ABSTRACT

Background and Purpose: The purpose of this study is to determine the feasibility of measuring concentration of cerebral metabolites in ischemic stroke patients using proton magnetic resonance spectroscopy and to compare these concentrations in contra lateral brain regions and in normal healthy volunteers. Also assessment of stroke severity, and prediction of outcome.

Methods: Both patients and control groups were studied with conventional MRI on 1.5 Tesla unit (Magnetom Symphony, Siemens, Version VA 12A using a head coil. Metabolic peaks used in the differentiation of different tissue types were as follows: NAA at 2.02 ppm, Cho at 3.22 ppm, Cr at 3.03 ppm, and lactate at 1.33 ppm. All spectroscopic examinations used TR of 1500ms, a voxel size of 1 x 1 x 1 mm and were acquired in 7 min. The data collected were examined as single spectra related to individual voxels. Data were analyzed using SPSS (statistical package for social sciences) version 10. Qualitative variables were presented as number and percentage chi square (X²) test was used for comparison between groups. Quantitative variables were presented as mean±SD. Students test was used for 2 groups comparison. ANOVA (F) test with Bonferroni multiple comparison was used for more than two groups comparison. Paersons correlation coefficient was used to calculate correlation between variables.

Results: The maximum lactate peak associated with a stroke are present immediately after stroke onset, whereas the NAA signal falls below its normal high value over a matter of days, presumably as debris from destroyed neurons is cleared. Our results showed appearance of lactate in infarcted site but did not appear in contralateral site or control subjects. However, as with NAA, patient total creatin and choline levels (in both infarct and contralateral region) were significantly lower than in normal voluntaries. This change is presumably related to global or bilateral perfusion deficits. Lesion lactate was highly correlated with both acute stroke severity (as assessed by NIHSS) and eventual clinical outcome (as measured by the Barthel index at the time of hospital discharge). Conclusion: Magnetic resonance spectroscopy (MRS) is a non-invasive in vivo method that allows the investigation of biochemical changes in cerebral ischemia. The application of MRS to the study of stroke has made possible dynamic studies of intracellular metabolism of cerebral ischemia. (Egypt J. Neurol. Psychiat. Neurosurg., 2006, 43(1): 141-157)

INTRODUCTION

Stroke continues to be a major public health problem that rank in the top of three causes of death in most countries following cardiac diseases and cancer-related deaths, and is responsible for a large proportion of the burden of neurological disorders, more often disabling than fatal.

MRS has been shown to be an effective noninvasive diagnostic tool that can be used to monitor serially biochemical and metabolic changes in diseases that affect the brain. MRS coupled with MRI techniques allows for the correlation of anatomic and physiologic information.

The normal (1H)- MRS spectrum

Figure (1). Normal proton MRS of brain using Stimulated echo acquisition (STEAM) sequence (echo time (TE)135 ms), shows choline
(Cho), Creatine (Cr), and N-acetyl aspartate (NAA). Fig. (1)

Major brain metabolites detected with MRS are N-acetyl aspartate (NAA), choline (Cho), creatine (Cr), and lactate (Lac).

Histochemical and cell culture studies have shown that specific cell types or structures have metabolites that give rise to {1H}-MRS peaks. A change in the resonance intensity of these marker compounds may reflect loss or damage to a specific cell type or compound. The acquisition of long echo data (TE=270ms, TR =3s) only allows the detection of NAA, Cr and Cho in normal brain, and lactate in regions of abnormality. T1 losses results in lower signal- to- noise and increases the complexity of quantitation methods. The acquisition of short echo time data {TE=30ms,TR=2s} reduces the effects of signal loss due to T2 relaxation and, therefore, provides spectra with increased signal- to- noise. In addition, short echo time spectroscopy detects additional resonances from metabolites with complex MR spectra such as myo-inositol, glutamate and glutamine. Signals from these metabolites cancel at long echo times due to phase modulation {'J-coupling'}. Whilst providing us with more information, short echo time data include a broad background signal consisting of low concentration metabolites, and macromolecules and lipids with short T2 relaxation times, which increases the difficulty of accurate peak area estimation. The chemical shift of the peaks are assigned with respect to water which has been removed from the spectra.

