapeutic intervention to prevent cognitive deterioration. Aim of the Work: The purpose of this study was to investigate the predictive factors for the development of post-stroke dementia and predicting patients liable to develop dementia for a prophylactic treatment. Methods: This study was carried out on 45 stroke patients their ages ranged from 50 to 70 years and were followed for 3 months for the occurrence of post-stroke dementia. Patients were subjected to full general, neurological and neurovascular examinations, stroke severity was assessed using NIH stroke scale, routine laboratory investigations. CT brain and clinical neuropsychological rating scales including MMSS, HIS, Blessed Dementia Stroke, Clinical Dementia Rating Scale, Global Deterioration Scale, and DSM-IV for vascular dementia. Results: Various significant predictors and risk factors were more prevalent in VaD. Of these data aging, male sex, and low level of education, uncontrolled hypertension, smoking, uncontrolled D.M, ischaemic heart diseases and hyperlipidemia. Moreover, certain radiological data e.g. left sided (dominant) hemispheric lesions, multiple and large sized lesions were significantly associated with the occurrence of post-stroke dementia. Conclusion: The only major cause in all causes of dementia that is preventable is post-stroke dementia. Education seems to be an important protective factor against vascular dementia. It appears that, modifiable cardiovascular risk factors may identify persons at risk for vascular dementia. Treatment of these factors and efforts aimed at prevention of stroke will lead to prevention and reduction of dementia associated with stroke. (Egypt J. Neurol. Psychiat. Neurosurg., 2007, 44(1): 259-270).

INTRODUCTION

The term "post-stroke dementia" includes all dementias revealed after a stroke, irrespective of their cause. Dementia may be the consequence of vascular lesions of the brain, associated Alzheimer's type pathology, white matter changes or the cumulative effect of these lesions1,2.

While it is known that ischemic stroke significantly increases the long-term risk of developing dementia among patients initially found to be non-demented, few studies have investigated the risk factors for incident dementia after ischemic stroke. However, even though the identification of significant risk factors could provide an avenue for targeted therapeutic intervention to prevent cognitive deterioration1.

The purpose of this study was to investigate the predictive factors and to establish a model for the development of post-stroke dementia and predicting patients liable to develop dementia for a prophylactic treatment.

PATIENTS AND METHODS

This study was conducted on 45 patients with acute cerebrovascular ischemic stroke and were followed up to detect the occurrence of dementia 3 months after stroke onset. They were 30 males and 15 females, ranging in age from 50 to 70 years.

After the follow up period, the patients were divided into two groups according to the results of the following neuropsychological scales:
1. Mini-mental scale3.
2. Blessed dementia scale3.
3. Clinical dementia rating scale5.
The Two Groups:
- **Group A**: (Demented group): which included 30 patients (20 males and 10 females) who developed cognitive impairment and post-stroke dementia in the follow up period.
- **Group B**: (Non-demented group): which included 15 patients (10 males and 5 females) who didn't develop cognitive impairment or post-stroke dementia in the follow-up period and they are considered as control group in this study.

Exclusion criteria:
1. Mini-mental scale less than 24.
2. Patients with transient ischaemic attacks.
3. Patients with other neurological disease as Parkinson’s disease.
4. Patients with Alzheimer disease and other diseases causing dementia.
5. Patients with severe medical co-morbidity e.g. cancer in the terminal stages.
6. Patients with organ failure as renal, hepatic, cardiac, etc…

Methods:
Subjects in this study were submitted to the following:
1. Careful history taking and thorough general and neurological examination.
2. Neurovascular examination.
3. Stroke severity is assessed by NIH stroke scale on admission.
4. Routine laboratory investigations:
   * Complete blood picture.
   * Blood sugar e.g. fasting and two hours post-prandial.
   * Renal function e.g. urea and creatinine.
   * Liver functions.
   * Serum uric acid.
   * Lipid profile including:
     Triglycerides, cholesterol, low-density lipoprotein and high-density lipoprotein.
5. Neuroimaging studies including CT and/or MRI brain.

