Clinical deterioration in vascular dementia: Role of new ischemic lesions, hypoalbuminemia and hyponatremia

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ABSTRACT

Vascular dementia is the second most common form of dementia. Particular features of vascular dementia include apathy seen early in the course of dementia, poor verbal fluency, preservation of speech and behavior, cognitive slowing, difficulty in shifting mental sets and impaired abstraction. Depression is common among patients with vascular dementia with a prevalence rate of 25-30% and significantly affects the prognosis and functioning, hence the need for efficient studying. Clinicians frequently regard deterioration in patients with vascular dementia as an inevitable outcome of the disorder with little hope for regression or recovery. A integral part of the diagnosis lies in the neuroimaging modalities particularly the MRI scans. However even the most updated MRI imaging modalities have proved quite deficient because although they help to differentiate vascular dementia from other causes yet they are unable to identify recent vascular insults and to differentiate them form older areas of ischemia. An important advance is the MRI, Diffusion-weighted imaging (DWI) which is useful in detecting focal brain ischemia in the acute stage and can differentiate recent from old strokes. The objective of our study was to investigate causes of deterioration in patients with vascular dementia known to have lacunar infarcts and Leukoaraiosis to sort out the different possible causes and thus provide basis for therapeutic intervention. WE studied 54 patients with the diagnosis of vascular dementia according to the DSM 4 criteria with evident lacunar infarcts and periventricular white matter hyperintensities on their MRI T2 weighted images. The patients were divided into three groups according to their mode of presentation. All patients had a thorough neurological and psychiatric evaluation, a Glasgow coma score, NIHSS Score, Cornell scale for depression in dementia and a functional outcome score for dementia, laboratory analysis and MRI images with Diffusion weighted images and adjustment of $P$-values to a low of 500 and a high of 1000. We concluded that deterioration in the level of consciousness was mainly caused by either a recent cerebrovascular event best diagnosed by MRI DWI with a high $P$-value, or by biochemical derangement commonly hypo albuminemia or hyponatremia. We also concluded that deterioration of cognitive functions in clear consciousness is caused by either a minor cerebrovascular accident or by a depressive episode while impairment in daily living activities in the absence of cognitive deficits would imply a primarily psychological cause. (Egypt J. Neurol. Psychiat. Neurosurg., 2004, 41(1): 401-412).

INTRODUCTION

Vascular dementia is the second most common form of dementia after Alzheimer disease (AD) according to Western statistical data from the United States and Northern Europe¹ but may indeed be the most common cause of dementia in the Middle East countries including Egypt and Saudi Arabia. Many subtypes of vascular dementia have been described to date. The most common types include multiinfarct dementia, vascular dementia due to a strategic single infarct, vascular dementia due to multiple lacunar lesions, vascular dementia due to hemorrhagic lesions, CADASIL andBinswanger disease.

Vascular disease produces either focal or diffuse lesions in the brain that cause cognitive decline. Common areas of the brain that are

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related to cognitive decline are the white matter of the cerebral hemispheres and the deep gray nuclei, especially the striatum and the thalamus. In many patients, both focal and diffuse disease are observed together and singular gyrus infarction\(^1\). In multiinfarct dementia, the combined effects of different infarcts produces cognitive decline by affecting the neural connections in addition to crucial subcortical substrates for cognition. In this case, small-vessel disease affects the deep penetrating vessels of the brain and produces 2 major syndromes; Binswanger disease and the lacunar state\(^1\).

Lacunar disease is due to small-vessel occlusions that produce small cavitory lesions within the brain parenchyma. These lacunes are found more typically in the internal capsule, deep gray nuclei, and white matter\(^3,4\). Binswanger disease (otherwise known as subcortical leukoencephalopathy) is due to diffuse and selective white-matter disease where vascular changes observed are fibrohyalinosis of small arteries and fibrinoid necrosis of the larger vessels inside the brain. These two common causes of vascular dementia (multiple lacunar strokes and Binswanger disease) commonly co-exist being caused by similar predisposing factors namely advanced age, hypertension, dyslipidemia, diabetes mellitus and smoking\(^3,4\).