N-acetyl aspartate (2.01 ppm)

The methyl resonance of N-acetyl aspartate (NAA) produces a large sharp peak at 2.01 ppm and acts as a neuronal marker as it is almost exclusively confined to neurons in the human brain, where it is found predominantly in the axons and nerve processes.

The interpretation of NAA signal in the brain of children is complicated by increases in the concentration during development, when it is thought to have a role in supplying acetyl groups for myelin synthesis. In adults, the concentration of NAA is known to vary in different areas of the brain. This can be overcome in the study of stroke patients by using the contralateral hemisphere for comparison.

Creatine (3.94 & 3.03 ppm)

Both creatine and phosphocreatine have signals at 3.94 ppm {methylene singlet} and 3.03 ppm {methyl singlet} which makes it impossible
to distinguish between the two compounds and therefore total creatine (Cr/PCr) signal changes are considered by [$^1\text{H}$]-MRS as one signal. Cr/PCr is found in both neurons and glial cells\textsuperscript{11}, and acts as a phosphate transport system and energy buffer within the cell. As the signal comes from the sum of creatine and phosphocreatine, little information can be gleaned about phosphocreatine metabolism\textsuperscript{12}. The complete absence of creatine signal probably reflects necrotic tissue\textsuperscript{13}.

**Choline (3.22ppm)**

The trimethylamine resonance of choline–containing compounds is present at 3.2 ppm and has been proposed as a marker of membrane damage\textsuperscript{14}. In normal brain, the choline (Cho) peak is thought to consist predominantly of glycerolphosphocholine and phosphocholine; both compounds are involved in membrane synthesis and degradation\textsuperscript{15}.

**Lactate (1.33 ppm):**

Lactate levels in the brain are normally low (less than 1 mM) such that it is usually not seen in the spectra of normal brain. The response of lactate typically indicates that non-oxidative carbohydrate catabolism is happening. Lactate has been detected in patients with stroke, some brain tumors, hypoxia, mitochondrial encephalopathies, and in epileptic foci immediately after a seizure.

**MRS in ischemic cerebral stroke:**

Magnetic resonance spectroscopy (MRS) is a non-invasive in vivo method that allows the investigation of biochemical changes\textsuperscript{7}. Large variations in the initial concentrations of Cho have been observed in the region of infarction\textsuperscript{1}.

The majority of the stroke studies have been carried out using proton [$^1\text{H}$]-MRS which allows the detection of N-acetyl aspartate (NAA), a neuronal marker. [$^1\text{H}$]-MRS changes in humans demonstrate that after an infarct, lactate appears, while NAA and total creatine are reduced compared to the contra lateral hemisphere\textsuperscript{6}.

Chemical shift imaging (CSI) or multivoxel MR spectroscopic imaging is a more advanced form of spectroscopy that uses phase encoding to subdivide a large volume of interest into smaller acquisition voxels, thereby allowing the study of large and heterogeneous areas of brain. The major disadvantages of CSI include complicated acquisition techniques, longer scan times, and a large volume of generated data. CSI is ideally suited to the study of stroke. However, the complexity of image acquisition and data processing often necessitates the availability of a dedicated spectroscopist and has limited the number of stroke studies carried out using CSI\textsuperscript{2}.

**SUBJECTS AND METHODS**

This study was conducted on 40 (22 males and 18 females, mean age 63.85±9.07) clinically diagnosed patients with an ischemic cerebral stroke admitted to Neurology Department, Mansoura University Hospital. The selection criteria were as follows:

- Patients with ischemic cerebral stroke.
- Patients were examined from 2h to 10 days following the onset of symptoms.
- Patients not having history suggestive of cerebral stroke.