6. Clinical and neuropsychological rating scales:
   a) Mini-mental scale:
      - This test consists of a variety of items that assess orientation to time and place, attention/concentration, language, constructional ability, and immediate and delayed recall. The maximum score is 30 points and scores less than 24 are considered abnormal.
      - Normal subjects achieve scores of 24 to 30 in individuals with at least complete primary school education, a score of 23 or less indicates mental decline, and a score below 17 indicates dementia.
      - It had been suggested that this test could be very useful clinically for rapid screening of those with cognitive deficits.
   b) Hachinski cerebral ischaemia scale:
      - It is used to classify dementia into Alzheimer, vascular or mixed dementia.
      - The original scale consists of 13 items considered typical of multi-infarct dementia (MID) each scale item was assigned a numeric value with double weighting applied to specific clinical features.
      - It is widely used index supposed to distinguish dementia arising from vascular disease from that due to Alzheimer’s disease.
      - A score of 7 or more is taken as indication of vascular dementia.
      - A score of 0-4 suggested Alzheimer’s dementia.
      - A score of 4-7 suggests mixed dementia.
   c) Blessed dementia scale:
      It assesses four main functional elements: cognition, personality change, apathy
withdrawal and self-care. Information is obtained from a relative in close contact with the subjects.

These scales were done in the acute stage and after 3 months, in addition to the following:

a) Clinical Dementia Rating Scale: It describes different stages of dementia including: questionable dementia, mild, moderate and severe dementia.

b) Global Deterioration Scale: It includes seven different stages: stages 1-4 are the pre-dementia stages, and then stages 5-7 are the dementia stages.

c) Diagnostic and statistical manual of mental disorders’ fourth edition (DSM-IV) for vascular dementia.

Results were tabulated and statistically compared between the patients and controls.

RESULTS

Demographic Data:

The mean age for group A (Demented group) was 60.37±6.6 years (range 50-70 years). 20 patients (66.7%) of them were males; whereas 10 (33.3%) were females. 10 (33.3%) of them were educated, whereas 20 (66.7%) were illiterate.

The mean age for group B (Non-demented group) was 61.73±6.87 years (range 50-70 years). 10 (66.7%) of them were males, whereas 5 (33.3%) were females. 3 (20%) of them were educated, whereas 12 (80%) were illiterate.

Clinical Neurological Findings:

* In group A (Demented): The neurological findings were:
28 patients (93.3%) had motor deficit, 23 (76.7%) had sensory deficit, 25 (83.3%) had facial nerve affection, and five (16.7%) had hypoglossal nerve affection. No one had coordination deficit. 17 patients (56.7%) had gait disturbance while 7 (23.3%) had bladder affection.

* In group B (Non-demented): The neurological findings were:
14 patients (93.3%) had motor deficit, 13 (86.7%) had sensory deficit and 13 (86.7%) had facial nerve affection, 2 (13.3%) had hypoglossal nerve affection and no one had coordination deficit. 7 (46.7%) had gait disturbance while 3 (20%) had bladder affection.

Neuroimaging Results:

Table (1) shows the neuroimaging findings in both groups of patients as regards lesions number, side, depth, site and size.

Vascular Risk Factors:

Fig. (1) illustrates the different risk factors in both groups of patients which were hypertension smoking, obesity, cardiac disease, D.M, hyperlipidemia, and hyperuricemia.

Stroke Severity:

The NIH Scales measures consciousness, visual fields facial palsy, motor arm affection, motor leg affection, sensory affection, language deficit, dysarthria, extinction, and distal motor affection.

Neuropsychological Results:

Table (3) shows comparison between the results of different neuropsychological scales in group A patients in acute and chronic stages:

* As regards M.M.S.E scale its mean was (28.39±1.83) and (21.3±5.77) in acute and chronic stages respectively.

* As regards Hachinski scale its mean was (8.13±1.04) and (9.93±1.43) in acute and chronic stages respectively.

* Blessed dementia scale its mean was (0.63±0.76) and (6.36±4.64) in acute and chronic stages respectively.
There is a highly significant differences between the two stages regarding the 3 mentioned scales (P<0.01).

Table (4) shows comparison between the results of different neuropsychological scales in chronic stages of group A patients and group B patients:
* As regards M.M.S.E scale its mean was (21.33±5.77) and (29.56±0.69) in group A and group B respectively.
* As regards Hachinski scale its mean was (9.93±1.43) and (8.26±0.79) in group A and group B respectively.
* Blessed dementia scale its mean was (6.367±4.64) and (0.66±0.72) in group A and group B respectively.
* As regards Global Deterioration scale its mean was (3.60±1.58) and (1.33±0.48) in group A and group B respectively.
* As regards Clinical Dementia Rating scale its mean was (1.01±1.03) and (0.00±0.00) in group A and group B respectively.

