MR Susceptibility Sign of the Middle Cerebral Artery in Imaging of Acute Stroke

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ABSTRACT

Purpose: To evaluate the accuracy of echo-planar T2*-weighted, compared to MRA and FLAIR in the detection of acute middle cerebral artery (MCA) thrombotic occlusion. Materials and Methods: 47 consecutive patients with acute stroke involving the MCA territory underwent MR imaging within 2 to 76 hours after clinical onset. MR examination included echo-planar T2*-weighted-, FLAIR, diffusion-weighted-imaging (DWI) and MR-angiography (MRA). The susceptibility sign on echo-planar T2*-weighted images, which is indicative of acute thrombotic occlusion involving the MCA, was assessed and compared to findings on MRA and axial FLAIR in all patients and to CT in 23 patients. National Institutes of Health Stroke Scale (NIHSS) score, which is a clinical scale assessment, was used for evaluating the neurological status of patients. Results: Forty seven (47) patients (29 males; age range 11-86 years (mean: 58.3 ± 14.7 yrs) and 18 females; age range 35-83 years (mean: 59.4 ± 12.7 yrs) with acute territorial MCA infarcts were included in this study. Out of these 47 patients, 10 had hyperacute MCA infarction (scanned 2-6 hours after ictus), 7 of those patients presented with very severe stroke (NIHSS score 21 or more) and 3 patients with severe stroke (NIHSS score 15-20). Thirty seven (37) patients were studied within 76 hours from ictus (acute to early subacute). Among this group and according to NIHSS score, 5 patients had mild to moderate stroke, 16 patients severe stroke; and 13 patients had very severe stroke. One patient who had very severe stroke with NIHSS score of 22, died. She had left MCA occlusion, presented 4 hours within onset of right hemiplegia and had a history of DM, HTN and COPD. Conclusion: Presence of the susceptibility sign on T2*WI proximal to the MCA bifurcation provides fast and accurate detection of acute proximal MCA thrombotic occlusion. It is considered a warning sign for rapid and efficient intervention for stroke treatment, including thrombolysis and can be used for follow up of thrombus evolution. (Egypt J. Neurol. Psychiat. Neurosurg., 2008, 45(1): 3-15)

INTRODUCTION

Stroke is a leading cause of mortality and morbidity in the developed world1. MR imaging has become an established tool for the evaluation of stroke in patients in the hyperacute (typically 0–6 hr from symptom onset) and acute (first few days after symptom onset) stages2. With their high sensitivity to acute cerebral infarction, the new magnetic resonance (MR) imaging techniques integrated in the initial imaging protocol are gaining an increasingly important role3. The role of MR is to establish a proper early diagnosis, coupled with accurate information about the intracranial vasculature and brain for guidance in selecting the appropriate therapy4. The presence of hyperattenuated middle cerebral artery sign on computed tomography used to indicate intraluminal clot with a high specificity but very low sensitivity5. Occlusion of intracranial arteries is visible as the lack of a flow void in bright cerebrospinal fluid on T2-weighted MR images. FLAIR MR images show intra-arterial
thrombus or slow arterial flow more clearly as a hyperintense vessel surrounded by hypointense cerebrospinal fluid. In that context several authors, like Flacke et al. and Rovira et al. have described the Middle Cerebral Artery (MCA) susceptibility sign, which was defined as presence of hypointensity within the MCA, on axial T2-weighted susceptibility-based gradient-echo images (T2*-weighted), in which the diameter of the hypointense signal within the vessel exceeded the contralateral vessel diameter. Diffusion MR imaging depicts early diffusion changes associated with cytotoxic edema following energy metabolism failure and disruption of ion homeostasis. Consequently the echoplanar diffusion weighted imaging (EP-DWI) and the T2-weighted susceptibility-based gradient-echo images (T2*-weighted) have gained increasing acceptance in the model imaging of acute stroke. Demonstration of an occluded intracranial artery at MR angiography is one of the generally accepted MR criteria to identify candidates for thrombolysis. But according to Ciccone et al., evidence is still required to support the clinical feeling that intra-arterial thrombolysis, which needs longer time and greater complexity, leads to a better outcome. Most of the published studies were performed between 3 and 6 hours after onset of clinical symptoms, not enough data exist about the diagnostic accuracy of imaging an acute thrombus with the susceptibility-based T2* sequence and the signal changes that may occur beyond the 6 hours window and over time.