The clinical features of vascular dementia caused by small vessel disease includes cognitive impairment developing acutely or subacutely, following an acute neurological event with a stepwise progression. Particular features of vascular dementia include apathy seen early in the course of dementia, poor verbal fluency, preservation of speech and behavior, cognitive slowing, difficulty in shifting mental sets, impaired executive motor functions and impaired abstraction.

Localizing features commonly occur with the lacunar strokes and include hemiparesis, bradykinesia, hyperreflexia, extensor plantar responses, ataxia, pseudobulbar palsy, swallowing difficulties, and gait disorders.\(^2,5\)

Particular features of multiple lacunar strokes are caused by the accumulation of multiple lacunar infarcts (the so-called lacunar state) usually causing a more readily recognized stepwise course, eventually causing a dementia syndrome characterized by a short-step gait, dysarthria, dysphagia, pseudobulbar manifestations, cognitive impairment, imbalance, and incontinence.\(^1\) In Binswanger disease the average age of onset is between the fourth and seventh decades, and a history of hypertension is present in 80% of patients. Patients show progressive motor, cognitive, mood, and behavioral changes over a period of 5-10 years. Mood and behavioral changes are observed early and, in some patients, may be the presenting feature. Patients may be apathetic or abulic. Intellectual deficits also are observed early in the disease, and patients frequently are described as confused, inattentive, and vague. Patients with Binswanger dementia often have early-onset urinary incontinence and gait disturbances.\(^6,12\)

Depression is common among patients with vascular dementia with a prevalence rate of 25-30%. Cognitive deficits associated with depression may be severe and indistinguishable from those associated with dementia, except that they may respond to antidepressant therapy. If left untreated depression may be a contributing factor to the institutionalization of dementia patients. Other psychiatric features include behavioral changes, psychotic symptoms particularly delusions, disturbance of sleep patterns and acute confusional states.

Neuroimaging plays a key rule in the diagnosis of vascular dementia and in differentiating it from other Dementia syndromes. Lacunar infarcts appear as small patches of hypo intensities on CCT and MRI T1 images and as hyper intense patches on T2 weighted images with a predominantly subcortical distribution commonly in the basal ganglia, thalamus, internal capsule and subcortical white matter of the cerebrum in addition to the brainstem.\(^13\)
Leukoaraiosis (LA) refers to bilateral and either patchy or diffuse areas of hypodensity of the cerebral white matter (WM) on CT or hyperintensity on T2-weighted MRI associated with cognitive impairment, mood disorders, disability, and dementia. Among patients with dementia of presumed vascular origin (VD), LA and multiple lacunes are detected by CT in 41% to 100% and by MRI in 64% to 100% of the cases. It is also worth noting that a large proportion of Alzheimer’s disease (AD) patients (ranging from 19% to 78% in CT-based studies and from 7.5% to 100% in MRI studies) have LA and ischemic changes similar to multiple lacunes, although the changes in the white matter and the lacunar infarcts are much less severe than in those patients with cerebrovascular disorders. A threshold exists between the extent of MRI-LA, lacunar infarcts and subtle cognitive impairment; among 100 volunteers participating in a single study, those in whom MRI-LA, lacunar infarcts involved an area greater than 10 cm² had significant decreases in cognitive performances involving frontal lobe abilities, attention, and speed of information processing. Two studies report that cognitive impairment is more severe in subjects with lacunar infarcts and MRI-LA than by those without LA. An increased prevalence of MRI-LA and lacunar infarcts has been recently described in a variety of psychiatric disorders.

Recent advances in MRI imaging provide further aid to the diagnosis and management of patients with vascular dementia. While one of the well known limitations of MRI imaging is the difficulty in differentiating between old and recent lacunar infarcts and white matter ischemic areas forming the LA patches thus making it difficult to judge the contribution of recent ischemia to the symptoms and signs of dementia.

Diffusion-weighted MRI (DWI) is useful in detecting focal brain ischemia even in the hyperacute (<6 hours) stage. Acute brain ischemia causes a rapid decrease in water diffusion, and ischemic regions appear hyperintense on DW images. The lesions disappear over 5 to 10 days (pseudonormalization) and reappear as hypo intense lesions thereafter. In addition to acute stroke, DWI may also provide information on the extent and elucidate the mechanism of Leukoaraiosis and lacunar strokes detecting hyperacute lesions not amenable to the conventional CT scans and MRI imaging.

