Imaging Prediction of the Prognosis in Carbon Monoxide Poisoning Encephalopathy: Magnetic Resonance Imaging Study

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Abstract. In order to explore the evaluation value of diffusion kurtosis imaging (DKI) on the prognosis of patients with carbon monoxide toxic encephalopathy, we analyzing the characteristics of early MRI and DKI images of patients with carbon monoxide poisoning encephalopathy. 19 patients with clinically confirmed acute carbon monoxide poisoning encephalopathy (5.2±1.25 days after poisoning) and 10 age-matched normal volunteers underwent routine MRI scan, of which 5 patients received DKI and MRI scan 3 weeks to 3 months after poisoning. The DKI parameters included fractional anapatiity (FA), mean diffusion (MD) and mean kurtosis (MK) were analyzed in different brain area between the case group and the normal control group. During the acute phase of carbon monoxide poisoning, MRI scans showed abnormal signal in bilateral frontal and temporal lobe cortex, subcortical white matter area and hippocampus, while bilateral cerebral white matter region and globus pallidus were showed in the patients with delayed encephalopathy after acute carbon monoxide poisoning (DEACMP). Two of the five patients were diagnosed with DECAMP, their early MK value was increased, while the early FA value of each lesion was decreased and MD value was increased in the globus pallidus during follow-up. The change of MK value might be correlated with the prognosis of lesions. Abnormal increase of MK value in early lesions may indicate poor prognosis.

Introduction

Acute carbon monoxide poisoning is the main cause of acute poisoning, which can lead to multiple organ damage, including brain, myocardial and other important organs, among which brain damage can lead to poor prognosis. Delayed encephalopathy after acute carbon monoxide poisoning (DEACMP) is a serious complication of carbon monoxide poisoning. Clinically, it is difficult to evaluate the degree of brain injury in these patients. The mental state, consciousness state and the concentration of carbon and oxygen hemoglobin of patients in acute phase are considered to be not helpful to predict their clinical behavior in chronic phase [1, 2]. In the early diagnosis of carbon monoxide toxic encephalopathy, imaging methods are of guiding significance to the changes of brain tissue morphology and function. At present, MRI imaging technology in common clinical imaging methods is of great significance in the diagnosis and prognosis of carbon monoxide toxic encephalopathy. Diffusional kurtosis imaging (DKI) is a new diffusion technique developed on the basis of DTI. DKI has good sensitivity and specificity in evaluating the changes of gray matter microstructure in the brain, and it can play a very important role in evaluating disease progression and treatment prognosis [3-9]. In this experiment, 19 patients with carbon monoxide poisoning were examined by conventional MRI and DKI. The conventional MRI and DKI images of carbon monoxide poisoning cases were analyzed to explore the imaging changes of carbon monoxide poisoning and the value of magnetic resonance diffusion kurtosis imaging in the diagnosis and prognosis of carbon monoxide poisoning.
Materials and Methods

Case Group

Nineteen patients underwent conventional MRI after acute carbon monoxide poisoning. Five of the 19 patients received DKI and MRI scan 3 weeks to 3 months after poisoning. Two of them were diagnosed as DEACMP due to recurrent neurological and psychiatric symptoms after false recovery. The other 3 patients were reexamined according to the doctor's advice after discharge from the hospital without obvious neurological and psychiatric symptoms during reexamination.

All patients met the following criteria: (1) carbon monoxide exposure and coma; (2) age 20-33 years old; (3) No history of craniocerebral trauma, no history of neurological and mental diseases, and serious heart, liver and kidney diseases, diabetes illness, etc.

Control Group. (1) 10 normal volunteers (4 males and 6 females, 22-27 years old, average 25±1.3 years old); (2) No history of nervous system diseases, brain trauma, loss of consciousness or mental disorders; (3) No obvious positive signs were found in clinical neurological examination; (4) No obvious abnormal signal foci were found in brain on MRI imaging sequences.

Methods and Parameters

U.S. GE signa Hi-Speed 3.0T superconducting magnetic resonance imaging system and its matched 8-channel orthogonal head coil are used.

Conventional MR Scan Sequence and Parameters: included axial T1WI, T2WI, sagittal or coronal T2WI. Scanning is performed according to the following sequence and parameters: T1WI: TR: Spin Echo (SE), TR 1750ms, TE 24ms, thickness 3.0mm, space 0.0mm, and flip angle 90; T2WI: Fast Spin Echo (FSE), TR 7000ms, TE 109.9ms, thickness 5.0mm, space 1.5mm, turning angle 90;

DKI Scan Sequence and Parameters: single excitation spin plane echo sequence (SE-EPI), TR 8000ms, TE 109.9ms, thickness 3.0mm, space 0.0mm, FOV 24×24cm, acquisition moment 256×256, NEX1, B value 0,1000, and 2000 s/mm², acquisition direction 15, and scanning time 3 minutes and 31 seconds.