The aim of this work

is to evaluate the accuracy of echo-planar T2* weighted magnetic resonance (MR) sequence in detection of acute middle cerebral artery (MCA) thrombotic occlusion, not limited to the 6 hour window and comparing it to the information obtained from MRA, axial FLAIR sequence, and EP-DWI as well as CT, if available.

MATERIALS AND METHODS

A total of 130 patients were enrolled in this prospective study over a period of 2 years, from 2005 – 2007. One hundred patients with acute supratentorial neurological deficit were referred from the Neurological Emergency Department to the department of MR imaging at Ain Shams Specialized Hospital (ASUSH) within 76 hours after stroke onset (defined as the last time the patient was known to have no neurological symptoms). Thirty patients suffering from neurological disease without any evidence of stroke on DWI were also subjected to the same study protocol, to be considered as a control group for the study. Individual consent was obtained from each patient. Patients were evaluated clinically by NIHSS score for assessment of level of consciousness, vision and gaze, facial palsy, extremity weakness, limb ataxia, sensory loss, language, dysarthria, and neglect. A patient with a completely normal neurological examination and normal mental status will have an NIHSS of 0. The maximum recordable NIHSS score is 42. Patients with an NIHSS score greater than 15-20 are considered to have a severe stroke, while 14 or less have mild to moderate stroke.

A group of 47 patients (29 males: age range 11-86yrs; mean 58.3±14.7 yrs and 18 females: age range 35-83yrs; mean 59.4±12.7 yrs) were selected for the evaluation of the T2* MCA susceptibility sign. Selection of the study group was based on the following criteria:

Inclusion criteria for this study were:
1. MR diagnosis of an acute infarct within the MCA territory based upon EP-DWI
2. MRI performed from 2 to 76 hrs after ictus
3. Complete study, including all required sequences
4. Good quality images
Excluded were:
1. Patients with unstable vital signs or with contraindications for MR study
2. Late subacute and chronic cases
3. Transient Ischemic Attacks (TIAs)
4. Extensive hemorrhage that obscures the susceptibility sign on T2*
5. Acute on top of chronic infarcts
6. Infarctions related to multiple vascular territories

Imaging Examinations:
CT scan of the brain was the initial examination in 22 patients (out of 46). MRI studies were performed at the MRI unit of the Ain Shams Specialized Hospital. The machine used was a 1.5 Tesla General Electric Superconducting Magnet System, (Signa Horizon LX Prospeed) with a standard head coil and echoplanar capabilities.

MRI Protocol: The acute stroke protocol which did not exceed 20 minutes for the total imaging time, including positioning, consisted of: T2*-weighted gradient-echo imaging, diffusion-weighted imaging, 3D time-of-flight (TOF) MR angiography, FLAIR, T2- and T1-weighted imaging. For axial gradient echo EPI T2*-weighted imaging a GR/20; TR/TE: 500/15 msec and matrix size : 265 × 192 was used. Slice thickness was 5 mm and the intersection gap was 2 mm, FOV : 24 × 24 mm, slice thickness: 5 mm, interslice gap: 0. Additional parameters for the acute stroke MR protocol were as follows: for 3D TOF MR angiography, a 3D/TOF/SPGR/20 was used to visualize the intracranial internal carotid system, including the middle cerebral and the anterior cerebral arteries, at the level of circle of Willis. The following imaging parameters were used: 37/6.9/fr (TR/TE), 20° flip angle, 22×16 mm FOV, 256 × 160/1 NEX matrix, 800 sections with a 1.4 mm effective thickness and 0 spacing that resulted in the coverage of the above mentioned arteries from base of skull till midway between vertex and Corpus Callosum on sagittal localizer. Maximum intensity projection images (MIP) were produced at 12 equally spaced intervals perpendicular to the left-right axis that covered a total of 180° of rotation (6° per interval). No IV contrast was used. For the axial FLAIR: TR 8000 / TE 147/Ef msec; matrix size: 256 × 128/1NEX, slice thickness: 5 mm, interslice gap: 2 mm, FOV: 24 × 24 mm, inversion time: 2000 msec. Routine use of axial T1WI (fse) with a TR/TE of 500-600 / 9Fr and axial T2WI (fse) with a TR / TE: 6000/ 90-110/Ef msec