Prognostic Significance: Vascular dementia has a guarded prognosis; the presence of LA and lacunar infarcts in patients with vascular dementia suggests an increased incidence of subsequent minor lacunar strokes and further increase in the severity of cognitive deficits. Other determinants include aging, arterial hypertension, presence of lacunes on CT, and electrocardiographic abnormalities. LA is associated only with a more rapid decline in visuospatial, associative, and psychomotor functions.

In this study we noted that patients with vascular dementia often showed clinical deterioration with disturbed consciousness, rapid and marked deterioration of cognitive functions and the occurrence of soft neurological signs with worsening of speech disorders, transient pseudobulbar palsy, confusion, apathy, further slowing of the executive motor functions and disturbance of praxis including worsening of gait disorder. This evident deterioration may be regarded as an inevitable consequence of dementia necessitating hospitalization. However careful and meticulous history taking and examination searching for subtle neurological, psychiatric signs as well as laboratory parameters together with the application of the most updated MRI Diffusoin Weighted Imaging modalities may provide a better understanding regarding the causes of clinical deterioration in these patients and form a basis for therapy during these periods of clinical deterioration.

**Aim of the study**

The objective of our study was to investigate and identify the possible causes of clinical deterioration in patients with vascular dementia specifically the subgroup of vascular dementia.
dementia patients with lacunar infarcts and Leukoaraiosis and thus provide basis for treatment plans and therapies specifically for these events of rapid acute deterioration in these patients.

**PATIENTS AND METHODS**

We studied 54 patients with the diagnosis of vascular dementia according to the DSM 4 clinical criteria with evident lacunar infarcts and periventricular white matter hyper intensities on their MRI T2 weighted images. All Patients were admitted to hospital because of significant recent deterioration in the level of consciousness, marked deterioration in their cognitive dysfunction or because of marked deterioration in their daily living activities particularly feeding difficulties, difficulty in walking and bowel control. We also included 20 patients with vascular dementia treated in the outpatient clinic with no evident deterioration as a control group.

We divided the patients with vascular dementia into three groups based on their presenting clinical features and type of recent deterioration:

**Group 1:** included patients with recent deterioration in the level of consciousness as evaluated by the Glasgow Coma Scale and Glasgow outcome scale, and this included 16 patients.

**Group 2:** included patients with intact level of consciousness and recent deterioration in cognitive functions as assessed by the Hierarchic Dementia scale for the assessment of cognitive functions in dementia, this group included 21 patients.

**Group 3:** included patients with recent deterioration in their daily living activities as assessed by the Structural interview for deterioration in daily living activities in dementia, this group included 17 patients.

All patients were submitted to the following investigations:

* A thorough general and clinical neuropsychiatric examination.
* Glasgow Coma Scale and Glasgow outcome scale for evaluation of the level of consciousness on admission and daily afterwards during the course of hospital stay.
* NIHSS for evaluation of recent manifestations of an acute stroke.
* Psychological testing including: Hierarchic Dementia scale for the assessment of cognitive functions in dementia, Structural interview for deterioration in daily living activities in dementia and Cornell scale for the assessment of depression in dementia.
* Neuroimaging Modalities including MRI scans using a Siemens high resolution 1.5 tesla imager capable of echo planar imaging with application of a special protocol including Axial and Coronal T1, T2 weighted images, FLAIR and Diffusion Weighted images with ADC mapping and higher P values of $P=500$, $P=1000$ and 0 with comparison between the various images for detection of recent ischemic insults including new lesions and recent ischemic changes in pre-existing lesions.
* Laboratory workup including a complete blood picture, C reactive protein, creatinine clearance, liver function tests, Chest X-rays, EKG, Thyroid function tests, Lipid profile, Blood sugar curve and glycosylated Hgb levels, Serum electrolytes including serum Na, K, Ca, Mg, Hematocreit value and serum albumin.

**RESULTS**

We studied 54 patients with vascular dementia brought by their relatives or caregivers to the emergency department of a leading hospital in KSA and to two of the leading hospitals in Cairo because of recent clinical deterioration in the 48-hour period prior to their clinical presentation. We also included 30 patients with known vascular dementia.
presenting for regular follow up in the outpatient departments of these hospitals and showing no evidence of recent deterioration.

