Diagnostic value of MRI diffusion-weighted imaging in evaluation of intracranial hemorrhage (ICH)

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Authors: S. Khedr; Jiddah/SA
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**Purpose**

To assess the diagnostic value of diffusion weighted imaging in patients with intracranial hemorrhage.

**Methods and Materials**

**Patients**

Patients among all consecutive patients admitted to our institution between March 2008 and Feb. 2011, we retrospectively selected those who fulfilled the following criteria: 1) sudden onset of acute neurologic symptoms due to an intracranial hematoma; 2) patients performed MRI(including DWI and GRE) and CT with time interval between the two examinations 2 hour.

61 patients (20 females and 41 males; mean age, 56 years; range, 19-83) fulfilled these criteria and constituted our study group. The median delay from clinical onset to MR imaging was 25 hours (minimum 3 hours; maximum 9 days). When a patient underwent sequential MR imaging, only the initial examination was considered for this study.

**Imaging Techniques**

MR examination was done for all patients using Magnetom symphony, syngo, 1.5 T machine. The conventional MR imaging protocol included (a) axial T1-weighted spin-echo (467/9 [repetition time (TR) msec/echo time (TE) msec]), (b) axial T2-weighted fast spin-echo (3417/102 [effective echo time]), and (c) axial FLAIR (10000/400/2200 [inversion time]). The parameters of conventional MR imaging were a 256 192 matrix, a 23-cm field of view, and a 5 mm/2 mm slice thickness/intersection gap. Singleshot, spin-echo, echo-planar DWI sequences were obtained by applying diffusion gradients in three orthogonal directions at each slice, with two diffusion weightings (b value= 0 and 900 or 1000 sec/mm2). Isotropic DWI was generated on-line by averaging three orthogonal-axis images. The DWI examination acquired 20 slices with parameters of 6500/96.8 (TR/TE), a 128 128 matrix, a 28-cm field of view, and 5-mm slice thickness with a 2-mm intersection gap. gradient-echo imaging (TR/TE = 450/20).

Computed tomographic scans were performed on Lightspeed scanner (General Electric). Images were acquired following the orbito-meatal plane with 3 mm thickness for the entire examination.

**Imaging Analysis**
All the MRI and CT examination were reviewed by experienced neuroradiologist blinded to the clinical information and all patient identifiers. Interpretations for each imaging modality (CT and MRI) for a single patient were performed on different days to avoid reader recognition or recall of findings from the other modality. The order of presentation of the films was randomized and differed for each modality.

Diffusion weighted imagings were analyzed for

- The presence of hemorrhage
- The age of hemorrhage; hyperacute, acute, early subacute or late subacute.
- Type of hemorrhage; parenchymal, intraventricular, subarachnoid, subdural, and epidural
- If it is parenchymal is it primary hemorrhage or hemorrhagic lesions(hemorrhagic arterial infarction or hemorrhagic venous infarction).
- Location of the hemorrhage; cortical, subcortical or basal ganglia.

**Quantitative analysis**

Was used to determine the apparent diffusion coefficient (ADC) of each ICH (hyper acute, acute, early subacute and late subacute) at its center, as seen at DWI. A region of interest (ROI) was carefully placed within the hematoma and also in contralateral normal white matter. The ROI was drawn as large as possible while using a circular or rectangular ROI on the workstation, and its area ranged from 14 to 302 mm². In each case, the radiologist measured the ROI and the ADC values were calculated. Relative ADC, which is the ratio of the ADC value for a lesion to that for normal contralateral white matter, was also calculated in each case. All data concerning ADC values for each stage are presented as means 1 standard deviation. In cases of hemorrhagic infarction the area of infarction was calculated.