Image Analysis:
The radiologists first reviewed the images of the examinations independently in a blinded fashion, followed by a final consensus reading. If the sign was present, the observers classified it according to its location into: horizontal M1-, genu or M2-segments.
The extent and involved territory of the acute infarct was established based upon the EP-DWI.

Data interpretation was based on the examination of the entire set of MR images and the corresponding sections on CT (if available), specifically targeting the course of MCA on axial images:
1. Identification of the susceptibility sign on T2* (present or absent).
2. Evaluation of MCA course on FLAIR.
3. Identification of hyperattenuated MCA (hyperdense MCA-sign) on CT (was possible in 23 out of 47 cases).

Obtained data were compared to the 3D reformatted MRA images, and the MRA axial source images of the targeted MCA. According to the MRA-appearance of the affected MCA, the following criteria had to be checked:
1. Pattern of MCA arterial flow signal interruption: abrupt, gradually tapering, irregular.
2. Site of thrombus: proximal / horizontal M1-segment, genu, M2-segment or no thrombus identified.
3. State of ipsilateral ICA (thrombosed or well visualized).
4. State of the entire visualized intracerebral arterial system.

**RESULTS**

Fourty seven (47) patients (29 males; age range 11-86 years (mean: 58.3±14.7 yrs) and 18 females; age range 35-83 years (mean: 59.4±12.7 yrs) with acute territorial MCA infarcts were included in this study. Out of these 47 patients, 10 had hyperacute MCA infarction (scanned 2-6 hours after ictus), 7 of those patients presented with very severe stroke (NIHSS score 21 or more) and 3 patients with severe stroke (NIHSS score 15-20). Thirty seven (37) patients were studied within 76 hours from ictus (acute to early subacute). Among this group and according to NIHSS score, 5 patients had mild to moderate stroke, 16 patients severe stroke; and 13 patients had very severe stroke. One patient who had very severe stroke with NIHSS score of 22, died. She had left MCA occlusion, presented 4 hours within onset of right hemiplegia and had a history of DM, HTN and COPD.

Table 1. The (T2*) susceptibility sign and (Flair) hyperintense vessel sign in 47 patients, grouped according to stage of MCA infarction.

<table>
<thead>
<tr>
<th>MRI</th>
<th>T2*+ (36/47)</th>
<th>FL+ (29/47)</th>
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<tbody>
<tr>
<td>Hyperacute</td>
<td>9/10 90%</td>
<td>7/10 70%</td>
</tr>
<tr>
<td>Not hyperacute</td>
<td>27/37 73%</td>
<td>22/37 59.5%</td>
</tr>
<tr>
<td>- Acute</td>
<td>13/17 76.5%</td>
<td>12/17 70.6%</td>
</tr>
<tr>
<td>- Early subacute</td>
<td>14/20 70%</td>
<td>10/20 50%</td>
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</table>