**Exclusion Criteria:**

- Patients with hemorrhagic cerebral stroke.
- Patients having history suggestive of cerebral stroke.
- Patients with contraindication for magnetic stimulation\textsuperscript{16}.
  1. Cardiac pacemaker, implanted neuro stimulators, cochlear implants.
  2. Ferromagnetic aneurysm clips.
  3. Metallic implants or foreign bodies with large component of iron or cobalt.
  4. Metallic fragments within the eye.
  5. Placement of a stent, coil or filler within the past 6 weeks.
  6. Relative contraindications:
     a. Claustrophobic patients.
Forty patients underwent chemical shift imaging (CSI). The region of interest was chosen to include the infarct region as well as the contralateral and other normal tissue, while avoiding bone, subcutaneous fat, or other materials that would complicate shimming and water suppression. All spectroscopic examinations used TR of 1500ms, a voxel size of 1 x 1 x 1 mm and were acquired in 7 min. Curve fitting was done automatically for all obtained spectra.

Metabolic peaks used in the differentiation of different tissue types were as follows: NAA at 2.02 ppm, Cho at 3.22 ppm, Cr at 3.03 ppm, and lactate at 1.33 ppm. The lactate assignment was made on the basis of an inverted doublet due to J-coupling at a TE of 135ms.

Patient spectra were interpreted qualitatively by inspection of the peaks and determination of any peak changes compared to the other control spectra of the same patient. Another approach is the quantitative approach using the integral ratios to detect the numerical values of different metabolites. We correlate the infarct volume, lactate peak, and other metabolites integral with NIHSS & BI score to detect stroke severity & prognosis.

Data were analyzed using SPSS (statistical package for social sciences) version 10. Qualitative variables were presented as number and percentage chi square (X²) test was used for comparison between groups. Quantitative variables were presented as mean±SD. Students test was used for 2 groups comparison. ANOVA (F) test with Bonferroni multiple comparison was used for more than two groups comparison. Paersons correlation coefficient was used to calculate correlation between variables.

RESULTS

There was a negative correlation (P=0.05, r=0.27) between NIHSS and NAA integral i.e.
the less NAA integral the more NIHSS score indicating neuronal loss. There was a weak negative correlation between NIHSS and Cho (P = 0.06, r = -0.11).

Cr showed a negative correlation with NIHSS of no significance (P = 0.22, r = -0.20). Direct moderate significant correlation (P = 0.003, r = 0.48) between BI and NAA integral.

No significant relation was detected between BI and Cho (P = 0.21, r = 0.37). Also, no detected significant relation between BI and Cr (P = 0.40, r = 0.43). Direct moderate significant correlation (P = 0.001, r = 0.53) between lesion volume and NIHSS i.e. the more lesion volume the more severe NIHSS. Inverse moderate significant correlation (P = 0.001, r = -0.57) between lesion volume and BI i.e. the more the lesion volume the less BI score i.e. more disabled.

Direct strong significant correlation was found between lesion volume and Lac peak (P = 0.000, r = 0.05) there is inverse moderate significant correlation between lesion volume and NAA integral (P = 0.002, r = -0.49), i.e. the more the lesion volume the less NAA integral. Inverse weak correlation with moderate significance between lesion volume and Cho. (P = 0.018, r = -0.37). Also, inverse weak correlation with moderate significance between lesion volume and Cr. (P = 0.008, r = -0.42).

Table 1. Mean and SD of integral of metabolites detected on MRS in patients group versus control group.