**Statistical Analysis**

In the initial sample we found that MRI was detecting hemorrhages not visualized on CT which was also supported by previous studies (5). So CT could not be used as criterion standard in this study. Therefore, the analysis plane of this study was to compare the DWI with conventional MRI sequences(T1,T2WIs, and FLAIR), GRE, and CT without assuming that one technique was inherently a criterion standard to assess the diagnostic value of diffusion weighted imaging versus CT and GRE in evaluation of intracranial hemorrhage. All data concerning ADC values for each stage are presented as means 1 standard deviation.
Results

<table>
<thead>
<tr>
<th>Stage of hematoma</th>
<th>No of patients</th>
<th>T1 WIs</th>
<th>T2 WIs</th>
<th>DWI</th>
<th>GRE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyper acute</td>
<td>3</td>
<td>isointense</td>
<td>hyperintense</td>
<td>Heterogenous</td>
<td>Iso or hyperintense</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>11</td>
<td>isointense</td>
<td>hypointense</td>
<td>Hypo</td>
<td>Hypointense</td>
</tr>
<tr>
<td>Small parenchymal hemorrhage</td>
<td>4</td>
<td>-</td>
<td>Hypo or Hypo</td>
<td>hypointense</td>
<td>hyperintense</td>
</tr>
<tr>
<td>Early subacute</td>
<td>7</td>
<td>hyperintense</td>
<td>hypointense</td>
<td>hypointense</td>
<td>hypointense</td>
</tr>
<tr>
<td>Late subacute</td>
<td>9</td>
<td>hyperintense</td>
<td>hyperintense</td>
<td>hyperintense</td>
<td>hyperintense</td>
</tr>
<tr>
<td>Subdural early</td>
<td>1</td>
<td>hyperintense</td>
<td>hypointense</td>
<td>hypointense</td>
<td>hypointense</td>
</tr>
<tr>
<td>late</td>
<td>4</td>
<td>hyperintense</td>
<td>hyperintense</td>
<td>hyperintense</td>
<td>hypointense</td>
</tr>
<tr>
<td>Intraventricular</td>
<td>4</td>
<td>hypointense</td>
<td>hypointense</td>
<td>hypointense</td>
<td>hypointense</td>
</tr>
<tr>
<td>Hemorrhagic arterial infarction</td>
<td>8</td>
<td>Heterogenous</td>
<td>Heterogenous</td>
<td>Heterogenous</td>
<td>Heterogenous</td>
</tr>
<tr>
<td>Hemorrhagic venous infarction</td>
<td>7</td>
<td>Heterogenous</td>
<td>Heterogenous</td>
<td>Heterogenous</td>
<td>Heterogenous</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>3</td>
<td>Hypo</td>
<td>hypointense</td>
<td>hypointense</td>
<td>hypointense</td>
</tr>
</tbody>
</table>

Table 1. Signal Intensities of intracerebral hematoma- according to the Various Stages Demonstrated on MR Images .

-Hyperacute blood was found in 3 cases , all were equally detected by diffusion weighted imaging, GRE, and CT

-Acute intracerebral hematoma was found in 11 cases, all were equally detected by diffusion weighted imaging, GRE, and CT.

-Small parenchymal hemorrhage (post traumatic) was found in 4 cases, all were detected by CT, 2 of them were missed by DWI and GRE.
Table 2 diagnostic accuracy of DWI, GRE and CT for detection of small intraparenchymal hemorrhage.

- Early subacute hematoma was found in 7 cases, all were equally detected by diffusion weighted imaging, GRE, and CT.

- Late subacute hematoma was found in 9 cases, all were equally detected by diffusion weighted imaging and GRE. 5 of these cases were detected by CT.

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>DWI</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>GRE</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>CT</td>
<td>55.5</td>
<td>100%</td>
<td>100%</td>
<td>92.8%</td>
<td>93.4%</td>
</tr>
</tbody>
</table>

Table 3 diagnostic accuracy of DWI, GRE and CT for detection of late subacute hematoma.

- Subdural hematoma was found in 5 cases (1 early and 4 late subacute), all were equally detected by diffusion weighted imaging, GRE, and CT.

