MR imaging as predictor of delayed posttraumatic cerebral hemorrhage

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The occurrence of delayed traumatic intracerebral hemorrhage or hematomas was predicted in four patients by T2-weighted magnetic resonance (MR) imaging. From June, 1986, through February, 1987, 42 patients with head injury were admitted to the Neurosurgical Service of the Seirei Mikatabara General Hospital. Cerebral contusion was suspected in six of these patients. Although the initial computerized tomography (CT) scans showed no cerebral parenchymal lesion, the initial symptoms were more serious than might have been expected from the initial CT findings and/or because their initial CT scans showed intracranial extracerebral hemorrhage. In all six, the initial CT scans were obtained within 2 hours after the injury and were followed by MR imaging. In four patients, T2-weighted MR images revealed areas of increased signal intensity in the cerebral parenchyma, where hemorrhagic changes were subsequently demonstrated by follow-up CT scans. In the remaining two, T2-weighted MR images showed no parenchymal lesion and subsequent CT scans confirmed the absence of hemorrhagic change; these two patients were discharged from the hospital without neurological deficits. It is concluded that MR imaging is useful in predicting delayed hemorrhages.

KEY WORDS • delayed traumatic intracerebral hematoma • head injury • computerized tomography • magnetic resonance imaging

Since the advent of computerized tomography (CT), the concept of delayed traumatic intracerebral hemorrhage or hematoma (DTICH) has been changed from its original description as "traumatische Spät-Apoplexie," which was proposed by Bollinger in 1891.5,6 In cases of DTICH, CT scans obtained shortly after head injury show little abnormality of the brain parenchyma, but subsequent CT scans reveal intracerebral hemorrhage with perifocal edema.5 Although many authors have studied and reported the mechanism of pathological changes in the traumatized brain,1,3,10,14 this subject is still controversial. Since magnetic resonance (MR) imaging has been used diagnostically it has been reported that T2-weighted MR images are more valuable than CT scans in detecting contused brains.4,6,8,11 Four cases of DTICH are reported in which T2-weighted MR imaging predicted late development of hemorrhagic changes.

Clinical Material and Methods

Between June, 1986, and February, 1987, 42 head-injured patients were admitted to the Neurosurgical Service of the Seirei Mikatabara General Hospital. Cerebral contusion was suspected in six of these patients, although the initial CT scans showed no cerebral parenchymal lesion. Suspicion of cerebral contusion was aroused because the initial symptoms were more serious than might have been expected from the initial CT findings and/or because their initial CT scans showed intracranial extracerebral hemorrhage. In four of the six patients, serial CT scans demonstrated DTICH. A diagnosis of DTICH was eventually made when, in the absence of cerebral parenchymal lesions on the initial CT scans, parenchymal high-density lesions appeared on subsequent CT scans. In all six patients, the initial CT scans were obtained within 2 hours after the injuries, immediately followed by T2-weighted MR images. The examinations were performed with a CT/T8800 scanner and an Acutsan MR system using a static magnetic field of 0.02 tesla. It took about 17 minutes to obtain eight slices of T2-weighted MR images (multislice spin-echo method; TR 2000 msec and TE 150 msec). With
both CT and MR imaging, a transaxial projection and a 10-mm slice thickness were obtained. During these examinations, 50 mg of thiopental and/or 5 mg of diazepam were administered intravenously as needed. A physician skilled in intubation and resuscitation was present in all cases during MR imaging. Magnetic resonance imaging was not performed in patients with implanted metals of unknown material such as intracranial aneurysmal clips, or in patients attached to mechanical life-support devices.