There is a significant difference between the two groups regarding all scales used (P<0.01 and P<0.05).

Relation between education and neuropsychological impairment:
Illiterate patients had a significantly low performance in various neuropsychological scales compared to educated patients.

Relation between laboratory findings and neuropsychological impairment:
Patients with high blood sugar, hyperlipidemia had a significantly bad performance in various neuropsychological tests than those with normal laboratory parameters.

Relation between the neuroradiological findings and the results of neuropsychological scales:
* As regards lesion site patients with temporoparietal lesions had the lowest mean of Hachinski scale, GDS, those with occipital lesions had the highest mean of Blessed dementia scale, those with parietal lesions had the lowest mean of M.M.S.E and those with temporal lesions had the highest mean of CDR Scale.
* As regards lesion side those with left sided lesions had greater level of neuropsychological impairments (in all scales used).
* As regards lesion number multiple lesions on C.T for MRI brain examination associated with greater cognitive impairment.
* As regards lesion size those with large and medium sized lesion sizes had more cognitive impairment.
* As regards lesion depth those with deep lesions with cortical extension had more severe cognitive decline.

Correlative Studies:
Correlation between age of the patients and duration (years of education) and neuropsychological scale (Tables 5 and 6).
* Regarding correlation between age of patients in years and neuropsychological scales (Table 5) shows a negative correlation between increasing age of the patient and decreasing scoring in M.M.S.E. While a positive correlation was noted between increasing age and scoring of Hachinski, BDS, GDS and CDR scales. All these correlations reached a significant value.
* Regarding correlation between years of education in years and neuropsychological scales (Table 6) a significant positive correlation was observed between duration of education and score and score of M.M.S.E. and a significant negative correlation with score of Hachinski, BDS, GDS and CDR scales.
Table 1. Neuroimaging findings in both groups of patients.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group A (Demented) (30)</th>
<th>Group B (Non-Demented) (15)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>1. Number:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>25</td>
<td>83.3%</td>
</tr>
<tr>
<td>Multiple</td>
<td>5</td>
<td>16.7%</td>
</tr>
<tr>
<td>2. Side:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>10</td>
<td>33.3%</td>
</tr>
<tr>
<td>Left</td>
<td>15</td>
<td>50.0%</td>
</tr>
<tr>
<td>Both</td>
<td>5</td>
<td>16.7%</td>
</tr>
<tr>
<td>3. Depth:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortical extension</td>
<td>12</td>
<td>40.0%</td>
</tr>
<tr>
<td>Deep only</td>
<td>12</td>
<td>40.0%</td>
</tr>
<tr>
<td>Both</td>
<td>6</td>
<td>20.0%</td>
</tr>
<tr>
<td>4. Location:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>2</td>
<td>6.7%</td>
</tr>
<tr>
<td>Parietal</td>
<td>14</td>
<td>46.7%</td>
</tr>
<tr>
<td>Temporal</td>
<td>8</td>
<td>26.7%</td>
</tr>
<tr>
<td>Parieto-temporal</td>
<td>4</td>
<td>13.3%</td>
</tr>
<tr>
<td>Occipital</td>
<td>2</td>
<td>6.7%</td>
</tr>
<tr>
<td>5. Size:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.5-1.5 cm</td>
<td>15</td>
<td>50.0%</td>
</tr>
<tr>
<td>&gt;1.5 cm</td>
<td>10</td>
<td>33.3%</td>
</tr>
<tr>
<td>Both</td>
<td>5</td>
<td>16.7%</td>
</tr>
</tbody>
</table>

Fig. (1): Vascular risk factors in both groups of patients.
Table 2. The NIH scale affection in both groups.