The mean age of our patients was 64 years (SD 8.7) while the mean age of the control group was 68 (SD 5.6) (p>0.05). The patients sample included 34 (63%) males and 20 (37%) females and the control group included 22 (55%) males and 18 (45%) females (p>0.05). The mean duration of illness from the start of symptoms as rated by the relatives was 4.2 SD 1.2 years in the patients group and 3.9 (SD 0.95) years in the control group (p>0.05). Both the patients and control groups were matched for age, sex and mean duration of illness (Table 1).

Of our patient’s group 52 (96%) had a history of hypertension with a mean systolic blood pressure of 160 (SD 9.8) and a diastolic blood pressure of 98 (SD 5.6) on presentation to the emergency department while in the control group 36 (90%) had a history of hypertension with a mean systolic blood pressure of 150 (SD 8.4) and a diastolic blood pressure of 96 (SD 6.2) on presentation to the outpatient clinic (p>0.05). Forty patients (74%) had a history of diabetes mellitus with a mean duration of 7.4 (1.2) years with a mean random blood sugar of 198 (SD 18) on presentation to the emergency department, while in the control group 28 patients (70%) had a history of diabetes mellitus with a mean duration of 6.2 (SD 2.1) years with a mean random blood sugar of 178 (SD 12) on presentation to the outpatient clinic (p>0.05). History of cigarette smoking was present in 30 (56%) patients and in 15 (37.5%) of the control group (p>0.05). History of dyslipidemia with a LDL cholesterol level more than 200 mg/dL and/or triglyceride level more than 150 mg/dL was present in 36 (66%) of the patients group and 15 (55%) of the control group (p>0.05). Both groups were matched for the studied risk factors for ischemic strokes and Leukoaraiosis (Table 1).

Group 1 included 16 patients who presented mainly with deteriorated level of consciousness and had a mean Glasgow Coma Score of 8 (SD 1.2) compared to the control group who had a Glasgow coma score of 13 (SD 0.8) (p<0.05). The mean NIHSS was 14 (SD 2.8) in those patients compared to 3 (SD 1.9) in the control group (p<0.05) (Table 2). Group 2 and 3 included 38 patients who presented mainly with either recent deterioration in their cognitive functions (group 2 including 21 patients), or deterioration in their daily living activities (group 3 including 17 patients). The mean Glasgow Coma Score was 13 (SD 0.8) in group 2 and 14 (SD 0.4) in group 3 compared to the control group who had a Glasgow coma score of 13 (SD 0.8) (p>0.05) indicating no statistically significant difference between either group and the control group regarding the level of consciousness. The mean NIHSS was 2 points (SD 2.1) in group 2 and 1 point (SD 0.8) in group 3 compared to 3 points (SD 1.9) in the control group (p>0.05) indicating no statistically significant difference between both groups and the control group on the recent stroke scale. The NIHSS is sensitive to the recent and subtle neurological symptoms and signs suggestive of a recent stroke.

Laboratory parameters: different parameters were studied including electrolytes thyroid hormone levels, Hematocrit value and exclusively serum albumin levels (Table 3). Although no statistically significant difference was found between the mean levels for the main serum electrolytes for the 3 groups of dementia compared to the control groups (Table 3) IN patients of group 1 presenting with deteriorated level of consciousness the Mean Serum sodium levels were found to be significantly low with a mean value of 118 (SD 4.3) compared to the control group whose mean values were 137 (SD 1.6) p<0.05 and compared to the groups 2 and 3 who had a mean serum sodium levels of 134 (SD 6.2) p<0.05 and 132 (SD 4.1) p<0.05. Also the mean serum albumin levels were found to be significantly low with a mean value of 118 (SD 4.3) compared to the control group whose mean values were 137 (SD 1.6) p<0.05 and compared to the groups 2 and 3 who had a mean serum sodium levels of 134 (SD 6.2) p<0.05 and 132 (SD 4.1) p<0.05 (Table 5).
Neuroimaging: Recent hyperacute infarcts on MRI diffusion weighted images were detected. Example for high P value images of an VD diseased brain are shown in figure below. with low P values of 500 and higher P values of 1000 in the 3 groups of patients. In group 1, 7 (43.7%) patients had abnormal hyper intense signals on low P values while 10 (62.5%) had abnormal hyper intense patches on high P values. In group 2, 5 (23.8%) patients had abnormal hyper intense signals on low P values while 8 (38%) had abnormal hyper intense patches on high P values. In group 3, 1 (5.8%) patients had abnormal hyper intense signals on low P values while 3 (17.6%) had abnormal hyper intense patches on high P values. In the control group, 3 (5.5%) patients had abnormal hyper intense signals on low P values while 7 (12.9%) had abnormal hyper intense patches on high P values. There was a statistically significant difference between the number of patients in group 1 and group 2 compared to the normal control group (p<0.05) regarding hyper intensities on MRI imaging. Group 1 showed a statistically significant difference regarding the distribution of the hyper intensities mainly involving the thalamic region and basal ganglia compared to group 2, group 3 and the control group (p values <0.05) while patients in group 2 showed a tendency to have periventricular hyper intensities in the subcortical frontal regions though the number of patients showed no statistical significance (Table 6).