Results

Table 1 summarizes the clinical course in the six patients described here. Among the six patients suspected of having DTICH according to our criteria, the diagnosis was confirmed in four. In these four cases, early CT scans failed to show any cerebral parenchymal lesions and only subsequent CT scans revealed hemorrhagic changes. The T₂-weighted MR images obtained immediately following the initial CT scans revealed

TABLE 1
Clinical summary of six cases presented in this study*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Cause of Injury</th>
<th>Site of Injury</th>
<th>Neurological Findings on Admission &amp; Interval</th>
<th>Initial Findings &amp; Interval</th>
<th>Subsequent CT Findings &amp; Interval</th>
<th>Neurological Outcome at Discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>56, M</td>
<td>traffic accident</td>
<td>lt temporal area</td>
<td>GCS: E2, M5, V2; SAH in bilat sylvian fissures, 75 min</td>
<td>ISI in bilat temporal &amp; rt frontal lobes, 105 min</td>
<td>3 DTICH in bilat temporal &amp; rt frontal lobes, 8 hrs 15 min</td>
<td>no MW, 43 days</td>
</tr>
<tr>
<td>2</td>
<td>25, M</td>
<td>fall from a height</td>
<td>occipital area</td>
<td>GCS: E4, M6, V4; SAH in bilat sylvian fissures, 110 min</td>
<td>ISI in rt temporal &amp; frontal poles, 140 min</td>
<td>2 DTICH in rt temporal &amp; frontal lobes, 19 hrs 50 min</td>
<td>no major deficit, 67 days</td>
</tr>
<tr>
<td>3</td>
<td>75, M</td>
<td>traffic accident</td>
<td>lt temporal area</td>
<td>GCS: E4, M6, V4; no MW, 40 min</td>
<td>acute EDH in lt temporal base, 110 min</td>
<td>ISI in rt temporal lobe, 140 min</td>
<td>no MW, 89 days</td>
</tr>
<tr>
<td>4</td>
<td>78, M</td>
<td>fall on the ground</td>
<td>lt temporal area</td>
<td>GCS: E2, M5, V2; no MW, 70 min</td>
<td>acute SDH at rt temporal area, swelling of rt hemisphere, 120 min</td>
<td>ISI in rt temporal &amp; frontal lobes, 160 min</td>
<td>2 DTICH in rt temporal &amp; frontal lobes, 25 hrs</td>
</tr>
<tr>
<td>5</td>
<td>75, M</td>
<td>traffic accident</td>
<td>lt temporal area &amp; face</td>
<td>GCS: E2, M6, V3; no lesion, 45 min</td>
<td>no lesion, 45 min</td>
<td>no lesion, 90 min</td>
<td>no lesion, 22 hrs</td>
</tr>
<tr>
<td>6</td>
<td>24, F</td>
<td>traffic accident</td>
<td>lt occipital area</td>
<td>GCS: E4, M6, V4; SAH in bilat sylvian fissure &amp; basal cistern, 50 min</td>
<td>no parenchymal lesion, 80 min</td>
<td>no parenchymal lesion, 19 hrs 30 min</td>
<td>no deficit, 9 days</td>
</tr>
</tbody>
</table>

* CT = computerized tomography; MR = magnetic resonance; GCS = Glasgow Coma Scale score (E = eye opening; M = motor response; V = verbal response); SAH = subarachnoid hemorrhage; ISI = increased signal intensity area(s); DTICH = delayed traumatic intracerebral hemorrhage; MW = motor weakness; EDH = epidural hematoma; SDH = subdural hematoma. All intervals were calculated from the time of injury.
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areas of high signal intensity in the cerebral parenchyma where subsequent CT scans showed cerebral contusion. All of these areas of increased signal intensity were contralateral and/or ipsilateral to the impact sustained by the patient and were thought to be coup or contre-coup injuries. In no case did the DTICH become larger after the second CT scan. The six patients were all treated conservatively because their intracranial hematomas were small. Three of these four cases are described briefly below.

In the remaining two patients, subsequent CT scans did not show any parenchymal lesion. In Case 5, the patient’s level of response improved 2 hours after the injury. He then developed a syndrome of inappropriate antidiuretic hormone, but was discharged from the hospital 37 days later without neurological deficit. In Case 6, the initial CT scan showed subarachnoid hemorrhage (SAH) but a subsequent CT scan revealed no parenchymal lesion, and the patient was discharged from the hospital 9 days after injury without neurological deficit.