<table>
<thead>
<tr>
<th>NIH scale</th>
<th>Group A</th>
<th></th>
<th>Group B</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>a) Consciousness</td>
<td>9</td>
<td>30.0%</td>
<td>5</td>
<td>33.3%</td>
</tr>
<tr>
<td>b) Questions</td>
<td>13</td>
<td>43.3%</td>
<td>5</td>
<td>33.3%</td>
</tr>
<tr>
<td>c) Commands</td>
<td>9</td>
<td>30.0%</td>
<td>5</td>
<td>33.3%</td>
</tr>
<tr>
<td>• Best gaze</td>
<td>0</td>
<td></td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>• Visual fields</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Score 1</td>
<td>12</td>
<td>40.0%</td>
<td>Score 1</td>
<td>6</td>
</tr>
<tr>
<td>Score 2</td>
<td>3</td>
<td>10.0%</td>
<td>Score 3</td>
<td>1</td>
</tr>
<tr>
<td>• Facial palsy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Score 1</td>
<td>14</td>
<td>46.7%</td>
<td>Score 1</td>
<td>7</td>
</tr>
<tr>
<td>Score 2</td>
<td>11</td>
<td>36.7%</td>
<td>Score 2</td>
<td>6</td>
</tr>
<tr>
<td>• Motor arm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Score 1</td>
<td>17</td>
<td>56.7%</td>
<td>Score 1</td>
<td>10</td>
</tr>
<tr>
<td>Score 2</td>
<td>9</td>
<td>30.0%</td>
<td>Score 2</td>
<td>3</td>
</tr>
<tr>
<td>Score 3</td>
<td>2</td>
<td>6.7%</td>
<td>Score 3</td>
<td>1</td>
</tr>
<tr>
<td>• Motor leg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Score 1</td>
<td>11</td>
<td>36.7%</td>
<td>Score 1</td>
<td>6</td>
</tr>
<tr>
<td>Score 2</td>
<td>12</td>
<td>40.0%</td>
<td>Score 2</td>
<td>6</td>
</tr>
<tr>
<td>Score 3</td>
<td>2</td>
<td>6.7%</td>
<td>Score 3</td>
<td>1</td>
</tr>
<tr>
<td>• Ataxia</td>
<td>0</td>
<td></td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>• Sensory</td>
<td>24</td>
<td>80.0%</td>
<td>13</td>
<td>86.7%</td>
</tr>
<tr>
<td>• Language</td>
<td>15</td>
<td>50.0%</td>
<td>8</td>
<td>53.3%</td>
</tr>
<tr>
<td>• Dysarthia</td>
<td>12</td>
<td>40.0%</td>
<td>6</td>
<td>40.0%</td>
</tr>
<tr>
<td>• Extinction</td>
<td>1</td>
<td>3.3%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>• Distal motor</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Score 1</td>
<td>19</td>
<td>63.3%</td>
<td>Score 1</td>
<td>10</td>
</tr>
<tr>
<td>Score 2</td>
<td>5</td>
<td>30.0%</td>
<td>Score 2</td>
<td>4</td>
</tr>
</tbody>
</table>

Table 3. Comparison between the results of different neuropsychological scales in acute group A patients and those of chronic patients.

<table>
<thead>
<tr>
<th>Neuropsychological scale</th>
<th>Group A</th>
<th></th>
<th>P-value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Acute patients</td>
<td>Chronic patients</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>&lt;0.01</td>
<td>H.S.</td>
</tr>
<tr>
<td>M.M.S.E</td>
<td>28.39±1.83</td>
<td>21.33±5.77</td>
<td>&lt;0.01</td>
<td>H.S.</td>
</tr>
<tr>
<td>Hachinski</td>
<td>8.13±1.04</td>
<td>9.93±1.43</td>
<td>&lt;0.01</td>
<td>H.S.</td>
</tr>
<tr>
<td>BDS</td>
<td>0.63±0.76</td>
<td>6.36±4.64</td>
<td>&lt;0.01</td>
<td>H.S.</td>
</tr>
</tbody>
</table>

H.S = Highly significant
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Table 4. Comparison between the results of different neuropsychological scales in chronic group A patients and those of chronic group B patients.

<table>
<thead>
<tr>
<th>Neuropsychological scale</th>
<th>Group A patients in the chronic stage</th>
<th>Group B patients in the chronic stage</th>
<th>P-value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean±SD</td>
<td>Mean±SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.M.S.E</td>
<td>21.33±5.77</td>
<td>29.56±0.69</td>
<td>&lt;0.01</td>
<td>H.S.</td>
</tr>
<tr>
<td>Hachinski</td>
<td>9.93±1.43</td>
<td>8.26±0.79</td>
<td>&lt;0.01</td>
<td>H.S.</td>
</tr>
<tr>
<td>BDS</td>
<td>6.36±4.64</td>
<td>0.66±0.72</td>
<td>&lt;0.01</td>
<td>H.S.</td>
</tr>
<tr>
<td>G.D.S.</td>
<td>3.60±1.58</td>
<td>1.33±0.48</td>
<td>&lt;0.01</td>
<td>H.S.</td>
</tr>
<tr>
<td>C.D.R.S.</td>
<td>1.01±1.03</td>
<td>0.00±0.00</td>
<td>&lt;0.05</td>
<td>S.</td>
</tr>
</tbody>
</table>

H.S. = Highly significant  S. = Significant

Table 5. Correlation between age of the patients with vascular dementia and different neuropsychological scales.