Psychological testing: Comparative study between patients of group 2 with recent deterioration in cognitive functions and control subjects showed a statistically significant difference between the 2 groups on Cornell scale for depression in dementia (Table 3). Comparative study between patients of group 3 with recent deterioration in daily living activities and control subjects showed a statistically significant difference between the 2 groups on Interview for daily living activities (IDLA) and on Cornell scale for depression (Table 4).

Fig. (1): MRI image (FLAIR) showing leucoariosis.
Fig. (2): MRI images A IR, B, C DWI showing hyper intensities of recent lacunars infarcts.

Table 1. Baseline criteria in patients and control groups.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Patients: 54</th>
<th>Control: 40</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age mean (SD)</td>
<td>64 (8.7)</td>
<td>68 (5.6)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Gender M:F NO (SD)</td>
<td>34: 20</td>
<td>22:18</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Duration of illness mean (SD)</td>
<td>4.2 (1.2)</td>
<td>3.9 (0.95)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Hypertension no (%) systolic</td>
<td>52 (96)</td>
<td>36 (36)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>mean (SD) diastolic mean (SD)</td>
<td>160 (9.8)</td>
<td>155 (8.4)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td></td>
<td>98 (5.6)</td>
<td>96 (5.2)</td>
<td></td>
</tr>
<tr>
<td>Diabetes no (%) mean blood</td>
<td>40 (74%)</td>
<td>28 (70%)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>glucose (SD)</td>
<td>198 (18)</td>
<td>178 (18)</td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>30 (56%)</td>
<td>15 (37.5)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>36(66)</td>
<td>22(55)</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Table 2. Comparison between the Glasgow Coma Score and NIHSS in the study groups.

<table>
<thead>
<tr>
<th>Study groups</th>
<th>GCS mean (SD)</th>
<th>P value</th>
<th>NIHSS mean (SD)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>8 (1.2)</td>
<td>&lt;0.05</td>
<td>14(208)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Group 2</td>
<td>13(0.8)</td>
<td>&gt;0.05</td>
<td>2(2.1)</td>
<td>&gt;0.05</td>
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<tr>
<td>Group 3</td>
<td>14(0.4)</td>
<td>&gt;0.05</td>
<td>1(0.8)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Control group</td>
<td>13(1.2)</td>
<td>&gt;0.05</td>
<td>3(1.9)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Group 1</td>
<td></td>
<td>&lt;0.05</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
DISCUSSION

Multiple ischemic lesions cause dementia usually with a step-like course. In most cases of VD there is a gradual development of the disease due to small-vessel involvement resulting in lacunar infarctions in the basal ganglia and diffuse changes in the white matter. These patients present diffuse periventricular hyperintensities on T2-weighted MRI known (the leukoaraiosis). We have used high P value analyzed diffusion MRI imaging to detect the recent white matter ischemic lesions in patients suffering of VD specifically during their periods of deterioration with the aim to use this method to differentiate between the disease states and to visualize recent minor ischemic episodes in the white matter (supposed to be more damaged in VD).