<table>
<thead>
<tr>
<th>Metabolites</th>
<th>Control Group</th>
<th>Diseased side</th>
<th>Contralateral side</th>
<th>Significant test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lac</td>
<td>-------- AB</td>
<td>2.1±1.1 AC</td>
<td>-------- BC</td>
<td>F=70.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>P=0.000</td>
</tr>
<tr>
<td>NAA</td>
<td>17.8± 1.4 AB</td>
<td>1.6± 0.4 AC</td>
<td>6.9± 0.9 BC</td>
<td>F= 2788.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>P=0.001</td>
</tr>
<tr>
<td>Cho</td>
<td>2.3± 0.5 AB</td>
<td>1.2± 0.4 AC</td>
<td>1.9± 0.5 BC</td>
<td>F= 53.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>P=0.001</td>
</tr>
<tr>
<td>Cr</td>
<td>7.4± 1.9 AB</td>
<td>1.4± 0.4 AC</td>
<td>4.5± 1.1 BC</td>
<td>F= 221.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>P=0.001</td>
</tr>
</tbody>
</table>

A, B, C significant difference between corresponding group by Bonferroni multiple comparisons.

Table 2. Correlation between metabolites detected on MRS & lesion volume on MRI with NIHSS & BI score.

<table>
<thead>
<tr>
<th></th>
<th>NIHSS r</th>
<th>NIHSS p</th>
<th>BI r</th>
<th>BI p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lac</td>
<td>0.5</td>
<td>0.000</td>
<td>-0.49</td>
<td>0.000</td>
</tr>
<tr>
<td>NAA</td>
<td>-0.27</td>
<td>0.05</td>
<td>0.48</td>
<td>0.003</td>
</tr>
<tr>
<td>Cho</td>
<td>-0.11</td>
<td>0.06</td>
<td>0.37</td>
<td>0.21</td>
</tr>
<tr>
<td>Cr</td>
<td>-0.20</td>
<td>0.22</td>
<td>0.43</td>
<td>0.40</td>
</tr>
<tr>
<td>Lesion volume</td>
<td>0.53</td>
<td>0.001</td>
<td>-0.57</td>
<td>0.001</td>
</tr>
</tbody>
</table>
Table (3): Correlation between lesion volume and Lac, NAA, Cho, and Cr.

<table>
<thead>
<tr>
<th></th>
<th>Lesion volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lac</td>
<td>r</td>
</tr>
<tr>
<td></td>
<td>0.5</td>
</tr>
<tr>
<td>NAA</td>
<td>-0.49</td>
</tr>
<tr>
<td>Cho</td>
<td>-0.37</td>
</tr>
<tr>
<td>Cr</td>
<td>-0.42</td>
</tr>
</tbody>
</table>

Negative moderate significant correlation between NAA and NIHSS.

Fig. (2): Correlation between NIHSS and NAA.

Direct moderate significant correlation between NAA and BI.
Fig. (3): Correlation between BI and NAA.

Direct moderate significant correlation between lesion volume and NIHSS.

Fig. (4): Correlation between lesion volume and NIHSS.

Inverse moderate significant correlation between lesion volume and BI.

Fig. (5): Correlation between lesion volume and BI.
Case (1)

A male normal volunteer aged 65 years

(A & B) Axial T1, T2- weighted magnetic resonance image.

C) The spectroscopic imaging region of interest each small square represents one voxel in the spectroscopic image.