Case Reports

Case 1

This 56-year-old man was struck on the left temporal area during an automobile accident; he lost consciousness and developed bleeding in the left ear. He was admitted to our service about 40 minutes later with a Glasgow Coma Scale (GCS) score of 9. Plain skull x-ray films revealed a left temporoparietal linear skull fracture. A CT scan obtained 75 minutes after the injury revealed SAH in both sylvian fissures but no parenchymal abnormalities (Fig. 1). The T2-weighted MR images obtained immediately after CT scanning revealed areas of increased signal intensity in both temporal lobes and the right frontal lobe (Fig. 2). A CT scan obtained 8 hours 15 minutes after the injury revealed DTICH in both temporal lobes and the right frontal lobe (Fig. 3).
Two days after the injury the patient became less responsive, with a GCS score of 5 to 7, and developed right-sided motor weakness. A CT scan showed enlarged areas of low density around the hematomas. The worsening of these symptoms was thought to be due to traumatic edema. A T$_2$-weighted MR image obtained 11 days after the injury revealed that the areas of increased signal intensity were larger than on the initial T$_2$-weighted images. The patient was treated conservatively and was discharged from the hospital 43 days later with no motor weakness. On T$_2$-weighted images obtained 8 months after the injury, the areas of increased signal intensity in the temporal lobes and in the right frontal lobe were larger than those seen on the initial T$_2$-weighted images (Fig. 4).

**Case 2**

This 25-year-old man fell from a height and struck his back and occipital area on the pavement. He was admitted to our service about 30 minutes after the injury with a GCS score of 14. Plain x-ray films revealed an occipital linear fracture, a compression fracture of the T-12 vertebra, and a right 12th rib fracture. A CT scan obtained 110 minutes after the injury suggested SAH in both sylvian fissures (Fig. 5). On T$_2$-weighted MR images obtained immediately following the initial CT scan, increased signal intensity was revealed in the right temporal and frontal poles representing contrecoup injuries (Fig. 6). The patient then became irritable and agitated, with a GCS score of 10 to 12. A CT scan obtained 19 hours 50 minutes after the injury revealed DTICH in the right frontal and temporal lobes (Fig. 7). He was treated conservatively.

Three days after the injury, T$_2$-weighted images showed enlarged areas of increased signal intensity in the right frontal and temporal lobes compared to the initial T$_2$-weighted MR images. In the right frontal high-intensity area, a focus of isointensity corresponding to the hematoma seen on the CT scans was observed. Subsequent T$_2$-weighted MR images obtained 65 days after the injury showed marked edema in the right frontal and temporal lobes.
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after the injury showed that the areas of increased signal intensity were larger than on the initial $T_2$-weighted images. The patient was discharged from the hospital 67 days after injury without any major neurological deficit.

**Case 4**

This 78-year-old man fell down and struck his left temporal area on the floor. He was admitted to our service about 70 minutes after the injury. Physical examination revealed a subgaleal hematoma over the left temporal area. Neurological examination showed a decreased level of response with a GCS score of 9; there was no motor weakness. Plain skull x-ray films revealed a left temporoparietal linear fracture. A CT scan obtained about 120 minutes after injury showed a thin subdural hematoma in the right frontal area and swelling of the right cerebral hemisphere (Fig. 8). About 160 minutes after the injury, $T_2$-weighted MR images were obtained which revealed increased signal intensity in the right frontal and temporal lobes corresponding to a contrecoup injury (Fig. 9). A subsequent CT scan about 25 hours after the injury showed right frontal and temporal DTICH with perifocal edema (Fig. 10).

The patient was treated conservatively. His level of response decreased during the first 3 days (GCS scores of 8 or 9), and then gradually improved. The $T_2$-weighted MR images obtained 18 days after the injury showed that the areas of increased signal intensity had become much larger, and 33 days after the injury $T_2$-weighted MR images showed that they had enlarged even further. He was discharged from the hospital 36 days after the injury without any motor weakness.