- In the group of the hyperacute MCA infarction, 9 out of 10 patients (90%) showed the MCA-susceptibility sign on T2*; the site of MCA thrombosis could be traced on FLAIR in 7 out of 10 patients (70%). The horizontal M1-segment was affected in all 10 patients. All patients in this group had severe to very severe stroke according to NIHSS score and one patient died.
- In the group of acute to early subacute MCA infarction (37 patients), the MCA-susceptibility sign was recognized on T2* in 27 out of 37 patients (73%), while in 10 patients the sign could not be identified (27%). The site of thrombus could be traced on Flair in 22 out of 37 patients (59.5%). Among this group of patients, there were 4 patients with mild to moderate stroke according to NIHSS score. This group was further subdivided into acute (up to 48 hours) and early subacute (up to 76 hours). Seventeen (17) patients belonged to the acute group. Here analysis of the images revealed that 13 out of the 17 patients (76.5%) in the acute group showed the MCA-susceptibility sign on T2*, in 4 patients (23.5%) the sign was not recognized. In the early subacute group (20 patients), the sign was recognized in 14 out of 20 patients (70%), it was not recognized in 6 patients (30%).
- Analysis of the images on Flair showed that the pick up rate of the hyperintense vessel sign (HVS) was higher in the acute group: 12 out of 17 (70.6%) and less in the later imaged patients (the subacute group): 10 out of 20 (50%)
- The left MCA was involved in 33 patients (70.2%) out of the total 47, while the right side was affected in only 14 patients (29.8%). Overall the M1-segment, proximal to the genu was involved in 35 patients (74.5%), the genu was involved in
7 patients (14.9%), the lenticulostriate artery in 2 patients (4.3%), and the M2 segment of MCA in 2 patients (4.3%). ICA involvement was noted in 15 out of 47 patients (31.9%).

- 23 patients had an initial CT of the brain. The hyperdense-MCA-sign was identified in 11 out of those (47.8%). All patients showing the hyperdense MCA-sign on CT, showed also the MCA-susceptibility sign on T2*.

Statistical Analysis

The SPSS statistics package (version 12.0 for Windows [Microsoft], Statistical Package for the Social Sciences) was used for subsequent statistical analysis.

To assess the diagnostic reliability of the presence or absence of the MCA susceptibility sign on T2*-weighted MR images and the statistical significance of the differences in patient age and sex and a positive MCA susceptibility sign were assessed using the Mann-Whitney U test. A p value of less than 0.05 was considered to indicate a significant difference.

MCA susceptibility sign on T2* weighted images in 47 Patients showed a p-value of < 0.01 which is an indication of its high significance. The Chi-square test value was 43.155

Table 2. Susceptibility Sign on MR Images in 47 Patients.

<table>
<thead>
<tr>
<th>Statistic</th>
<th>All Patients (47)</th>
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<tbody>
<tr>
<td>Sensitivity</td>
<td>76.6%</td>
</tr>
<tr>
<td>Specificity</td>
<td>100 %</td>
</tr>
<tr>
<td>Negative predictive value</td>
<td>73.2%</td>
</tr>
<tr>
<td>Positive predictive value</td>
<td>100 %</td>
</tr>
<tr>
<td>Accuracy</td>
<td>85.7%</td>
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The hyperintense vessel sign on Flair images in 47 Patients showed a p-value of <0.01, the Chi-square test value was 36.86. The relationship between sex and stroke showed a p-value of >0.05, indicating no significant relationship between sex and stroke.

Fig. (1): (a) axial T2*WI showing MCA-susceptibility sign and site of thrombus in a case of hyperacute infarction (arrow) (b) 3D TOF of the same patient showing the abrupt loss of arterial flow signal at proximal left MCA (arrow) (c) axial Flair of another patient showing the hyperintense MCA vessel sign (arrow).
Fig. (2): (a) hyperacute left MCA infarction seen as hyperintense signal on coronal DWI (b): axial T2*WI shows no MCA-susceptibility sign (c) 3D TOF, collapsed image, of the same patient showing the abrupt loss of arterial flow signal at origin of left MCA (arrow).