<table>
<thead>
<tr>
<th>Neuropsychological scales</th>
<th>r-value</th>
<th>P-value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.M.S.E.</td>
<td>-0.621</td>
<td>&lt;0.05</td>
<td>S.</td>
</tr>
<tr>
<td>Hachinski ischemic scale</td>
<td>+0.338</td>
<td>&lt;0.05</td>
<td>S.</td>
</tr>
<tr>
<td>BDS</td>
<td>+0.752</td>
<td>&lt;0.05</td>
<td>S.</td>
</tr>
<tr>
<td>G.D.S.</td>
<td>+0.421</td>
<td>&lt;0.05</td>
<td>S.</td>
</tr>
<tr>
<td>C.D.R.S.</td>
<td>+0.543</td>
<td>&lt;0.05</td>
<td>S.</td>
</tr>
</tbody>
</table>

Table 6. Correlation between the duration of education in patients and different neuropsychological scales.

<table>
<thead>
<tr>
<th>Neuropsychological scales</th>
<th>r-value</th>
<th>P-value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.M.S.E.</td>
<td>+0.721</td>
<td>&lt;0.05</td>
<td>S.</td>
</tr>
<tr>
<td>Hachinski ischemic scale</td>
<td>-0.415</td>
<td>&lt;0.05</td>
<td>S.</td>
</tr>
<tr>
<td>BDS</td>
<td>-0.391</td>
<td>&lt;0.05</td>
<td>S.</td>
</tr>
<tr>
<td>G.D.S.</td>
<td>-0.625</td>
<td>&lt;0.05</td>
<td>S.</td>
</tr>
<tr>
<td>C.D.R.S.</td>
<td>-0.543</td>
<td>&lt;0.05</td>
<td>S.</td>
</tr>
</tbody>
</table>

**DISCUSSION**

Not all individuals with stroke develop dementia, therefore it is important to determine the risk factors for ischaemic vascular dementia. Moreover, risk factors for ischemic vascular dementia include demographic characteristics, atherogenic and non-atherogenic risk factors, and stroke related risk factors\textsuperscript{11}.

Our study provides information about the diagnosis, different risk factors, and predictors of post-stroke dementia. Certain host characteristics were relevant to vascular dementia specifically older age, male sex and fewer years of education\textsuperscript{12,13,14}.

Previous studies found that the proportion of stroke patients who developed post-stroke dementia depended mostly on the age range, length of the follow-up and diagnostic criteria\textsuperscript{15,16}.
Gorelick et al.\textsuperscript{17} stated that, men are thought to be at higher risk of vascular dementia than women. The incidence of vascular dementia is higher in men because the incidence of stroke generally is about 30\% higher in men than in women\textsuperscript{15}. In our study, we had 66.7\% of cases were males while 33.3\% of cases were females, however, non-statistical difference between the patients and control group was due to selection of control subjects regarding age, and sex to be matched with patients group. On the other hand, Kohmen et al.\textsuperscript{19} reported that men had a high prevalence of VaD than women in most age groups.

The present study revealed a statistical significance between the educated and the ignorant in the results of the neuropsychological scales. These results were in accordance with that reported by Dantigues et al.\textsuperscript{20}; Stern et al.\textsuperscript{21}; Gorelick et al.\textsuperscript{17}; Tatemichi et al.\textsuperscript{12}; Pohjasvaara et al.\textsuperscript{16}. This was explained by the fact that more years of education likely relates to larger function cognitive reserve and thus a greater ability to remain functionally competent despite an increasing burden of cerebrovascular disease among patients with more education\textsuperscript{11,14,20}. On the contrary lower level of education leads to lower functional brain reserve and increase susceptibility to cognitive effect of cerebrovascular insults\textsuperscript{13,22-29}.

Many vascular risk factors seems to be a risk for developing vascular dementia and post-stroke dementia namely hypertension, coronary heart disease, cardiac arrhythmia, heart failure, diabetes mellitus, cigarette smoking, hypercholesterolemia, obesity, higher hematocrit value, alcohol abuse\textsuperscript{6,30}.