Clinicians frequently regard deterioration in patients with vascular dementia as an inevitable outcome of the disorder with little hope for regression or recovery. Clinical deterioration often causing the patient to be prompted to the hospital by the caregivers or family is often regarded as due to progressive non-salvageable loss of neural cells and therefore implying little hope for recovery or effective therapeutic intervention. Furthermore neurological examination could hardly detect further localizing symptoms and signs of clinical significance other than those already present on the initial clinical evaluation apart from deterioration of consciousness. Regular psychiatric evaluation is often difficult to interpret and is over masked by cognitive and neurological disability. Neuroimaging studies are often ambiguous in identifying recent from old ischemic lesions on CT scans or regular MRI scans adopting the routinely applied T1, T2 and FLAIR modalities.

Our study demonstrated that clinical deterioration in these patients was not homogenous and could be segregated into three main clinical patterns with significant overlap. The first pattern (group 1 patients in our study) was the deterioration in the level of consciousness, the second (group 2) was the
deterioration in cognitive functions with no evident impairment in the level of consciousness and the third (group 3) was the deterioration in the daily living activities with no evident disturbance of cognition nor consciousness.

Investigations of patients of group 1 dementia who presented mainly with deterioration of their level of consciousness as noted by the Glasgow Coma Scale in comparison to the control group showed that their MRI scans utilising the standard imaging protocols with T1W, T2W images and FLAIR showed no significant recent changes on presentation to hospital with no evident recent ischemic regions identified. These patients however had increased abnormal findings on their MRI scans with diffusion weighted images and best identified by high P values (of 1000) that had otherwise passed unnoticed with the conventional MRI scans or CT scans. The cause was that preexisting lesions particularly the periventricular hyperintensities on T2 weighted images and FLAIR usually mask the more recent hyperacute lesions. We were able to discriminate hyperacute lesions occurring within less than 48 hours form older regions of more than 1 week duration which is a point of importance in managing these patients as patients with a recent acute ischemic stroke. We also noted that in patients with LA they tended to have increased signal intensity on diffusion weighted images than the normal control group which implies a state of chronic hypo perfusion in these patients.

Another important feature were the laboratory parameters showing significantly lower levels of serum albumin in these patients compared to the control group and to our other two subgroups of dementia patients. The low serum albumin levels may be due to nutritional factors that are commonly encountered in those patients living at home among their families and suffering of pseudobulbar palsy, generally negative attitudes with refusal of food or pseudobulbar palsy and bradykinesia of the bulbar and cranial nerve movements responsible for deglutition.

Investigations of patients of group 2 dementia who presented mainly with deterioration of their level of cognitive functions with no impairment of their consciousness showed that those patients had either severe depression as assessed by Cornell Depression scale for Dementia or abnormal findings on their MRI diffusion weighted images with a predominantly subcortical periventricular and frontal recent ischemic areas. Our results are concomitant with many authors (Lundquist 1997, Alexopoulos 1993, Devanand 1997, William et al. 2000). Alexopoulos (1993) reported that a number of clinically depressed patients with cognitive impairment can proceed to develop irreversible dementia. Early recognition of depression in vascular dementia patients and early management will prevent further deterioration in cognitive functions.

Investigations of patients of group 3 dementia who presented mainly with deterioration of their daily living activities in absence of deterioration in consciousness or cognitive functions showed that their was a statistically significant difference between these group 3 patients and the control group in their performance on their Cornell scale of depression whereas no differences were found between the two groups regarding the abnormalities on MRI scans with diffusion weighted images or laboratory tests.

We concluded that deterioration in patients with vascular dementia is not always an inevitable sequel of the disorder and should be investigated meticulously with the aim of identifying possible reversible causes of deterioration and administration of prompt therapeutic measures.

REFERENCES


الملخص العربي

أسلوب تدهور مرضى العته الناتج عن اعتلال الأوعية الدموية الدقيقة للمخ،
دور الجلطات الصغيرة وانخفاض نسبة الصوديوم الأمونيوم في الدم

يعتبر العته الناتج عن اضطراب وقصور الدورة الدموية للمخ ثاني أسباب العته شهوة، ومن الأعراض المميزة لهذا النوع من العته وجود تقلبات في الحالة وردة في أعراضها كقلام والأفعال بصورة مرضية، وصعوبة التغير من نقطة أخرى. وتعاني نسبة من المرضى بالعنة من اكتشاف نسيء من علاجات العئة وتعتبر

20% - 30% من المرضى ويتزامن أعراض الالتهاب تتأثر في أمراض العينة الكاذبة المتجمعة للمرضي وتعتبر عامل مساعد في العنة.