Fig. (3): (a) CT brain, showing hyperdense MCA sign at rt MCA (white arrow) in a 76 ys female with hyperacute stroke, missed by outside readers. The left MCA is also abnormal (black arrow), due to atherosclerosis (b) high density at horizontal left MCA, in another patient with hyperacute (6 hs) infarction (arrow), missed by outside readers (c) Axial T2*WI showing the susceptibility sign distal to the left MCA bifurcation (arrow).

Fig. (4): (a) Axial T2*WI showing the susceptibility sign in a 2 hour hyperacute stroke patient as it develops in horizontal left MCA. (arrow) (b) Signal changes start to develop at the axial DWI (arrow).
DISCUSSION

Findings of this study indicate the overall high sensitivity (76.6%) and specificity (100%) (see table 2) of echo-planar T2*-weighted MR imaging in detection of hyperacute intracranial thrombotic occlusion proximal to the MCA bifurcation (Fig. 1a) and its significant association with the presence of MRA findings of occlusion of the corresponding artery segment (Fig. 1b). Using the high sensitivity of MR angiography as the reference standard was chosen based upon the results of studies performed by Flacke et al.3 and Kim et al.9, who found that the results of MRA are highly accurate and are comparable to digital subtraction angiography regarding proximal MCA trunk thrombotic occlusion. The possibility of misdiagnosing a subtotal occlusion as a total, described by Hirai T et al.10, was taken into consideration, although it does not affect decision making regarding thrombolytic therapy.

In the group of the hyperacute MCA infarction, 9 out of 10 patients (90%) showed the MCA-susceptibility sign on T2*, all had proximal MCA thrombotic occlusion (Fig 4 & 5). This is in keeping with Rovira et al6, who showed a 96% accuracy in proximal MCA and 83% in distal occlusion. The diagnostic accuracy according to our study was 90% for the hyperacute patient group. In the group of acute to early subacute MCA infarction (37 patients), the MCA-susceptibility sign was recognized on T2* in 27 out of 37 patients (73%). Ten patients did not show the MCA-susceptibility sign on T2* (27%). Further separation and subdivision of this group revealed that 13 out of 17 patients, who had an acute infarction, showed the MCA-susceptibility sign on T2* (76.5%). The early subacute (up to 76 hours) cerebral infarction group included 20 patients. The sign was recognized in 14 out of 20 patients (70%). This reveals a decreasing reliability of the T2* susceptibility sign over time, starting from hyperacute through acute until subacute stages.
This may be due to partial recanalisation of the thrombosed MCA, which may be spontaneous or drug induced.

The signal loss on T2*, that helps identify the susceptibility sign, is explained by severe T2 shortening of an acute clot, representing the magnetic susceptibility differences that arise from intracellular deoxy-hemoglobin, which is present in high concentrations in acute thrombo-embolism (Fig. 3c). This magnetic susceptibility effect produces a non uniform magnetic field leading to a rapid dephasing of proton spins, which results in signal loss, seen on T2* susceptibility-weighted images. In one of the 10 patients with hyperacute MCA occlusion (a female with acute onset of right sided hemiparesis), the MCA-susceptibility sign could not be identified, (Fig. 2 a,b,c). The reason for this false negative result may have been related to the patient’s clinical history, which disclosed longstanding chronic obstructive pulmonary disease (COPD) and might have been the cause for an altered oxy-/deoxy-hemoglobin level in blood, producing a rather uniform magnetic field. Lack of the magnetic susceptibility effect leads to loss of the MCA-susceptibility sign on T2*WI in this particular case.