D) CSI (TE: 270 ms) proton MR spectra show resonance assigned to (Cho, 3.20 ppm), (Cr, 3.03ppm) and (NAA, 2.01) with integral 2.50 μmol/g, 6.81 μmol/g and 18.67μmol/g respectively.
Case (2)
A male patients aged 63 years presented with right- sided hemiparesis, NIHSS (5), BI (100).
(A & B-) Axial T1 and T2 WIS : show left parietal lesion volume (10 cm³).
(C &D-) CSI (TE : 270 ms , 135 ms) in the diseased side.
(E & F-) CSI (TE : 270 ms , 135 ms) in the contralateral side.
There is reduction in NAA 9.80 μmol/g, Cho 2.59 μmol/g and Cr 2.47 μmol/g peaks in the diseased side in comparison to that of contralateral and control one. Also an inverted large lactate doublet (Lac) is observed at 1.33 ppm, denoting anaerobic glycolysis. The phase reversal at 1.33 ppm on long echo TE spectrum confirms the presence of lactate. The integral of NAA, Cho and Cr are 9.8 , 2.50 and 2.47 respectively and Lac peak at 0.6 which are correlated with lesion volume (10 cm³), mild NIHSS (5) and more improved BI (100).
Case (3)
A male patient aged 65 years presented with right-sided hemiparesis, NIHSS (6), BI (85).
(A&B) Axial T1 and T2 WIS show left parietal lesion volume (25 cm³).
(C & D) CSI (TE: 270 ms, 135 ms) in the diseased side.
(E & F) CSI (TE: 270 ms, 135 ms) in the contra lateral side.
There is obvious reduction in NAA 8.80 μmol/g, Cho 3.22 μmol/g and Cr 4.59 μmol/g peaks in the diseased side in comparison to that of contra lateral and control one. Also an inverted large lactate doublet (Lac) is observed at 1.33 ppm, and not appearing in the contra lateral side. The integral of NAA, Cho and Cr are 8.80 μmol/g, 3.22 μmol/g and 4.59 μmol/g respectively and Lac peak at 1.4 which are correlated with lesion volume (25 cm³), mild NIHSS (6) and improved BI (85).
Case (4)
A male patients aged 70 years presented with left-sided hemi paresis, NIHSS (18), BI (50).
(A & B-) Axial T1 and T2 WIS: show right high parietal lesion volume (66 cm$^3$).
(C & D-) CSI (TE : 270 ms, 135 ms) in the diseased side.
(E & F-) CSI (TE : 270 ms, 135 ms) in the contra lateral side.
There is a marked reduction in NAA 2.72 μmol/g, Cho 2.85 μmol/g and Cr 1.83 μmol/g peaks in the diseased side in comparison to that of contra lateral and control one. Also an inverted large lactate doublet (Lac) is observed at 1.33 ppm, denoting anaerobic glycolysis. The phase reversal at 1.33 ppm on echo TE spectrum confirms the presence of lactate. The integral of NAA, Cho and Cr are 2.72 μmol/g, 2.85 μmol/g and 1.83 μmol/g respectively and Lac peak at 1.9 which are correlated with lesion volume (66 cm$^3$), severe NIHSS (18) and disabled BI (50).
**Case (5)**

A male patient aged 67 years presented with left-sided hemiparesis, NIHSS (22), BI (15).

(A & B-) Axial T1 and T2 WIS: show left parietal lesion volume (140 cm³).

(C & D-) CSI (TE: 270 ms, 135 ms) in the diseased side.

(E & F-) CSI (TE: 270 ms, 135 ms) in the contra lateral side.

There is severe reduction in NAA 2.60 μmol/g, Cho 2.05 μmol/g and Cr 1.53 μmol/g peaks in the diseased side in comparison to that of contra lateral and control one. Also an inverted large lactate doublet (Lac) is observed at 1.33 ppm, denotes anaerobic glycolysis. The phase reversal at 1.33 ppm on echo TE spectrum confirms the presence of lactate. The integral of NAA, Cho and Cr are 2.60 μmol/g, 2.05 μmol/g and 1.53 μmol/g respectively and Lac peak at 2.4 which are correlated with lesion volume (140 cm³), severe NIHSS (22) and more disabled BI (15).
DISCUSSION

Stroke is the third most common cause of death and the most common cause of adult disability. A stroke is rapidly developing clinical symptoms and signs of focal and at times global loss of brain functions with symptoms lasting more than 24 hours or leading to death, which is thought to be due to inadequate blood supply to a part of the brain or spontaneous hemorrhage into or over the brain substance.

The prognosis of stroke is extremely variable, and it is difficult to predict the clinical outcome of the patients at the time of presentation. Better prediction of outcome would allow treatment to be targeted at those most likely to benefit.

MRI allows precise localization of the region of infarction and is very sensitive to the early changes of cerebral ischemia. However, MRI provides mainly anatomic information about the size and site of the lesion and gives no information about the biochemical changes and severity of ischemia occurring within the region of imaging abnormality.