- Intraventricular hematoma was found in 4 cases, all were equally detected by diffusion weighted imaging, GRE, and CT.

- Hemorrhagic arterial infarction was found in 8 cases, all were equally detected by DWI, and GRE. 5 of these cases were detected by CT.

- Hemorrhagic venous infarction was found in 7 cases, all were detected by GRE, 1 case missed by DWI, and 3 cases missed by CT.

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>DWI</td>
<td>93.3%</td>
<td>100%</td>
<td>100%</td>
<td>97.8%</td>
<td>98.3%</td>
</tr>
<tr>
<td>GRE</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>CT</td>
<td>60%</td>
<td>100%</td>
<td>100%</td>
<td>88.4%</td>
<td>90.1%</td>
</tr>
</tbody>
</table>

Table 4 diagnostic accuracy of DWI, GRE and CT for detection of hemorrhagic brain lesions.

- Subarachnoid hemorrhage was found in 3 cases, all were detected by CT. One case was missed by GRE and 2 cases were missed by DWI.
<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>DWI</td>
<td>33.3%</td>
<td>100%</td>
<td>100%</td>
<td>96.6%</td>
<td>96.7%</td>
</tr>
<tr>
<td>GRE</td>
<td>66.6%</td>
<td>100%</td>
<td>100%</td>
<td>98.2%</td>
<td>98.3%</td>
</tr>
<tr>
<td>CT</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Table 5 diagnostic accuracy of DWI, GRE and CT for detection of subarachnoid hemorrhage.

<table>
<thead>
<tr>
<th>stage</th>
<th>Numbers of patients</th>
<th>ADC value</th>
<th>Relative ADC</th>
</tr>
</thead>
<tbody>
<tr>
<td>hyperacute</td>
<td>3</td>
<td>0.53±0.06(0.46-0.63)</td>
<td>0.71±0.11(0.69-0.88)</td>
</tr>
<tr>
<td>acute</td>
<td>11</td>
<td>0.56±0.18(0.41-0.89)</td>
<td>0.73±0.16(0.56-1.08)</td>
</tr>
<tr>
<td>Early subacute</td>
<td>7</td>
<td>0.54±0.25(0.19-0.71)</td>
<td>0.71±0.31(0.22-0.98)</td>
</tr>
<tr>
<td>Late subacute</td>
<td>9</td>
<td>0.57±0.11(0.37-0.74)</td>
<td>0.70±0.18(0.45-0.98)</td>
</tr>
</tbody>
</table>

Note.- Relative ADC equals the ratio of the ADC value of hematoma to that of normal contralateral white matter.

ADC value and relative ADC were presented as means 1 standard deviation. ADC value (10⁻³ mm²/sec)

Table 6. Apparent Diffusion Coefficient (ADC) Value and Relative ADC of Intracerebral Hematoma according to Various Stages Demonstrated on DWI.

The ADC value for hemorrhagic arterial infarction was 0.2 × 10⁻³ mm²/sec-0.3 × 10⁻³ mm²/sec

The ADC value for hemorrhagic venous infarction was 0.53 × 10⁻³ mm²/sec-0.6 × 10⁻³ mm²/sec