ونمو العنة في مرضي المخ بالсоедин المغناطيسي مع وجود قياس معدل الامساك المرن لجزئيات الماء وتصوير تلك الظهريات في مجال فحص المخ بالأشعة المغناطيسية مع وجود قياس معدل الامساك المرن لجزئيات الماء وتصوير تلك الظهريات في مجال فحص المخ بالأشعة المغناطيسية.

وهذه الظاهرة قد تؤدي إلى تدهور في حالة الرئة، وهو ما كان قد ممكنًا قبل عدة سنوات، حيث كان مرض المخ بالأشعة المغناطيسية كاملاً كبيرًا في نظرية تفسير تلك الجلطات الصغرى خاصة مع وجود عدة جلطات قوية قد يصل عددها إلى 10% وزيادة حاجيات بكتيرات المخ مما يؤدي إلى انتشار جلطات بكتيرات المخ الساقية في حالة المحبطات، وسبب فقدان الرئة المغناطيسية-tellid، ولكن التطور في مجال فحص المخ بالأشعة المغناطيسية ساهم في التحسن في قدرة

الفحص لتحديد عمر جلطات المخ الصغيرة ومعرفة وفرصة الحدث من القرب.

وقد تحدث التهاب العينة من مرضى العنة الناتج عن اعتلال الأوعية الدموية الصغرى للبخ (موجود جلطات صغيرة مقدمة ونماذج على بطنية المخ) وذلك أثناء الفترة التي يعانون فيها من تدهور وضع مهذ في المستوي الوعي أو في أعراض العنة أو في القدرة على القيام بأعمالهم اليومية وذلك لبيان معرفة أسباب التدهور تهيئا لعمل وطرق العلاجات للمرضي أثناء تدهورهم.

وقد تم حصص عينة من 54 مريضًا يعانون من العنة الناتج عن اعتلال الأوعية الدموية للبخ، وذلك وجد عدد جلطات صغيرة (وحروفات بالبخ مع وجود تسببات) ونماذج صغرى حول بطنية المخ، وتم تقسيم هؤلاء المرضى إلى ثلاث مجموعات تطبيقًا للحالة الأولية: نقل مريضي ethernet، ونقل مريضي بحث اكتساب العينة وقياس الإصابة بالالتباس، ونقب وفقًا للعامة للإصابة بالالتباس مع ضبط معدلات بحث 500 و1000 وحوالي 30

كما تم عمل تحليل بالدم عند المريض وقياس نسبة الصوديوم بالدم بالإضافة إلى نسبة الهيدروكربونات ووظائف الكبد وفروض الدم الخاص بالدمه. وقد أثبت البحث أن التدهور الأولي لالتقاط العينة وتحول في حالة اكتساب النسيء هو وجود لجفة أو عدد جلطات '+' مع ضبط المريض، وهذا لا يظهر إلا عند استخدام فحص المخ بالأشعة المغناطيسية ومن النتائج تأثير العينة على معدلات جلطات mri.

النقطة الأولى ووجد في النتائج الأولى هو وجود مسألة نوعية مستمرة محلولية على السام جلادوف ووجد أن نسبة هو

وجود لجفة أو عدد جلطات '+' مع ضبط المريض، وهذا لا يظهر إلا عند استخدام فحص المخ بالأشعة المغناطيسية، ومن النتائج تأثير العينة على معدلات جلطات mri.

النقطة الأولى وجدت في النتائج الثانية هو وجود التهاب العينة والحركة مع عدم وجود تغير في مستوى السام و

وجد أن نسبة هذه المريض يحملن في جلطات صغيرة نشطة بالبخ ووجد نهاية اكتساب النسيء مما يدل على اكتساب نوعية مع ضبط المريض، ووجد اضطرابات في الانتشار المعدن أو مواقع نوعية في القياس المحدد حيث المريض الأول وضع هذه البحث أساس

لمعرفة أسباب التدهور عند مرضى العنة وتشخيص العلاج والعلاج.

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