In our study, hypertension was present more in demented cases than the control group (83.3\% versus 80\%), a difference which is statistically insignificant (P\textgreater{}0.05).

This is in accordance with the results of Meyer et al.\textsuperscript{31}, who reported that hypertension is the most frequent vascular risk factor among patients with multi-infarct dementia and occurred in 66\% of cases in his study. This is to be expected since hypertension is the most potent risk factor for stroke. Recently, Skoog et al.\textsuperscript{32} related high midlife blood pressure to a higher risk of dementia later in life.

Interestingly, in the series of Guo et al.\textsuperscript{33} the demented patients showed lower arterial blood pressure values at the time of diagnosis, which was not the case in the series of Skoog et al.\textsuperscript{32}. In another study the mean arterial blood pressure was lower in the demented group and low blood pressure has been related to the severity of dementia\textsuperscript{6,33}.

So, our results were in agreement with the reports of Meyer et al.\textsuperscript{31} and Dunbabin et al.\textsuperscript{34}, who found that therapy of hypertension as a risk reducing factor is certainly useful but should not be too aggressive since the induced hypotension may have a negative influence on cognition\textsuperscript{35}. We reported that in controlling hypertension, avoidance of hypotension is strongly advocated, as poor autoregulation in VaD patients increases its deleterious effects on cerebral blood flow.

In our study diabetes was present in a similar number in both groups (66.7\% of cases in both groups). There was no statistical significance. But our results are not in accordance with the results obtained by Katzman et al.\textsuperscript{36}, who found that diabetes was a significant predictor for the development of vascular dementia in non-demented elderly population, and, also it did not correspond with the results of Censori et al.\textsuperscript{37}, who found that diabetes was significantly associated with post-stroke dementia. Perhaps hyperglycemia in the acute phase of stroke aggravates cellular damage through tissue acidosis\textsuperscript{38,39} and diabetes causes, small vessel disease that impairs recovery from the acute ischemic changes\textsuperscript{39}.

Smoking, also, is found to be a significant atherogenic risk factor. It was present in 83.3\% of demented cases in comparison to 53.3\% of controls group, with a highly significant p value (P\textless{}0.05).

Our results are in accordance with the results obtained by Rogers et al.\textsuperscript{40,41}, they have shown that untreated hypertension and cigarette smoking...
are independent risk factors associated with abnormal and excessive decreases in cerebral perfusion and that control of hypertension or cessation of smoking independently restore cerebral perfusion toward normal. If both risk factors coexist and both are controlled, greater cognitive benefits and protection against further stroke may be expected.

Obesity was present in the control group much more than the demented group 33.3% versus 13.3% with a statistical significance; P-value <0.05. This finding was in accordance with the results obtained by Dollear et al.42, who found that, generally, the patients with multi-infarct dementia (MID) were leaner than the multi-infarcts controls and had a higher frequency of death during follow-up.

Hyperlipidemia was present in a similar number in both groups (33.3% of cases in both groups), which is statistically insignificant. However, several studies have suggested that hyper-cholesterolemia and hypertriglyceridemia are associated with impaired cognitive function. Better performance in mental status testing may be related to higher levels of high-density lipoprotein cholesterol, and a form of dementia due to extremely high levels of plasma lipids has been reported. Lowering of the total serum cholesterol level may improve cognitive performance.43,44,45

Ischemic heart disease (IHD) were present much more in vascular demented group than the control group (26.7% versus 13.3%). Our findings are also in accordance with those of Bornstein et al.46, who reported that, patients with ischaemic heart disease tend to be more cognitively impaired than those with non-ischaemic disease particularly in the areas of memory and concentration. Gorelick et al.17 stated history of myocardial infarction could be a predictor of multi-infarct dementia (MID) through its role as a marker or factor that predispose to stroke.

Radiological Features (CT Data) showed that the side of the lesion (the affected hemisphere) in our study, i.e. left hemisphere (the dominant hemisphere in our cases) was affected in most of the demented cases. In agreement with our study, Tatemichi et al.12 and Gorelick et al.17 showed that, left hemispheric stroke localization was also more common in demented patients in their clinical trials on 283 patients.

As regards the location of the lesion in our study, there was non-specific location more common in demented patients than non-demented (control group). However, Ladurner et al.47 stated that the distribution of the mainly small infarcts in demented patients was predominant in the basal ganglia and the watershed areas. In addition to the higher number of infarcts in the demented patients, they also showed a higher proportion of infarcts in the thalamus as compared with the non-demented patients.