Images for this section:
Fig. 1: Fig.1. Hyperacute hematoma in the left frontotemporal region with intraventricular extension appearing hyperdense in CT(a), isointense in T1WIs(b), heterogenous hyperintense in T2WIs(c), heterogenous hyperintense in DWI with peripheral hypointense rim (arrow in d), heterogenous hypointense in ADC(e) and heterogenous hyperintense in GRE(f). The peripheral hypointense rim is more obvious in GRE.
Fig. 2: Acute right frontotemporal hematoma appearing hyperdense in CT(a), isointense in T1WIs(b), hypointense in T2WIs(c), FLAIR(d), GRE (e), DWI (f), and ADC(g). It is surrounded by perifocal brain oedema.
Fig. 3: Fig.3. CT (a,b), FLAIR(c,d), T1WIs(e,f), T2WIs(g,h), DWI(l,j), ADC(k,g), GRE(m,n). Left frontal small areas of hemorrhage appearing hyperdense in CT(b) hyperintense in FLAIR(d), not seen in T1 and T2WIs, appearing hyperintense in DWI(j), hypointense in ADC(l) and GRE(n) interpreted as old hematoma. There is area of diffusion restriction (contusion) in the splenium of corpus callosum only seen in DWI(i), ADC(k) and FLAIR(c).
Fig. 4: Late subacute hematoma in the right cerebellar hemisphere appearing hypodense in CT(a), hyperintense in T1WIs(b), T2WIs(c), FLAIR(d), DWI(e), hypointense in ADC(f) and heterogenous hyperintense in GRE(g).

Fig. 5: Late subacute hematoma in the right cerebellar hemisphere appearing hypodense in CT(a), hyperintense in T1WIs(b), T2WIs(c), FLAIR(d), DWI(e), hypointense in ADC(f) and heterogenous hyperintense in GRE(g).
Fig. 14: Fig.8. Axial CT(a), T1WIs(b,c), T2WIs(d,e), FLAIR(f), DWI(g,h), ADC(l,j), GRE(k). Left sided subarachnoid hemorrhage seen by CT, T1, T2, FLAIR, GRE. The left subarachnoid hemorrhage appears as area of ischemia on DWI and ADC. Lt occipital late subacute hematoma seen by DWI, T1, T2, GRE and not seen by CT. Note that the late subacute hematoma in DWI is surrounded by hypointense rim. Left internal carotid and middle cerebral artery occlusion by MRA(l).

Fig. 13: Fig.8. Axial CT(a), T1WIs(b,c), T2WIs(d,e), FLAIR(f), DWI(g,h), ADC(l,j), GRE(k). Left sided subarachnoid hemorrhage seen by CT, T1, T2, FLAIR, GRE. The left subarachnoid hemorrhage appears as area of ischemia on DWI and ADC. Lt occipital late subacute hematoma seen by DWI, T1, T2, GRE and not seen by CT. Note that the late subacute hematoma in DWI is surrounded by hypointense rim. Left internal carotid and middle cerebral artery occlusion by MRA(l).
**Fig. 12:** Fig.8. Axial CT(a), T1WIs(b,c), T2WIs(d,e), FLAIR(f), DWI(g,h), ADC(I,j), GRE(k). Left sided subarachnoid hemorrhage seen by CT, T1,T2,FLAIR,GRE. The left subarachnoid hemorrhage appears as area of ischemia on DWI and ADC. Lt occipital late subacute hematoma seen by DWI, T1, T2, GRE and not seen by CT. Note that the late subacute hematoma in DWI is surrounded by hypointense rim. Left internal carotid and middle cerebral artery occlusion by MRA(l).

**Fig. 11:** Fig.7. Right occipital hemorrhagic venous infarct appearing hypodense in CT(a), The hemorrhagic area appears hyperintense in T1WIs (b), hypointense in T2 WIs(c), FLAIR(d), DWI (e), ADC(f), and GRE(g). There was marked attenuation of the superior sagittal sin in MRV(h).
**Fig. 10:** Fig.7. Right occipital hemorrhagic venous infarct appearing hypodense in CT(a), The hemorrhagic area appears hyperintense in T1WIs (b), hypointense in T2 WIs(c), FLAIR(d), DWI (e), ADC(f), and GRE(g). There was marked attenuation of the superior sagittal sin in MRV(h).
Fig. 9: Left frontotemporal hemorrhagic arterial infarct appearing hypodense in CT(a). The hemorrhagic area is seen in the lentiform nucleus appearing slightly hyperintense in T1WIs (b), hypointense in T2WIs, GRE and ADC(d,e,f). Left middle cerebral artery occlusion in MRA(g)
**Fig. 8:** Fig. 6. Left frontotemporal hemorrhagic arterial infarct appearing hypodense in CT(a). The hemorrhagic area is seen in the lentiform nucleus appearing slightly hyperintense in T1WIs (b), hypointense in T2WIs, GRE and ADC(d,e,f). Left middle cerebral artery occlusion in MRA(g)