No false positive signs of MCA-susceptibility in acute thrombo-embolism were encountered in this study which is in keeping with the findings of Rovira et al., who does not even see any need for a confirmation by MRA, once the susceptibility sign is detected. This is a point that can be challenged, as including the MRA in the routine work-up of a stroke patient is essential at our institution, not only does it show the site of acute vascular occlusion, but it also gives the clinician an idea about the overall status of the anterior and posterior cerebral circulation, which are key factors in the further management and prognosis. Combining various imaging techniques improves the diagnostic capability which may have an impact on clinical outcome, a point that has been proved by Hacke, who showed good clinical outcome of thrombolytic drug therapy in patients with acute stroke who were selected on the basis of imaging criteria. Our findings show a tendency for having severe stroke in the acute stage when MCA susceptibility sign is present, but this did not indicate a high expectation of bad prognosis in those patients presented in acute stage. This was also noticed by Flacke et al., who described the radiological findings of susceptibility changes in MCA in 23 patients with acute MCA ischemic stroke and concluded that the presence of MCA susceptibility sign correlated positively with the initial presentation but yet, was not a predictor for clinical outcome in acute stage. Tomsick et al., investigated the relationship between the hyperdense MCA sign and the neurological deficit assessed by NIHSS score. They included 55 patients with hyperacute ischemic stroke, treated by thrombolysis within 90 minutes and assessed by CT scan and compared the baseline initial NIHSS score comparing those with and without hyperdense MCA sign, then after 2 hours and 3 months residual neurological deficit. They found that all patients with hyperdense MCA sign (33% of total patients) had a median baseline NIHSS score 19.5 “range 15-21”. Non of those patients with positive hyperdense MCA sign had an NIHSS score less than 10, while patients lacking this sign had an NIHSS score of 10. In their study, patients with hyperdense MCA sign had a greater pretreatment neurological deficit which matches our results, as all hyperacute patients had an NIHSS score of severe to very severe stroke. Our study documents that the MCA susceptibility sign in T2* weighted image is associated with large neurologic deficits, i.e. “severe to very severe NIHSS score”
For the detection of the susceptibility sign, we used 5-mm section thickness and fast two-dimensional echo-planar T2*-weighted MR imaging. This technique demonstrated high sensitivity in depiction of hyperacute thrombotic occlusion within the proximal MCA. This susceptibility-based sequence has been introduced into our routine stroke protocol and is used to rule out hyperacute parenchymal hemorrhage, as therapy decisions are required immediately in these patients, which means that CT brain is not always performed prior to MR imaging as a tool to rule out hemorrhage. In this study 23 Patients (out of 47) had an initial CT of the brain, 11 out of those (47.8%) showed the hyperdense MCA-sign on CT. Flacke et al.3, reported a 54% pick up rate for the hyperdense sign on CT. Petitti 13 described the hyperdense sign as being an indirect sign of cerebral ischemia that corresponds to occlusion of the middle cerebral artery by an intraluminal clot from a thrombus or an embolus. The attenuation of flowing blood, which is approximately 40 HU, is linearly related to the hemoglobin concentration. In a thrombus, heme is an extrusion of serum, and the attenuation of a thrombus at CT is about 80 HU 13. False positive signs can be obtained in cases of calcified atherosclerosis and high hematocrite values14. According to Bastianello et al. 15, the hyperdense MCA-sign on CT is a transient phenomenon, due to the mobile nature of the clot. This study showed that the sign was completely missed and not reported in 6 out of 11 cases, due to lack of experience at other facilities, and had to be corrected by the study group on subsequent image evaluation. Only two patients of the hyperacute group had an initial CT of the brain, both showed the hyperdense MCA-sign (Fig.3a,b), unfortunately the sign was missed by outside readers, which indicates the need for higher expert reading.

In the group of acute infarction 7 out of 17 patients had a CT of the brain before the MRI study. Three patients showed the hyperdense MCA-sign on CT (3 / 7= 42.8%), which means that the CT was negative in 4 / 7 cases (57%). Compared to the MCA-susceptibility sign on T2*, which was identified in 13 out of 17 patients (76.5%). Thirteen patients out of the subacute group had an initial CT of the brain. Seven out of those (7 / 13 = 54%) did not show the hyperdense MCA-sign and in 6 patients (6 / 13 = 46%) it could be identified. This is in keeping with Flacke et al., 2000, who proved the higher sensitivity of T2*WI compared to CT in the detection of an MCA thromboembolism.