In our results no specific site is more common in the demented patients. Thus far, no specific stroke location has been identified that plays a particularly critical role in the development of dementia.48

Those with multiple lesions showed lowest mean in Mini-Mental Scale Examination (MMSE) and highest for Blessed dementia and other scales the same is for large lesion >1.5 cm. But no statistical significance between patients and controls was detected.

An observation supported by the study of Desmond et al.14, who suggested that the cumulative burden of cerebrovascular lesions is of importance. That cumulative burden could result from the number of such lesions, with multiple strategically located infarcts causing cognitive impairment and cognitive decline.

Pohjasvaara et al.16 and Barba et al.11 added that a single explanation for post-stroke dementia is not adequate and the multiple factors including host characteristics, clinical, stroke related, and lesion related radiological factors has been reported to predict dementia in stroke patients. In addition to the following battery of tests to help for diagnosis and scaling of dementia.
References


التنبؤ بالعته بعد جلطة المخ

يشتمل عنة ما بعد السكتة الدماغية على كل أنواع العنة التي تحدث بعد السكتة الدماغية بعض الطرق عن سابقتها، كما أن التشخيص المبكر عنة ما بعد السكتة الدماغية ذو أهمية قصوى إذ أنه من ضمن الآليات القليلة للعنة التي يمكن معالجاً.

اشتملت هذه الدراسة خمسة وأربعون مريض معاون من جلطة المخ في سن يتراوح ما بين 50-70 عام، ومنها مجموعة صبطة تشمل 15 شخصة للمقارنة. وقد تم إجراء التالي للمرضى والمجموعة المضادة:

(أ) المرحلة المبتدئة لحول الجلطة:

1. فحص كلينيكي شامل للجهاز العصبي وفحصات عملية كاملة.
2. أعراض بالكيميائية والرئتين المغناطيسي على المخ.
3. فحصات إكلينيكية وسبيولوجية وتشمل: مقياس القدرة الممتعه، مقياس الجلطة الدماغية لوليشتافين، مقاييس نوع العنة (مقايي هامينيكس)، مقياس بنفسه للعنة.

(ب) المرحلة المزمنة:

كل الفحوصات السابقة تم إعدادها بعد ثلاثة أشهر من بداية حدوث الجلطة مع إضافة التالي: مقياس التهدر الكلي، مقياس تقسيم العنة الإكلينيكي، مقياس تشخيص نوع العنة (DSM-IV).

من هذه الدراسة وجد أن بعض العوامل الدماغية توجد بصورة كبيرة في عنة ما بعد السكتة الدماغية عنها في أوقات العنة الأخرى. من هذه العوامل وجدنا أن الҚيم في السن عامع خطر لا يمكن تقلبه. وجد أيضاً أن الرجال أكثر عرضة من النساء للعنة ما بعد السكتة الدماغية، كما وجد أيضاً ونسبة عالية ذات دلالة إحصائية أن التعليم يمثل عامل هام للحماية من هذا العنة.

من هذه الدراسة تبين أن ارتفاع ضغط الدم ومرض السكر وقصور شرايين القلب وارتفاع نسبة الدهون بالدم عرضه لتهدور الوظائف العقلية عن المرضى الذين لا يعانون من هذه العوامل.

من دراستنا لأشعة المخ المقطعية لعنة المريض وجدنا أنه: (1) النصف الرخوي الأيسر (السائد) (50% من المرضى) أصاب في معظم حالات العنة أكثر من النصف الأيمن. (2) المرضى ذو الإصابات المتعددة وكبيرة الحجم أكثر عرضة للإصابة بمرض العنة.

تستعرض هذه الدراسة أن عنة ما بعد السكتة الدماغية هو الوحيد من بين الآليات الكبرى للعنة الذي يمكن معالجها في هذه الدراسة أن التعلم عمل وقائي ضمن هذه العنة، كما وجدنا أيضاً أن وجود عوامل الخطر الوقائي القابلة للعثور على الأشخاص المعرضين لخطر الإصابة عنة ما بعد السكتة الدماغية، ابلا، هذه العوامل والجهود التي تهدف لمجنع السكتة الدماغية سوف يؤدي لمجنع أو تقليل حدوث العنة المصاحبة للسكتة الدماغية.