**Fig. 7:** Fig. 5. Left sided subacute subdural hematoma appearing heterogenous hypodense in CT(a), hyperintense in T1 and T2WIs (b,c), hypointense in DWI(d), hyperintense in ADC(e), and heterogenous hyperintense in GRE(f).
Fig. 6: Fig.5. Let sided subacute subdural hematoma appearing heterogenous hypodense in CT(a), hyperintense in T1 and T2WIs (b,c), hypointense in DWI(d), hyperintense in ADC(e), and heterogenous hyperintense in GRE(f).

Fig. 15: Fig.8. Axial CT(a), T1WIS(b,c), T2WIs(d,e), FLAIR(f), DWI(g,h), ADC(l,j), GRE(k). Left sided subaracnoid hemorrhage seen by CT, T1,T2,FLAIR,GRE. The left subarachnoid hemorrhage appears as area of ischemia on DWI and ADC. Lt occipital late subacute hematoma seen by DWI ,T1,T2,GRE and not seen by CT. Note that the late subacute hematoma in DWI is surrounded by hypointense rim. Left internal carotid and middle cerebral artery occlusion by MRA(l).
**Fig. 16:** Fig.9. Left sided subarachnoid appearing hyperdense in CT(a), hyperintense in T1WIs(b), hypointense in T2WIs(c), FLAIR (d), DWI (e), ADC(f) and GRE(g).

**Fig. 17:** Fig.9. Left sided subarachnoid appearing hyperdense in CT(a), hyperintense in T1WIs(b), hypointense in T2WIs(c), FLAIR (d), DWI (e), ADC(f) and GRE(g).
Conclusion

Discussion

Neuroimaging plays a crucial role in the evaluation of patients presenting with acute stroke symptoms. While patient symptoms and clinical examinations may suggest the diagnosis, only brain imaging studies can confirm the diagnosis and differentiate hemorrhage from ischemia with high accuracy. This differentiation is critical in making acute treatment decisions, including patient eligibility for thrombolytic therapy. Although noncontrast CT has been considered the criterion standard for assessing intracerebral hemorrhage, formal studies have never been performed to validate the accuracy of this technique compared to the true criterion standard, pathology (6,7).

In the current study we had 61 patients with intracranial hemorrhage, 10 of them were missed by CT, 5 cases were missed by DWI, and 3 cases were missed by GRE.

As regarding the hyper acute hematoma in the current study we had 3 cases (figure 1), all were equally detected by DWI, GRE and CT. At DWI the hyper acute hematoma showed heterogenous hyperintense core, hypointense rim surrounded by perifocal brain oedema. The central hyperintensity has been attributed to intracellular oxyhemoglobin and the hypointense rim to early intracellular deoxyhemoglobin at the periphery of a hematoma. This characteristic hypointense rim has been reported to occur within the first few hours of hemorrhage and in patients with acute neurologic symptoms is valuable for differentiating between acute ischemic stroke and hemorrhage (7,8 ). This hypointense rim was more obvious in GRE than in DWI in the current study (figure 1). The signal intensity of hyperacute ICH observed at DWI was consistent with the findings of previous studies (9, 10). The MR features of hyperintensity at the core of a hematoma and focal variable hypointensity were consistently found in all patients with hyperacute ICH. Hypointensity within a hyperacute hematoma, revealed by DWI, may be an important feature for differentiating hemorrhage from infarction in the practical clinical setting of hyperacute stroke. The focal hypointensity seen at DWI within a hyperacute hematoma may caused by unclotted liquid separated from a retracted clot (9,10). Previous studies have suggested that the cause of hypointensity within a hyperacute hematoma, seen on DWI may be the early presence of paramagnetic deoxyhemoglobin (11). The biophysical explanation for decreased ADC at the core of a lesion in hyperacute hematoma is shrinkage of extracellular space due to resorption of plasma with clot retraction (12), which causes high viscosity.