The presence of hyperintense vessels on FLAIR images is thought to indicate the presence of slow flow or stasis in small arteries, veins, or collateral vessels and, according to Maeda et al.16, most frequently observed at the Sylvian fissure. The mechanism of increased intravascular signal intensity on FLAIR images in acute ischemia is most likely due to a combination of slow flow (intravoxel phase dispersion, TOF effects), flow-related enhancement (slow, but not static flow), and clot signal intensity (oxyhemoglobin) in small arteries, veins, and collateral vessels17. Analysis of the Flair images in this study showed the site of thrombus in 29 out of 47 patients (61.7%). But in addition, an overall increased intensity of cerebral blood vessels on Flair was noted, which correlates well with the statement of Kamran et al.18, who have noted this phenomenon in the setting of acute ischemia. The diagnostic accuracy of the hyperintense vessel sign (HVS) on Flair was highest in the hyperacute group (70%) (Fig. 1c), followed by the acute group: 12 out of 17 (70.6%) and less in the later imaged patients (the subacute group): 10 out of 20 (50%). Comparison showed that the diagnostic accuracy of the susceptibility-based T2* sequence in the hyperacute stage proved to be more sensitive than the hyperintense vessel sign (HVS) on Flair, while in the acute and
early subacute stage results of Flair and T2* were very close. However, in this context, it has to be mentioned that patients with atherosclerosis and/or slow flow in their MCA showed similar signal changes to a thrombosed artery and that the T2*W sequence was always inspected first, followed by the Flair. Makkat et al.2, mentioned the problem of missing the HVS sign on Flair, if not actively looking for it, which makes it not very reliable. Without the presence of an MRA- and the T2*- guide, the Flair sequence is often misleading, especially in patients with chronic cerebrovascular disease. The results of this study show that the prognostic value of the hyperintense vessel sign (HVS) on Flair needs to be further evaluated with a large prospective study in acute and chronic settings to become clinically useful. A similar suggestion has been made by Wolf17. We agree with Schellinger et al.19 that the use of such signs could be additional or confirmatory information as regards to vessel patency, hemodynamic status and thrombus composition, thus improving diagnostic and prognostic strength of stroke MR imaging, but is not necessarily specific for thrombotic occlusion.

The susceptibility sign on T2* can be hindered by susceptibility artifacts like dental implants, which were the source of artifacts at the skull base in the study performed by Flacke et al.3, obscuring the area of the MCA trunk. This problem was not encountered in this study, but is an important factor that has to be kept in mind. Other problems that have to be considered are susceptibility changes of small blood clots in the proximal part of the MCA and within the distal internal carotid artery, which may be underestimated as transverse views display them in only cross section1.

The findings of this study show no correlation between bad prognosis and the MCA susceptibility sign, which is in agreement with Flacke et al.3. On the basis of our observations, the presence of the MCA susceptibility sign as a single prognostic factor has no association with poor prognosis in spontaneous MCA stroke, as has been reported by Tomsick et al.12. We do not agree with Kim et al.9 in that appearance of this sign was associated with an unfavorable clinical outcome. In their study a favorable clinical outcome were a low baseline NIHSS score on admission and the site of the occlusion (M2 occlusion). In this study, only one patient died, who had very severe stroke with NIHSS score of 22. The patient had left MCA occlusion, presented 4 hours within onset of right hemiplegia and had a history of DM, HTN and COPD and did not show the MCA susceptibility sign on T2* (Fig. 2b).