Image Analysis and Data Measurement

The DKI parameters, including fractured animation (FA), mean diffusion(MD), and axial diffusion(AD) were generated by Functool software. Referring to conventional MRI and DWI images, the region of interest is taken from FA, MD and MK images. The region of interest includes left and right semi-oval centers, basal ganglia, frontal lobe, temporal lobe, hippocampus and cerebellar hemisphere. The FA, MD and MK data measured at various parts of 5 patients in the early stage were averaged and compared with the corresponding parts of the normal control group.

Results

Conventional MRI Findings of Acute Homogenized Carbon Poisoning Brain

19 cases of acute carbon monoxide poisoning showed symmetrical hypointensities T1WI and hyperintensities in T2WI, FLAIR and DWI signals in bilateral frontal and temporal lobe cortex, subcortical white matter area and hippocampus. MRI scan of 5 patients was reexamined from 3 weeks to 3 months after poisoning. Abnormal signals seen in bilateral frontal and temporal cortex, hippocampus and subcortical white matter areas in acute phase were significantly improved compared with before, suggesting that intracranial carbon monoxide poisoning lesions of the 3 patients (patient C, D, E) tended to recover, as shown in (Figure. 1). Abnormal signals in bilateral frontal lobe and temporal lobe cortex, hippocampus and subcortical white matter areas of other 2 DEACMP patients (patient A, B) were also significantly improved compared with the former, leaving symmetrical abnormal signals in bilateral globus pallidus (Figure. 1).
Changes of DKI Parameters and Lesion Evolution in Patients with Carbon Monoxide Toxic Encephalopathy

Compared with the normal control group, the early MK value of the lesion (bilateral frontal lobe, temporal lobe and hippocampus) was decreased, while in the 2 cases (DEACMP patient) with globus pallidus softening, the early MK value of the lesion was increased. The early FA value of each lesion was decreased and MD value was increased in all the 5 patients (Table 1). During follow-up, it was found that the globus pallidus showed obvious high signal in MD and low signal in MK, suggesting that the lesion in the globus pallidus region of the patient tended to infarction, as shown in (Figure. 2).

Table 1. DKI Parameter values of globus pallidus in 5 patient.

<table>
<thead>
<tr>
<th></th>
<th>MK</th>
<th>FA</th>
<th>MD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control Group</td>
<td>0.8587±0.0125</td>
<td>0.3534±0.053</td>
<td>0.6771±0.2257</td>
</tr>
<tr>
<td>Patient A</td>
<td>1.0943±0.016↑*</td>
<td>0.3851±0.054</td>
<td>0.8324±0.039</td>
</tr>
<tr>
<td>Patient B</td>
<td>0.9568±0.078↑*</td>
<td>0.2167±0.073↓</td>
<td>0.8275±0.042</td>
</tr>
<tr>
<td>Patient C</td>
<td>0.7147±0.074↓*</td>
<td>0.2745±0.041↓</td>
<td>0.8886±0.041↑*</td>
</tr>
<tr>
<td>Patient D</td>
<td>0.716±0.035↓*</td>
<td>0.250±0.013↓</td>
<td>0.8770±0.026↑*</td>
</tr>
<tr>
<td>Patient E</td>
<td>0.7344±0.017↓*</td>
<td>0.3346±0.013</td>
<td>0.9394±0.033↑*</td>
</tr>
</tbody>
</table>

Diffusion kurtosis imaging derived values based on the patient and corresponding areas in healthy controls. Data are means (of voxels) ± inter-subject SD. *P<0.05 compared with controls

5 Comparison of DKI Parameters in Patients with Acute Carbon Monoxide Poisoning

The MK value of globus pallidus of acute carbon monoxide poisoning patients A and B were both higher than that of normal control group, FA value was lower than that of normal control group, MD value was higher than that of normal control group. MK and FA values in bilateral frontal lobe, temporal lobe and hippocampus of patients A and B were lower than those in normal control group,
while MD values were higher than those in normal control group (Table 1); The MK and FA values of globus pallidus, frontal lobe, temporal lobe and hippocampus of the three patients (patient C, D, E) with acute carbon monoxide poisoning were lower than those of the normal control group, while the MD values were higher than those of the normal control group.