In the current study, we had 11 cases of acute hematoma (figure 2) and 7 cases of early subacute hematoma all were equally detected by DWI, GRE and CT.

In our study, the cores of lesions found in acute and early subacute hematomas were markedly hypointense at DWI as well as on T2-weighted images. This hypointensity has been attributed to the magnetic field inhomogeneity caused by paramagnetic
intracellular deoxyhemoglobin in acute hematoma (13) and paramagnetic intracellular methemoglobin in early subacute hematoma(14) .

In our study, the cores of acute and early subacute ICHs showed reduced ADC values compared with normal contralateral white matter

-Small post traumatic parenchymal hemorrhage was found in 4 cases in our study, all were detected by CT(100% sensitivity, 100 specificity, 100% accuracy). 2 cases were missed by DWI and GRE (50% sensitivity, 100 specificity, 96.7% accuracy). These 2 cases were interpreted as small ischemic foci on DWI(appear hyperintense) and as old hematoma by GRE(appear hypointense). In the all 4 post traumatic patients in the current study, small areas of restriction on DWI seen in the splenium of corpus callosum (secondary to trauma) not seen by CT or GRE (figure 3).

Physicians should be aware that in cases of small hemorrhages, it may be difficult to make an exact distinction between acute and chronic hemorrhage based on GRE images alone. A noncontrast CT may be necessary in these cases to determine hemorrhage age. With acute medium-large hemorrhages, the characteristic appearance of mixed signal intensity and the surrounding hyperintensity due to edema is very specific and will make the age of the hemorrhage apparent. However, small hemorrhages may have similar characteristics to calcifications and intravascular thrombus and have minimal edema making the determination of hemorrhage age as well as the distinction of hemorrhage versus nonhemorrhage more difficult. (15)

Late subacute hematoma was found in 9 cases in the current study, all were equally detected by diffusion weighted imaging and GRE( figure 4,8). 4 of these cases were missed by CT appearing hypodense. In all 9 patients with hematomas at this stage, diffusion-weighted, T1- and T2-weighted, and FLAIR images showed marked hyper intensity (with hypointense rim at DWI), While GRE demonstrated heterogeneous hyperintensity. Our data showed that in late subacute hematoma, the lesion core showed that the ADC value was lower than that of normal white matter. At the late subacute stage of hematoma, red blood cell lysis occurs and the compartmentalization of methemoglobin is lost, resulting in the elimination of the inhomogeneous susceptibility effect (16). In addition, the intracellular contents are distributed in the extracellular space, possibly causing high viscosity. Other biological changes at this stage include high cellularity resulting from the infiltration of inflammatory cells and macrophages . All these changes may affect molecular diffusion and the ADC of a hematoma(16,17).

-We had 5 cases of Subdural hematoma (1 early subacute and 4 late subacute), all were detected by diffusion weighted imaging, GRE, and CT. However we found that the DWI signal intensity of late subacute subdural hematoma was hypointense(figure 5) compared to intraparenchymal late subacute hematoma. This may be explained by the fact that the subacute subdural hematoma is almost always mixed (acute, early and late subacute), and the hypointensity caused by the paramagnetic by paramagnetic intracellular deoxyhemoglobin and paramagnetic intracellular methemoglobin (13,14).
We had 4 cases of intraventricular hematoma (figure 1), all were detected by diffusion weighted imaging, GRE, and CT. The DWI signal intensity of intraventricular hematoma was low signal intensity in all cases.