Another MR technique, in vivo 1H MR spectroscopy (1H MRS), allows the noninvasive study of the biochemical changes that accompany cerebral infarction and has the potential to allow measurement of the severity of ischemic damage.

In infarction, all metabolites (N-acetyl aspartate (NAA), choline (Cho), creatine (Cr) and lactate (Lac)) levels may change relative to normal brain, and contra lateral brain metabolism may differ from that in healthy subjects because stroke patients often have extensive bilateral vascular disease.

In this study, we aimed at clarifying the value of MRS in measuring changes in cerebral metabolites in ischemic cerebral stroke patients and compare their concentrations with those in contralateral brain regions and in normal healthy volunteers.

This study was conducted on 22 males representing 55% of the examined sample of patients, and 18 females representing 45%. It reflected the higher incidence in males as reported by Smith et al. who stated that there is a small excess of males.

The mean age of the selected group was 63.85±9.07 with most of the studied group lying in category between 56-65 years (9 males and 10 females), followed by group 66-75 years (8 males and 4 females) and only 2 subjects more than 75 years (one male and one female) These finding were consistent with results obtained from study of Warlow, who stated that the incidence between male and female is most prominent in middle to old age, disappearing in the very elderly and probably less prominent in the young.

Control subjects in this study were selected to be as much as possible age (t=0.14, P=0.9) and sex (X2=0.45, P=0.5) matching to the patients group.

The most common site of ischemic infarction detected by conventional brain MRI in this study was the right parietal lobe (47.5%) followed by the left parietal lobe (37.5%). Left occipital lobe was affected in 2.5% of patients while the right & left internal capsules and left basal ganglia were the site of infarction in 12.5% of patients.

These findings were consistent with results obtained from studies of Shaper et al., Williams et al., Petroff et al. and Smith et al. In contrast, Matthews et al. demonstrated left occipital lobe (32%) is most common site of ischemic stroke.

When all patients were considered together and compared with healthy age and sex matched control subjects, the lactate appear while NAA and creatine are reduced in infracted areas compared with the contralateral hemisphere (p=0.001) and normal individuals This finding was similar to Gillard et al., who stated that lactate levels in the brain are normally low (less than 1 mM) such that it is usually not seen in the spectra of the normal brain. The presence of lactate typically indicates that non-oxidative carbohydrate catabolism is happening as occurring in ischemic stroke.

Matthews et al. also found Lac. Peak was higher in infarction than contralateral or normal
individual (P<0.001), NAA become lower in infarction when compared with contralateral or normal individual (P>0.001), also Cho and Cr was lower as NAA with (P>0.005), (P>0.002), respectively.

Maheshwari et al.\textsuperscript{27} stated that marked difference in Lac. Peak or NAA, Cho, Cr integral with (P>0.001), (P>0.002), (P>0.007), and (P>0.003), respectively.

In contrast, infarct choline (1.3±0.4 µmol/g), was not significantly different from contralateral choline (1.3±0.3 µmol/g), infarct creatine level was slightly (but not statistically significantly) lower than contralateral total creatine.\textsuperscript{28} However, NAA level in both infarct (P=0.005) and contralateral (P=0.01) regions) was significantly lower than in normal volunteer. The average lactate concentration (1.3 µmol/g) and can become extremely high (30-50 µmol/g) as the residual blood flow continues to supply glucose, which may be converted to lactate, to the ischemic tissues.

Our observation, that there was reduced NAA integral in cerebral infarcts, is consistent was the result of Wardlaw et al.\textsuperscript{29}, who found NAA is generally accepted as a neuronal marker, in attempting to interpret the meaning of NAA reduction, it is useful to consider its biological significance, it has been suggested that NAA is actively degraded by enzymes within the injured neurons in the first days or hours following infarction\textsuperscript{31}.