Discussion

The central nervous system is most sensitive to hypoxia and is firstly affected after CO poisoning. CT examination after acute carbon monoxide poisoning mainly shows acute cerebral edema, narrowing and disappearance sulcus of gyrus, and reduction of transverse diameter of third ventricle. MRI findings in the early stage of acute carbon monoxide poisoning encephalopathy showed that the lesions were mostly located in cerebral cortex and basal ganglia, mainly implicating globus pallidus. Pathologic changes are similar to hypoxic-ischemic encephalopathy, mainly manifested by cerebral vasospasms followed by dilation, increased permeability, cerebral edema and varying degrees of focal encephalomalacia or necrosis. Cerebral edema and ischemia can cause secondary cerebral circulation disorder, which causes ischemic encephalomalacia and aggravates cerebral hypoxia. The most common lesions are basal ganglia, especially globus pallidus, and semiovale, where axonal destruction, myelin sheath breakdown and dissolution occur in. The magnetic resonance examination of acute carbon monoxide poisoning patients observed in this study is mainly located in bilateral frontotemporal parietal cortex, subcortical white matter region, hippocampus, globus pallidus, etc. However, the pathological changes of cerebral lesions take a certain time, so patients can pass a period of false recovery from coma to wakefulness, and then suffer from related neuropsychiatric symptoms, namely delayed encephalopathy after acute carbon monoxide poisoning (DEACMP). However, in the 2 confirmed DEACMP patients, the main manifestation was the reduction of white matter lesions during follow-up review, suggesting that possible matter demyelination, and the formation of globus pallidus malacia foci can be observed, suggesting that it is irreversible damage. Some studies believe that patients with globus pallidus lesions and white matter lesions involved have severe clinical manifestations and poor prognosis. The results of study suggested that MRI scan could detect the lesions and have certain value in judging prognosis.

Diffusion Weighted Imaging (DWI) has been applied to the evaluation of brain injury after CO poisoning in the imaging examination of carbon monoxide poisoning encephalopathy, which can objectively and quantitatively represent the diffusion of water molecules in tissues. Cytotoxic edema of acute ischemic lesion is manifested as DWI high signal area and decreased ADC value. In this case, DWI is also applicable to acute or subacute stage of CO poisoning [10]. Previous studies have suggested that DWI is more sensitive to cytotoxic edema after white matter injury than conventional magnetic resonance imaging [11-12]. In subacute phase, compared with DWI of globus pallidus and white matter, globus pallidus shows low signal area and high ADC value, while white matter shows high signal area and low ADC value [12]. Some researchers thinks that necrosis of globus pallidus may occur earlier than white matter. Therefore, white matter injury is not only attributed to hypotensive ischemic changes such as globus pallidus injury, but also to progressive demyelination. Diffusion Tensor Imaging (DTI) can be used to judge the disease condition and evaluate the prognosis of CO toxic encephalopathy [13-15]. DTI can reflect the directivity of water molecule diffusion and the pathological changes of brain microstructure that cannot be reflected by conventional MRI. Diffuse characteristics of DTI (FA) can accurately reflect the different pathological changes of the microstructure and quantify the degree of brain injury. FA value reflects the direction of diffusion of water molecules and provides information about cell arrangement and structural integrity of white matter bundles [10, 16]. Since DTI can only detect the structure of white matter, it cannot provide more information in gray matter regions. In order to evaluate brain damage caused by CO poisoning (WM and GM), a new sequence, diffusion peak imaging (DKI), developed on the basis of DTI, was used to evaluating the changes of gray matter microstructure in the brain, and it can play a very important role in evaluating disease progression and treatment prognosis[4-9].
Reviewing the 5 patients in this clinical study, there was no abnormal signal in the globus pallidus region during the routine MRI in the acute phase. However, after 3 weeks of subsequent visit, it was found that the bilateral globus pallidus of both patients A and B of DECAMP showed obvious necrosis and softened changes, while the other 3 patients (patient C, D, E) did not show changes of the globus pallidus. Therefore, we believe that conventional magnetic resonance imaging can only reflect the changes in signal and morphology of the lesions at the time of examination, but it cannot predict the future evolution of the lesions and reveal some potential hidden lesions. However, after DKI scan of specific region of these 5 patients, it was found that the MK values measured by patients A and B with necrosis and softening of the globus pallidum were significantly higher, while the MK values measured by D, E and F with no necrosis or softening of the globus pallidum did not significantly change compared to the control group. The FA value of globus pallidus in the early stage of 5 patients all decreased, while the MD value increased. These two indexes (FA, MD) have no specificity for the subsequent lesion evolution. The MK value has higher accuracy than FA value in reflecting the changes of gray matter structure, which is consistent with the literature.

Conclusion

Conventional MRI of patients with carbon monoxide poisoning can reflect the changes in signal and morphology of the lesions, but cannot identify the pathological evolution basis of the lesions, and cannot provide a prediction basis for the development trend of the lesions. DKI scan shows that the abnormal increase of the MK value of the globus pallidus in acute phase may have a certain correlation with the necrosis and softening of the globus pallidus during the chronic phase of the patients. However, as the research object sample was a case study, in order to further explore and prove whether the increase of local MK value is related to the occurrence of local necrosis and softening in the chronic phase of the patient, further experimental proof is needed.

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