We had 8 cases of hemorrhagic arterial infarction (figure 6) all were detected by DWI, GRE, and 3 cases were missed by CT.

We had 7 cases of hemorrhagic venous infarction (figure 8), all were detected by GRE, 1 case missed by DWI, and 3 cases missed by CT.

In our study all cases of hemorrhagic arterial and venous infarction appeared heterogeneous on DWI showing areas of low signal intensity within areas of restricted diffusion. We found that the areas of diffusion restriction in hemorrhagic arterial infarction were more bright (low ADC value $0.2 \times 10^{-3} \text{ mm}^2/\text{sec}$-$0.3 \times 10^{-3} \text{ mm}^2/\text{sec}$) than hemorrhagic venous infarction (ADC value $0.53 \times 10^{-3} \text{ mm}^2/\text{sec}$-$0.6 \times 10^{-3} \text{ mm}^2/\text{sec}$). These findings were in agreement with previous studies (18,19) and was explained by the presence of prominent vasogenic edema associated with mild cytotoxic edema(20) . The hemorrhagic areas in hemorrhagic arterial and venous infarctions gave the signal intensity of early subacute hematoma on DWI, T1WIs, and T2WIs.

These results are supported by recent case reports of "CT negative intracerebral hemorrhages." It is possible that hemorrhagic transformation of ischemic infarcts is an underrecognized phenomenon(17).

The implication of this finding for the neuroimaging evaluation of acute stroke patients who are candidates for thrombolytic therapy is unclear. In the National Institute of Neurological Disorders and Stroke (NINDS) trial, intravenous tPA was shown to be effective based on CT enrollment criteria (19). While it may be hypothesized that patients with MRI evidence of hemorrhagic transformation are at higher risk of developing symptomatic hemorrhage if treated with thrombolytics, it is also possible that overall this group of patients may receive net benefit from therapy(19,21).

We had 3 cases of Subarachnoid hemorrhage in our study, All were detected by CT (figure 8,9). One case was missed by GRE and 2 cases were missed by DWI. The one positive case of subarachnoid hemorrhage on DWI showed area of low signal intensity at the left temporal sulci surrounded by area of ischemia(figure 8), however the 2 negative cases there were areas of diffusion restriction at the regions of subarachnoid hemorrhage(figure 9). These findings were in agreement with previous studies(22,23).

Our study may have implications for the imaging evaluation of patients with acute stroke symptoms. Our findings support prior studies suggesting that MRI DWI is as accurate as CT for the detection of hyperacute , medium and large sized acute, and early sub acute hemorrhage. One important caveat is that with small hemorrhages, blood that appears as acute on CT may appear as ischemic foci on DWI and as chronic hemorrhage on GRE MRI . A noncontrast CT may be required to confirm the diagnosis in these cases. Our
study suggests that DWI and GRE MRI may be able to detect regions of hemorrhagic arterial and venous infarction not evident on CT. Our findings suggesting that DWI was nearly as accurate as CT for the detection of early subacute hemorrhage and subdural hematoma. Our study confirms the superiority of DWI for detection of late subacute subdural. CT was superior to DWI in detection of subarachnoid hemorrhage.

**The limitations of this study**

include the lack of histopathological confirmation and the small number of cases. Although a complete understanding of the underlying biophysical basis may require further studies with a large population, our data suggest that the appearance of intracerebral hematomas on diffusion-weighted images is influenced not only by ADC values but also by magnetic susceptibility and T2 shine-through effects. In addition, our study corroborates the key features of evolving intracerebral hematomas, as depicted by conventional MR imaging.

**Conclusion**

Due to its advantages in delineating ischemic pathophysiology, in combination with the findings suggesting equivalency to CT for detecting acute hemorrhage, MRI with DWI and GRE may be acceptable as the sole imaging technique for acute stroke at centers with expertise in interpreting these findings.

**References**

**REFERENCES**


**Personal Information**