Miller\textsuperscript{15} introduced the concept of axonal injury, and underlined the fact that signal at 2.01 ppm includes contributions from N-acetylated groups other than those of NAA.\textsuperscript{3} suggested that the slight NAA reduction observed in acute lesions could reflect a less severe axonal loss or axonal shrinkage caused by ischemic edema.

Interestingly, we found that NAA was lower in brain regions contralateral (p=0.001) to the infarction compared with the same regions in the control subjects. This suggest that stroke patients may also suffer from low-level global ischemic changes or other perfusion abnormalities resulting in neuronal loss\textsuperscript{3}. As regard the creatine and choline levels, in this study, infarct total creatine and choline were lower than contralateral total creatine and choline levels (p=0.001). Moreover, as with NAA, patients total creatine and choline levels in both infarct and contralateral regions were significantly lower than in normal volunteers (p=0.001). This finding was similar to what proved by Bonavita et al.\textsuperscript{30} and Wardlaw et al.\textsuperscript{29}.

Similarly, Saunders et al.\textsuperscript{3}, Ford et al.\textsuperscript{31}, Van Gijn\textsuperscript{32}, Graham et al.\textsuperscript{1}, Bliamire et al.\textsuperscript{33}, and Saunders et al.\textsuperscript{3} found that in 14 patients with cerebral infarct total creatine and choline levels were slightly (but not statistically significantly) lower than contralateral total creatine and choline levels during ischemia. They claimed that reduction in NAA in an infarct region is more marked than the reduction in Cr and this is thought to reflect the increased sensitivity of neurons to ischemia.

As shown in this study, lesion lactate was highly correlated with both acute stroke severity (as assessed by NIHSS) and eventual clinical out come (as measured by the Barthel index at the time of hospital discharge).

We also noticed that lesion NAA was not correlated with the NIHSS (p=0.005) but correlated with lesion volume. NAA was most significantly correlated with stroke functional out come (p=0.003), reflected in the Barthel index score.

So the stronger correlation are with appearing lactate signal rather than NAA decline\textsuperscript{1}, this difference is consistent with our observation that the maximum lactate peak associated with a stroke are present immediately after stroke onset, whereas the NAA signal falls below its normal high value over a matter of days\textsuperscript{1}, presumably as debris from destroyed neurons is cleared.

The lactate signal, therefore, might be expected to provide the earliest MRS-measurable indicator of ultimate lesion severity. Since high concentrations of lactate are produced during the period of initial ischemic injury\textsuperscript{34} before significant lesion infiltration by leukocytes\textsuperscript{35}, its level and anatomic extent of permanent cerebral injury, reflected in functional out come.
Lesion choline and creatine was not correlated with NIHSS and BI, but correlated with lesion volume.

Also; NAA concentration (P=0.002, r=-0.49) correlated inversely with moderate significance with lesion volume so; the more the lesion volume the less NAA concentration.

Lesion volume also correlated well with the measures of lesion severity (NIHSS) (r=0.53, p=0.001) and clinical out come (BI) (r=-0.53, p=0.001).

Lesion volume predicted clinical out come. The corresponding values for NAA integral also predict clinical out come. However, if both measurements were used, the sensitivity to predict out come was high. These Finding were consistent with result obtained from studies of Peteira et al. 36.

Measurement of the NAA integral provides objective and quantitative information about the biochemical function of neurons remaining in the core of the infarct 3.

Our results confirm that 1HMRS provides a method for assessing the severity of cerebral infarction at presentation (NIHSS) and at follow-up (BI score).

Patients with a low NAA concentration made a poor recovery while, who with a higher NAA concentration were more likely to recover fully, which indicated that they had less-severe ischemia and a greater amount of viable neuronal tissue.

Our study confirms a report by Ford et al. 34, who studied a small cohort of patients over time and found that the patients who did well had relatively normal levels of Lac, NAA, Cho and Cr. Our finding are consistent with those of Wardlaw et al. 29, who also reported that clinical outcome was related to lesion volume and peak of lactate and integral of NAA, Cr and Cho.