**Discussion**

Many authors have described the mechanism and clinical features of DTICH, especially in the period after the advent of CT. Snow, et al., reported

![Fig. 6. Case 2. Magnetic resonance $T_2$-weighted images obtained immediately after the initial computerized tomography scan revealing areas of increased signal intensity (arrows) in the right frontal and temporal poles.](image1)

![Fig. 7. Case 2. Computerized tomography scan obtained about 20 hours after the injury showing a delayed traumatic intracerebral hemorrhage where the $T_2$-weighted magnetic resonance images obtained on admission had shown increased signal intensity.](image2)

![Fig. 8. Case 4. Computerized tomography scan obtained about 120 minutes after the injury showing a thin layer of subdural hematoma (arrows) and a slight midline shift. No parenchymal lesion is observed.](image3)
five patients in whom the CT scans were normal while MR imaging revealed parenchymal nonhemorrhagic contusions. They stated that both the hemorrhagic and the nonhemorrhagic contusions appeared isointense with the brain on MR imaging during approximately the first 72 hours, and they recommended CT scanning during this period. We disagree with them on this point: we have found that T$_2$-weighted MR images can detect contusions as early as 2 hours after injury. Perhaps in the series of Snow, et al., the TR and TE were not long enough to detect small amounts of increased water content in their T$_2$-weighted MR images. Han, et al., reported T$_2$-weighted MR images of brain contusion more than 8 hours after the injury and concluded that MR imaging clearly demonstrated the abnormally increased brightness of contused brain of T$_2$-weighted spin-echo studies and that this brightness was due to increased mobile hydrogen content, for instance as a result of edema. In experimental studies of brain trauma, Smith, et al., reported that perivenous extravasations and petechiae began to appear within 5 to 6 minutes, and edema and swelling of the surrounding cortex occurred within 15 minutes after the injury. Similarly, in experimental studies of DTICH, Aruga, et al., reported that small petechiae were seen 5 minutes posttrauma and became larger with microscopic retention of interstitial water as early as 30 minutes after the injury. It is most likely that the areas of increased signal intensity in our cases were mainly caused by increased water content which CT scans could not detect as changes in x-ray attenuation properties. If petechiae and perifocal edema were larger, they might appear as high- and low-density areas, respectively, in CT scans, but they can be too small to be detected in the early stages or their intermixture may appear as isodense.

Many authors have reported that some DTICH's were secondary to cerebral contusions. Gudeman, et al., regarded them as an epiphenomenon in damaged brains subjected to further insults, and our cases would fit with their hypothesis. If DTICH is seen on T$_2$-weighted MR images only, it might be considered an enlarging brain contusion with hemorrhage. In fact, early contusions appeared as areas of increased signal intensity on T$_2$-weighted MR images. These areas of high signal intensity became larger in a few days, during which time CT scans showed hemorrhagic changes. The areas of increased signal intensity became smaller in the chronic stage, but even in the patient with the longest follow-up period, these areas of increased signal intensity were much larger than they appeared on the initial T$_2$-weighted images. In addition, these areas seemed to be larger than those of hypertensive intracerebral hematomas in the chronic stage. Traumatic hematomas cannot be entirely responsible for the large areas of increased signal intensity; the contusions themselves appear to be responsible.

To predict the occurrence of DTICH, some authors have tried contrast-enhanced CT scanning and have reported correct prognosis in some cases. However, the intravenous administration of contrast medium, which generally contains an anticoagulant agent such as ethylenediaminetetra-acetic acid (EDTA), to patients who might have hemorrhagic changes is controversial. Some authors have reported that DTICH's were found after surgical removal of epidural, subdural, or intracerebral hematomas. In the surgical management of head injuries it is helpful to know the number, location, and size of contusions which are "silent" on early CT scans. This diagnosis is possible on T$_2$-weighted MR images in the acute stage.

In our Cases 5 and 6 no abnormal intensity was found even in T$_2$-weighted MR images, and these patients did not develop DTICH. However, we cannot be sure that small parenchymal contusions were not missed in the T$_2$-weighted MR images due to relatively low spatial resolution. In the same period there were no cases studied by T$_2$-weighted MR images in which the initial CT scans showed small hemorrhagic changes.
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that became larger on subsequent CT scans. If the initial CT scans had been performed later, our Cases 1 to 4 might have shown small enlarging hemorrhagic changes. There were no cases in which the initial T2-weighted MR images failed to detect contusions that appeared later.

Our series of cases is small; however, new techniques to obtain MR images in a shorter time after injury will make its use in the diagnosis of brain trauma easier and more common even in the acute stage. It is possible that MR imaging in the acute stage of brain trauma may change the classifications of brain trauma itself.

References

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