In the study performed by Rovira et al.6, most of the patients were examined beyond the 3-hour window and always no later than 6 hours after the onset of symptoms. Also, no serial MR examinations were performed in the first days. Therefore, they could not establish the minimum time needed to study an acute thrombus with the susceptibility-based T2* sequence. According to our study the MCA-susceptibility sign on T2*WI was picked up as early as 2 hours after the ictus, which was the earliest presentation we had (Fig. 4a). To Rovira et al.6, the signal changes that may occur over time were unknown and they could not ensure that the high level of diagnostic accuracy obtained in their study (71%) will be reproduced in patients in whom the MR examination is performed within the first 3 hours or beyond the 6 hours after symptoms onset. The results of this study indicate that the MCA-susceptibility sign on T2*WI in the hyperacute patient group showed a 90% pick up rate. In acute and subacute cerebral infarctions it is also a reliable sign, but its sensitivity diminishes with time, to reach 76.5% in the acute and 70% in the subacute cerebral infarction group. The susceptibility sign on T2*WI is of high significance, showing an overall p-value of < 0.01 and a Chi-square
value of 43.16. All clinical observation was based on spontaneous stroke evolution after treatment with antiplatelet and heparin. In all 47 patients, the susceptibility sign as detected at CT (if available) and its MRI correlate were ipsilateral to the clinically involved hemisphere. Although we could not confirm that patients with a positive susceptibility sign were admitted earlier than other patients with hyperacute infarction, on the basis of our observation, the presence of MCA susceptibility sign as a single factor can predict the severity of stroke. From our study, all patients presenting in the hyperacute stage had a severe to very severe stroke, according to NIHSS score which should an alarm for rapid and efficient stroke treatment intervention like thrombolysis. Therapy proposals are diverse and variable. In a very recent editorial20, the hope was expressed to be able, within a few years, to select the ideal patient for intravenous thrombolysis and the ideal patient for other intra-arterial recanalization techniques, or even a combination of both21.

Conclusion

It is advised to use the echo-planar T2* WI as one of the essential sequences in the MRI protocol for all cerebrovascular stroke patients, because diagnosis of the MCA susceptibility sign is a sensitive and specific sign of acute thromboembolic occlusion. It is useful in the evaluation, management decisions, prognosis and follow up of patients with recent stroke. The MCA susceptibility sign can be used not only as a reliable indicator of acute thromboembolic occlusion, but also in monitoring the immediate effectiveness of therapy, whether intraarterial or intravenous thrombolysis, in patients with hyperacute ischemic stroke. Further studies are needed for follow up of the surviving group of patients who showed the MCA susceptibility sign within 3 to 6 months, for prediction of the prognostic value of this sign.

REFERENCES


الملخص العربي

حساسية الرنين المغناطيسي في تصوير الشريان المخى الأوسط

في حالات السكتة الدماغية الحادة

الغرض من البحث:
تقيم دقة الفحص بالمقارنة Echo-Planar T2* في حالات السكتة الدماغية الحادة بالمقارنة بفحص شرايين المخ MRA و Flair في الكشف على الانسداد الحاد في الشريان المخى الأوسط.

المواد والطرق:
تضمن البحث 47 مريضاً على التوالي مصابين بالسكتة الدماغية الحادة والتي نتجت عن انسداد شرايين المخ الوسطى ولقد تم فحصهم بالرنين المغناطيسي في غضون فترة تتراوح بين ساعتين و 76 ساعة من بداية الإصابة و تم Echo-Planar T2* و Flair و MRA للفحص بوجود علامة الحساسية في شرايين المخ الوسطى ومتت مقارنتها بنتائج فحص شرايين المخ بالرنين المغناطيسي (MRA) و فحص عن طريق Flair Score.

النتائج:
لقد استخلصنا من هذا البحث أن وجود علامة الحساسية في فحص Echo-Planar T2* في الشريان المخى الوسطى هو علامة على الانسداد الحاد لهذه الشرايين مما يجعلها تستوجب علامة مفيدة سرعة التدخل العلاجي.