Conclusion

Magnetic resonance spectroscopy (MRS) is a non-invasive in vivo method that allows the investigation of biochemical changes in cerebral ischemia.

The application of MRS to the study of stroke has made possible dynamic studies of intracellular metabolism of cerebral ischemia.

It is of agent benefit to correlate the risk factors of stroke with brain metabolites, especially the decrease of these metabolites in non infarct area & to follow up these changes and whether. It is permanent or temporarily changes.

REFERENCES


References:


الملخص العربي

السكتة الدماغية هي مصطلح إكلينيكي لوصف فقدان الجهاز الدوائي النموذج الدماغي لمنطقة معينة من المخ والتي تؤدي إلى حدوث جلطة صمام وغيرها من فقدان الوظائف العصبية للمنطقة مع تغير في نسبة أيض بعض العناصر والذي يمكن قياسه بواسطة الرنين المغناطيسي الطبي.

وتتعرض السكتة الدماغية في ثالث سبب للوفاة ومن أهم أسباب الإصابة بين البالغين في العالم مما يؤدي إلى تكلفة عالية.

في الغالب، تؤدي إلى خفض الدخل القومي بنسبة كبيرة وحرمان المجتمع من أسمائهم في التنمية.

الיך يعتمد على الأكسدة للجلوكوز كمصدر للطاقة لثبيت احتياجات المخ لعملية الإصابة إذا قلت نسبة الإيداد الدموي بالمي إلى حد معين، تنشئ سلسلة من العمليات البيولوجية التي تترتب على حسب فترة نقص الإيداد الدموي من قصور موقت بالدورة الدموية إلى الاحتشاء المدني.

تهدف تلك الدراسة إلى قياس تركز بعض نواتج الأيض المخى في حالات الاحتشاء المخى بواسطة الرنين المغناطيسي الطبي ومقارنتها بالأجزاء المقابلة من المخ وفي بعض المتطوعين الأصحاء.

تتغطى تلك الدراسة على 40 مريضاً عالون من مرضى السكتة الدماغية تم اختيارهم من بين المرضى قسم طب المخ والأعصاب مستشفى جامعة المنصورة وقد رجحت نتيجة الفحص الإكلينيكي لهم وجود المرض. وقد تم دراسة هؤلاء المرضى إلكلينيكياً بالإضافة إلى الرنين المغناطيسي التقليدي والرنين المغناطيسي الطبي.

أظهرت النتائج هذه الدراسة أن متوسط الفئة العمرية التي تتكافأ فيها الإصابة بالمرض تتراوح بين 56 إلى 75 سنة بينما تقل نسبة حدوث المرض قبل 56 سنة أو بعد 75 سنة.

أظهر الفحص بالرنين المغناطيسي التقليدي وجود أعلى معدل لمكان الإصابة هو مكان انتشار الشريان المخى المتوسط الأيمن تتم الأضرر ثم الخلفي وبعض الحالات بالكبسولة الداخلية اليمنى ثم الخلفى ثم العقدة القاعدية اليسرى.

أوضح الفحص بالرنين المغناطيسي الطبي اختلافاً محظياً في نسبة المواد الكيمائية بالمغناطيسي في المقاطعة المصحبة بالمقارنة بالمناطق المقابلة لها بالذات الأخر وأيضاً لوحظ اختلاف نسبة المواد الكيمائية بالمناطق المقابلة لمناطق الإصابة بالمغناطيس بالأشخاص المصابين كما وجد علاقة طفيفة بين مساحة الإصابة ودرجة الإصابة الناتجة عن المرض وعلاقة عكسية بين درجة الإصابة التي تسببها المرض ونسبة بعض هذه المواد (خاصة نسبة أن أستر أسبارتين و الثلاثيات) مما يشير إلى فائدة الرنين المغناطيسي الطبي في تحديد درجة الإصابة المتوقعة.